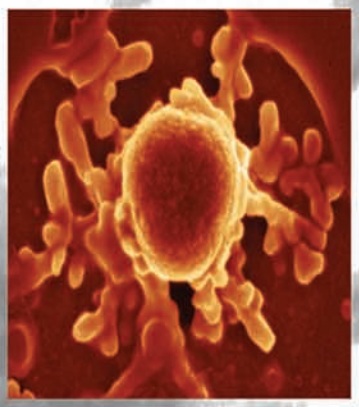
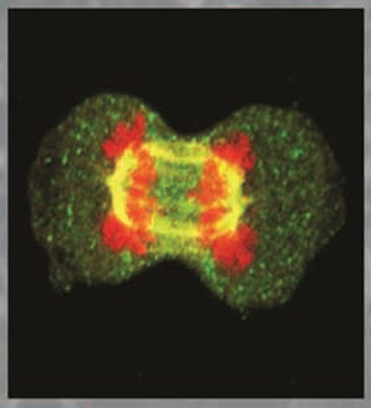
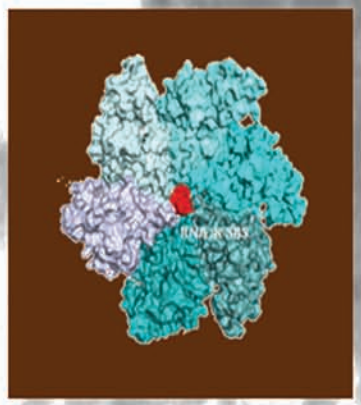
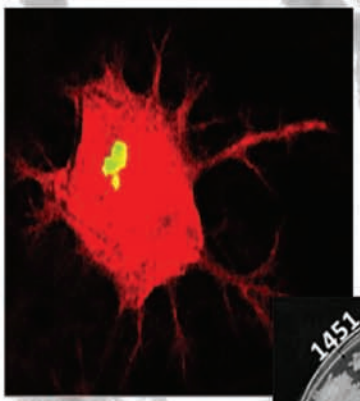
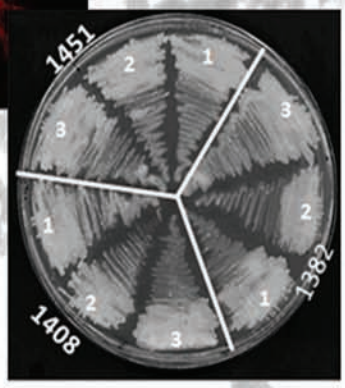
वार्षिक प्रतिवेदन 2015-16
ANNUAL REPORT 2015-16









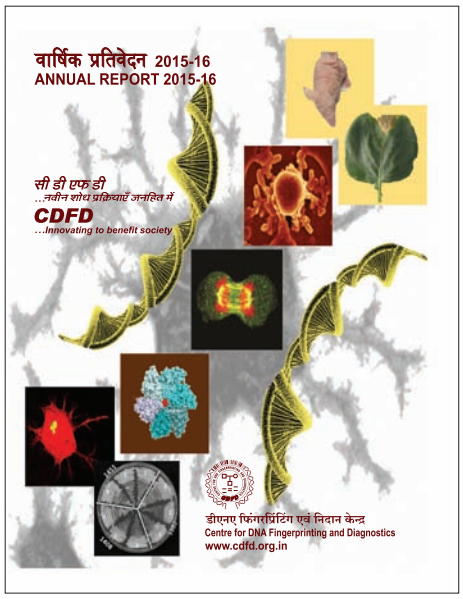


सी डी एफ डी
...नवीन शोध प्रक्रियाएँ जनहित में

CDFD
...Innovating to benefit society

डीएनए फिंगरप्रिंटिंग एवं निदान केन्द्र
Centre for DNA Fingerprinting and Diagnostics
www.cdfd.org.in

मुख्य आवरण पृष्ठ का विवरण
Description of the Front Cover Page



इस तस्वीर की पृष्ठभूमि में चूहे की एक न्यूरोब्लास्टोमा कोशिका (न्यूकरो२ए) दर्शाई गई है।
(स्रोत : अभिकलनात्मक एवं कार्यात्मक जीनोमिकी प्रयोगशाला, सीडीएफडी)।

अन्य चित्रों में निम्नलिखित शामिल हैं :

1. ओंकोजेनिक कोशिकाओं का फॉक्स एन1एनयू चूहों में त्वचा के नीचे इंजेक्शन। (स्रोत : प्रयोगशाला जंतु सुविधा, सीडीएफडी)
2. Δएक्सकएसएसए उत्परिवर्ती में रोगजनकता की कमी होती है और इनकी वृद्धि गोभी के अंदर होती है। (स्रोत : पादप रोगाणु अंत:क्रिया प्रयोगशाला, सीडीएफडी)
3. एक बड़े प्रियोन के समान एचवायपीके के एन्यूलर ओलिगोमर द्वारा इसकी परिधि पर हंटिंगटिन समुच्चयों का क्रम (अभिकलनात्मक एवं कार्यात्मक जीनोमिकी प्रयोगशाला, सीडीएफडी)
4. अर्धसूत्री विभाजन के दौरान तर्कु उपकरण के साथ वीडीआर5 का जुड़ाव। यहां एनाफेज चरण दिखाया गया है। (स्रोत : कोशिका चक्र नियमन प्रयोगशाला, सीडीएफडी)
5. कार्टून में आरएचओ हेक्सामर का बंद कॉम्प्लेक्स। (सीसी) दर्शाया गया है (पीडीबी कोड : 1 पीवीओ)। (स्रोत : अनुलेखन प्रयोगशाला, सीडीएफडी)
6. पेरिन्यूक्लियर हंटिंगटिन समुच्चय दर्शाने वाली एक मानव आईएमआर32 सेल लाइन तथा एक्टोपिक रूप से अभिवक्ति एचवायपीके (स्रोत : अभिकलनात्मक एवं कार्यात्मक जीनोमिकी प्रयोगशाला, सीडीएफडी)
7. आनुवंशिक झिल्ली में उगाई गई कॉलोनियां का प्रतिनिधित्व जिससे ऐसे क्लोन अलग किए गए जो एटीसी द्वारा प्रेरण पर एम. स्मेगमेटिस की वृद्धि का संदमन करते हैं। (स्रोत : अनुलेखन प्रयोगशाला, सीडीएफडी)

The background of the image represents a mouse neuroblastoma cell (Neuro2a) [Source: Laboratory of Computational & Functional Genomics].

The other images comprise the followings:

1. Subcutaneous injection of oncogenic cells into FoxN1^{nu} mice. [Source: Laboratory of Animal Facility]
2. Δ*xssA* mutant are deficient in virulence and growth inside cabbage. [Source: Laboratory of Plant-Microbe Interaction, CDFD]
3. A large prion like annular oligomer of HYPK sequestering Huntingtin aggregates at its periphery. [Laboratory of Computational & Functional Genomics, CDFD]
4. WDR5 associates with the spindle apparatus during mitosis. Anaphase stage is shown here. [Source: Laboratory of Cell Cycle Regulation, CDFD]
5. Cartoon showing the closed complex (CC) of the Rho hexamer (PDB code: 1PVO). [Source: Laboratory of Transcription, CDFD]
6. A human IMR32 cell line showing perinuclear Huntingtin aggregate and ectopically expressed HYPK. [Source: Laboratory of Computational & Functional Genomics]
7. Rrepresentation of the grown colonies in genetic screen to isolate the clones that inhibit growth of *M. smegmatis* upon induction by ATC. [Source: Laboratory of Transcription, CDFD]

(मुख्य आवरण पृष्ठ का चित्रांकन अभिकलनात्मक एवं कार्यात्मक जीनोमिकी प्रयोगशाला के वरिष्ठ अनुसंधान अध्येता श्री देबाशिष के घोष द्वारा किया गया है।)

(The main cover page above has been designed by Senior Research Fellow Mr. Debasish K Ghosh of the Laboratory of Computational & Functional Genomics.)

सी डी एफ डी
CDFD

वार्षिक प्रतिवेदन

अप्रैल 2015 से मार्च 2016 तक

ANNUAL REPORT

April 2015 to March 2016

डी एन ए फिंगरप्रिंटिंग एवं निदान केन्द्र

नामपल्ली, हैदराबाद - 500 001

Centre for DNA Fingerprinting and Diagnostics

Nampally, Hyderabad - 500 001

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अधिदेश
Mandate

अधिदेश

सीडीएफडी सोसाइटी के संगम ज्ञापन तथा नियम एवं विनियमों में बताए गए अनुसार डीएनए फिंगरप्रिंटिंग एवं निदान केंद्र की स्थापना जिन उद्देश्यों के लिए हुई वे निम्न प्रकार हैं :

i. पितृत्व विवाद, आप्रवास और अस्पतालों में नवजात शिशुओं की अदला-बदली जैसे मामलों में निजी पक्षों सहित विविध अभिकरणों के लिए पर्याप्त अदायगी पर डीएनए प्रोफाइलिंग और उससे संबंधित विश्लेषण का वैज्ञानिक अनुसंधान करना।

ii. अपराध अन्वेषण अभिकरणों को डीएनए फिंगरप्रिंटिंग और उससे संबंधित विश्लेषण तथा सुविधाएं प्रदान करना।

iii. अपराध अन्वेषण और परिवार मामलों में डीएनए प्रोफाइल विश्लेषण और उससे संबंधित तकनीकों के साक्ष्य संबंधी मूल्य को समझने में पुलिस कर्मियों, न्यायिक वैज्ञानिकों, वकीलों तथा न्यायपालिका की सहायता करना।

iv. आनुवंशिक अव्यवस्थाओं को संसूचित करने हेतु डीएनए नैदानिक विधियां सिद्ध करना और इस प्रकार के संसूचन के लिए संपरीक्षाएं विकसित करना।

v. पादप और जंतु कोशिका माल, कोशिका लाइनों के प्रमाणीकरण के लिए डीएनए फिंगरप्रिंटिंग तकनीकों का उपयोग करना और ऐसे प्रयोजनों के लिए आवश्यकतानुसार नई संपरीक्षाएं विकसित करना।

vi. डीएनए फिंगरप्रिंटिंग तकनीकों पर प्रशिक्षण प्रदान करना।

vii. मूलभूत, अनुप्रयुक्त अनुसंधान एवं विकास कार्य करना।

viii. देश में चिकित्सा संस्थाओं, जन-स्वास्थ्य अभिकरणों और उद्योग को परामर्शी सेवाएं प्रदान करना।

ix. केंद्र के उद्देश्यों से संगत क्षेत्रों में विदेशी अनुसंधान संस्थाओं एवं प्रयोगशालाओं और अन्य अंतरराष्ट्रीय संगठनों के साथ सहयोग करना।

x. अनुसंधान छात्रों को स्नातकोत्तर उपाधियों के लिए पंजीकृत कर सकने के प्रयोजन हेतु उच्चतर अधिगम के मान्यता प्राप्त विश्वविद्यालयों एवं संस्थाओं के साथ संबंध स्थापित करना।

xi. भारत सरकार, राज्य सरकारों, देश में स्थित पूर्व संस्थाओं / न्यासों, व्यक्तियों और अन्य गतिविधियों के लिए अंतरराष्ट्रीय संगठनों सहित विदेशी स्रोतों से आर्थिक सहायता प्राप्त करना।

xii. केंद्र सरकार के पूर्व अनुमोदन से प्रशिक्षण कार्यक्रमों, वैज्ञानिक अनुसंधान और अन्य गतिविधियों के लिए अंतरराष्ट्रीय संगठनों सहित विदेशी स्रोतों से आर्थिक सहायता प्राप्त करना।

xiii. केंद्र की गतिविधियों को चलाने के लिए जैसा आवश्यक या सुविधाजनक हो, कोई भी संपत्ति चल या अचल या भवनों एवं निर्माणों को निर्मित करने, सुधार करने, परिवर्तित करने, गिरा देने या मरम्मत करने हेतु उपहार, क्रय, विनियम, पट्टा, भाडे पर लेने द्वारा या अन्यथा किसी भी तरह अर्जित करना।

xiv. केंद्र के प्रयोजन हेतु, भारत सरकार और अन्य प्रोनोटों, विनियम पत्रों या अन्य परक्राम्य लिखतों को आहरित करना और स्वीकार करना, तैयार करना और पृष्ठांकित करना, बट्टा करना और परक्रामण करना।

xv. केंद्र को सौंपी गई निधियों या धन को निवेश करने के लिए, ऐसी प्रतिभूतियों को खोलने या ऐसे तरीके से, जो कि समय-समय पर शासी परिषद द्वारा निर्धारित किए जाते हैं, इस प्रकार के निवेश को विक्रय या पक्षांतरण करना।

xvi. उक्त सभी उद्देश्यों या उनमें से किसी उद्देश्य की प्राप्ति के लिए सभी ऐसे अन्य विधिसम्मत कार्य, जैसा आवश्यक, प्रासंगिक या सहायक हो, करना।

xvii. केंद्र के उद्देश्यों को वास्तविक बनाने के लिए प्रोफेसरों, अन्य संकाय पदों, अभ्यागत अध्येतावृत्तियों सहित अध्येतावृत्तियों, अनुसंधान एवं संवर्ग पदों, छात्रवृत्तियों आदि को संस्थापित करना।

xviii. केंद्र के वैज्ञानिक एवं प्रौद्योगिकीय कार्य के लिए प्रयोगशालाओं, कार्यशालाओं, भंडार, पुस्तकालय, कार्यालय और अन्य सुविधाओं को स्थापित करना।

xix. तकनीकी जानकारी को उद्यमकर्ताओं और उद्योगों से प्राप्त या उनको अंतरण करना, और

xx. पेटेंटों, डिजाइनों एवं तकनीकी जानकारी जो कि केंद्र द्वारा विकसित की गई हो, को पंजीकृत करना और केंद्र के हित में ऐसे पेटेंटों / डिजाइनों / तकनीकी जानकारी के किसी भाग को अंतरण करना।

MANDATE

The objectives for which the Centre for DNA Fingerprinting and Diagnostics (CDFD) was established, as enumerated in Memorandum of Association and Rules and Regulations of CDFD Society, are as follows:

i. To carry out scientific research pertaining to DNA profiling and related analysis in civil cases like paternity disputes, immigration, and exchange of newborns in hospitals, for various agencies including private parties, on appropriate payment;

ii. To provide DNA fingerprinting and related analysis and facilities to crime investigation agencies;

iii. To assist police personnel, forensic scientists, lawyers and the judiciary in understanding the evidential value of the DNA profile analysis and related techniques in crime investigation and family matters;

iv. To establish DNA diagnostic methods for detecting genetic disorders and to develop probes for such detection;

v. To use DNA fingerprinting techniques for the authentication of plant and animal cell material, cell lines and to develop new probes where necessary for such purposes;

vi. To provide training in DNA fingerprinting techniques;

vii. To undertake basic, applied and developmental R & D work;

viii. To provide consultancy services to medical institutions, public health agencies and industry in the country;

ix. To collaborate with foreign research institutions and laboratories and other international organizations in fields relevant to the objectives of the Centre;

x. To establish affiliation with recognized universities and institutions of higher learning for the purpose of enabling research scholars to register for post-graduate degrees;

xi. To receive grants, donations and contributions in cash or in other forms from the Government of India, State Governments, Charitable Institutions/Trusts, individuals and industry within the country;

xii. To receive, with the prior approval of the Central Government, monetary assistance from foreign sources including international organizations for training programmes, scientific research and other activities;

xiii. To acquire by gift, purchase, exchange, lease, hire or otherwise howsoever, any property movable or immovable or to construct, improve, alter, demolish or repair buildings and structures as may be necessary or convenient for carrying on the activities of the Centre;

xiv. For the purpose of the Centre, to draw and accept, make and endorse, discount and negotiate Government of India and other Promissory Notes, Bills of Exchange, Cheques or other Negotiable Instruments;

xv. For investing the funds of or money entrusted to the Centre, to open such securities or in such manner as may from time to time be determined by the Governing Council and to sell or transpose such investment;

xvi. To do all such other lawful acts as may be necessary, incidental or conducive to the attainment of all or any of the above objectives;

xvii. To institute Professorships, other faculty positions, fellowships including visiting fellowships, research and cadre positions, scholarships, etc. for realizing the objectives of the Centre;

xviii. To establish, maintain and manage laboratories, workshops, stores, library, office and other facilities for scientific and technological work of the Centre;

xix. To acquire or transfer technical know-how from/to entrepreneurs and industries; and

xx. To register patents, designs & technical know-how that may be developed by the Centre and transfer any portion of such patents/designs/technical know-how in the interest of the Centre.

निदेशक का संदेश
From the Director’s Desk

निदेशक का संदेश

अपने सहयोगियों और अपनी तरफ से, मैं यहां वर्ष 2015-16 के लिए सीडीएफडी की वार्षिक रिपोर्ट प्रस्तुत कर रहा हूँ। केंद्र में दो प्रकार की विशिष्ट गतिविधियों को संयोजित किया जाता है i) कानून प्रवर्तन एजेंसियों के लिए मानव डीएनए रूपरेखा के क्षेत्र में सेवाएं, आनुवंशिकी विकारों के लिए नैदानिक परीक्षण, शुद्धता के लिए बासमती चावल के विश्लेषण, और ii) आधुनिक जीव विज्ञान के विभिन्न विषयों में बुनियादी अनुसंधान भी संलग्न हैं।

डीएनए फिंगरप्रिंटिंग सेवा प्रयोगशाला (एलडीएफएस) से प्राप्त लगभग 400 मामलों को न्याय पालिका द्वारा तथा राज्य और संघीय सरकारों की कानून प्रवर्तन और जांच एजेंसियों द्वारा अग्रेषित किया गया था। एलडीएफएस जैव प्रौद्योगिकी विभाग के समन्वय से डीएनए विधेयक को अंतिम रुप देने में सक्रिय रूप से शामिल रही।

नैदानिकी प्रभाग द्वारा विभिन्न आनुवंशिकी रोगों के लिए 4859 रोगियों को आनुवंशिकी मूल्यांकन प्रदान किए गए। निजाम्स् इंस्टीट्यूट ऑफ मेडिकल साइंसेस, हैदराबाद, सीडीएफडी में चिकित्सा आनुवंशिकी विभाग में नए निधिकरण के समन्वय से चिकित्सा आनुवंशिकी में डीएनबी कार्यक्रम का आयोजन सफलतापूर्वक किया गया है और क्लिनिकल साइटोजेनेटिक्स और क्लिनिकल आण्विक आनुवंशिकी में अध्येतावृत्ति कार्यक्रम चलाए गए हैं। इनके अलावा विभिन्न लाइसोसोमल भण्डार विकारों के नए उत्परिवर्तनों के आण्विक विश्लेषण किए गए। मानव एक्सोसोम विश्लेषणों द्वारा दुर्लभ आनुवंशिक विकारों वाले परिवारों पर भी कार्य किया गया।

आण्विक आनुवंशिकी प्रयोगशाला ने रेशम कीट में लिंग निर्धारण के आण्विक आधार पर अनुसंधान जारी रखा। पुनः उन्होंने ड्रोसोफिला, डी मेंडिबुलर में नोड्यूलर समजात की भूमिकाओं को अनुलेखन कारक, एनएफKबी में समझाया है।

क्रोमैटिन जीवन विज्ञान और एपिजेनेटिक्स प्रयोगशाला फिशन ईस्ट सिरटुइन एचएसटी4 की डीएनए द्विगुणन और क्षति में भूमिकाएं समझने में संलग्न रही।

अभिकलनात्मक जीव विज्ञान प्रयोगशाला द्वारा विकार से ग्रस्त हिस्सों का सरेखित करने के लिए एक नए प्रतिस्थापन स्कोरिंग मेट्रिक्स के सूत्रण के लिए और प्रोटीन के विकार ग्रस्त हिस्सों में पाए गए मिससेंस उत्परिवर्तनों के कार्यात्मक प्रभाव का अनुमान लगाने की नई विधि द्वारा प्रयास किए गए। एक युक्ति संगत डेटा बेस और एक सॉफ्टवेयर सूट का विकास कैंसर के रोगियों तथा स्वस्थ व्यक्तियों के सांस, मूत्र और लार के नमूनों से वाष्पशील चयापचय यौगिकों पर जानकारी जमा करने हेतु किया गया।

प्रोटियोमिक मार्गों का उपयोग करते हुए कोशिका मृत्यु तथा कोशिका उत्तरजीविता प्रयोगशाला द्वारा 143 मानव फॉस्फेटेज़ के एक विस्तृत अंतः क्रियात्मक नेटवर्क का मानचित्रण किया गया है। इन विश्लेषणों को नई कोशिकीय प्रक्रियाओं के साथ अनेक फॉस्फेटेज़ जोड़ने में इस्तेमाल किया गया और इससे कैंसर सहित विभिन्न मानक रोगों से आनुवंशिक तौर पर जुड़ी प्रोटीन-प्रोटीन अंतः क्रियाओं का पता लगाया गया।

आण्विक ओंकोलॉजी प्रयोगशाला ने निम्नलिखित पक्षों पर अध्ययन किए हैं। i) पीएआर कॉम्प्लेक्स में पीएआर6जी की भूमिका समझना ii) सुझाया गया कि Ca2+/NF- T सिग्नलिंग को Wnt- रेक्टल कैंसर में समृद्ध बनाया जाये आबादी में नए एचईडी से पैदा होने वाले उत्परिवर्तनों का लाक्षणीकरण किया गया।

अनुलेखन प्रयोगशाला द्वारा एनमूएसजी की सहायता से रो आश्रित अनुलेखन समापन के मॉड्यूलन का आण्विक आधार प्रकट किया गया। इन्होंने माइकोबैक्टीरिमम प्रजाति

को मारने में सक्षम माइको बैक्टीरियो फेज जीनों का अलग करने की विधियों की भी रिपोर्ट की है।

कोशिका सिग्नलिंग प्रयोगशाला से प्रदर्शित किया गया है कि आईपी7 और जीन आईपी6के1 विभिन्न शरीर क्रियात्मक मार्गों में शामिल है, जैसे कैंसर कोशिकाओं का कीमोटेक्सिस, मोटर प्रोटीन डायनिन की गतिशीलता।

ड्रोसोफिला तंत्रिका विकास प्रयोगशाला द्वारा एक विनियामक विशिष्ट पहचान के साथ केंद्रीय तंत्रिका तंत्र के अग्र - पश्च अक्ष के साथ अनुलेखन कारकों के हॉक्स परिवार के कार्यों का आण्विक आधार प्रदर्शित किया गया। इन्होंने हॉक्स जीन, विकृत के स्व विनियमन पर अंतर्दृष्टि पर फोकस किया है।

कवक रोगाणु जनन प्रयोगशाला में प्रदर्शित किया गया है कि रोग जनक यीस्ट कैंडिडा ग्लैब्रेटा दो तनाव प्रतिक्रियाशील माइटोजन से सक्रिय बनाए गए प्रोटीन काइनेज CgHog1 और CgSlt2 के सक्रिमण द्वारा आमरन के उच्च बाह्य स्तर पर प्रतिक्रिया देता है और काइनेज आमरन के होमियोस्टेसिस के रखरखाव, जैविक और अजैविक सतहों का पालन करने तथा सी. ग्लैब्रेटा के रोग जनक होने में महत्वपूर्ण है।

आण्विक कोशिका जीव विज्ञान प्रयोगशाला के अध्ययनों से IRAK3, MKP-1 और MAPK सिग्नलिंग कास्केड टीएलआर२ के बीच एक संबंध होने का संकेत मिला जो तपेदिक में प्रो तथा एंटी इंफ्लेमेटरी साइटोकाइन प्रतिक्रिया पर निमंत्रण में एक महत्वपूर्ण भूमिका निभाता है। पुन: इन्होंने दर्शाया है कि एम. ट्यूबरकुलोसिस का पीई11 प्रोटीन इसके गैर रोगाणु जनक सेरोगेट एम. स्मेग्मेटिस की अभिव्यक्ति से एक प्रारूपिक रोग जनक माइकोबैक्टीरिया सहित बढ़ी हुई कोशिका भित्ति की अखण्डता, पर्यावरण तनाव की प्रतिरोधकता, उन्नत उत्तरजीविता के गुण मेजबान के अंदर प्रदर्शित कर सकता है।

स्तनधारी आनुवंशिकी प्रयोगशाला द्वारा कार्सिनोजेनेसिस और विकास में डीएनए मेथिल ट्रांसफरेज Dnmt3l a और Dnmt2 की भूमिका को समझा है। प्रयोगशाला द्वारा एपिजेनेटिक बदलावों को भी पहचाना गया है जो एम. ट्यूबरकुलोसिस के साथ चुनौती देने पर मेजबान कोशिका में होते हैं।

पादप सूक्ष्मजीव अंत: क्रिया प्रयोगशाला के अनुसंधान में पहली बार यह प्रदर्शित किया गया है कि पादप रोगाणु जेंथोमोनाज़ कैम्पेस्ट्रीस पीवी. कैम्पेस्ट्रीस द्वारा जेंथोफेरिन उत्पाादित किया जाता है, जो आयरन की अल्प मात्रा वाली परिस्थितियों और रोग जनकता के तहत वृद्धि के लिए आवश्यक है। पुन: इन्होंने पादप सुरक्षा प्रतिक्रिया के उत्प्रेरक के रूप में कोशिका-कोशिका सिग्नलिंग अणु डीएसएफ के कार्य को दर्शाया है।

प्रतिरक्षा विज्ञान प्रयोगशाला द्वारा यह अभिज्ञात किया गया है कि उन्नत ग्लाइकेशन अंतिम उत्पाद (एजीई) जहां मधुमेह के रोगियों में इसका जमाव होता है और बढ़ती उम्र के लोगों में इससे शोथ, एपॉप्टॉसिस, मोटापा और आयु संबंधी विकार साइटोकाइन आईएल-8 माध्यित कोशिका मृत्यु होती है, एनएफ-केबी और एपी-1 द्वारा शोथकारी प्रतिक्रिया बढ़ती है, लाइपोजेनेसिस और ऑटोफेगी बढ़ जाती है।

जीवाणु आनुवंशिकी प्रयोगशाला के अनुसंधानकर्ता ई.कोलाई को एक मॉडल तंत्र के रूप में लेकर बैक्टीरिया के शरीर क्रिया विज्ञान में एक pppGpp कोशिकीय अलार्मोन में K^+ आमरन परिवहन और भूमिका की प्रतिक्रिया को समझने में शामिल हैं।

कोशिका चक्र नियमन प्रयोगशाला द्वारा इस प्रक्रिया को समझा गया है कि आरबीपी2 किस प्रकार पॉकेट प्रोटीन 130 के साथ एच3के4 डिमेथिलेशन करता है और ई2एफ प्रतिक्रियाशील जीनों की जीन अभिव्मक्ति का रिप्रेशन होता है।

प्रतिवेदनाधीन अवधि के दौरान सीडीएफडी ने प्रो. डेविड राइक, जेनेटिक्स विभाग, हार्वड मेडिकल स्कूल, यूएसए और प्रो. रणजीत चक्रवर्ती, आण्विक और चिकित्सा आनुवंशिकी विभाग, यूनिवर्सिटी ऑफ नोर्थ टेक्सास हेल्थ साइंस सेंटर, टेक्सास यू एस ए के सार्वजनिक व्याख्यानों के आयोजन द्वारा डीबीटी की 30वीं वर्षगांठ मनाई।

इस वर्ष भी पिछले वर्ष के समान सीडीएफडी के अनेक संकाय सदस्यों और अध्येताओं को प्रतिष्ठित पुरस्कार और सम्मान प्राप्त हुए हैं। इनमें से कुछ वेलकम ट्रस्ट / डीबीटी इंडिया एलायंस वरिष्ठ अध्येतावृत्ति, राष्ट्रीय महिला जैव

सांख्यिकी पुरस्कार, डीबीटी, भारतीय राष्ट्रीय विज्ञान अकादमी, अंतरराष्ट्रीय अनुसंधान अनुदान जो मानव अग्रणी विज्ञान कार्यक्रम (एचएफएसपी) है और इंडियन इम्यूनोलॉजी सोसायटी द्वारा डॉ. जी पी तलवार यंग साइंटिस्ट पुरस्कार प्रदान किए गए। इस प्रतिवेदनाधीन अवधि के दौरान नौ अनुसंधान अध्येताओं को पीएचडी की उपाधि प्रदान की गई। अनेक पोस्ट डॉक्टरल अध्ये्ता, परियोजना सहयोगी और ग्रीष्मकालीन प्रशिक्षु सीडीएफडी में कार्य करते हैं तथा ये केंद्र के विकास में एक अहम भूमिका भी निभाते हैं।

उप्पल में स्थायी परिसर लगभग जाने के लिए तैयार है। हमारा प्रशासन शीघ्र ही नए परिसर से काम करेगा। प्रयोगशाला खण्ड का निर्माण भी पूरी तेजी से जारी है।

मैं इस अथक सहयोग के प्रति आभार व्यक्त करता हूं जो इसकी गतिविधियों के लिए शासी परिषद, अनुसंधान क्षेत्र पैनल-वैज्ञानिक सलाहकार समिति, शैक्षिक/वित्तीय/भवन समितियों तथा साथ ही बायोटेक्नोलॉजी विभाग की ओर से प्रदान किया गया। मैं डीबीटी के सभी सदस्यों और अधिकारियों को उनके द्वारा दिए गए समर्थन हेतु धन्यवाद देता हूं।

मैं सीडीएफडी परिवार के प्रति भी अपना हार्दिक आभार व्यक्त करता हूं जिसने केंद्र के जारी कार्यक्रमों तथा विकास में एक अहम भूमिका निभाई जिसके बिना कोई प्रगति संभव नहीं होती।

रंजन सेन
प्रभारी निदेशक

31 मार्च 2016

Director's Message

On behalf of my colleagues and myself, here I present the Annual Report of the CDFD for the year 2015-16. The Centre uniquely combines two kinds of activities; i) services in the areas of Human DNA profiling for law-enforcement agencies, diagnostics tests for genetic disorders and analysis of basmati rice for purity, and ii) cutting edge basic research in various disciplines of the modern biology.

The laboratory of DNA Fingerprinting and Services (LDFS) received ~400 cases forwarded by the judiciary and law enforcement and by the investigation agencies of the State and the Federal Governments. LDFS was also actively involved in coordinating with the Department of Biotechnology, CDFD to finalize the draft Bill for enactment by the Parliament of India.

The Diagnostics division provided genetic evaluation to 4859 patients for various genetic diseases. In collaboration with the newly founded Medical Genetics department of at the Nizam's Institute of Medical Sciences, Hyderabad, CDFD is successfully conducting a DNB program in Medical Genetics and a fellowship program in Clinical Cytogenetics and Clinical Molecular Genetics. In addition to these, molecular analyses of novel mutations of different lysosomal storage disorders were performed. Human exosome analyses of the families having rare genetic disorders were also undertaken.

The Laboratory of Molecular Genetics continued their research into the molecular basis of the sex determination in the silk worm. Further they have deciphered the roles of Nodular homologue in Drosophila, Dmnodular, in regulating the transcription factor, NF-κB.

The Laboratory of Chromatin Biology and Epigeneticsis involved in understanding the roles of fission yeast sirtuin Hst4 in DNA replication and damage.

Efforts were made by the Laboratory of Computational Biology to formulate a new substitution scoring matrix suitable for aligning disordered regions as well as a new method for predicting the functional impact of missense mutations found in disordered regions of proteins. A relational database and a software suite were developed to store the information on the volatile metabolite compounds detected from breath, urine and saliva samples of cancer patients as well as the healthy individuals.

Using proteomic approaches, the Lab of Cell Death & Cell Survival has mapped a detailed interaction network of the 143 human phosphatases. These analyses have linked several phosphatases with new cellular processes and unveiled protein-protein interactions genetically linked to various human diseases including cancer.

The Laboratory of Molecular Oncology has undertaken studies in the following aspects. I) Elucidating the role of PAR6G in the PAR complex, ii) suggested that Ca^{2+}/NFAT signalling to be enriched in Wnt- rectal cancer and iii) characterized novel HED causing mutations in the Indian population.

Laboratory of Transcription has deciphered the molecular basis of modulation of Rho-dependent transcription termination by NusG. They also reported methodologies to isolate mycobacteriophages genes capable of killing Mycobacterium species.

The Laboratory of Cell Signalling had demonstrated that the IP7 as well as the gene *IP6K1* is involved in various physiological pathways, such as, chemotaxis of cancer cells, dynamics of motor protein dyenein.

Laboratory of Drosophila Neural Development studied the molecular basis of functions of Hox family of transcription factors in regulating specific identity along the anterior posterior axis of the central nervous system. They have focused to get insights into the autoregulation of the Hox gene, Deformed.

Laboratory of Fungal Pathogenesis has demonstrated that the pathogenic yeast *Candidaglabrata* respond to high external iron levels via activation of two stress-responsive mitogen-activated protein kinases, the CgHog1 and the CgSlt2, and that the CgHog1 kinase

is pivotal to maintenance of iron homeostasis, adherence to biotic and abiotic surfaces and virulence of *C.glabrata.*

Studies from the laboratory of Molecular Cell Biology hinted to an existence of a link between IRAK3, MKP-1 and MAPK signalling cascades downstream of TLR2 that plays an important role in dictating the pro- and anti-inflammatory cytokine responses in tuberculosis. Further they shown that the expression of the PE11 protein of *M. tuberculosis* in a non-pathogenic surrogate *M. smegmatis* could confer its properties akin to typical virulent mycobacteria including increased cell wall integrity, resistance to environmental stress, improved survival inside host.

The Laboratory of Mammalian Genetics has dissected out the role of DNA methyltransferases Dnmt3l and Dnmt2 in carcinogenesis and development. The laboratory has also identified epigenetic changes that the host cell undergoes when challenged with *M. tuberculosis.*

Research from the laboratory of Plant Microbe interaction has demonstrated for the first time that the plant pathogen *Xanthomonascampestrispv. campestris* produces xanthoferrin, which is required for growth under low-iron conditions and virulence. Further, they have shown that the cell-cell signalling molecule DSF act as an elicitor of the plantdefence response.

The Laboratory of Immunology had identified that the advanced glycation end products (AGE) that accumulate in diabetic patients and aging people causing inflammation, apoptosis, obesity and age-related disorders are due to cytokine IL-8 mediated cell-death, increased inflammatory responses by NF-κB. and AP-1, increased lipogenesis and autophagy.

Researchers from laboratory of Bacterial Genetics are involved in understanding the mechanism of K^+ ion transport and roles of cellular alarmonepppGpp in bacterial physiology using *E. coli* as model system.

The laboratory of cell cycle regulation has delineated the mechanism of how RBP2 interacts with the pocket protein p130 to bring about H3K4 demethylation and repression of gene expression of the E2F responsive genes.

During this reporting period, CDFD has celebrated 30th anniversary of DBT by organising public lectures by Prof David Reich, Department of Genetics, Harvard Medical School, USA and Prof Ranajit Chakraborty, Department of Molecular and Medical Genetics, University of North Texas Health Science Center, Texas.

Like previous years, this year too several of the CDFD faculty members and scholars have been recipients of prestigious awards and honours. Few of them include, Wellcome Trust/ DBT India Alliance Senior Fellowship, National Women Bioscientist award by Department of Biotechnology, Fellow of the Indian National Science Academy, International Research Grant by Human Frontier Science Program (HFSP) and Dr G.P. Talwar Young Scientist award by Indian Immunology Society etc. During this period nine research scholars were conferred with Ph D degree. Many postdoctoral fellows, project associates and summer trainees were trained at CDFD, who also played a vital role in the Centre's Development.

The permanent campus at Uppal is almost ready to be occupied. Our Administration will soon be operated from the new campus. The construction of Laboratory Block is also progressing in full swing.

I take this opportunity to acknowledge the unfettered co-operation which the Centre has received for its activities from the Governing Council, Academic/Finance/Building and Research Area Panels-Scientific Advisory Committees and most importantly from the Department of Biotechnology (DBT). I wish to thank all the members and officials of DBT for supporting us.

I also express my sincere thanks to all the members of the CDFD family for their support without which we would not have made any progress.

Ranjan Sen
In-charge Director

March 31, 2016

सेवाएँ
Services

LABORATORY OF DNA FINGERPRINTING SERVICES

Faculty	Madhusudan Reddy Nandineni	Staff Scientist
Other members	SPR Prasad	Senior Technical Officer
	Ch V Goud	Technical Officer
	Devinder Singh Negi	Technical Officer
	Devinder Kumar	Technical Officer
	Sanjukta Mukerjee	Technical Officer
	S. Naveen Chandra	Technical Officer (Till September, 2015)
	Neelima Thota	Technical Officer
	Pooja Tripathi	Technical Officer
	Kiranmai Joshi	Technical Officer
	Girnar Vijay Amrutrao	Technical Assistant
	Shruti Dasgupta	Technical Assistant
	Chandra Shekhar Singh	Technical Assistant
Coordinator	DP Kasbekar	Haldane Chair

Objectives

1. To provide DNA fingerprinting services in cases forwarded by law-enforcing agencies / judiciary of State and Federal Governments, relating to murder, sexual assault, paternity, maternity, child swapping, deceased identification, organ transplantation, etc.,
2. To develop human resources skilled in DNA fingerprinting, to cater to the needs of State and Federal Government agencies;
3. To impart periodical training to manpower involved in DNA fingerprinting sponsored by State and Federal Government agencies;
4. To provide advisory services to State and Federal Government agencies in establishing DNA Fingerprinting facility;
5. To create DNA marker databases of different populations of India.

Summary of services provided until the beginning of the reporting year (upto March 31, 2015)

A total of 559 cases were received for DNA fingerprinting examination during the previous reporting period (2014 - 2015). Of these, 280 cases were related to identification of deceased, 101 cases were related to paternity / maternity, 151 cases were pertaining to sexual assault (rape), 13 cases were related to murder and 14 cases were pertaining to biological relationship (organ transplantation). Eighteen States, Union Territories of India and one foreign country (East Timor) have availed DNA fingerprinting services of CDFD during this period. Madhya Pradesh forwarded the highest number of cases (197), followed by Andhra Pradesh (103), Telangana (79), Chhattisgarh (40), Odisha (18 cases received at ILS, Odisha out of 29), Uttar Pradesh (29), Punjab (26), Goa (15), Tamil Nadu (13), Karnataka (6), Puducherry (5), Kerala (4), Maharashtra (3), Delhi (2), Jammu & Kashmir (1), West Bengal (1) and East Timor (1).

Details of services provided in the current reporting year, (April 1, 2015 – March 31, 2016):

Breakup of the cases during this reporting period is given below under following heads:

Biological Relationship	19
Identity of Deceased	162
Murder	19
Paternity/Maternity	98
Sexual Assault (Rape)	99
Total number of cases	**397**

Prominent cases during April 1, 2015 to March 31, 2016

1) Cases from National Investigation Agency (NIA) involving national security and public safety
2) DNA profiling of relatives of deceased Indians in Fly Dubai Aircraft crash in Russia
3) Terror attack on BSF convoy in Udhampur District, Jammu & Kashmir
4) Sexual assault case of two women tourists from Delhi in Goa

Deposition of evidence in Courts of Law

During this reporting year, the DNA experts defended their reports in 25 cases in various Honorable Courts throughout the country.

Training:

Training in DNA fingerprinting procedures was provided to Senior Scientific Officers from Forensic Science Laboratory, Haryana, Madhuban during 22.06.2015 to 26.06.2015

Summary of the State-wise breakup of DNA Fingerprinting Cases

Name of the State	Biological relationship	Identity of deceased	Maternity / Paternity	Murder	Sexual Assault (Rape)	Total No. of Cases
Andaman & Nicobar			2			2
Andhra Pradesh		22	4	1		27
Bihar		1	1			2
Chhattisgarh		23	21	1	4	49
Delhi		1				1
Goa		9	9		1	19
Haryana		1			1	2
Himachal Pradesh		1				1
Karnataka	1		4			5
Kerala		2	1			3
Madhya Pradesh	1	51	35	17	72	176
Maharashtra			3			3
Odisha			1			1
Puducherry		2	2		1	5
Punjab			2		19	21
Rajasthan			1			1
Tamil Nadu	16					16
Telangana	1	45	9			55
Uttar Pradesh		2			1	3
West Bengal		2				2
East Timor			3			3
Total No. of Cases.	**19**	**162**	**98**	**19**	**99**	**397**

A total of 397 cases were received for DNA fingerprinting examination during the current reporting period (2015 - 2016). Of these, 162 cases were related to identification of deceased, 99 cases were pertaining to sexual assault (rape), 98 cases were related to paternity / maternity, 19 cases were related to murder and 19 cases were pertaining to biological relationship (organ transplantation). Twenty States and Union Territories of India and one foreign country (East Timor) have availed the DNA fingerprinting services of CDFD during this period. Madhya

Pradesh forwarded the highest number of cases (176) followed by Telangana (55), Chhattisgarh (49), Andhra Pradesh (27), Punjab (21), Goa (19), Tamil Nadu (16), Puducherry (5), Karnataka (5), Kerala (3), Maharashtra (3), East Timor (3), Uttar Pradesh (3), Andaman & Nicobar (2), Bihar (2), Haryana (2), West Bengal (2), Delhi (1), Himachal Pradesh (1), Odisha (1) and Rajasthan (1), (Fig.1).

Revenues generated:

During this reporting period, an amount of **73,38,639 /-** (Rupees Seventy three lakhs thirty eight thousand six hundred and thirty nine only) has been received towards DNA fingerprinting analysis charges, which is inclusive of service charge as levied by Govt. of India.

The cases involving identification of the deceased (41%), paternity (24%), sexual assault (25%), murder (5%) and biological relationship (5%) constituted the bulk of the cases received (Fig.2).

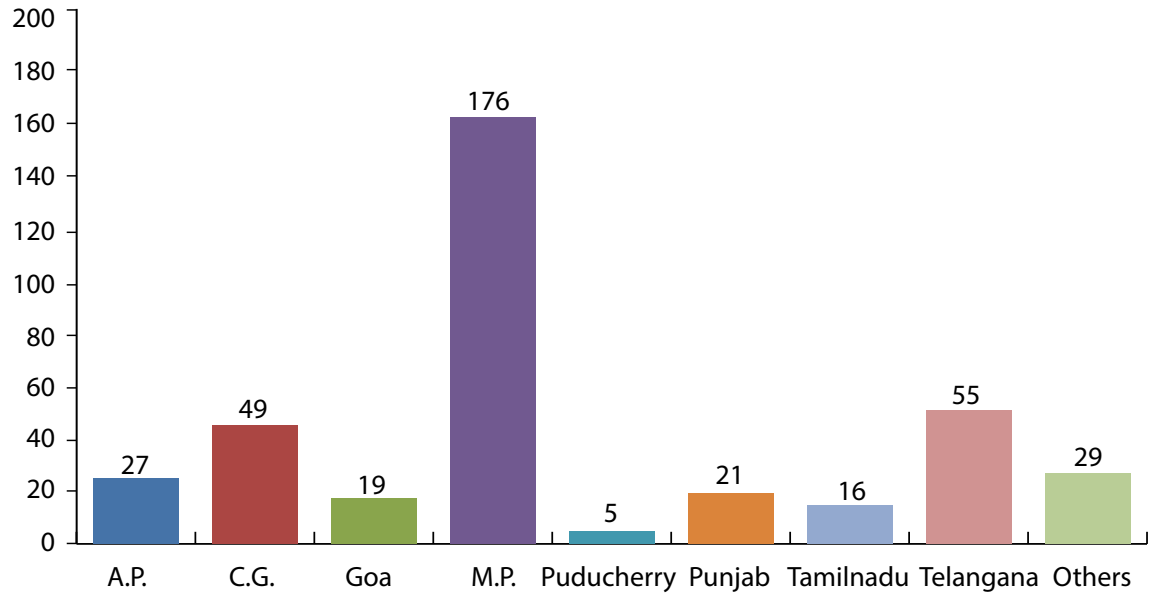

Figure 1. State-wise number of cases received

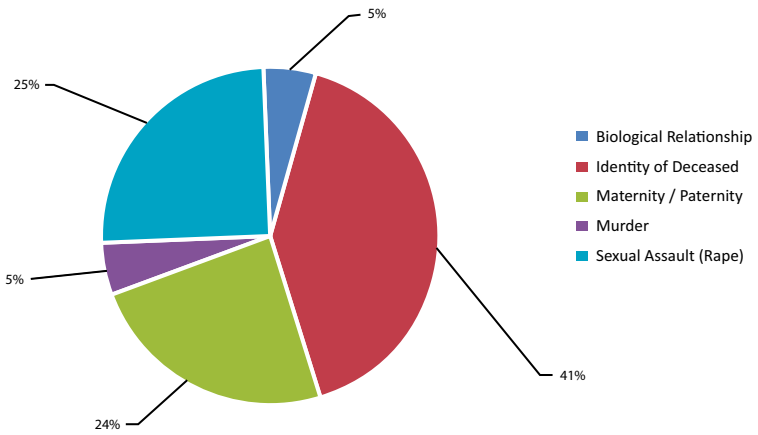

Figure 2. Types of cases received (%)

DIAGNOSTICS DIVISION

Faculty	Ashwin Dalal	Staff Scientist
Adjunct Faculty	Prajnya Ranganath	Associate Professor, NIMS
	Shagun Aggarwal	Associate Professor, NIMS
PhD Students	Anusha Uttarilli	Senior Research Fellow
	Ashish Bahal	Junior Research Fellow (till May 2015)
	Anjana Kar	Senior Research Fellow
	Deshpande Dipti Vijayrao	Junior Research Fellow
Other Members	Aneek Das Bhowmik	Research Associate
	Maria Celestina Vanaja	Research Associate
	Vineeth VS	Research Associate (since June 2015)
	Sowmya Gayatri	SIAMG Fellow (till Feb 2016)
	Avinash Pagdhune	SIAMG Fellow (since September 2015)
	Krishna Reddy CH	SIAMG Fellow (since September 2015)
	Divya Matta	Project Junior Research Fellow (till August 2015)
	P Divya	Project Junior Research Fellow (since August 2015)
	P Rajitha	Technical Officer III
	Angalena R	Senior Technical Officer
	Dutta Usha Rani	Technical Officer
	M Muthulakshmi	Technical Officer
	A Sobhan Babu	Technical Officer
	S Jamal Md Nurul Jain	Technical Officer
	S Vasantha Rani	Technical Officer
	C. Krishna Prasad	Technician
	R. Sudheer Kumar	Technician

Objectives

1. To conduct genetic evaluation for patients/ families with genetic disorders;
2. To develop new methods and assays for genetic analysis and engage in research on chromosomal and single gene disorders;
3. To act as national referral center for analysis and quality control of genetic tests for few genetic diseases; and
4. To impart training in genetic evaluation of patients with genetic disorders.

Details of services provided in the current reporting year (April 1, 2015-March 31, 2016)

Clinical Genetics

A total of 4859 patient samples were analyzed for genetic testing, during the year 2015-16. These consisted of patients with chromosomal disorders, monogenic disorders, mental retardation, congenital malformations, inborn errors of metabolism, and other familial disorders. The SIAMG fellowship program for training in Clinical Cytogenetics and Clinical Molecular Genetics has been initiated in collaboration with Society

for Indian Academy of Medical Genetics. One student each joined for the fellowship program and one student completed the fellowship in Clinical Cytogenetics during 2015-16.

The Department of Medical Genetics established at Nizam's Institute of Medical Sciences, Hyderabad is functioning successfully. A total of 3354 patients were examined and counseled in the unit during 2015-16. In addition antenatal ultrasonograms were done in 306 cases, antenatal invasive procedures (chorionic villus sampling and amniocentesis) in 119 cases and foetal autopsies were conducted in 82 foetuses. A 3 year training program for Diplomate of National Board (DNB) in Medical Genetics initiated with affiliation to National Board of Examinations, New Delhi is running successfully.

Genetic investigations done during 2015-16

Investigation	Total cases	Positives
Cytogenetics	1626	117 (7%)
Proband	1446	107 (7.3%)
Prenatal	180	10 (5.5%)
Molecular Genetics	2324	850 (36.5 %)
Proband	2175	807 (37 %)
Prenatal	149	43 (29 %)
Biochemical Genetics	909	255 (28.0%)
Proband	893	248 (27.7%)
Prenatal	16	7 (43.75%)

Cytogenetics

Disease	Abnormality	No of cases
Down	47,XY,+21	39
Syndrome	47,XX,+21	14
	46,XX,rob(13;21) +21	1
	46,XX,rob(21;21) +21	1
	46,XY,rob(21;21)+21	1
	46,XX,rob(13;14)+21	1
	47,SC,+21	2
Edward syndrome	47,XX,+18	1
Patau Syndrome	47,SC,+13	1
Turner syndrome	Monosomy X (45,X)	4
	mos 45,X/ 46,X,r(X)	1
	mos 45,X/46,X,i(X)	2
	mos 46,X,del(X)(p21p22.3)/45,X	1
	46,X,i(X)(q10)	1
	mos 46,XY/46,XX	1
Klinefelter	47,XXY	5
Syndrome	47,SC, XXY	1
Triple X Syndrome	47,XXX	1

Structural chromosomal abnormalities

Inversions	
46,XX,inv(3)	1
46,X,inv(Y)	2
46,XX,inv(15)(q21.3q24)	1
46,XY,inv(9)	1
Duplications	
46,XY,add(1q36)	1
46,XX,15p+	3
46,XY,15p+	1
Marker	
mos 47,XY,+marker/46,XY	1
Translocations	
46,XY,t(5;10)(p15;q24)	2
46,XY,t(13;15)(q22;q22)	1

46,XX,t(4;13)(q31;q14)	1
46,XX,t(11;22)(q23;q11.2)	1
46,XX,t(11;13)(q24;q12)	1
46,XX,t(1;9)(p36.1;p23)	1
46,XX,der(4),t(4;13)(p31;q14)mat	1
45,XX,rob(13;14)(q10;q10)	1
46,XX,t(15;16)(q11.1;q11.1)	1
46,SC,t(15;16)(q11.1;q11.1)mat	1
46,SC,der(5),t(5;11)(p15.1;p11.2)pat	1
46,SC,der(15),t(9;15)(p13;p11)pat	1
45,SC,t(13;14)(q11.1;q11.1)pat	1
46,SC,t(5;10)((p15;q24)pat	1
Polymorphic variants	13

Fluorescence *in situ* Hybridization (FISH)

Disease/translocation	Probe	No of tests
Prader-Willi Syndrome	SNRPN(15q11)/PML(15q24)	4
1p36 deletion syndrome	1p36 probe	2
Di-George Syndrome	TUPLE(22q11.2)/ARSA(22q13)	6
Marker chromosomes	WCP-11, WCP-13, 9, 18 SE(X)/(Y), Acro-p-arm	10
Spectral karyotyping		4

Quantitative Fluorescent PCR (QF-PCR)

MLPA	Cases	Positives
Prenatal (Aneuploidy)	83	6
Postnatal (Microdeletion syndromes)	125	10

Biochemical Genetics

Disease/Test	Positives
Urine & Blood Metabolic Screening tests (N=225)	**74**
Amino acid disorders (N=201)	**51**
Non Ketotic Hyperglycinemia	16
Hyperornithinemia	5
Tyrosinemia	2
Phenylketonuria	3
MSUD	3
Hyperprolinemia	3
Other amino acid disorders	19

Disease/Test	Positives
Lysosomal storage disorders (N=467)	**123**
Hurler syndrome(39)	16
Hunter syndrome(33)	17
Sanfilippo B (23)	7
Morquio A disease (32)	12
Maroteaux Lamy syndrome (9)	3
Sly disease (13)	1
GM1-Gangliosidosis (74)	4
Gaucher disease (49)	6

Krabbe disease (24)	4
Pompe disease (13)	2
Niemann Pick disease (42)	19
Mucolipidosis(9)	8
Metachromatic Leukodystrophy (59)	15
Fabry disease(12)	4
Hexosaminidase A/B (36)	
Tay Sachs disease	1
Sandhoff disease	4

Prenatal diagnosis (16)	**7**
Mucolipidosis (1)	0
Sanfilippo B (1)	0
Metachromatic Leukodystrophy (1)	1
Gaucher disease (2)	1
Hurler syndrome (2)	1
Maroteaux Lamy syndrome (1)	0
Morquio A disease (2)	2
GM1- Gangliosidosis (2)	1
Niemann Pick disease (4)	1

Molecular Genetics

Name of disorders	**No of cases**	**Positive**	**Negative**		
DMD/BMD	294	181	113		
DMD Carrier Analysis	69	22	47		
Spinal Muscular Atrophy	163	72	91		
SMA Carrier Analysis	47	23	24		
		Normal	**Homozygous**	**Heterozygous**	**Compound Heterozygous**
β thalassemia and Sickle cell anemia	421	30	228	68	95
Factor V Leiden	276	269	01	06	NA
Factor II mutation	191	191	0	0	NA
Cystic Fibrosis	114	101	05	08	NA
Pancreatitis	22	18	03	01	NA
Connexin 26	18	12	02	04	0
Achondroplasia	12	08	0	04	NA
Hemophilia	11	08	01	02	NA
Gilbert Syndrome	39	05	30	04	NA
LHON disease	2	2	0	0	NA
Leigh disease	3	3	0	0	NA
MTHFR	13	08	0	05	NA
Triplet Repeat Disorders		**Positive**	**Negative**		
Friedrichs Ataxia	59	14	45		
Myotonic Dystrophy	72	44	28		
Huntington Disease	56	34	22		
SCA Panel (1,2,3,6 &7)	104	36	68		
DRPLA	13	0	13		
Spinobulbar Muscular Atrophy (SBMA)	2	1	1		
Fragile X Syndrome	174	15	159		

NA- Not applicable

MOLECULAR GENETICS--PRENATAL DIAGNOSIS

Prenatal Diagnosis	No of cases	Positive	Negative		
DMD	09	01	08		
Spinal Muscular atrophy	42	10	32		
Cystic Fibrosis	8	0	8		
Myotonic dystrophy	2	0	2		
Fragile X Syndrome	1	1	0		
Hemophilia	2	0	2		
		Normal	Homozygous	Heterozygous	Compound Heterozygous
β thalassemia	84	11	24	43	06
Connexin	1	0	1		

II. Diagnostics Research

Project 1: Human exome sequencing for identification of novel genes in rare mendelian disorders

Summary of work done until the beginning of this reporting year (upto March 31, 2015)

Single gene disorders are rare by themselves but collectively they are an important cause of morbidity and mortality. The identification of genes for single gene disorders has value, not only in prenatal diagnosis and genetic counselling of affected families, but also in basic research towards understanding gene functions and mechanisms of disease. Till date more than 3000 genes causing single gene disorders have been identified using classical linkage analysis methods but still a large number remains to be characterized. The availability of massively parallel sequencing technologies have made it possible to identify gene for a particular disease using just a few affected individuals. Over the past years of providing services in clinical genetics, we have identified several interesting novel disorders and syndromes with a single gene pattern of Mendelian inheritance. We plan to employ exome sequencing to identify novel genes in such families.

Details of work done in the current reporting year (April 1, 2015 – March 31, 2016)

We have performed exome sequencing for two families with rare autosomal recessive disorders last year and identified disease causing variant in *BHLHA9* gene in a family with Camptosynpolydactyly (OMIM: 607539) and in *BUB1B* gene in another family with two female siblings affected with microcephaly, macular degeneration, Wilm's tumour and short stature. During the reporting year we have recruited a family with three siblings (including two male and one female) affected with intellectual disability, ptosis and polydactyly, born out of consanguineous marriage. Array CGH for identification of common homozygous region was done in all the affected individuals. This helped us in narrowing to five common homozygous regions of 16 Mb size containing 228 genes. Exome sequencing using Illumina NGS platform followed by mapping of reads to reference genome (hg19) and detection of variants was done. Filtering of known SNPs, 1000G variants (MAF≥0.01), ExAC variants (MAF≥0.01) and in-house exome database variants revealed 6 common homozygous variants among the siblings. Among these six variants, c.879G>A in *ARMC9* gene was predicted to be disease causing and thus considered as candidate gene for the disorder. c.879G>A is a synonymous variant altering the splicing site in exon 8 of *ARMC9* gene. *ARMC9* gene codes for Armadillo repeat containing 9 protein, which is an interacting partner of *SIAH1* (Siah E3 ubiquitin protein ligase 1) and *CMTM5* (CKLF like MARVEL trans-membrane domain containing protein family 5). Little is known about *ARMC9* and interaction with *SIAH1* indicates that it may be a part of ubiquitination pathway. Sanger sequencing and validation of variant has been done in all affected individuals and parents, which shows autosomal recessive segregation pattern. Functional characterization is being planned to characterize the mutation and its effects on protein function.

Project II: Clinical, biochemical and molecular analysis of lysosomal storage disorders

Summary of work done until the beginning of this reporting year (upto March 31, 2015)

Lysosomal storage disorders are a heterogeneous group of disorders associated with specific lysosomal enzyme deficiency. The diagnosis in most of these disorders is based on enzyme assay. There is a large amount of overlap in enzyme levels among carriers and normal people; hence it is very difficult to detect carriers by enzyme assay. Mutation detection is helpful for carrier detection and accurate prenatal diagnosis. Our study aims to characterize the clinical features, biochemical parameters and molecular defects in various lysosomal storage disorders

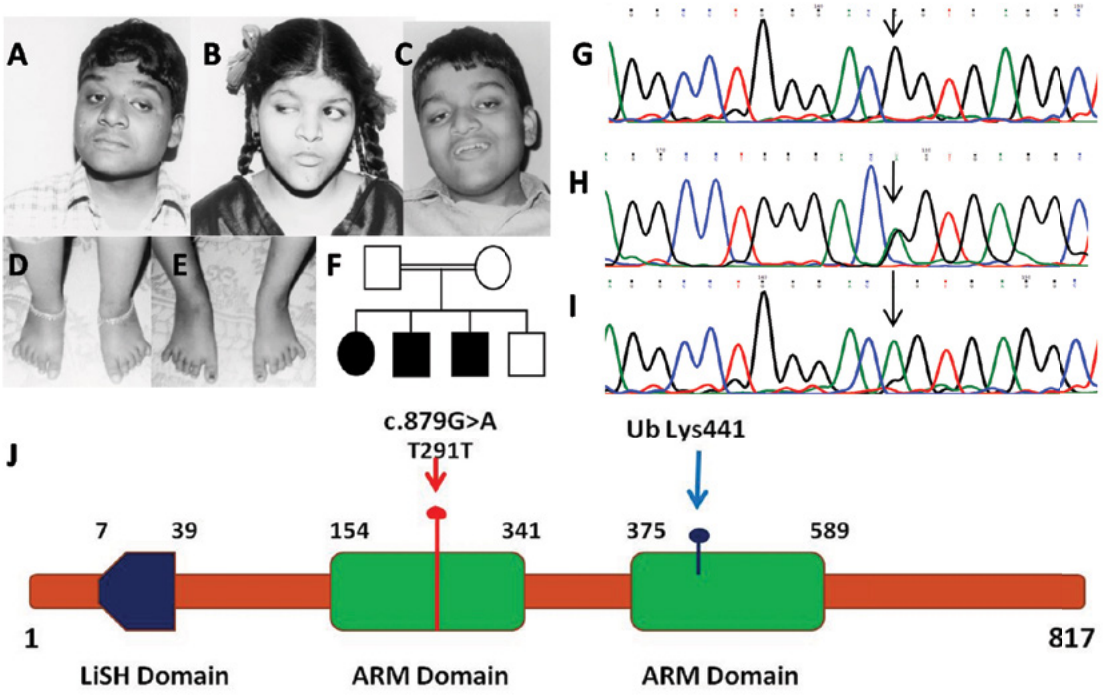

Figure 1. Candidate gene identification in family with rare autosomal recessive disorder.

A – E : Photograph of affected children showing down slanting palpebral fissure, ptosis and bilateral postaxial polydactyly.

F : Pedigree of family with mental retardation, ptosis and polydactyly

G – I : Sanger sequencing chromatogram of Control (Normal), Parent (Heterozygous) and patient (homozygous showing c.879G>A indicated by arrows.

J : Schematic illustration of *ARMC9* with location of ARM domains. Mutation c.879G>A indicated by red arrow and ubiquitination site at Lys441 indicated by blue arrow, which may be altered due to splicing defect caused by mutation.

Details of work done in the current reporting year (April 1, 2015 – March 31, 2016)

Over last six years we have been able to identify mutations in more than 250 patients with different lysosomal storage diseases (LSDs) (Table 1). This study has revealed the mutation spectrum in patients with LSDs in the Indian population.

Lysosomal Storage Disorder	Gene	Number of cases	Total mutations	Novel mutations
Niemann-Pick disease types A & B	SMPD1	81	60	26
Metachromatic leukodystrophy	ARSA	79	56	23
Mucopolysaccharidosis I	IDUA	31	22	15
Mucopolysaccharidosis II	IDS	33	20	7
Mucopolysaccharidosis VI	ARSB	38	24	18
Sialidosis	NEU1	5	3	3
Total		**250**	**185**	**92**

Table 1. Data sheet showing mutation analysis for LSDs

During the reporting year, we have done mutation analysis for 64 patients as shown in Table 2 which further revealed the mutation spectrum of these diseases. This was done as part of a National Task Force on Lysosomal Storage Diseases funded by Indian Council of Medical Research and Department of Health Research.

Lysosomal storage Disease	Gene	No. of patients
Sialidosis	NEU1	5
I-Cell disease	GNPTAB/GNPTG	23
Niemann Pick Disease	SMPD1	28
Mucopolysacharidosis Type VI	ARSB	8
Total		**64**

Table 2. Data sheet showing mutation analysis for LSDs in NTF-LSD project

In addition, we have started a new project this year on development of a next generation sequencing based assay for mutation analysis for lysosomal storage disorders. While Sanger sequencing is very useful for sequence analysis of small genes, when applied for large genomic regions it becomes time consuming and laborious, requiring multiple PCR reactions for generating amplicons for sequencing. The development of high throughput massively parallel sequencing strategies in recent years has revolutionized the concept of sequence analysis and has made sequencing of large genomic segments far more feasible and much less time-consuming. In the present project we plan to amplify about 5 kb fragments of genomic DNA from specific lysosomal storage disease gene and then pool the samples for next generation sequencing based analysis. Pooling of samples from different individuals with different affected genes will help to decrease the cost of sequencing significantly. We have standardized Long PCR for following genes: ARSA, SMPD1, IDUA, NEU1, and are analyzing the results received from first run of the multiplexed NGS reaction.

Publications

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2. Gupta N, Benjamin M, Kar A, Munjal SD, Sarangi AN, Dalal A, Aggarwal R (2015). Identification of Promotor and Exonic Variations, and Functional Characterization of a Splice Site Mutation in Indian Patients with Unconjugated Hyperbilirubinemia. ***PLoS One*** 10(12):e0145967.
3. Das Bhowmik A, Rangaswamaiah S, Srinivas G, Dalal AB. (2015) Molecular genetic analysis of trinucleotide repeat disorders (TRDs) in Indian population and application of repeat primed PCR. ***European Journal of Medical Genetics*** 58(3):160-167.
4. Stephen J, Girisha KM, Dalal A, Shukla A, Shah H, Srivastava P, Kornak U, Phadke SR. (2015) Mutations in patients with osteogenesis

imperfecta from consanguineous Indian families. ***European Journal of Medical Genetics*** 58(1):21-27.

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* Partial work done in CDFD

APEDA-CDFD CENTRE FOR BASMATI DNA ANALYSIS

Members	VV Satyavathi	Technical Officer
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	VLN Reddy	ANGRAU, Hyderabad
	A Srividya	ANGRAU, Hyderabad
Coordinator	G R Chandak	Director

Objectives

1. Testing the purity of Basmati samples received from Export Inspection Council (EIC), Ministry of Commerce, Government of India, Basmati rice exporters from India, and other countries; and
2. Molecular dissection of a QTL governing grain size in Basmati rice.

Summary of the work done until the beginning of this reporting year (upto March 31, 2015)

The work undertaken in earlier years under objective 2 has been summarized in the first part of the corresponding description below.

Details of progress made in the current reporting year (April 1, 2015 - March 31, 2016)

Objective 1: Testing the purity of Basmati samples received from Export Inspection Council (EIC), Ministry of Commerce, Govt. of India, Basmati rice exporters from India and other countries.

During the period under report, a total of 209 Basmati samples were analyzed and the number of samples indicating the percentage of adulteration with non-basmati rice is shown in Figure 1.

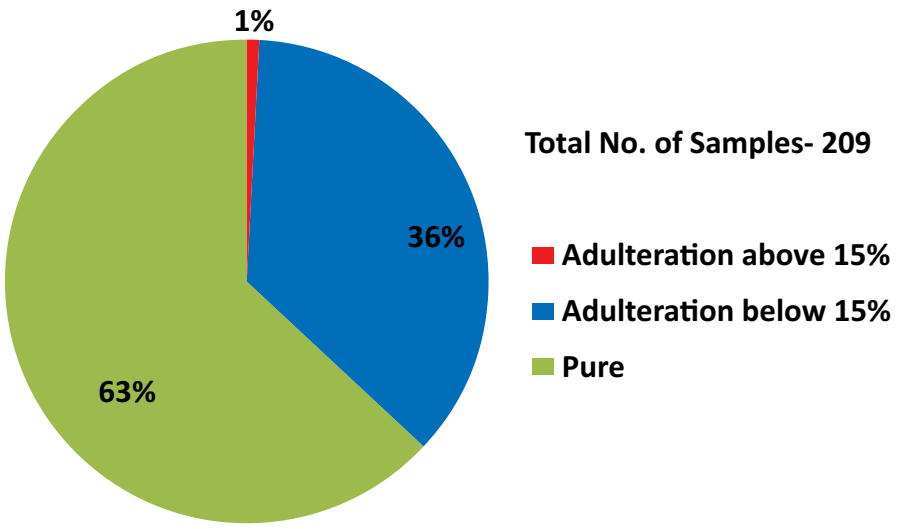

Figure 1. Basmati samples analyzed at APEDA-CDFD Centre in the current reporting year.

The protocol for DNA testing of Basmati rice was initially developed for detection of adulteration using eight Simple Sequence Repeats (SSRs) marker assay with eleven notified Basmati varieties. In view of the complexities and challenges arising in adulteration testing, efforts are being made to further expand as well as fine tune the present protocol as mentioned below:

i) Updating the database of Basmati varieties

At present twenty varieties of Basmati rice have been notified by Department of Agriculture and Cooperation (DAC) under Seeds Act, 1966. We have extended our method of multiplexed eight markers panel analysis for identification of all the twenty notified varieties to generate a comprehensive database.

ii) Single grain analysis for varietal identification

On the unknown rice samples, where the sample was predominantly one variety, the identification using our standardized method is in good agreement. However, for identification of rice varieties in samples of complex mixtures, single grain analysis is now being used.

iii) Increase the number of markers (SSRs) & employ SNPs for better resolution of complex mixtures and varietal identification

With the constant release of new rice varieties, it becomes imperative to incorporate more number of SSR markers in the present assay. The SSRs that are highly discriminatory between the various rice varieties are being identified.

Objective 2: Molecular dissection of a QTL governing grain size in Basmati rice.

Grain size is one of the most important characters that determine the quality of Basmati rice from consumers as well as traders point of view. Though many genes governing grain size have been identified in *indica* and *japonica*, little work has been done in Basmati rice. Ninety six diverse rice germplasm viz. aromatic (27), *indica* (45), *japonica* and javonica (19) and aus (5) groups; which differ significantly for grain size traits were screened with a total of 55 SSR markers.

During the period under report, association mapping has been carried out with three SSRs, RM 6024 (grain breadth), RM1237 and RM18582 (grain length breadth ratio), which were identified as 'constitutive QTL' markers associated with grain size. Fine mapping was carried out using 39 SSR markers by screening 410 F2 populations derived from a cross between Jaya and Basmati370. About 7 polymorphic markers in the marker interval RM6024-RM18582 accounting for 18 percent polymorphism were identified. The QTLs for grain size, thousand grain weight and panicle number were found to be clustered in the region RM6024-RM18550 with a physical distance of 268 kb which is novel and unique to Basmati. The candidate gene prediction by semiquantitative PCR, qTELLER and nonsynonymous SNPs revealed that zinc finger transcription factors, cytochrome p450 (brassinosteroid signaling) and tetratricopeptide like proteins in the QTL cluster were involved in regulating grain length whereas ubiquitin mediated protein, degradation proteins and cytokinin oxidase 1 were involved in grain breadth.

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*work done outside CDFD

शोध
Research

LABORATORY OF BACTERIAL GENETICS

Studies on gene regulation, transcription termination, and amino acid and ion-transport in *Escherichia coli*

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	Abhijit A Sardesai	Staff Scientist
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	Nalini Raghunathan	Senior Research Fellow
	Rajeshree Sanyal	Senior Research Fellow
	Nida Ali	Senior Research Fellow
	Ravish Sharma	Senior Research Fellow
	J Mallikarjun	Junior Research Fellow
	Sayantan Goswami	Junior Research Fellow
	Swati Dubey	Junior Research Fellow
	Vani Singh	Junior Research Fellow(since June 2015)
	Prajakta Tathe	Junior Research Fellow (since July 2015)
Other Members	V K Mishra	Staff Scientist
	K Anupama	Staff Scientist
	J Krishna Leela	Technical Officer
	T S Shaffiqu	Technical Officer
	Vimala Allada	Research Associate
	P Hima Bindu	Research Associate
	Saswat Mohapatra	Research Associate (Since Dec.2015)

The Laboratory of Bacterial Genetics comprises three faculty groups engaged in research on several aspects of the physiology and genetics of *Escherichia coli*, and is majorly supported by the Department of Biotechnology as a Centre of Excellence for Microbial Biology. The work undertaken in this reporting year is described below under the following objectives:

Objectives

1. To understand the pathology of RNA-DNA hybrids (R-loops) and the mechanisms of their avoidance;
2. Studies on essentiality and oligomerization features of RNase E in *E. coli*;
3. Delineation of a cryptic pathway for potassium translocation in *E. coli*;
4. Studies on basic amino acid export in *E. coli*;
5. To understand the genetic interactions between (p)ppGpp and the tm-RNA system leading to modulation of transcriptional polarity by (p)ppGpp.
6. To determine the role of (p)ppGpp in modulation of cell division;
7. Consequences of glycerol stasis in the *glpD* mutant of *E.coli*;
8. Does basal (p)ppGpp modulate chromosomal replication?

Summary of work done until the beginning of this reporting year (upto March 31, 2015)

The work undertaken in earlier years on each of the objectives has been summarized in the first parts of the corresponding descriptions below.

Details of progress made in the current reporting year (April 1, 2015-March 31, 2016)

1. Occurrence of pathological R-loops and their consequences in *E. coli.*

Our laboratory has for several years been pursuing the hypothesis that nascent transcripts in *E. coli* are prone to re-anneal with the upstream template DNA strand to generate pathological RNA-DNA hybrids or R-loops which can act to impede transcription and replication. According to this model, R-loop occurrence is avoided or minimized by engagement of the nascent transcripts with translating ribosomes (ie., transcription-translation coupling), and in the absence / failure of such coupling by the termination of transcription mediated by proteins Rho and NusG. We had shown earlier that the lethality of knockout mutations in *rho* or *nusG* can be rescued by ectopic expression of UvsW, an R-loop helicase from phage T4; and that R-loops (as detected by a bisulphite-sensitivity assay) are distributed genome-wide with several defined hotspots in the bacterial cells, including those inferred to be generated from antisense transcripts.

R-loops are known also to be sites of aberrant chromosomal replication initiation (that is DnaA- and *oriC*-independent), which is referred to as constitutive stable DNA replication (cSDR). Based on our finding that R-loops are distributed genome-wide, we have earlier suggested that cSDR origins are also widespread, each however only with a very small and stochastic firing potential.

In the current year, we have compared the genomic positions of R-loop prevalence, that have been detected by us in the bisulphite-sensitivity assay, with two other published datasets: (i) an algorithmic prediction of R-loop forming sequences in the *E.coli* genome (Jenjaroenpun et al., Nucleic Acids Research, 2015, 43:10081), and (ii) an identification (Peters et al., Genes and Development, 2012, 26:2621-2633) of approximately 900 antisense transcripts whose abundance is increased in presence of the Rho inhibitor bicyclomycin (ie., these are transcripts that are normally not present because of the action of Rho in terminating their synthesis).

In the former dataset, twenty six R-loop prone sequences had been computationally predicted in the *E.coli* genome (without prior knowledge of whether or not they are transcribed), and we have found that eleven of them exactly match the strand-specific hotspots of bisulphite sensitivity that were detected in our earlier studies. We believe that this indeed provides strong support for the notion that bisulphite sensitivity indeed is a marker of R-loop prevalence in the cells.

Comparison of the bisulphite-sensitivity data with the second dataset of Peters et al led to a very interesting finding: that the prevalence of Rho-sensitive antisense transcription (as detected in RNA-Seq experiments) is inversely correlated with the propensity for bisulphite sensitivity, that is, the loci exhibiting high bisulphite sensitivity were less likely to be represented in the antisense transcription dataset and vice versa. We suggest that this counterintuitive result may be explained by a model positing that antisense transcripts from a very highly bisulphite-sensitive locus immediately form R-loops and consequently inhibit further transcription, so that their abundance in the RNA-Seq experiments would be low; on the other hand, R-loop formation would be less prevalent from antisense transcripts at the loci that are not bisulphite-sensitive, and these transcripts would therefore be detected by RNA-Seq. One prediction from this model, which we intend to test in future experiments, is that the present RNA-Seq approaches tend to underestimate the propensity for antisense transcription in E. coli, which can be more accurately assessed by performing RNA-Seq in *rho* or *nusG* knockout strains expressing the R-loop helicase uvsW.

The additional studies currently being undertaken in this component of the project are directed towards (i) determining the various situations in which cSDR can be detected and the genetic requirements for cSDR under these conditions; (ii) understanding the mechanism(s) of cSDR, including through next-generation-sequencing-experiments; (iii) quantification of R-loop prevalence in different strains with the aid of the monoclonal antibody S9.6 that is specific for RNA-DNA hybrids; and (iv) employment of in vitro transcription approaches to examine Rho-dependent transcription termination in the presence of nucleoid-binding proteins.

2. Essentiality and oligomerization features of RNase E in *E. coli.*

RNase E is an endonuclease that is essential for viability in *E. coli*, which functions both for stable RNA processing as the rate-limiting enzyme for

mRNA degradation. The salient features of RNase E are that (i) it is a homotetramer of a polypeptide of 1061 amino acid residues; (ii) its catalytic activity resides in the N-terminal half of the protein, with the C-terminal half being dispensable for viability; and (iii) its activity is modulated by the nature of the 5'-end of the substrate, being maximal on 5'-monophosphorylated RNA. The crystal structures of the tetrameric N-terminal half of RNase E in both apo-form and with bound RNA have been determined, which indicate that (i) the 5'-RNA end is recognized by a pocket in the enzyme (that includes residues R169 and T170) which is distinct from the active site; and (ii) the RNA is so positioned that its 5'-end is in one subunit of the oligomer while the endonucleolytic scission would take place in an adjacent subunit.

We had previously shown that an RNase E variant with truncation of its C-terminal half along with an R169Q substitution in its 5'-end recognition pocket is lethal. Work undertaken by us in the current year suggests that this lethality can be suppressed by perturbations that reduce the expression of stable RNA in the cells; these perturbations include (i) increase in basal ppGpp levels, (ii) introduction of "stringent" RNA polymerase mutations; (iii) over-expression of the protein DksA; and (iv) reduction in the number of ribosomal RNA operons in the genome from the normal seven to three or to two. We hypothesize that the reduced stable RNA levels under these conditions minimize the need of RNase E to process them, so that the need of the enzyme for mRNA degradation can now be adequately met.

In related studies, we have also shown that the co-expression in the same cell of two RNase E variant polypeptides that are individually lethal- one with a 5'-end recognition pocket mutation and the other with a catalytic active site mutation - is able to confer viability. These findings are in support of the model proposed from the crystal structure data that substrate 5'-end recognition and cleavage occur in different subunits of the oligomer.

3. A cryptic pathway for potassium translocation in *E. coli.*

Research in this project is directed towards examination of a physiological link between the phosphoenol pyruvate dependent phosphotransferase system comprising PtsP-PtsO-PtsN and K^+ ion metabolism in *E. coli*. Absence of PtsN the terminal phosphoacceptor of the PtsP-O-N phosphorelay in *E. coli* leads to a potassium sensitive growth phenotype (K^S) as the external K^+ concentration ($[K^+]_e$) is increased above 1 mM. Studies on the K^S of the Δ*ptsN* mutant have shown that its growth inhibition by $[K^+]_e$, paradoxically correlates with cellular K^+ limitation that is mediated by YcgO, a predicted inner membrane protein belonging to the CPA1 family of proteins that mediate monovalent cation/proton antiport. Accordingly, the K^S is alleviated by the absence of YcgO. Furthermore overexpression of *ycgO* also yields a K^S that is similar in many respects to that displayed by the Δ*ptsN* mutant,implicating YcgO to be the mediator of the K^S. Overall our studies are consistent with a model (schematically depicted in Fig.1) which postulates that K^S in the Δ*ptsN* mutant occurs due to K^+ limitation resulting due to unfettered K^+ efflux mediated by YcgO, owing to the absence of dephospho-PtsN with K^+ efflux being additionally stimulated by $[K^+]_e$. Repression of the high affinity Kdp K^+ uptake system by $[K^+]_e$ is thought to contribute to the maintenance of K^+ limitation in the Δ*ptsN* mutant and it is assumed that the magnitude of K^+ efflux via YcgO is lower than the flux of K^+ uptake occurring separately through the Trk, Kup and the fully activated TrkF systems It is speculated that YcgO mediated K^+ limitation may be an output of a response to certain stress(es) which by modulating the phosphotransfer capacity of the PtsP-O-N phosphorelay, leads to growth cessation and stress tolerance.

Earlier we had isolated, after transposon mutagenesis, chromosomal suppressors of the K^S of the Δ*ptsN* mutant. Genetic studies on one of the suppressor mutants have shown that absence of a small integral membrane protein YajC alleviates the K^S. Previously we have also observed that the Δ*ycgO* mutation which suppresses the K^S of the Δ*ptsN* mutant, yielded a K^+ related growth phenotype and affected cellular K^+ content only in the absence of PtsN indicating that ordinarily YcgO activity is rendered cryptic in *E. coli*, probably by dephospho-PtsN. In principle any suppressor mutation of the K^S can mediate its effect by either directly altering cellular K^+ pools or may exert its effect on cellular K^+ pools only in the Δ*ptsN* mutant. The Δ*ycgO* mutation thus exerts its suppressive effects by the latter mechanism. A tester strain allows one to distinguish between the two possibilities mentioned above and our studies show that the Δ*yajC* mutation, like the Δ*ycgO* mutation,exerts

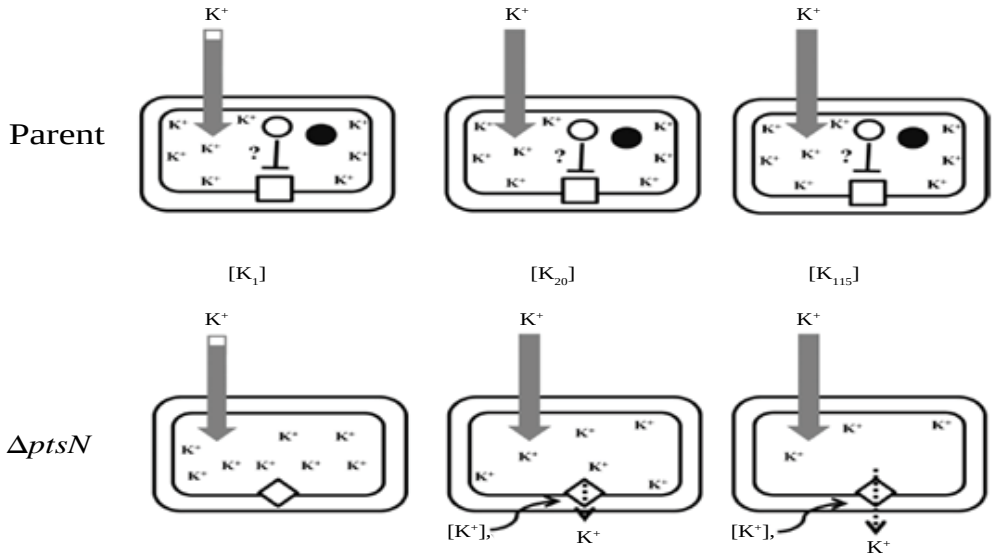

Figure 1. A model for K^+ limitation mediated by YcgO in the Δ*ptsN* mutant. Scheme depicting cellular K^+ content in the parent strain (top panel) and its reduction in the Δ*ptsN* mutant (bottom panel). K^+ content in the two strains is shown in media with $[K^+]_{es}$ of 1 ($[K_1]$), 20 ($[K_{20}]$), and 115 ($[K_{115}]$) mM. A double-colored arrow represents the contribution to cellular K^+ content due to K^+ uptake mediated by the Kdp (white) and the Trk plus Kup (gray) transporters, whereas a single-colored gray arrow represents K^+ uptake occurring via the Trk and Kup transporters and reflects the repression of the Kdp system by $[K^+]_e$. The contribution of the TrkF activity to the cellular K^+ content in strains bearing all K^+ uptake systems is considered to be negligible. K^+ efflux mediated by YcgO is represented as a dashed arrow, and its stimulation by $[K^+]_e$ is represented by a wavy arrow. The heights of the arrows representing K^+ uptake and efflux are proportional to their K^+ transport fluxes. Dephospho-PtsN, presumed to fetter YcgO, and the phosphorylated form of PtsN are represented as open and solid circles, respectively. The open square and diamond, respectively, represent the fettered and unfettered states of YcgO.

its suppressive effect only in the absence of PtsN. In addition the K^S of *ycgO* overexpression was also substantially suppressed by the Δ*yajC* mutation and, unlike that seen for the case of *ycgO*, overexpression of *yajC* did not lead to the K^S. These observations suggest that YajC may function as a positive regulator of YcgO activity. Current studies are directed towards testing the notion that YajC may interact with YcgO and whether the suppression by absence of YajC correlates with elevation of K^+ content in the Δ*ptsN* mutant. In addition, whether PtsN displays a phosphorylation state dependent interaction with YcgO is also being studied.

4. Studies on basic amino acid export in *E. coli.*

Towards studies on regulation of basic amino acid export in *E. coli*, we have previously reported genetic and physiological studies on the ORFs *yggA* (*argO*) and *ybjE (lysO*) that encode the L-arginine (Arg) and L-lysine (Lys) exporters ArgO and LysO respectively in *E. coli*. The ortholog of ArgO in *C. glutamicum*, LysE exports both Arg and Lys whereas ArgO ordinarily mediates export only of Arg. Our studies have shown that under conditions where expression of *argO* is dissociated from the repressive effect of Lys on its expression, which occurs via the ArgP transcriptional factor, the Lys export potential of ArgO is detectable, indicating that ArgO also bears a capacity (albeit latent) to mediate Lys export.

Proteins belonging to the LysE family are widely distributed, in many bacteria and contain on an average of 200 to 220 amino acid residues. Predictions of their topology are supportive of a 6 transmembrane (TM) helical arrangement. While LysE and ArgO remain the best functionally characterized members of the LysE family, there is an absence of structural information pertaining to them.

Towards determination of the mechanism of Arg export by ArgO, we had previously undertaken an analysis of its topology in *E. coli* using alkaline phosphatise fusion reporters, which provided limited information on the topological disposition of ArgO in the cytoplasmic membrane. Recently, we have used cysteine accessibility studies in situ to construct a detailed topological map of ArgO. For this purpose we have constructed a

set of 25 functional ArgO variants each bearing a single cysteine substitution at a specific position along the length of ArgO, and have determined the three possible locations for the cysteine residue namely periplasmic, cytoplasmic or intramembrane using protein PEGylation. Our studies indicate that ArgO assumes an N_{In}-C_{Out} configuration potentially forming a five transmembrane helix bundle flanked by an indispensable N-terminal cytoplasmic domain (NTD) and a dispensable short C-terminal periplasmic region (CTR). Mutagenesis studies implicate a pair of conserved aspartate residues, located near the cytoplasmic and periplasmic edges of the cytoplasmic membrane to play a pivotal role in facilitating transmembrane Arg flux.

We had earlier also isolated a set of *argO* mutants encoding proteins bearing amino acid substitutions that impair ArgO function in vivo. Furthermore we had isolated their derivatives bearing compensatory amino acid alterations, which implicated a role for interhelical interactions in the Arg export mechanism. Using the membrane permeable crosslinker disuccinimidylsuberate we have obtained evidence that ArgO may function *in vivo* as a monomer, highlighting thus the requirement for intramolecular interactions in ArgO as opposed to interactions across multiple ArgO monomers in the formation of an Arg translocating conduit.

Further studies in this regard are directed towards reconstitution of ArgO and LysO mediated Arg, Lys export respectively in proteoliposomes to obtain insights into the mechanistic basis of amino acid export mediated by the two exporters.

5. Genetic interactions between (p)ppGpp and tm-RNA (SsrA)/SmpB : Modulation of transcriptional polarity by (p)ppGpp.

The nucleoside derivative (p)ppGpp is an important signal of the status of growth physiology in bacteria. In work described by us in earlier reports, the synthetic lethal phenotype observed during the combined deficiency of (p)ppGpp and either SsrA or SmpB (explained below) was genetically and biochemically characterized and this led to proposal of the following model. An increased rate of transcription elongation in the $ppGpp^0$ strain (*ΔrelA ΔspoT*) uncouples transcription and translation resulting in mRNA segments between the RNA polymerase (RNAP) and the lead ribosome to be exposed. The exposed segments of mRNA become the target for ribonucleases and the transcription termination factors Rho/NusG, leading to the generation of truncated mRNAs. Ribosomes stalling on truncated mRNAs result in the generation of non-stop ribosome complexes and make ribosome rescue by the trans-translation machinery (SsrA and SmpB) essential for survival. The proposal that the transcription elongation rate is enhanced in the $ppGpp^0$ strain is based on the suppression and accentuation,respectively, of the $ppGpp^0$ ssrA synthetic lethality by the RNAP mutations *rpoB8* and *rpoB2.*

In the context of this model, it was also important to rule out the possibility of regulation of transcription initiation by (p)ppGpp contributing to the suppression. Towards this, a stable RNA ($tRNA^{ARG5}$)-encoding construct was fused to *lacZ* and used as reporter to compare transcription efficiencies between wild type and $ppGpp^0$ strains, by Northern blotting. With the reporter fused distal to the lac promoter, a 4-fold increased polarity was evident in the $ppGpp^0$ strain. To rule out effects on transcription initiation, a promoter-proximal fusion was also made and the efficiency of transcription measured; no difference was evident.

Since the synthetic lethality was individually suppressed by over-expression of the (p)ppGpp accessory factor DksA (but not $DksA^{NN}$ mutated at its conserved aspartate residues), as well as by the the stringent and slow moving RNAP mutants *rpoB*L571P and *rpoB8* respectively, their effects on transcription initiation and elongation was studied in the $ppGpp^0$ strain. Over-expression of DksA (but not $DksA^{NN}$) and *rpoB*L571P moderately increased the synthesis of full-length transcript without affecting the efficiency of transcription initiation. The rpoB8 mutant failed to improve full length transcript levels. Since over-expression of DksA (but not $DksA^{NN}$) or the stringent *rpoB* mutants alter transcription initiation from stringent promoters (positively or negatively regulated), their effects on transcription elongation noted here can be argued to be indirect; however, this is an unlikely explanation since effects arising from redistribution of RNAP would be seen on initiation as well.

Since the *rpoB8* mutant fails to increase full-length transcripts while it rescues synthetic lethality, it can be argued that the reduction in full-length transcripts per se is not the cause of lethality. It is accordingly proposed that in the $ppGpp^0$ strain, the reduction is the consequence

of both premature transcription termination as well as the generation of truncated mRNA through ribonucleases, while in the ppGpp^0 *rpoB8* strain it follows from the reduced rate of transcription elongation. The consequences for translation, therefore, are very different in the two strains. While the ppGpp^0 strain would have increased non-stop ribosome complex following the arrest of ribosomes at non-stop mRNA, in the ppGpp0rpoB8 strain increase in RNAP coupling with lead ribosome would alleviate the generation of non-stop mRNA and non-stop ribosome complex. It follows that the SsrA/SmpB-mediated trans-translation is essential in the ppGpp^0 strain but dispensable in the ppGpp^0 *rpoB8* mutant.

6. Modulation of cell division by (p)ppGpp.

In previous studies, we had documented the synthetic lethality of ppGpp^0 with mutation in *lon* (encoding the ATP-dependent Lon protease). Our studies had suggested that SulA-mediated inhibition of the cell division protein FtsZ could be the probable cause of lethality (since SulA is a natural substrate of Lon protease), although we did not find evidence for increased *sulA* expression in the (p)ppGpp^0 strain.

Based on genetic and molecular studies carried out during the current year, we propose that (i) basal levels of (p)ppGpp are required to sustain normal cell division in *E.coli* during growth in rich medium through the positive regulation of FtsZ ; and (ii) basal SulA level set by Lon protease is important for insulating cell division against both a decrease in FtsZ concentration and against conditions that can increase the susceptibility of FtsZ to SulA as seen in a ppGpp^0 strain. Work is in progress to understand the mechanism of regulation of FtsZ by basal (p)ppGpp.

7. Genetic and molecular characterization of glycerol stasis in the *glpD* mutant of *E.coli*.

It has been reported that cells lackingglycerol-3-Pdehydrogenase (encoded byglpD)undergo growth stasis following the addition of glycerol or glycerol-3-P in growth media lacking glucose, and that glucose can reverse this effect through a mechanism different from catabolite repression; the mechanism remains uncharacterized. A separate study has implicated depletion of nucleotides particularly that of ATP to be responsible for the growth arrest.

In the course of our earlier studies, that showed the existence of cross-talk between transketolase activity (involved in the pentose-phosphate shunt pathway) and glycerol metabolism, we identified that supplementation of ribose or other pentose sugars and pyrimidine (but not purine) nucleosides could individually rescue the glycerol induced growth stasis contingent on the synthesis of ribose-5-P. The rescue by ribose, but not by glucose, was abolished when *glpK*(encoding glycerol kinase) expression was made constitutive through a non-native promoter placed upstream of *glpK* on the chromosome but not when expression from the native promoter was made constitutive through a mutation in the transcriptional repressor *glpR*.

In the current year, we explored the link between the growth arrest induced by these sugars (glucose or ribose) and the intracellular concentration of nucleotides to ask if we could identify any correlation between the levels of nucleotides, *glpK* expression and the sugars used to rescue growth arrest. Our results show that ATP and GTP levels are reduced following the addition of glycerol, although the response is not instantaneous as reported. More interestingly, the metabolite that shows almost instantaneous disappearance or reappearance, respectively, following glycerol supplementation or growth rescue by addition of sugars, is phosphoribosylpyrophophate (PRPP). Further studies are in progress to understand how glycerol addition causes PRPP depletion which is then rescued by the addition of the sugars.

8. Does basal (p)ppGpp modulate chromosomal replication?

In a recent report (Ferrulo & Lovett, PLoS Genetics, 2008, 4:e1000300), inhibition of colony formation by (p)ppGpp accumulation was shown to be relieved in *seqA or dam mutants* (Dam methylates the A residue in palindromic GATC sites in DNA, and SeqA binds duplex DNA with hemi-methylated GATC sequences that would occur soon after passage of the replication fork). It was proposed that DNA methylation and SeqA binding to non-origin loci is necessary to enforce a full stringent arrest, affecting both initiation of replication and chromosome segregation. We were unable to reproduce the rescue of growth inhibition using a *ΔseqA* allele and the same plasmids used in that study for over-expression of (p)ppGpp. Preliminary results from our study

shows that increase in basal (p)ppGpp improves the growth of *seqA* mutant in rich medium and that synthetic lethality is observed in the ppGpp^0 *ΔseqA* strain under some growth conditions. The latter result suggests that (p)ppGpp is required for the growth of *seqA* mutants. Studies to further characterize the phenomenon are in progress which could help in understanding the role of basal (p)ppGpp in chromosomal replication.

Publications

1. Gowrishankar J (2015). End of the beginning: elongation and termination features of alternative modes of chromosomal replication initiation in bacteria. ***PLoS Genetics***11: e1004909.
2. Nazir A and Harinarayanan R (2016). Inactivation of cell division protein FtsZ by SulA makes Lon indispensable for the viability of ppGpp^0 strain of *Escherichia coli*. ***Journal of Bacteriology***198: 688-700.
3. Pathania A and Sardesai AA (2015). Distinct paths for basic amino acid export in *Escherichia coli*: YbjE (LysO) mediates export of L-lysine. ***Journal of Bacteriology*** 197: 2036-2047.
4. Vimala A and Harinarayanan R (2016). Transketolase activity modulates glycerol-3-phosphate levels in *Escherichia coli*. ***Molecular Microbiology.***

Other Publications

1. Gowrishankar J and Nandineni MR (2016). Why India is rooting for its DNA identification Act. ***Nature India*** doi:10.1038/nindia.2016.47.

LABORATORY OF CELL CYCLE REGULATION

Elucidating the role of effector proteins in G1 to S phase progression

Faculty	Shweta Tyagi	Staff Scientist
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	Zaffer Ullah Zargar	Senior Research Fellow
	Swathi Chodisetty	Senior Research Fellow
	Amit Mahendra Karole	Senior Research Fellow
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	Mallikharjuna Rao	Project JRF

Objectives

1. Identification of new effector proteins involved in regulation of E2F-responsive promoters; and
2. Study of chromatin modifying proteins in cell cycle regulation.

Project 1: Identification of new effector proteins involved in regulation of E2F-responsive promoters.

One of the major roles of E2F proteins is to regulate the transition from G1 to S phase. However, how E2Fs affect passage into S phase is still poorly understood. In this project we aim to identify new effector proteins involved in regulation of E2F-responsive promoters and better understand how these effectors influence transcriptional regulation during G1 to S phase progression.

Summary of work done until the beginning of this reporting year (up to March 31, 2015)

We showed that bacterially expressed GST-E2F4 fusion could pull down RBP2 from HeLa cell nuclear extract (NE). We were able to map the RBP2-interacting domain in E2F4 to the C-terminal Transactivation domain (TAD).

Details of the progress made in the current reporting year (April 1, 2015 –March 31, 2016)

In order to map the domain in E2F4 which associates with RBP2, we made C-terminal GST fusion of E2F4 truncations. The 76 amino acid Transactivation domain (see Fig 1A) was able to pull down RBP2 from the HeLa cell NE, indicating that this domain was sufficient for interaction with RBP2 (Fig 1B panel a lane 5).

E2Fs including E2F4 are known to interact with the pocket proteins via their Transactivation domain (Shan et al, Proc Natl Acad Sci. 1996, see Fig 1A). RBP2 was discovered in a screen for cellular proteins that bind to the retinoblastoma gene product (Rb binding protein) and has been reported to interact with p107 (Defeo-Jones et al., Nature 1991; Kim et al., Mol. Cell. Biol. 1994). Therefore, we wanted to ascertain that the E2F4-RBP2 interaction observed here was direct or via the pocket protein associated with E2F4. In our previous results, we observed that E2F4-RBP2 interaction is maximum in early G1 phase and p130 associates with E2F4 at this time. Therefore, we also probed the immunoblot with p130 antisera. As expected, p130 was present in the GST-E2F4 TAD pull down (Fig 1B panel b lane 5).

In order to establish that the RBP2 interaction with E2F4 was being mediated by the pocket protein (p130 here), we took advantage of the conservation of pocket protein-binding domain in E2Fs (Lee et al., Genes Dev. 2002; Shan et al, Proc. Natl. Acad. Sci. 1996) and created two sets of amino acid mutations in the GST-E2F4 TAD, M1 (Tyr 392 His) and M2 (Asp 404 Gly, Leu 405 Pro, Phe 406 Leu, and Asp 407 Gly) (Fig 1A). Both sets of amino acids mutations have been shown to abolish the E2F-pocket protein interaction previously (Lee et al., Genes Dev. 2002; Shan et al, Proc. Natl. Acad. Sci. 1996). p130 associated with the wild type GST-E2F4 TAD but not GST-E2F4 TAD M1 and GST-E2F4 TAD M2 (Fig 1B panel b, compare lane 5 with lane 3 and 4). These results are consistent with the conservation of E2F-pocket protein-binding and the interaction of pRb with other E2Fs (Lee et al., Genes Dev. 2002; Shan et al, Proc. Natl. Acad. Sci. 1996). The blot was probed for RBP2 interaction. Only wild type GST-E2F4 TAD (Fig 1B, panel a) interacted with RBP2.

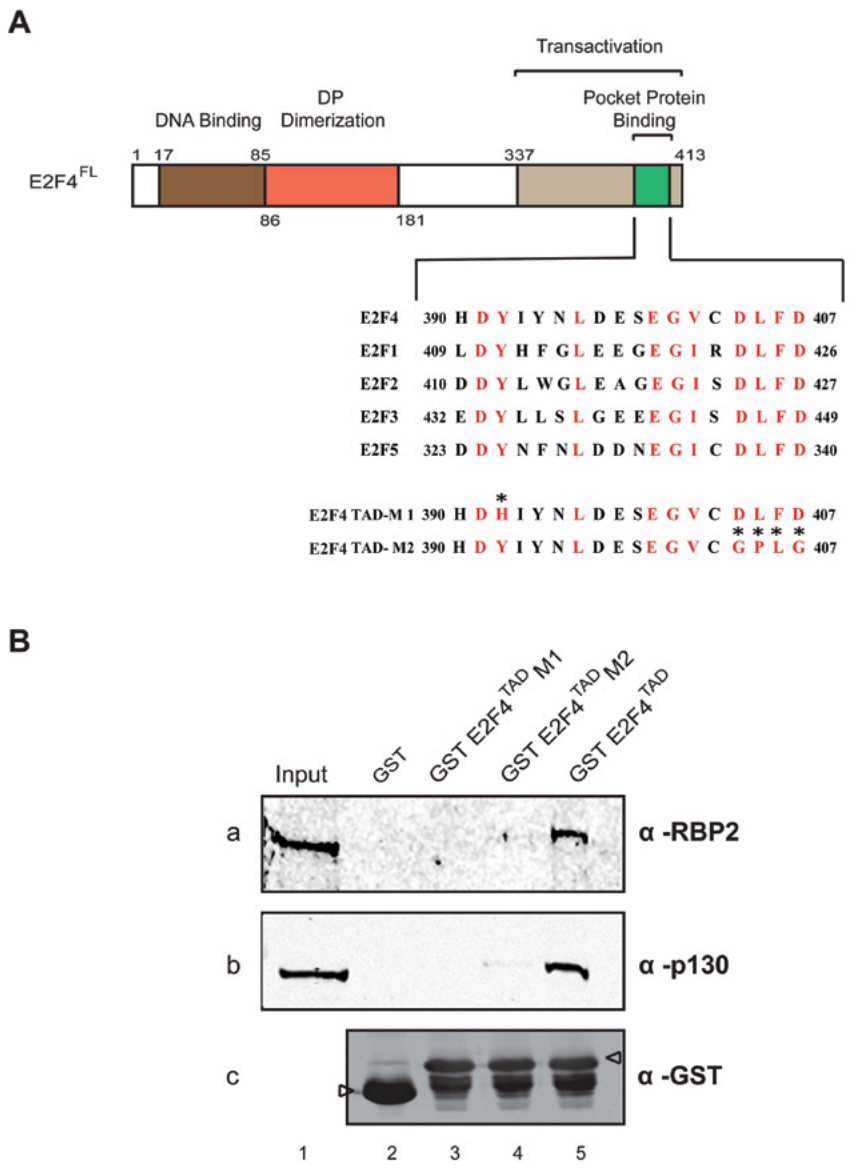

Figure 1. RBP2 interacts with E2F4 via pocket protein p130.

(A) Schematic structure of E2F4 protein. Functional domains are indicated on top. Numbers indicate amino acids. Conserved pRb binding residues (in red) for proteins E2F1 to E2F5 are shown. Asterisk indicates residues which have been mutated in GST-E2F4^{TAD}. FL, full length; TAD, transactivation Domain.

(B) The Interaction between E2F4 and RBP2 is mediated by p130. GST-E2F4^{TAD} (wild type, lane 5) and its mutant forms GST-E2F4^{TAD} M1 (Y 392 H, lane 3) and GST-E2F4^{TAD} M2 (D 404 G, L405 P, F 406 L, and D 407 G, lane4) were used for pull down experiment from HeLa cell NE. The blot was probed with anti-RBP2 (panel a), p130 (panel b) and GST (panel c) antibody.

Our results suggested that p130 is mediating the interaction between RBP2 and E2F4 proteins. To test this further, we fused the T/E1A interacting domain of p130 to C-terminal of GST and used this fusion protein to pull down RBP2 from HeLa cell NE as described above. p130 was able to pull down RBP2 robustly and specifically (Fig 2A). RBP2 is a large protein with multiple domains like PHD, bromo domain etc. (See Fig 2B). In order to map the domain of RBP2, which interacted with p130, we created five fragments of RBP2 protein as shown in the schematic in Fig 2B and expressed them as C-terminal fusions of GST protein. Out of these only fragment 5 of RBP2 (D5, see Fig 2B, lower panel lane 7) was able to pull down p130. Fragment 5 contains the

PHD3 domain of RBP2 as well as the leucine-X-cysteine-X-glutamic acid motif (LxCxE, where X is any amino acid).The proteins having LxCxE motif are known to associate with pocket proteins; and both pRb and p107 associate with RBP2 via the LxCxE motif (Kim et al., Mol. Cell. Biol. 1994). Interestingly, the mutation in LxCxE motif in RBP2 is sufficient to abrogate its interaction with p107 but not pRb. The region in RBP2 which contributes to pRb interaction in LxCxE mutant background has been mapped to 15kDa fragment. This 15kDa fragment is independent of LxCxE motif but present in our D5 fragment. Therefore, to test if binding of

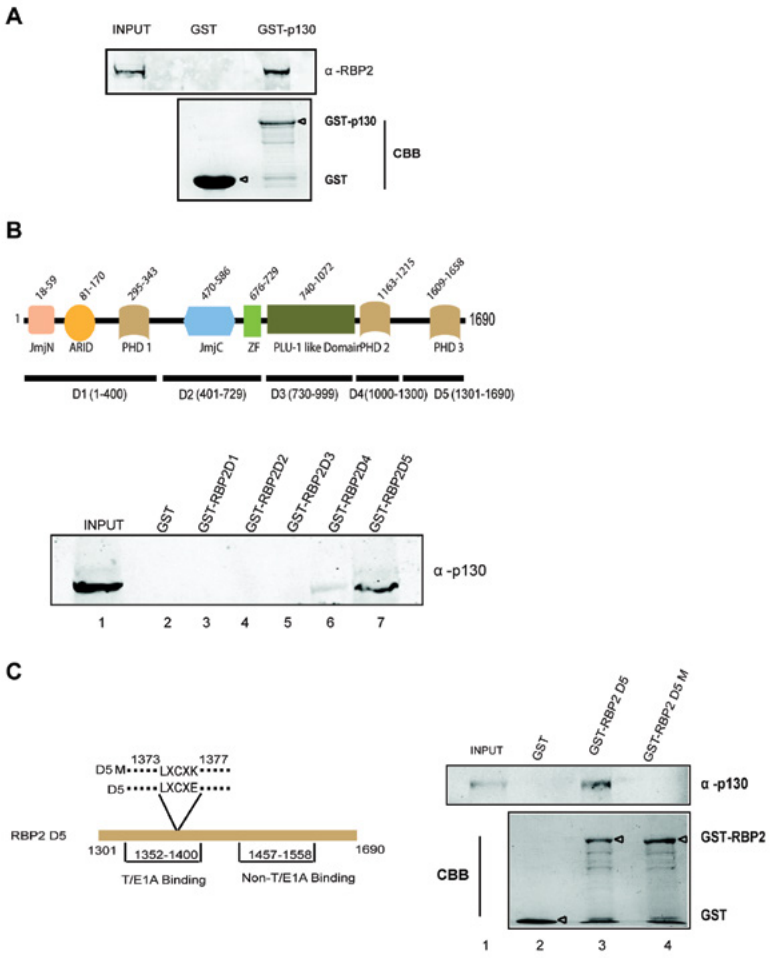

Figure 2. The interaction between p130 and RBP2 is direct and LxCxE dependent.

(A) p130 interacts with endogenous RBP2. GST and GST-fusion of pocket domain of p130 (GST-p130 T/E1A) were purified and used for pull down experiment using HeLa cell NE. The blot was probed with anti-RBP2 antibody (top panel). Bead-bound GST or GST-p130 T/E1A proteins stained with Coomassie Brilliant Blue (CBB) are shown in bottom panel.

(B) Schematic structure of RBP2 protein. Upper panel: Functional domains are indicated at the bottom. Deletions of RBP2 used in this study are indicated below the domains in bold lines. All deletions were fused to C-terminal of GST. Numbers on top indicate amino acids. JmjN/JmjC, N/C-terminal Jumonji domain; ARID, AT-rich interacting domain, PHD, plant homeodomain; ZF, zinc finger. Lower panel: Mapping of p130- interacting domain in RBP2. GST and GST-tagged deletions of RBP2 were used for pull down experiment from HeLa cell lysate. The blots were probed with anti-p130 antibody.

(C) The interaction between RBP2 and p130 is LxCxE motif dependent. On the right, the position of LxCxE motif is shown in deletion RBP2 D5. RBP2 D5 mutant was created by changing glutamic acid 1377 to lysine. GST-RBP2 D5 (wild type, lane 3) and its mutant GST-RBP2 M (E1377K, lane 4) were used for pull down experiment from HeLa cell NE. The blot was probed with anti-p130 (panel a) antibody. Bead-bound GST or RBP2 D5 proteins stained with CBB are shown in bottom panel.

RBP2 to p130 was LxCxE motif-dependent, we mutated glutamic acid in this motif to lysine (here E1377K), a mutation which is known to abrogate the LxCxE mediated interactions (Kim et al., Mol. Cell. Biol. 1994). As shown in Fig 2C, the RBP2 D5 (E1377K) mutant could not pull down p130 like the wild type, indicating that in its interaction with RBP2, p130 behaves like p107 (Kim et al., Mol. Cell. Biol. 1994, this study).

Based on these results we postulate that RBP2 may be recruited to E2F-responsive promoters by pocket protein p130 and we will test this hypothesis by performing chromatin immunoprecipitation experiments for RBP2 in the presence and absence of p130.

Project 2: Study of chromatin modifying proteins in cell cycle regulation

Histone 3 lysine 4 trimethylation is linked to active gene expression, but its precise role in cell cycle regulation is now being uncovered. We have shown that MLL-family of H3K4 histone methyltransferases is linked to cell cycle regulation by interacting with E2F1. In this project, we are looking at other roles of this chromatin-modifying complex that may influence cell cycle regulation.

Summary of work done until the beginning of this reporting year (up to March 31, 2015)

SET 1 family members have overlapping as well as unique functions. In order to clarify if other SET members participated or duplicated the functions of MLL in cell cycle progression we undertook further studies. Our experiments revealed that loss of Set1A, MLL2 and MLL3 resulted in pronounced and almost similar loss in cell proliferation like MLL depletion. In contrast, when assayed for mitotic defects, only Set1A RNAi displayed obvious phenotype, and not MLL2 or MLL3 siRNA-treated samples indicating that the mitotic role was unique for MLL and Set1A.

Details of the progress made in the current reporting year (April 1, 2015 –March 31, 2016)

To identify the mechanism of how MLL complex may regulate M-phase progression, we studied the subcellular localization of its components in the cell. For this we performed immunofluorescence (IF) staining against endogenous WDR5 in U2OS cells, using commercially available affinity-purified polyclonal antibody. As expected, during interphase, majority of WDR5 localized to the nucleus, though some protein was also

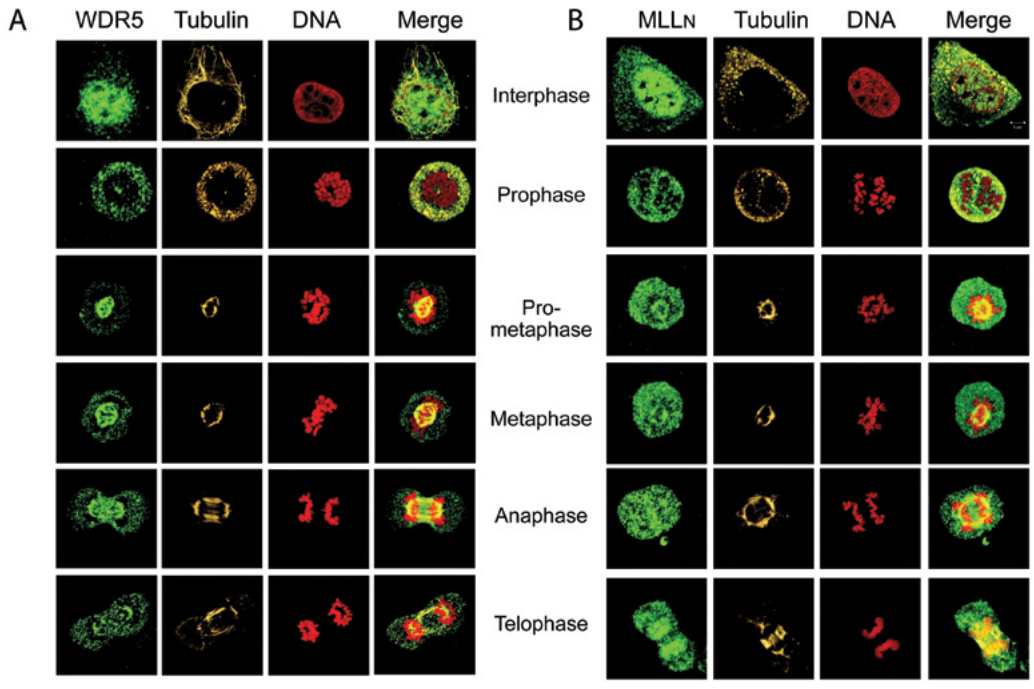

Figure 3. WDR5 and MLL associate with the spindle apparatus during mitosis. Interphase and mitotic U2OS cells stably expressing H2B-mcherry were analyzed by immunofluorescence staining with anti-WDR5 (A) or MLL_N (B) (green) and anti-alpha tubulin (amber) antibodies. Different stages of mitosis were identified based on the DNA and tubulin staining.

visible in the cytoplasm (Fig 3). We also stained for WDR5 in mitotic cells. The different stages of mitosis were determined by staining for DNA and alpha tubulin. In agreement with previous reports, we could not detect WDR5 on condensed chromosomes during mitosis. Instead, to our surprise, WDR5 was found associated with the spindle apparatus in pro-metaphase through telophase and it was only during cytokinesis that the chromatin localization of WDR5 was restored.

In order to confirm that the spindle staining of WDR5 was not due to non-specific staining of the polyclonal antibody, we performed two additional experiments. Firstly, we stained for endogenous WDR5 with two different polyclonal antibodies in addition to the one described above. Both antibodies could detect WDR5 on the spindle. Secondly, we knocked down WDR5 protein by siRNA experiments as described previously and performed IF staining against endogenous WDR5. Consistent with the reduced levels of WDR5, the staining of WDR5 was reduced on the spindle in WDR5 siRNA-transfected samples as oppose to control siRNA-transfected samples.

WDR5 is one of the core components of MLL HMT complex. However, WDR5 is also found in other complexes. In order to determine, if WDR5 occurred as a part of MLL HMT complex or any other complex on the spindle, we stained for MLL_N subunit. Just like WDR5, MLL also localized to the spindle during mitosis. Consistent with the localization of WDR5 and MLL_N, we could detect MLL_C subunit and RbBP5 on the spindle during pro-metaphase and metaphase stages in U2OS cells (data not shown). We could also detect WDR5 and RbBP5 on the spindle in HeLa and NIH3T3 cells, suggesting that this was not a cell specific occurrence.

We are now in the process of understanding, how spindle localization of MLL complex proteins affects mitotic progression and the exact role of MLL in spindle organization, if any.

LABORATORY OF CELL DEATH & CELL SURVIVAL

Functional protein networks controlling cellular pathways

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	Debjani Bhattacharya	Project-JRF
	Bhavya K	Project- JRF (till August 2015)
	Nanci Rani	Technical Assistant

Objectives

1. To dissect the functional network of phosphatases regulating cellular pathways.
2. To understand the cellular functions of canonical and non-canonical ubiquitination.

Summary of work done until the beginning of this reporting year (upto March 31, 2015)

Phosphatases play a critical role in nearly every cellular process, including metabolism, gene transcription, translation, cell-cycle progression, protein stability, signal transduction, and apoptosis. We initiated our studies on functional phosphatase network with the identification of interacting partners of every phosphatase in the cell. In this work we have already identified and characterized WWP2, PNUTS and TOPK as novel functional partners of PTEN (Maddika *et al*., Nature Cell Biol. 2011, Kavela *et al.*, Cancer Res. 2013). Recently, we identified a new cellular function for PTEN where we have shown that PTEN via interacting with Rab7 functions in endosome maturation (Shinde SR & Maddika S., Nature Communications., 2016). In addition to PTEN interactome, we also started developing networks of other cellular phosphatases. We identified PPM1G as a molecular switch that controls differential cellular role of E3 ligase WWP2, by controlling the transition between monomeric WWP2 and WWP2/WWP1 heterodimer (Chaudhary N & Maddika S., Mol Cell Biol 2014). Additionally, we have shown that PPM1G controls cell adhesion by interacting with alpha-catenin at cell junctions.

Details of progress in the current reporting year (April 1, 2015 - March 31, 2016)

Theme 1. Functional studies on phosphatase networks

Currently, we are focused on actively expanding the network of all the available phosphatases in cell. We cloned 143 human protein phosphatases in a gateway compatible triple tagged (SBP-Flag-S protein) vector and each of them was individually expressed in HEK293T cells. Protein complexes were isolated by tandem affinity purification and interacting proteins were identified by using LC-MS/MS analysis. A total of 76773 interactions were obtained from 143 phosphatase purifications. After filtering out the common contaminants using control GFP purification and eight different non-phosphatase purifications, we used Significance Analysis of Interactome (SAINT) algorithm to score protein-protein interactions. By using a SAINT score cut off of 0.9 and with spectral count above 3, we identified 6596 high confident interactions (HCIs) mediated by 2112 proteins (HCIPs) and 143 purified phosphatases (Figure 1). A

comparison of our data with iRefIndex, a source of protein-protein interactions curated from various primary interaction databases, revealed that 6325 interactions by 1956 HCIPs were previously uncharacterized thus accounting for 95% of novel interactions in the list. With inputs from computational biology group, we annotated these phosphatase interactions to KEGG pathways. Our enrichment analysis revealed association of phosphatases with nearly 83 different cellular pathways. As expected several already known functions associated with phosphatases were enriched in our analysis. For example, CDC25 phosphatases were found to interact with cell cycle proteins, dual specific phosphatases (DUSPs) interact with proteins in various immune signaling pathways and receptor tyrosine (PTPR) phosphatases interact with proteins in export, sorting and degradation pathways. In addition to known associations, several novel functions have been enriched in the analysis. For instance, atypical DUSP (ADSP) phosphatases interact with proteins in DNA replication and repair pathways. Further to understand how phosphatases are involved in disease pathways and to find components of biochemically related proteins linked to particular disease phenotype we integrated the information of these altered genomic loci into phosphatase interaction network. We used OMIM annotated disease linked genes and analysed for interaction of phosphatases with these disease linked genes. We identified 474 disease-linked proteins that interact with 138 phosphatases and form a network of 1637 interactions. We also matched phosphatase interactome to COSMIC (cancer gene census) dataset that contain genes mutated in human cancers. Out of 143 phosphatases analyzed, 107 phosphatases associated with cancer-linked proteins. Overall, we identified 90 interactors in phosphatase interactome that are genetically linked to various types of tumors forming a total of 289 interactions.

In addition to mapping the phosphatase network, we simultaneously started to characterize several of putative functional interactions of these purified phosphatases. To this end, we made significant progress in understanding multiple novel phosphatase interactions in the lab. The data generated from some of the exciting interactions has been presented below.

1.1. PHLPP facilitates kinetochore assembly by regulating SGT1

PHLPP is a tumor suppressor phosphatase that plays critical roles in cell survival. We found

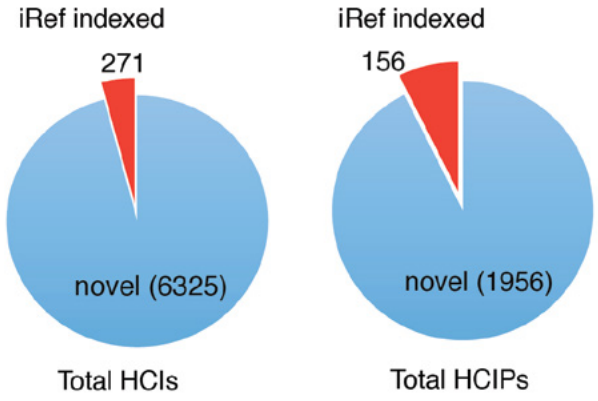

Figure 1. Analysis of human protein phosphatase interactome. High confidence interactions (HCIs) mediated by phosphatases and interacting proteins (HCIPs) identified in our study was compared with iRefIndex and the distribution of known and novel interactions was plotted.

SGT1 as one of the interesting candidates in PHLPP1 interaction list. SGT1 is a kinetochore protein that plays an important role in human kinetochore assembly. We confirmed that SGT1 specifically interacts with PHLPP1. As SGT1 is required for proper kinetochore assembly, we next tested the effect of PHLPP1 on this function. We found that depletion of PHLPP1 results in severe loss of outer kinetochore markers (Figure 2A), reminiscent of phenotypes from SGT1 loss in cells. Loss of PHLPP1 leads to defective kinetochore-microtubule attachment (Figure 2B & 2C) followed by delay in mitotic progression. In conclusion, we assigned a new role for PHLPP1

in kinetochore assembly based on its interaction with SGT1. Currently, we are trying to understand how PHLPP1 and SGT1 are mechanistically linked to control kinetochore assembly.

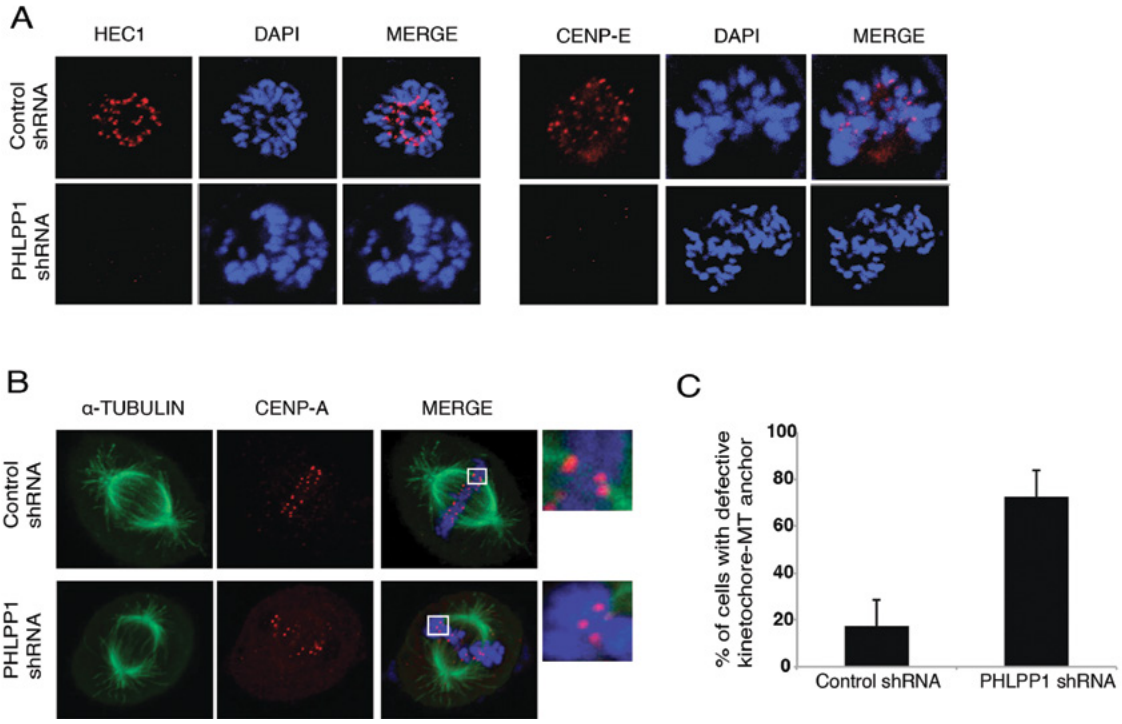

Figure 2. PHLPP1 is required for kinetochore assembly.

(A) Outer kinetochore proteins (HEC1 and CENP-E) were detected by immunofluorescence in control and PHLPP1 depleted cells.

(B) Attachment of microtubules to kinetochores was determined by co-staining with CENP-A and alpha-tubulin.

(C) Quantification of data from figure 2B was shown.

1.2. PTPN5 regulates cytokinetic abscission by interacting with Mob1a

PTPN5 also known as STEP is a non-receptor tyrosine phosphatase that is mainly expressed in the brain regions such as striatum, cortex, and hippocampus. We uncovered several novel PTPN5 associated proteins among which an uncharacterized interaction with Mob1a was found. Mob1a is a conserved co-activator of NDR and LATS family of kinases in Hippo signaling pathway and acts as a tumor suppressor. In addition, Mob1 is shown to be functionally important for cytokinesis during mitotic exit. We confirmed specific interaction of PTPN5 with Mob1a (Figure 3A). We found that PTPN5 dephosphorylates Mob1a at Y26 residue (Figure 3B). Functionally, we have demonstrated that PTPN5 via interacting with Mob1a participate in the control of cytokinesis during mitotic exit. PTPN5 depleted cells progressed through mitosis similar to control cells but took longer time to accomplish abscission. While control cells disassembled their midbodies and completed cytokinetic abscission by 45 minutes of entry in to mitosis, PTPN5 depleted cells showed defective cytokinesis with unseparated midbodies for longer hours (Figure 3C). Mechanistically, we have shown that PTPN5 controls midbody abscission through regulating Mob1A localization via its dephosphorylation. Mob1A readily localizes to midbodies, whereas its phosphomimetic mutant Y26D fails to do so, suggesting that Mob1A dephosphorylation at this site by PTPN5 is critical for its midbody localization.

Theme 2: Roles of canonical and non-canonical ubiquitination in cells

Ubiquitination is an ATP-dependent, highly ordered multistep enzymatic process, which results in covalent attachment of ubiquitin to the substrate. Ubiquitin linked to the substrates serves as a molecular tag that marks proteins for either degradation by proteasome dependent pathway or to function in wide variety of processes in a proteasome independent manner. In this project we are interested in studying both the canonical and non-canonical functions of ubiquitination in cells.

2.1. Role of K63 ubiquitin linkage in Wnt pathway

WWP2 is an oncogene that we earlier identified as an E3 ligase that degrades its substrates such as PTEN and p73 by transferring K48 ubiquitin linkages. In our quest for additional functional cellular substrates of WWP2, we found Dvl2 as its novel interacting protein. Dvl2 is an important player in the transduction of Wnt signaling pathway. We found that WWP2 ubiquitinates Dvl2 but interestingly does not lead to its degradation. By using various ubiquitin K-R mutants, we demonstrated that WWP2 ubiquitinates DVL2 via K63 linkage. In our functional experiments we found that WWP2 is required for activation of Wnt signaling pathway. Currently, we are trying to map the sites of ubiquitination on DVL2 and their mechanistic importance in Wnt pathway.

2.2. Non-canonical K27 ubiquitin linkage in protein secretion

While studying the role of ubiquitination in extracellular protein secretion, we used YB-1 as a model protein and identified the indispensable role of ubiquitination in this process. Importantly, we discovered HACE1 as YB-1 interacting E3 ligase that has the ability to generate functional K27 linked non-canonical ubiquitin linkages on its substrate. K27 ubiquitin linkages on YB-1 are necessary for its interaction with Tumor Susceptibility Gene 101 (TSG101), a component of the Multi Vesicular Body (MVB) pathway, which facilitates its secretion. Intriguingly, the secreted YB-1 unlike intracellular YB-1 displayed a strong EMT suppressor function. In summary, we identified a novel functional role for non-canonical ubiquitin linkages in mediating protein secretion.

In this theme, currently we are actively expanding the array of unknown cellular functions mediated by non-canonical ubiquitin chains by performing proteomic analysis using various ubiquitin mutants.

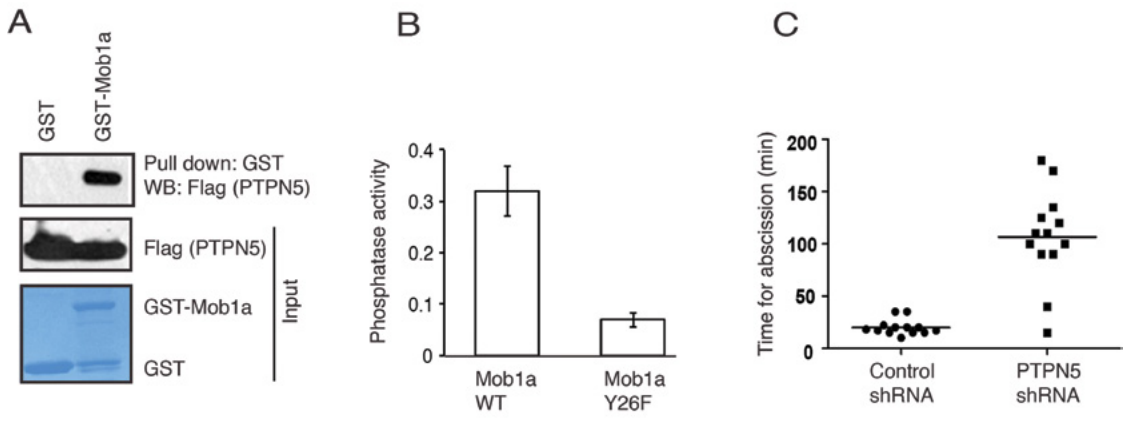

Figure-3: PTPN5 controls cytokinetic abscission

(A) Interaction of PTPN5 with Mob1a was assessed by immunoblotting after performing pull down assay with GST or GST-Mob1a fusion protein using 293T cell extract.

(B) *In vitro* phosphorylated wild type Mob1a or Y26F mutant were used as substrates in a phosphatase release assay to assess PTPN5 phosphatase activity.

(C) Time taken by each cell from mitotic entry to separation of midbodies after cytokinesis was calculated using live cell imaging and the data was plotted for control and PTPN5 depleted cells. Cells taking longer than 200 minutes for separation were not included in the analysis.

2.2. Identification of new functional E3 ligase complexes and their substrates

E3 ligases are critical proteins in the final step of the ubiquitination process where they recruit ubiquitin charged E2 enzymes along with specific substrates. In this work, we aim to identify new complexes for E3 ligases by using proteomics approach and further characterize their substrates by using human protoarrays. In one example, we identified that proteins containing LisH domain assemble E3 ligase complex to regulate specific protein substrates. Currently, we are trying to understand the functional importance of these E3 ligase-substrate complexes.

Publications

1. Palicharla VR & Maddika S (2015). HACE1 mediated K27 ubiquitin linkage leads to YB-1 protein secretion. ***Cellular Signalling.*** 27(12): 2355-62.
2. Kapoor R, Arora S, Ponia SS, Kumar B, Maddika S & Banerjea AC (2015). The miRNA miR-34a enhances HIV-1 replication by targeting PNUTS/PPP1R10, which negatively regulates HIV-1 transcriptional complex formation. ***Biochemical Journal*** 470(3): 293-302.
3. Shinde SR & Maddika S (2016). PTEN modulates EGFR late endocytic trafficking and degradation by dephosphorylating Rab7. ***Nature Communications*** 7: 10689.

LABORATORY OF CELL SIGNALLING

Investigating the role of inositol pyrophosphates in eukaryotic cell physiology

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Objectives

Inositol pyrophosphates are derivatives of inositol that contain pyrophosphate or diphosphate moieties in addition to monophosphates. They include diphosphoinositol pentakisphosphate (PP-IP_5, or IP_7) and bis-diphosphoinositol tetrakisphosphate ($[PP]_2$-IP_4 or IP_8), which participate in diverse biological functions, including DNA recombination, vesicular trafficking, rRNA transcription and osmotic regulation. The beta phosphate group of inositol pyrophosphates can be transferred to pre-phosphorylated serine residues on proteins to form pyrophosphoserine. Pyrophosphorylation occurs on several proteins within the cell, including proteins involved in ribosome biogenesis, vesicular trafficking and glycolysis. 5PP-IP_5 (5-IP_7) is synthesised from inositol hexakisphosphate (IP_6) and ATP by IP_6 kinases. Mammals have three isoforms of IP6 kinase, IP6K1, IP6K2 and IP6K3, whereas *Saccharomyces cerevisiae* have a single IP_6 kinase, Kcs1.

Our aim is to understand the molecular mechanisms by which various cellular phenomena are regulated by inositol pyrophosphates. We utilise *S. cerevisiae,* mammalian cell lines, and knockout mouse strains as model systems to investigate the signalling and metabolic pathways that are altered when inositol pyrophosphate levels are perturbed. In particular, we focus on the following objectives:

1. Investigate the cellular functions of mammalian inositol hexakisphosphate kinase 1 (IP6K1);
2. Understand the molecular details of protein pyrophosphorylation by inositol pyrophosphates; and
3. Study the role of inositol pyrophosphates and IP_6 kinases in whole animal physiology.

Summary of work done until the beginning of this reporting year (upto March 31, 2015)

To understand the cellular functions of IP_7 in mammals, we use mouse embryonic fibroblasts (MEFs) derived from *Ip6k1* knockout (*Ip6k1*$^{-/-}$) embryos, which have 70% reduced levels of IP7 compared with wild type (*Ip6k1*$^{+/+}$) MEFs. A gene expression microarray analysis conducted on these MEFs revealed 374 up-regulated and 888 down-regulated genes in cells lacking IP6K1. Pathway analysis tools predicted that the 'regulation of the actin cytoskeleton' is altered in *Ip6k1*$^{-/-}$ MEFs. Our investigations showed that *Ip6k1*$^{-/-}$ MEFs spread more slowly on fibronectin

coated surfaces compared with their *Ip6k1*$^{+/+}$ counterparts.

While examining the role of inositol pyrophosphates in vesicular trafficking, we observed a delay in trafficking of endocytosed transferrin from early endosomes to the endosomal recycling compartment in *Ip6k1*$^{-/-}$ MEFs when compared to *Ip6k1*$^{+/+}$ MEFs. Cells lacking IP6K1 also showed a fragmented Golgi morphology, slower migration of phagosomes towards the perinuclear region and an impaired rate of vesicle movement. These defects were reversed upon the expression of catalytically active but not inactive IP6K1. Since all these trafficking processes are driven by the motor protein dynein, we hypothesized that dynein function may be regulated by IP_7-mediated pyrophosphorylation.

To study the role of inositol pyrophosphates in whole animals, we established a colony of *Ip6k1*$^{+/-}$ heterozygous mice and bred them to obtain wild type and knockout litter-mates. We have reported that *Ip6k1*$^{-/-}$ male mice are sterile due to azoospermia, the absence of mature spermatozoa in the epididymides. We observed that IP6K1 is expressed to high levels in late pachytene and diplotene spermatocytes and in round spermatids. While following the first wave of spermatogenesis, we noted that *Ip6k1*$^{-/-}$ testes display a delay in the completion of meiosis and a major defect in spermiogenesis, the differentiation of round to elongated spermatids.

Details of progress made in the current reporting year (April 1, 2015 - March 31, 2016)

Project 1: Cellular functions of mammalian inositol hexakisphosphate kinase 1 (IP6K1)

To determine whether the role of IP6K1 in regulating actin cytoskeleton dependent cellular functions extends to cancer cells, we stably expressed shRNA directed against *Ip6k1* in HeLa and HCT116 human cancer cell lines. We obtained HeLa cells with an approximately 80% decrease in IP6K1 expression and HCT116 cells with 60% knockdown (Figure 1A). Analysis of the soluble inositol polyphosphate profile in these cells revealed different patterns in HeLa and HCT116 cells (Figure 1B, C). HeLa cells showed a substantial reduction in the inositol pyrophosphate PP-IP_4 but no change in IP_7, whereas HCT116 cells showed a significant reduction in IP_7 and a slight decrease in PP-IP_4. Both cell lines displayed a marginal reduction in IP_6 but no change in IP_5. The reduced levels of IP_6 upon depletion of IP6K1, while unexpected, is supported by the high rate of metabolic turnover reported for inositol pyrophosphates, and suggests that cells may evolve compensatory mechanisms in an attempt to maintain the ratio of inositol pyrophosphates to their precursor inositol polyphosphates. Depletion of IP6K1 in both HeLa and HCT116 cells resulted in a significant decrease in chemotactic migration towards serum-rich medium over a period of 24 h (Figure 1D-F). We also performed a wound healing assay on confluent monolayer cultures to look at collective cell migration, and observed reduced migration in IP6K1 depleted HeLa and HCT116 cells (Figure 1G-I). Together, our data show that the depletion of IP6K1 lowers chemotactic and collective migration in these cancer cell lines. To investigate the in vivo significance of these observations, we will study the tumourigenesis potential of cells with lowered IP6K1 levels.

Project 2. Inositol pyrophosphates regulate dynein-dependent vesicular trafficking

We examined whether the function of the motor protein dynein is regulated by IP_7-mediated pyrophosphorylation. Dynein is a 1.6 MDa multi-protein complex composed of two heavy chains that drive movement on microtubules, two intermediate chains (IC) that bind to the heavy chains and to vesicles, and additional light intermediate and light chains. By mass spectrometry, we identified that the mouse dynein intermediate chain IC-2C is phosphorylated by CK2 on three Ser residues, one of which, Ser 51, is also a consensus site for IP_7-mediated pyrophosphorylation. A fragment of IC-2C containing the N-terminal 111 amino acid residues is pyrophosphorylated by IP_7 in vitro subsequent to phosphorylation by CK2 (Figure 2A). The same protein fragment with a single substitution of Ser 51 with Ala is not pyrophosphorylated, confirming that Ser 51 is the only residue targeted by IP_7 in the IC-2C N-terminus. We used an indirect 'back-phosphorylation' strategy to determine whether endogenous IC is pyrophosphorylated by endogenous IP_7. We immunoprecipitated IC from *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ MEFs and used these proteins as substrates in an in vitro pyrophosphorylation reaction using radiolabelled IP_7 (Figure 2B). We observed a complete abrogation of 5[β^{32}P]-IP_7 mediated pyrophosphorylation of native IC from *Ip6k1*$^{+/+}$ MEFs, suggesting that this protein is heavily pyrophosphorylated in vivo.

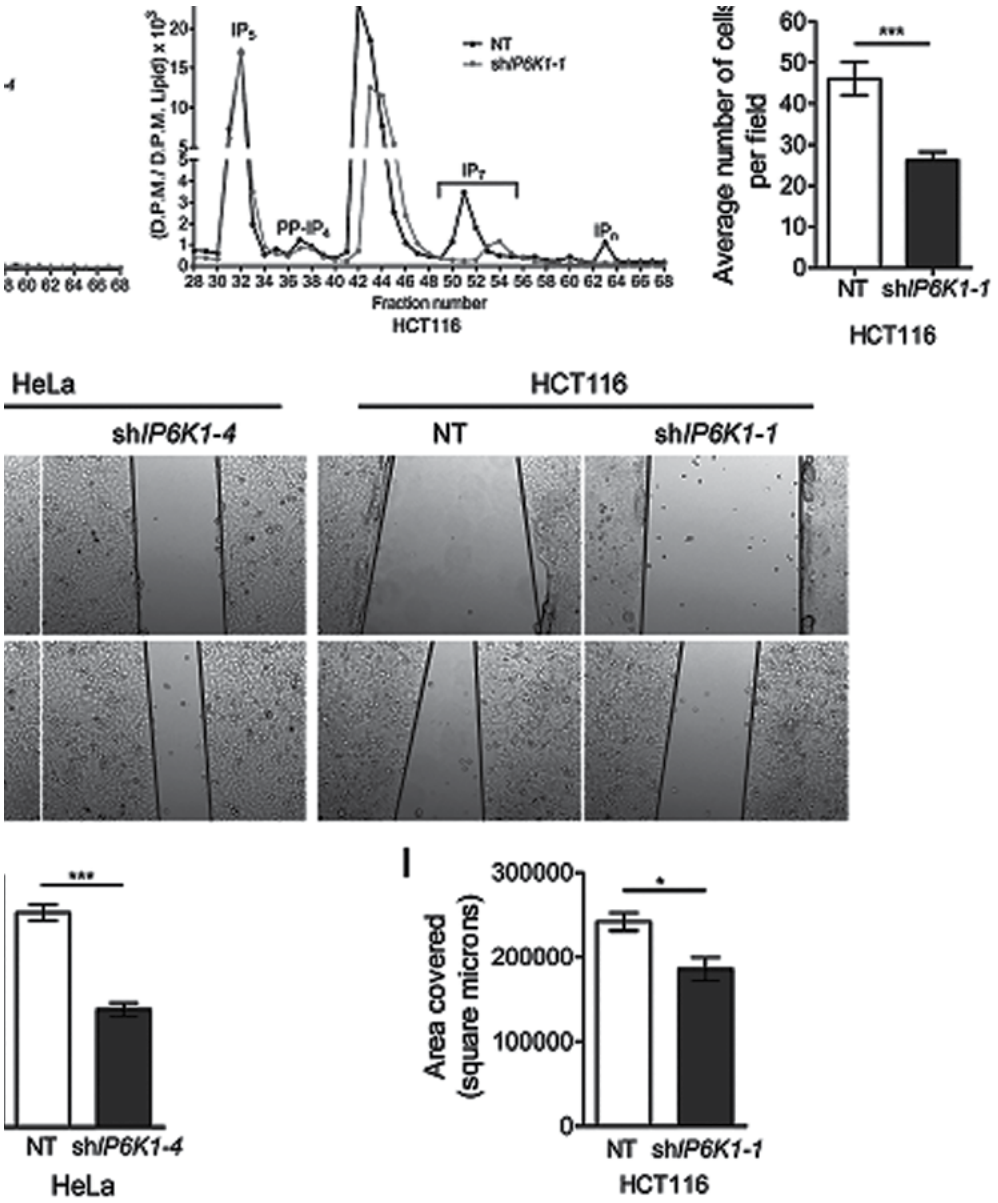

Figure 1. IP6K1 depletion reduces cancer cell migration. (A) Immunoblot to detect IP6K1 in lysates from HeLa and HCT116 cell lines that stably express the indicated shRNA (NT, non-targeting control; sh*IP6K1-1* and sh*IP6K1-4*, two different shRNA sequences directed against human IP6K1). The percentage knockdown of IP6K1 expression is indicated. Data are mean ± SEM from three independent experiments. (B, C) HPLC profile of [^{3}H] inositol labelled HeLa NT and sh*IP6K1-4* (B) and HCT116 NT and sh*IP6K1-1* (C) cell lines. Soluble inositol phosphate counts were normalized to the total lipid inositol count for each sample. Peaks corresponding to IP_5, PP-IP_4, IP_6 and IP_7 are indicated. Data are representative of two independent experiments. (D) Transwell migration was assessed in the indicated cells lines. Cells that migrated towards serum-rich medium 24 h after seeding were visualized by staining with DAPI. Scale bars represent 50 µm. (E, F) Quantification of (D); the bar graphs show the average number of cells migrated per field in HeLa (E) or HCT116 (F); data represents mean ± SEM (n = 127 and 134 fields respectively for NT control and sh*IP6K1-4* expressing HeLa; n = 152 and 186 fields respectively for NT control and sh*IP6K1-1* expressing HCT116 cells) compiled from three independent experiments and was analysed using the non-parametric two-tailed Mann-Whitney test. (G) Scratch wound healing assay on confluent monolayers to monitor collective cell migration in the indicated cell lines. Representative images are shown for the indicated time points. Black lines overlaid on the images mark the edges of the wound. (H, I) Quantification of area covered after 18 h in HeLa (H) or HCT116 (I) cells. Data represents mean ± SEM from three independent experiments and was analysed using a two-tailed unpaired Student's t-test. *** P≤0.001; * P≤0.05,

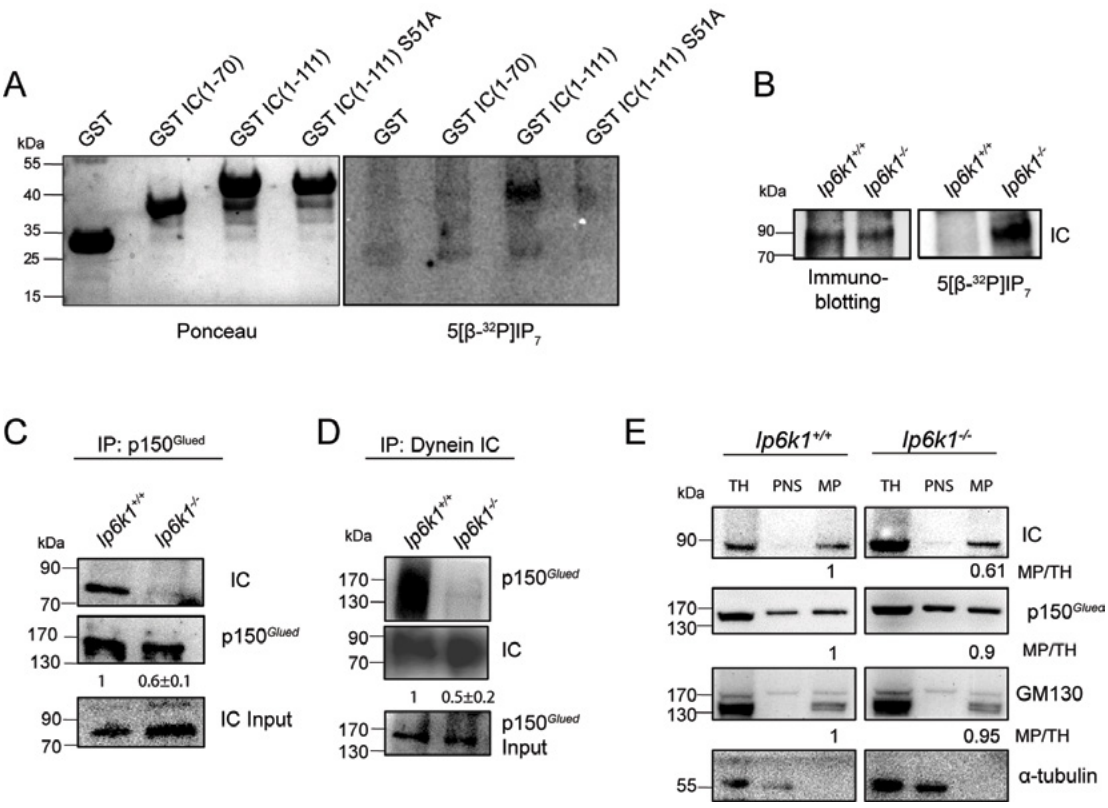

Figure 2. Effect of IP_7-mediated pyrophosphorylation on dynein intermediate chain. (A) Bacterially expressed and purified GST or GST-tagged IC(1-70), IC(1-111) and IC(1-111)S51A were pre-phosphorylated with CK2 and unlabeled ATP and incubated with 5[β-^{32}P]IP_7. Proteins were resolved using NuPAGE and transferred to a PVDF membrane. Pyrophosphorylation was detected by phosphorimager scanning (right) and the proteins were detected by Ponceau S staining (left). (B) Back-pyrophosphorylation of endogenous IC by IP_7. Native IC immunoprecipitated from *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ MEFs was incubated with 5[β-^{32}P] IP_7. Proteins were resolved using NuPAGE and transferred to a PVDF membrane. Pyrophosphorylation was detected by phosphorimager scanning (right) and proteins were detected by Western blotting (left). (C, D) Co-immunoprecipitation of dynein IC and p150^{Glued} from *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ MEFs. Protein extracts were cross-linked with a thiol-cleavable cross-linker, followed by immunoprecipitation of p150^{Glued} and IC. Representative immunoblots of co-immunoprecipitation of IC with p150^{Glued} (C) and p150^{Glued} with IC (D). The levels of co-immunoprecipitated IC or p150^{Glued} were normalized to the level of the immunoprecipitated partner. The fold change in the extent of co-immunoprecipitation in *Ip6k1*$^{-/-}$ compared to *Ip6k1*$^{+/+}$ MEFs is indicated as mean ± SEM from three independent experiments. (E) Subcellular fractions from *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ MEFs prepared by differential centrifugation were resolved on a 4-12% NuPAGE gel, and immunoblotted to detect dynein IC and p150^{Glued} in total homogenate (TH), post-nuclear supernatant (PNS) and membrane pellet (MP). Increased amount of protein was loaded in the *Ip6k1*$^{-/-}$ fractions to enable visualization of the dynein IC. GM130 was used as a membrane marker and α-tubulin was used as a loading control. The level of each protein in the MP fraction was normalized to its levels in TH. The fold change in protein levels in *Ip6k1*$^{-/-}$ compared to *Ip6k1*$^{+/+}$ MEFs is indicated below each blot.

Conversely IC from *Ip6k1*$^{-/-}$ MEFs was robustly pyrophosphorylated in vitro, implying that loss of IP6K1 leads to diminished pyrophosphorylation of IC inside cells. The dynein motor is known to bind vesicles by interacting with the multi-subunit protein complex, dynactin. One of the main sites of association of these protein complexes is an interaction of the dynein intermediate chain with the p150^{Glued} subunit of dynactin. We conducted co-immunoprecipitation assays of IC and p150^{Glued}, and noted a significant decrease in the extent of their interaction in extracts from *Ip6k1*$^{-/-}$ MEFs compared with *Ip6k1*$^{+/+}$ MEFs (Figure 2C, D). To monitor whether the decrease in dynein-dynactin interaction leads to reduced dynein recruitment on vesicle membranes, extracts from *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ MEFs were subjected to differential centrifugation. The amounts of IC, p150^{Glued}, and Golgi matrix protein GM130 in the membrane fraction were normalised to their levels in the total homogenate. The membrane-enriched fraction from *Ip6k1*$^{-/-}$ MEFs showed reduced amounts of IC compared with *Ip6k1*$^{+/+}$ MEFs (Figure 2E). In contrast, the levels of

p150^{Glued} and GM130 were unchanged in the same extracts.

In summary, our study has identified inositol pyrophosphates as novel regulators of dynein function. Cells with reduced levels of inositol pyrophosphates exhibit defects in dynein-dependent vesicle transport. Inositol pyrophosphate-mediated serine pyrophosphorylation of IC promotes its interaction with the p150^{Glued} subunit of dynactin, thereby facilitating attachment of the dynein motor to vesicles. A manuscript describing this work is currently under revision.

Project 3. Physiological role of IP^7 in mice: Regulation of spermatogenesis by IP6K1

To investigate whether delayed meiotic progression in *Ip6k1*$^{-/-}$ male mice is due to defects in meiotic recombination, we stained spermatocyte spreads to detect the DNA double strand break (DSB) marker phosphorylated histone H2AX (γ-H2AX). Spermatocytes in the different stages of prophase I were identified by synaptonemal complex protein 3 (SCP3) staining. Equal γ-H_2AX staining was observed in leptotene spermatocytes from *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ testes (Figure 3A), marking the successful initiation of meiotic recombination by Spo11-induced DNA DSBs. In *Ip6k1*$^{+/+}$ pachytene spermatocytes γ-H_2AX was only observed in the XY body, where it is known to coat the sex chromosomes that do not participate in synapsis, but persistent γ-H2AX staining was seen throughout *Ip6k1*$^{-/-}$ pachytene spermatocytes (Figure 3B). We detected unrepaired DNA by in situ TUNEL labelling in pachytene spermatocytes from *Ip6k1*$^{-/-}$ but not *Ip6k1*$^{+/+}$ testes (Figure 3C). However, immunostaining of testis sections with the apoptotic marker cleaved caspase 3 revealed that *Ip6k1*$^{-/-}$ spermatocytes do not undergo apoptosis despite the presence of DNA breaks (Figure 3D). By staining testes sections to detect the secondary spermatocyte marker, histone H3 phosphorylated on Ser10 (H3S10), we observed that the number of secondary spermatocytes were unchanged in *Ip6k1*$^{-/-}$ seminiferous tubules. This suggested that despite its involvement in maintaining meiotic germ-line genome integrity, the loss of IP6K1 does not affect the completion of meiosis. Since *Ip6k1*$^{-/-}$ spermatocytes complete meiosis while carrying DNA breaks, we examined post-meiotic cells to determine whether this DNA damage persists. γ-H2AX foci were clearly seen in round spermatids in *Ip6k1*$^{-/-}$ but not *Ip6k1*$^{+/+}$ testis sections (Figure 3E). *Ip6k1*$^{-/-}$ round spermatids were also positive for in situ TUNEL labelling, but did not contain cleaved caspase 3 (Figure 3F), indicating that they still do not undergo apoptosis. We also examined elongating spermatids in the same testis sections. DNA in elongating spermatids is known to undergo Topoisomerase IIβ-mediated breaks as part of their developmental programme. By stage XII, these breaks are repaired in *Ip6k1*$^{+/+}$ tubules, but *Ip6k1*$^{-/-}$ tubules continue to remain TUNEL positive even at stage II-III (Figure 3G). These *Ip6k1*$^{-/-}$ tubules also stained positive for cleaved caspase 3 (Figure 3H), indicating that the elongating spermatids undergo apoptosis and are eventually lost. These data suggest that the persistence of unrepaired DNA breaks in round spermatids may lead to improper nuclear condensation of *Ip6k1*$^{-/-}$ elongating spermatids, and contribute to azoospermia observed in mice lacking IP6K1.

Publications

1. Thota SG, Unnikannan CP, Thampatty SR, Manorama R and Bhandari R (2015). Inositol pyrophosphates regulate RNA polymerase I-mediated rRNA transcription in *Saccharomyces cerevisiae.* ***Biochemical Journal*** 466: 105-114.

2. Thota SG and Bhandari R (2015). The emerging roles of inositol pyrophosphates in eukaryotic cell physiology. ***Journal of Biosciences*** 40: 593-605.

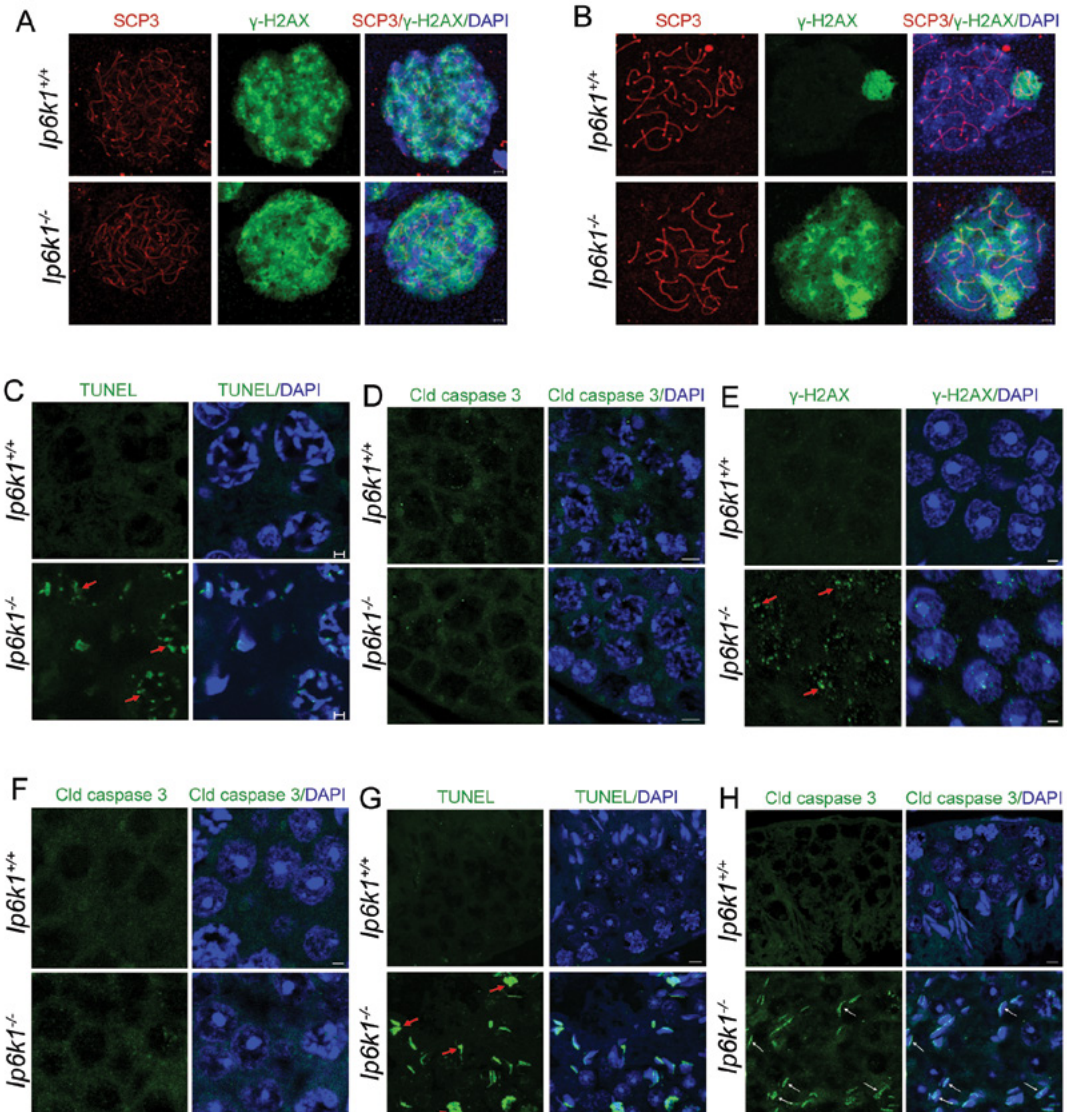

Figure 3. Loss of IP6K1 causes meiotic and post meiotic genomic instability. (A, B) Immunolabelling of surface spreads of primary spermatocytes from *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ testes with the DNA double strand break marker, γ-H2AX (green) and synaptonemal complex protein 3, SCP3 (red). Nuclei were counterstained with DAPI. Scale bars = 2μm. (C) TUNEL staining (green) in pachytene spermatocytes of *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ testes cross sections. Arrows indicate TUNEL positive *Ip6k1*$^{-/-}$ spermatocytes. Scale bars = 2μm. (D) Cleaved caspase 3 (green) staining in *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ testes suggesting that *Ip6k1*$^{-/-}$ spermatocytes do not undergo apoptosis despite carrying DNA breaks. Scale bars = 5μm. (E) Immunostaining of *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ round spermatids with γ-H2AX (green). Post-meiotic round spermatids in *Ip6k1*$^{-/-}$ mice exhibit DNA damage (arrows). Spermatid nuclei were counterstained with DAPI. Scale bars = 2μm. (F) Immunolabelling of cleaved caspase 3 (green) in *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ round spermatids. Cleaved caspase 3 is not detected in *Ip6k1*$^{-/-}$ round spermatids although they exhibit DNA damage. Spermatid nuclei were counterstained with DAPI. Scale bars = 2μm. (G) TUNEL (green) staining of *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ testes cross sections. Arrows indicate intense TUNEL staining in *Ip6k1*$^{-/-}$ elongating spermatids. Spermatid nuclei were counterstained with DAPI. Scale bars = 5μm. (H) Cleaved caspase 3 (green) staining in *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ testes cross sections indicating apoptotic elongating spermatids (arrows) in *Ip6k1*$^{-/-}$ testes. Spermatid nuclei were counterstained with DAPI. Scale bars = 5μm.

LABORATORY OF CHROMATIN BIOLOGY AND EPIGENETICS

Understanding functions and regulation of Sirtuin family protein deacetylases

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Objectives

Reversible acetylation/deacetylation of proteins regulates numerous important cellular processes. The Sirtuin family of protein/histone deacetylases (HDAC) are conserved enzymes that require NAD+ to deacetylate proteins. Sirtuins carry out a broad range of crucial cellular functions ranging from transcriptional silencing to DNA damage response, cell cycle regulation, metabolism and aging etc. Their molecular functions in DNA metabolic processes such as DNA replication and repair have not been studied extensively. During some of these processes, the expression level of specific sirtuins is known to alter, indicating conditional regulation of these proteins. However, the mechanism of regulation of sirtuin expression under many of these conditions remains elusive.

Our aim is to understand the molecular functions and mechanism of regulation of sirtuins during DNA damage response and repair. Since fission yeast *S. pombe* is more closely related to higher eukaryotes and sirtuins are conserved from yeast to mammals, we use fission yeast *S. Pombe* as a model systems to study sirtuin biology. Fission yeast, *S. pombe* has three Sirtuins, Sir2, Hst2 and Hst4. Deletion analysis and other studies have shown that all these genes function in transcriptional silencing. However, deletion of only *hst4*+ gene, not *sir2*+ and *hst2*+ genes, show interesting phenotypes of slow growth, elongated morphology, fragmented DNA and DNA damage sensitivity indicating it could have additional functions. These phenotypes are very useful tools to uncover novel signaling pathways where Hst4 could be functioning. We focused on the following objectives:

1) Understanding the molecular functions of sirtuin family NAD+ dependent histone/ protein deacetylases.
2) Investigating the molecular mechanism of regulation of fission yeast sirtuin Hst4.

Project 1: To decipher novel functions of sirtuin family NAD+ dependent histone deacetylase Hst4 of fission yeast.

Summary of work done until the beginning of this reporting year (upto March 31, 2015)

We had previously reported that deletion of fission yeast *S. Pombe* sirtuin hst4+ causes slow growth, elongated morphology, DNA fragmentation phenotypes, hyperacetylation of histone H3 lysine56 and S phase delay. To decipher novel functions of Hst4, a slow growth and DNA damage sensitivity phenotype suppressor screen has been carried out. Among the suppressor genes identified by this screen are a few genes encoding proteins involved in DNA replication. These genetic interactions indicated that Hst4 may be involved in regulation of DNA replication. Interestingly, one among these is Mcl1, an orthologue of budding yeast Ctf4, a DNA polymerase alpha interacting protein, crucial for DNA replication and sister chromatid cohesion. These genetic interactions indicated that Hst4 could be involved in regulation of DNA replication. To decipher the function of Hst4 in DNA replication, we are studying interaction of Hst4 with Mcl1. The phenotypes of hst4Δ mutants are mainly attributed to increased H3K56Ac levels. We have observed that the H3K56ac levels remain unchanged on over

expression of the suppressor gene indicating that it does not simply reduce H3K56ac levels by recruiting another deacetylase. The phenotypes of the H3K56R and H3K56Q mutants which mimic constitutive deacetylated and acetylated states respectively are similar to hst4Δ mutants. We have shown that Mcl1 expression could not suppress the phenotypes of these mutants. These results suggested that recovery of hst4Δ phenotypes by overexpression of Mcl1 is not dependent on H3K56 acetylation.

The phenotypes of hst4Δ mutants such as slow growth, elongated morphology and DNA damage sensitivity are similar to that of mcl1Δ mutants. To examine whether hst4 and mcl1 interact epistatically or exhibit synthetic lethality, the individual hst4Δ mutant and mcl1Δ were crossed to generate a double mutant. The double deletion mutants were viable and showed growth rate and MMS sensitivity similar to that of hst4Δ mutants. These results show that Mcl1 might act in the same pathway downstream of Hst4. Since it functions in DNA replication, we are currently investigating potential function of Hst4 in DNA replication.

The hst4Δ mutants show delayed S-phase. The delay in S-phase might be due to elevated and persistent levels of H3K56 acetylation resulting in firing of dormant origins or could be because Hst4 is involved in regulation of replisome by targeting one or more replisome components or combination of both. Mcl1 is crucial for DNA replication as it couples DNA polymerase to helicase. Therefore, to test whether mcl1 recovered the S-phase delay in hst4Δ mutants, the wild type and hst4Δ mutant strains were arrested in G2 and progression through the cell cycle was monitored using flow cytometry. The results showed that overexpression of Mcl1 could partially rescues the S-phase delay of hst4 deletion mutants; however the rate of progression was slower than the wild type. This data indicate

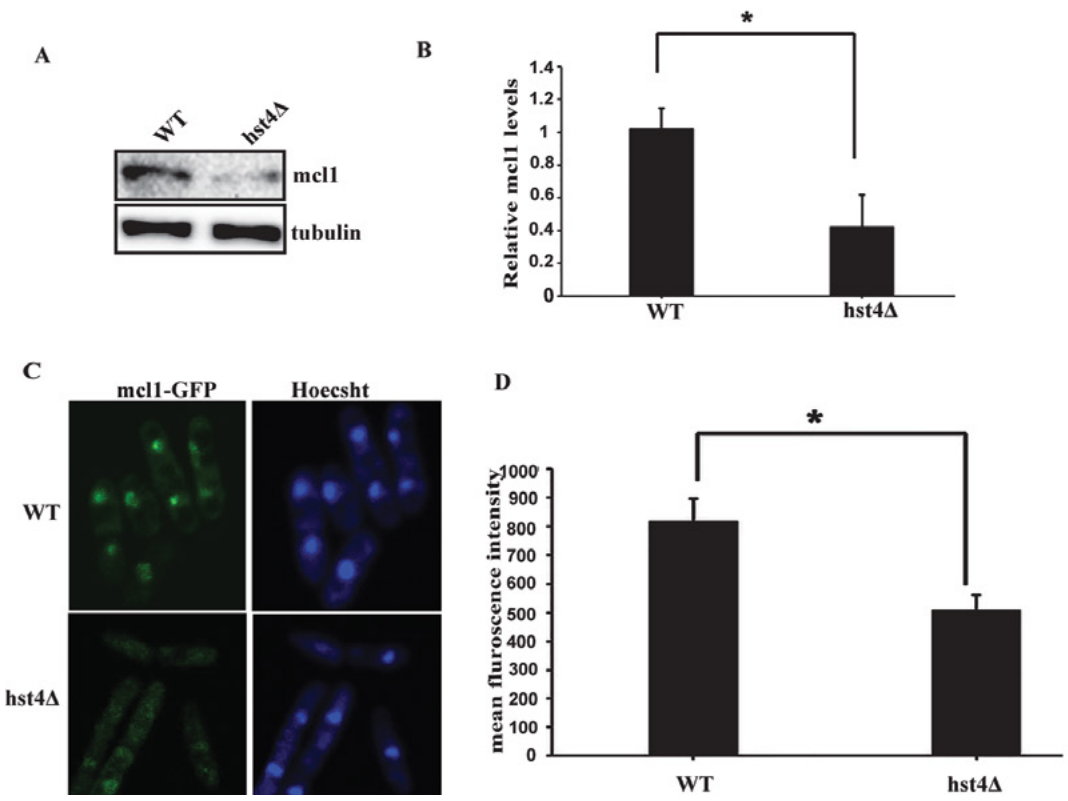


Figure 1. Hst4 regulates mcl1 expression. (A) Western blot showing expression of Mcl1. WT and hst4Δ strains were grown, whole cell lysates were prepared and the levels of Mcl1 were monitored by western blotting using anti-Mcl1 antibody. B) Qunatification of protein levels. C and D) Mcl1 levels determined by fluorescence microscopy E) Over-expression of Hst4 restores Mcl1 expression in hst4Δ mutants.

Hst4 affect S phase progression by regulating Mcl1 and the partial recovery might be due to hyperacetylated chromatin in hst4Δ mutants which may impede DNA replication process.

Details of progress made in the current reporting year (April 1, 2015 - March 31, 2016)

DNA replication is very tightly regulated process. The coupling between CMG helicase and DNA polymerases is a crucial determinant for DNA replication control. In *S. cerevisiae,* it has been shown that Mcl1 homolog, Ctf4 functions in coupling the DNA polymerase alpha and the helicase. Also, Ctf4 is a major target of H3K56 acetylation pathway. Our earlier (unpublished) data showed that fission yeast ortholog of Ctf4, Mcl1 overexpression could suppress hst4Δ mutant phenotypes and H3K56 acetylation is not required for this suppression. Therefore, we hypothesized that Mcl1 levels might be low in hst4Δ mutants resulting in the slow S phase progression. To examine if Mcl1 expression is altered in hst4Δ mutants, we checked Mcl1 levels in wild-type and hst4Δ mutants via western analysis and observed two fold lower amounts of Mcl1 in hst4Δ mutants compared to the wild-type cells (Fig.1A, 1B), suggesting that Hst4 is regulating mcl1 expression. Next, the expression of Mcl1 in WT and hst4Δ mutant yeast strains bearing endogenous GFP-tagged mcl1 gene was checked using fluorescence microscopy. This data confirmed a decrease in Mcl1 level in hst4Δ mutants (Fig.1C,1D). To further confirm regulation of Mcl1 by Hst4, we tested whether over expression of Hst4 will increase expression of Mcl1. Over expression of Hst4 or Mcl1 was carried out in hst4Δ mutants. The results presented in fig. 1E showed that Hst4 is required for expression of Mcl1. To check whether deletion of hst4 affect the expression other replication proteins. Next we tested the expression of other replication proteins such as Pol1; sub-unit of DNA polymerase α that binds to Mcl1, Mcm complex; helicase component, PCNA; clamp loader in hst4Δ mutants. We did not observe any significant differences in the expression of other replication proteins. Collectively, these results reveal that sirtuin Hst4 is specifically regulate the expression of Mcl1. Currently, we are working on understanding the mechanism of regulation of Mcl1 and investigating whether Hst4 affect the process of DNA replication by regulating the coupling of DNA polymerase and helicase via Mcl1.

Project 2: Understanding the molecular mechanism of regulation of fission yeast sirtuin Hst4.

Details of progress made in the current reporting year (April 1, 2015 - March 31, 2016)

This is a new activity, which aims to understand the molecular mechanism of regulation of fission yeast sirtuin Hst4. HDACs are known to be regulated in different ways and the mechanism of regulation is often determined by specific function dependent signal for regulation. The sirtuins family HDAC, Hst4 of *S. pombe* has been shown function in maintenance of genome stability. Deletion of Hst4 causes variety of DNA damage phenotypes. The expression of Hst4 oscillate in normal cell cycle as well as when cells are exposed to DNA damage. The timely oscillation of these proteins is important for maintaining genomic integrity. However, the molecular machinery for the degradation of Hst4 in S phase as well as during DNA damage is not known. As Hst4 is known to play an important role in maintaining genomic integrity, its regulation kinetics is needed to be studied to understand the role of chromatin during DNA damage and the regulation of these pathways in fission yeast. This project is aimed at investigating mechanism of regulation of Hst4 during DNA damage stress and also, to gain further insights into the replication stress associated DNA damage pathway in fission yeast.

To investigate the mechanism of regulation, *in vivo* protein stability of Hst4p was monitored by cycloheximide treatment which is a protein synthesis inhibitor. Wild type cells were grown till mid log phase in rich medium and cycloheximide was added at the concentration of 100 microgram/ml and cells were collected at different time points and immunoblotted. In asynchronous cultures consisting largely of G2 population, Hst4p is stable till 60 minutes and its half-life is between 30 and 60 minutes (Figure 2A). The post-translational mechanism of degradation of proteins is mainly mediated by ubiquitination. As the half-life of hst4 was found to be less in the asynchronous population, we hypothesized the role of ubiquitination in the degradation of Hst4. In order to check the role of proteosome in the regulation of Hst4, half life assay was done in the wildtype and proteosome mutant (mts2-1) strain using cycloheximide as discussed above. As shown in Fig.2B Hst4 levels were stabilized in proteosome mutant significantly as

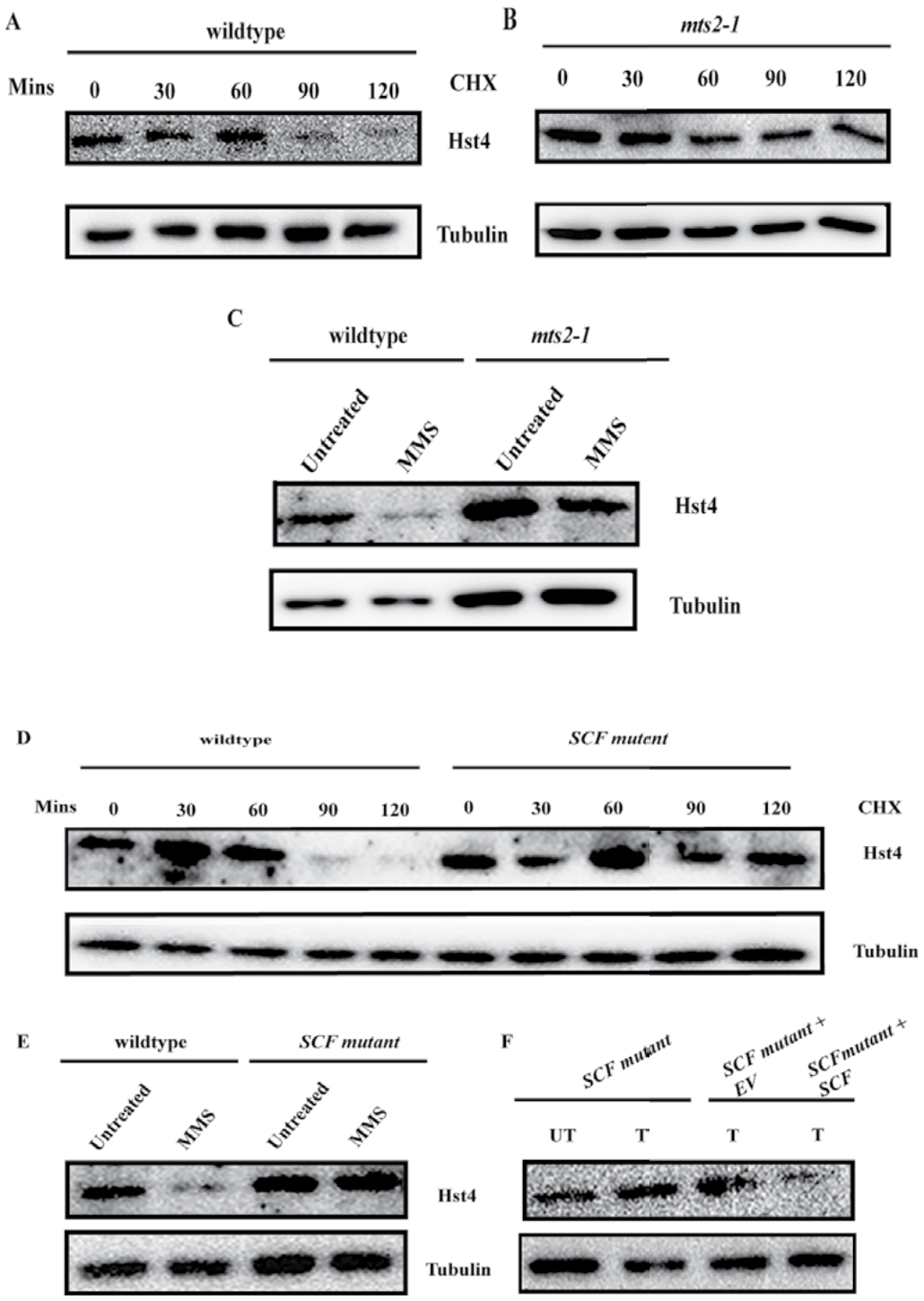


Figure 2. Fission yeast sirtuin Hst4 is regulated by ubiquitin ligase SCF mediated proteolysis in response to DNA damage. (A) and (B) Western blot showing stability of Hst4 in proteasome mutant (mts2-1), Wild type and mts2-1 strains (26S proteasome mutant) were grown and treated with cycloheximide for indicated time and Hst4 level detected by western blotting (C) Stabilization of Hst4 levels on DNA damage in mts2-1 strain. **Hst4 is regulated by SCF ubiquitin ligase** (D) Wild type and SCF mutant strains was grown and treated with cycloheximide for indicated time and Hst4 level detected by western blotting (E) Stabilization of Hst4 levels on DNA damage in SCF mutant strain. (F) Rescue of degradation by overexpression of SCF component in its mutant background.

compared to wild type. Further the levels of Hst4 on DNA damage were also been checked in the mutant strain. Fig.2 C shows stabilization of Hst4 in *mts2-1* strain during MMS treatment as compared to wild-type strains. Thus, these results show that Hst4 is regulated by ubiquitin mediated proteosomal degradation.

E3 ligases are the most important in ubiquitination as they specify the substrates targeted for ubiquitination. The SCF ubiquitin ligase is a conserved E3 ligase which regulates the expression of many cell cycle proteins which in turn regulates the G1/S switch. To study the role of SCF ubiquitin ligase in the regulation of hst4, stability of Hst4 protein was determined in SCF mutant strain (Fig 2D). Hst4 was stabilized in SCF mutant significantly as compared to wild type. This was comparable to the stability of Hst4 observed in proteosomal mutants (2B). Hst4 is known to be down regulated when cells are exposed to DNA damaging agent MMS (Methy methane sulphonate). To examine if decrease in level of Hst4 on DNA damage is also mediated through SCF ubiquitin ligase, Hst4 levels were determined in SCF mutant by western blot. The level of Hst4 did not decrease on MMS treatment in SCF mutant (2E). Further, the degradation of hst4 was rescued by the plasmid complementation of SCF component back in the null background. (Fig 2F). Collectively, these results show that Hst4 is regulated by ubiquitin ligase SCF mediated proteolysis in response to DNA damage. Work is underway to determine whether degradation of Hst4 on DNA damage is phosphorylation dependent as SCF complex recognize phosphorylated protein and if the degradation of Hst4 is mediated by DNA damage checkpoint proteins.

Publications

1. Reddy ER, Yellanki S, Medishetty R, Konada L, Alamuru NP, Haldar D, Parsa KVL, Kulkarni P and Rajadurai M (2015). Red Fluorescent Organic Nanoparticle Bioprobes: A Photostable Cytoplasmic Stain for Long Term In Vitro and In Vivo Visualization. ***Chem Nano Mat.*** 1: 567–576.

Other Publications

2. Haldar D (2016). Emerging epigenetic therapy of cancer. ***Spinco Biotech Cutting Edge*** 5 (10): 9-14.

LABORATORY OF CHROMOSOME STRUCTURE & DYNAMICS

Investigating the role of chromosome dynamics in microbial diversification

Faculty	Mohan C Joshi	Ramalingaswami Fellow
PhD Students	Bharat Chandra Dash	Junior Research Fellow
Other Members	Divya Matta	Technical Assistant

Objectives

Research in my lab is aimed at understanding how (a) nucleoid structure & organization is modulated during cell-cycle; and (b) cohesion regulated homologous recombination processes in *E. coli.* The long-term goal of my lab is to understand how chromosome dynamics dictates genetic diversity in bacteria.

Details of progress made in the current reporting year (August 20, 2015 - March 31, 2016)

Project. 1.Nucleoid structure & organization is modulated during cell-cycle

Single locus studies (FROS or FISH) have demonstrated that *E. coli* chromosome is highly dynamic and fluidic entity. However, spatial mapping of *E. coli* genome in high-resolution during cell-cycle (G0-S-M) remains an uphill task for cell-biologist. We have been working on "multi-color FISH" (Fluorescence *In Situ* hybridization) based approach to address this challenge. The approach integrates genetics, biochemical & high-end fluorescence imaging techniques with a MATLAB based image analysis software (Figure 1).

We are writing a PYTHONcode for our existingImage Processing software (MATLAB based) as well as adding new image processing and quantification plugin to streamline image quantification process.It is noteworthy to mention

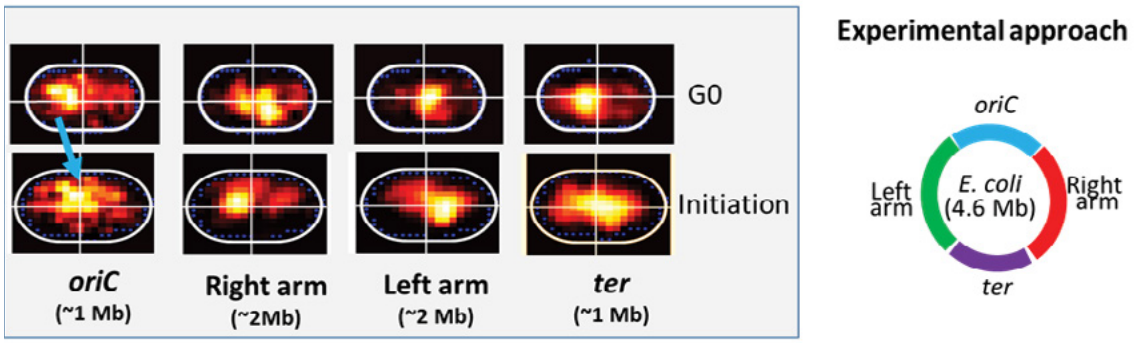


Figure 1. High-Resolution mapping of *E. coli* nucleoid using GANGA: Spatial localization of four different regions (left) of *E. coli* nucleoid within the cell (schematic, right).

here thatsuchPYTHON basedfree license software can be used in various imaging process related to eukaryotic organismsthat exists within CDFD.

Project 2:Chromosome cohesion mediated regulation of Homologous recombination

Homologous recombination (HR) is the major source of antibiotic-resistant gene expansion in pathogenic microbes. HR processes are conserved in all organisms, playing an important role in genomic maintenance during repair of DNA double strand breaks (DSBs) and reactivation of stalled replication fork. However, HR can also induce genomic instability via gene conversion, crossing over and mutation incorporation (under stress), thereby resulting in gene translocations, deletions, amplifications, inversions and loss of heterozygosity. Therefore HR plays a pivotal role in maintaining the equilibrium between genomic integrity and genetic diversity. Although HR is an extensively studied process, it remains unclear how this equilibrium is regulated during DNA repair. Recent data including our own suggested

that chromosome cohesion is an evolutionary conserved process and bacteria may also utilize a cohesion dependent mechanism for DSB repair. Therefore, *E. coli* provides a highly tractable and mutable model to test the role of cohesion in HR dependent DSB repair.

The focus will be on understanding whether/how (i) cohesion timing along the genome influences the efficiency of DSB repair; (ii) cohesion timing along the genome regulates accumulation of spontaneous and stress-induced mutation; and (iii) cohesion promotes genomic integrity and dictates the hot-spots for alteration along the genome, in *E. coli.* This knowledge will be insightful in understanding the mechanism underlying microbial diversity.

We are developing *E. coli* strains, in which a unique restriction enzyme cute site (I Sce-1) will be introduced at different loci across genome. This will be achieved using linear DNA recombineering technique, which allows target specific insertion of linear DNA across genome.For all of these genetic loci we have experimentally determined the cohesion timing. These strains will be verified using PCR,subsequently gene encoding for Isce-1 enzymeunder the control of arabinose promoter will be introduced into these strains using P1 based transduction method. We have designed primers for following genetic loci; *rfaJ, oriC&psd* and will be optimizing PCR and linear DNA recombineering method to generate these strains.

LABORATORY OF COMPUATIONAL BIOLOGY

Computational studies on protein structure, function and interactions

Faculty	HA Nagarajaram	Staff Scientist
PhD Students	Rachita HR	Senior Research Fellow (Till October, 2015)
	Suryanarayana Seera	Senior Research Fellow
	V A Ramesh	Senior Research Fellow
	Rakesh Trivedi	Senior Research Fellow
	Arijita Mitra	Junior Research Fellow
	K Guruprasad	Junior Research Fellow (since July 2015)
Other Members	Dr U S Raghavender	SERB-DST Young Scientist (Since January 2016)
	Rahul S Dhakne	Project JRF
	Rajkishore Mohapatra	Project JRF (till Feb.2016)

Collaborators (The New Indigo Project):

Srikanth Rapole	NCCS, Pune
Jochen Schubert	University of Rostock, Germany
Jose Camara	University of Madeira, Portugal

Objectives

1 Sequence and structural analyses on disease causing mutations in human proteins

2 Investigations on the evolution and conformational heterogeneity of instrinsically disordered regions in proteins

3 The New Indigo Project

   a. Multivariate analysis of the volatile compounds (VOCs) detected from the breath and urine samples of breast, lung and colon cancer patients and healthy individuals, as a means to identify potential cancer biomarkers; and

   b. Development of a database of VOCs detected by collaborators and a web portal hosting the database and other information related to this project

Summary of work done until the beginning of this reporting year (upto March 31, 2015)

1. Domains and motifs that mediate physical interactions between human and viral proteins were identified and studied. It was found that some of the viral proteins harbour ELMs (eukaryotic linear motifs) that interact with their binding domains in human proteins.

2. Structural analysis of known IDPs complexed with their interacting partners was carried out and it was found that most of the disordered regions in IDPs adopt helical structures when complexed with other human proteins.

3. HANSA was retrained using a new HUMSVAR dataset. We further explored usefulness of network centrality values of human proteins as additional features in HANSA.

4. A web portal was developed to host various information and also a database of volatile compounds detected in the breath, urine and saliva samples of breast and lung cancer patients.

Details of progress made in the current reporting year (April 1, 2015 - March 31, 2016)

Project 1: Prediction of pathogenic effect of missense mutations: Incorporation of additional features into HANSA.

1. Having found that the human proteins harbouring disease mutations are associated with high centrality values in the protein-protrein interaction network (PPIN) as compared with the human proteins horbouring neutral mutations, we trained a new SVM model with 13 features (10 features used in HANSA along with the three network centrality values degree, betweeness and closeness as additional features) using the new Humavar dataset that comprises of 22,196 disease mutations from 1852 proteins and 21,151 neutral mutations from 8791 proteins and was subjected to 5-fold cross validation. The ROC plot generated for the new SVM model is shown in Fig.1.

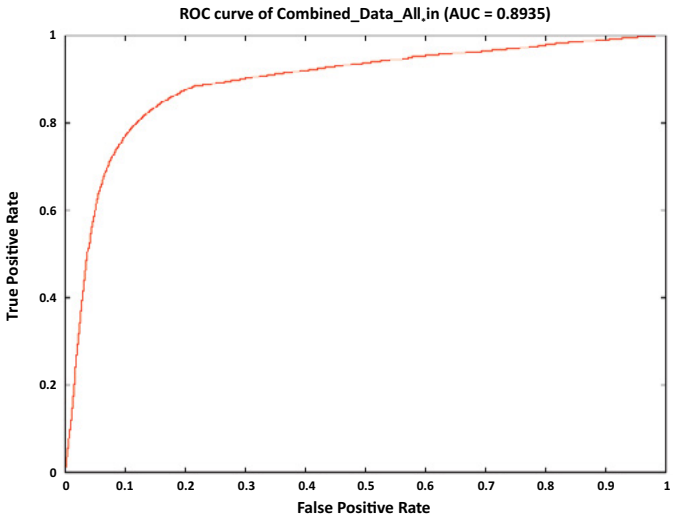


Figure 1. ROC plot obtained from the 5 fold cross validation of the new HANSA trained with additional features based on the network centrality measure of the proteins harbouring disease and neutral mutations. The area under the curve (AUC) value is 0.9.

2. All the structure-based methods for prediction of functional impact of missense mutations including HANSA are based on the premise that, disease-causing mutations destabilize folded proteins harbouring those mutations. These methods, therefore, cannot be used on mutations found in proteins enriched with disordered regions. Hence we setforth to build a new SVM-based method for predicting the functional impact of missense mutations in disordered regions. Our dataset for building a SVM model comprises of 1722 disease causing and 6101 neutral mutations found in the disordered regions of 408 and 6101 human proteins respectively. We have considered, initially, sequence conservation based and amino-acid based features at the mutation sites for building SVM-models. For estimating the sequence conservation at the mutation sites we have implemented Jensen-Shannon divergence (JSD) information theoretic approach. SVM training and testing are underway.

Project 2: Computational Studies on Intrinsically Disordered Proteins (IDPs): Construction of substitution scoring matrix specific to disordered regions.

1) Universal substitution scoring matrices such as BLOSSUM have been built using conserved regions in aligned proteins and, therefore, these matrices mostly encapsulate information pertaining to the amino acid substitutions that typically happen in structurally ordered regions. Such matrices are inappropriate for database searches of evolutionally related sequences or for sequence alignments of disordered regions in proteins. It is known that disordered regions are enriched with polar/charged/Gly/Pro amino acid residues and hence it is logical to expect amino acid residue substitutions in the conserved disordered regions to be different from those that are represented in BLOSSUM/PAM matrices. For this reason, we started building a substitution scoring matrix exclusively for disordered regions in proteins and also a tool that can automatically employ ordered/disordered matrix based on the type of the sequences that are aligned.

2) We first setout to collect human proteins enriched with disordered regions. Our search for human proteins having at least one disordered region of >=30 residues resulted in about 9000 proteins. Domains were identified in these proteins and their orthologs from higher mammalian species were identified using PSI-BLAST. Disordered regions in the orthologs were identified and their multiple sequence alignments (MSA) were carried out. From the aligned blocks of disordered regions we intend to calculate substitution frequencies of amino acid residues specific to the disordered blocks as well as the proposed substitution scoring matrix. Further work is under progress.

Project 3: An attractive and promising strategy for early cancer diagnosis through the assembly of the human cancer volatome (The New Indigo Project).

1) A relational database was developed to store the volatiles detected by our collaborating partners, who analyse breath, urine and saliva samples collected from breast and lung cancer patients and also from controls using GC-MS. This database has been designed in such a way that it holds patient's (as well as control's) demographic information as well as the known physicochemical, pathways and other relevant biological information (collected from various databases available on the public domain) of the detected metabolites. We have created user-friendly Q&A, data input help files etc., to help our collaborators to store data in this database. The database can be accessed after user authentification with userid and password and its access is currently limited to the project collaborators.

2) The number of volatile metabolite compounds typically detected from breath, urine and saliva samples of cancer patients count over 100. However, the number of patients and controls used in these studies are typically far less than the number of the compounds detected and hence can lead to spurious correlations. Therefore, statistical analyses of these data for biomarker discovery pose some challenges. Additionally, the data are also often confounded with missing values as a consequence of experimental issues. We, therefore, started to build a software suite based on R-platform to incude all the necessary tools such as missing value imputation, multivariate analysis tools, supervised and unsupervised methods, data dimensionality reduction methods etc. This will be hosted along side the HCV database on the project webportal.

Future plans and directions

1. Continuation of studies on IDPs harboring disease causing mutations.
2. Classification and analysis of disordered regions in proteins.
3. Studies on tissue-wise PPI networks integrated with drug-protein interaction data.

Publications

1. Rachita HR and Nagarajaram H A (2015) Molecular principles of human virus protein-protein interactions ***Bioinformatics*** 31: 1025-1033.
2. Bidcho A M, Dalal A, Trivedi R, Shukla A, Nampoothiri S, Sankar V H, Danda S, Gupta N, Kabra M, Hebbar S A, Bhat R Y, Matta D, Ekbote A V, Puri R D, Phadke S R, Gowrishankar Aggarwal K S, Ranganath P, Sharda S, Kamate M, Datar C A, Bhat K, Kamath N, Gopinath P M, Verma I C, Nagarajaram H A, Satyamoorthy K, Girisha K M (2015) Recurrent and novel GLB1 mutations in India ***Gene*** 567: 173-181.
3. Radha Rama Devi A, Ramesh V A, Nagarajaram H A, Satish S.P.S, Jayanthi U, Lokesh L (2016) Spectrum of Mutations in Glutaryl-CoA Dehydrogenase gene in GlutaricAciduria Type I - Study from South India ***Brain & Development*** 38: 54-60.
4. Chaudhary A K, Mohapatra R, Nagarajaram, H A, Ranganath P, Dalal A, Dutta A, Danda S, Girisha K, Bashyam M D (2016) The novel missense EDAR p.L397H mutation causes autosomal dominant hypohidrotic ectodermal dysplasia. ***Journal of European Academy of Dermatology and Venearology*** *(In Press)* DOI: 10.1111/jdv.13587.

LABORATORY OF COMPUTATIONAL AND FUNCTIONAL GENOMICS

Computational and functional genomics of biological organisms

Faculty	Akash Ranjan	Staff Scientist
PhD Students	Mr. Rohan Misra	Senior Research Fellow
	Mr. Bhavik Sawhney	Senior Research Fellow
	Mr. Ajit Roy	Senior Research Fellow
	Mr. Suhail Yousuf	Senior Research Fellow (till October 2015)
	Mr. Rajendra Kumar Angara	Senior Research Fellow
	Mr. Abhishek Kumar	Senior Research Fellow
	Mr. Debasish K Ghosh	Senior Research Fellow
	Mr. Shailesh Kumar Gupta	Junior Research Fellow
	Mr. Vijay Kumar M J	Junior Research Fellow (Since February 2016)
Collaborators	Anthony Addlagatta	CSIR-IICT, Hyderabad, India.
	V. Vindal	University of Hyderabad, India.

1. Identification of novel class of small RNA molecules from *Plasmodium falciparum:* tRNA derived RNA fragments

Summary of work done until the beginning of this reporting year (upto March 31, 2015)

Previously, we have annotated tRNA-modifying enzymes of *P. falciparum* through comparative genomics approach and hypothesized *P. falciparum* apicoplast tRNA-guanine transglycosylase as putative target for chemotherapeutic intervention against the parasite. Furthermore, *P. falciparum* adenosine deaminase acting on tRNA (ADAT) was functionally characterized and the complex was observed to differentially act on different tRNA molecules. In addition, small RNA molecules were sequenced from the intraerythrocytic stage of *P. falciparum* 3D7 and it was observed that the parasite contains canonical tRNA fragments (tRF5, tRF3 and tRF1). *P. falciparum* consists of two more species of tRNA fragments and based on the site of cleavage, we named them as tRF4, which originate from D loop and extend till the anticodon loop, and tRF2, which consists of sequence between the anticodon loop and T loop. tRNA halves of approximately 35 bases in size were abundantly present among the small RNA populations in *P. falciparum* intraerythrocytic stage.

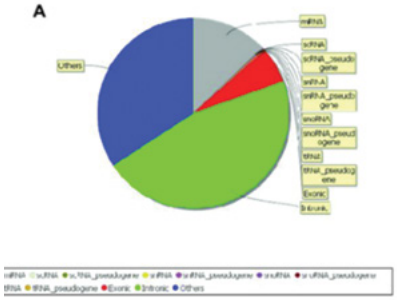
Detail of the work done in the current reporting period (April 1, 2015 – March 31, 2016)

Human miRNAs are abundantly present in *P. falciparum* small RNA library

To examine the possibility that small RNAs of human origin were present in the small RNA population that were derived from the intraerythrocytic stage of *P. falciparum* life cycle, the small RNA library was mapped to human genome. Interestingly, mapping of the small RNA library to human genome revealed that it majorly contained small RNA molecules that had originated from introns, followed by those that were generated from mi-RNAs (Fig. 1A and 1B). Within the human miRNA populations, mir-486 and mir-451a were found to be abundantly present in parasite (Fig. 1C and 1D). The Integrative Genomics Viewer (IGV) was utilized to visualize the alignment of miRNA, mir-486 to human genome (Fig. 2A) and likewise, the alignment of all the other human miRNAs were mapped to determine the mismatches at the base pair resolution. Northern blot analysis with oligonucleotides that were complementary to human miRNAs, mir-486 and mir-451a, suggested the stable existence of these miRNAs in the small RNA population of asynchronous culture of intraerythrocytic stage of *P. falciparum* 3D7. To rule out these miRNAs as potential

contaminant, the parasite was treated with RNaseA after saponin lysis of RBC and northern blot was repeated. Approximately, 21-bp bands corresponding to both human miRNAs were visible in the blot of *P. falciparum* small RNA species (Fig. 2B).

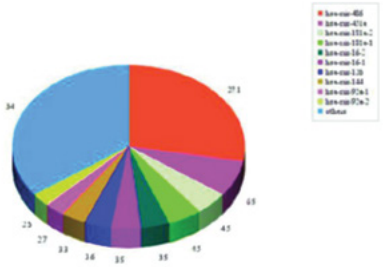
A



B

Region	Reads	Proportion (%)
Exonic	283387	6.04
Intronic	2166642	46.17
tRNA	15922	0.34
scRNA	3025	0.06
snoRNA	6996	0.15
miRNA	603824	12.8
tRNA pseudogene	2997	0.06
Others	1607894	34.26

C

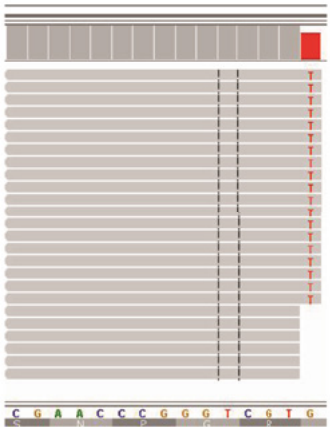


D

Gene Type	Reads	miRNA
hsa-mir-486	164442	miRNA
hsa-mir-451a	42203	miRNA
hsa-mir-181a-2	27780	miRNA
hsa-mir-181a-1	27465	miRNA
hsa-mir-16-2	24098	miRNA
hsa-mir-16-1	23666	miRNA
hsa-mir-126	22031	miRNA
hsa-mir-144	20387	miRNA
hsa-mir-92a-1	16908	miRNA
hsa-mir-92a-2	15851	miRNA

Figure 1. Mapping of small RNA library on human genome. (A) Proportion of small RNAs in *P. falciparum* small RNA library that were originated from different regions of human genome. (B) Tabulated comparison of number of reads of small RNAs that were generated from human genome and was present in the small RNA library of *P. falciparum*. Human miRNAs are abundantly present in *P. falciparum* small RNA library. (C) Pie chart of percentage of top ten most abundant human miRNAs that were detected in *P. falciparum* small RNA library. (D) Tabulated summary of number of reads of the ten most abundant human miRNAs found in the intraerythrocytic stage of *P. falciparum.*

A



B

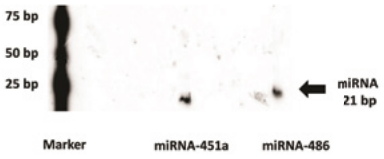


Figure 2. Human miRNAs in *P. falciparum* (A) Snapshot of IGV viewer. Schematic representation of alignment of human mir-451a on human genome using IGV viewer. The colored vertical bars at the bottom denote nucleotides in the reference sequence in the standard color designation (A-Green, T-Red, G-Brown and C-Blue). The gray horizontal bars on the top of the reference indicates the alignment of reads to reference sequence and the position indicates the location of the reference sequence in the genomic context, with the forward directions indicating the top strand of the alignment. The gray color indicates the perfect alignment while the colored areas represent the variations from the original sequence with the color indicates the identity of dissimilar nucleotide. (B) Northern blot analyses of human miRNAs. An approximately 21 bp band corresponding to mir-451a and mir-486 in the northern blot of small RNA population of *P. falciparum* was detected by using end-labelled antisense oligonucleotides against respective miRNAs. Lanes: 1-marker; 2&3-Blot probed with miRNAs mir-451a and mir-486.

2. Characterization of potential ligand of HosA, a MarR like transcription regulator in pathogenic *Escherichia coli*

Summary of work done until the beginning of this reporting year (upto March 31, 2015)

Previously, we have characterized the *in vivo* functional activity of HosA, a MarR like transcription regulator in pathogenic *Escherichia coli,* as regulator of non-oxidative Hydroxyarylic Acid Decarboxylase operon. In this study, we had identified the palindromic transcription regulation site (in PecdB), which is modulated by HosA along with detailed analysis of consensus site. Regulation of nonoxidative HAD operon is mediated by HosA and this seemed to be very crucial in regulating genes responsible for degradation process of hydroxyarylic acids.

Detail of the work done in the current reporting period (April 1, 2015 – March 31, 2016)

Identification of 4-HBA as small molecule regulator of HosA

We have identified 4-hydroxy benzoic acid (4-HBA) as the small molecule regulator of HosA. 4-HBA mediates induction of PecdB activity through selective derepression of HosA mediated repression. Any intracellular increase in 4-HBA concentration modulates the repression caused by HosA. Further, an increase in transcript level of non-oxidative HAD operon upon exposure to 4-HBA was observed in accordance with increase in derepression of HosA mediated repression on exposure to 4-HBA in heterologous *E. coli* strain MC4100 (Figure 3).

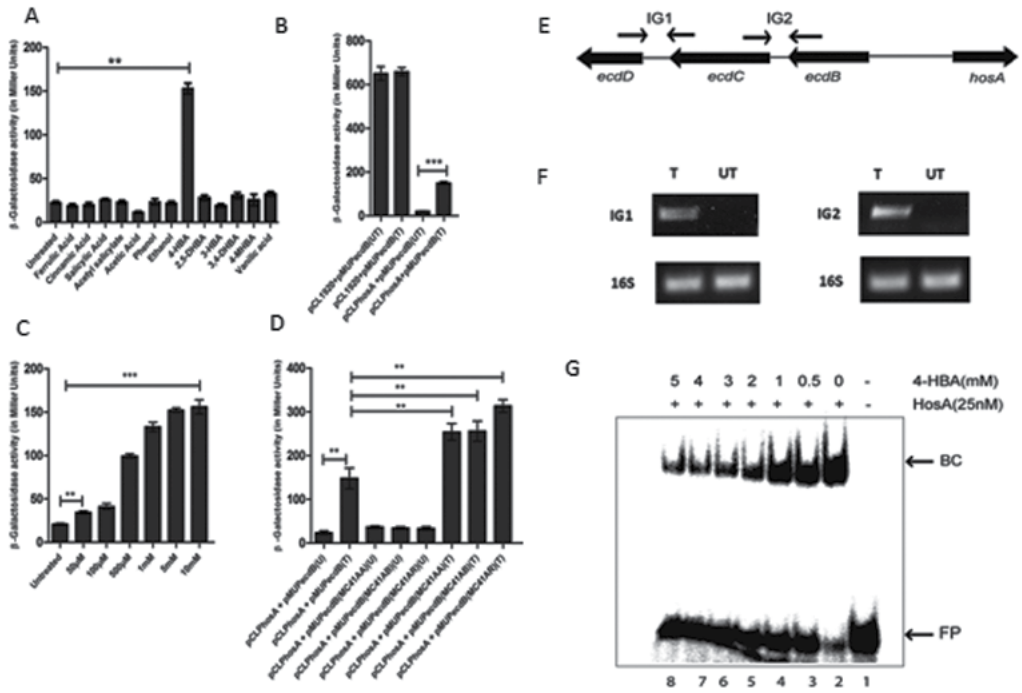


Figure 3. . Ligand identification of HosA: Effect of 4-HBA on expression level of HAD operon and HosA-DNA interaction. (A) β-Galactosidase assay showing the ability of different aromatic and nonaromatic compounds to cause the derepression of HosA mediated repression of PecdB. (B) β-Galactosidase assay showing the effect of 4-HBA on the promoter activity of PecdB, with and without the presence of HosA. (C) β-Galactosidase assay showing the effect of varying concentration of 4-HBA (50 μM-10 mM) on derepression of HosA mediated repression of PecdB. (D) Effect of 4-HBA (1 mM) on HosA mediated repression of PecdB in different 4-HBA exporter knockout strains as shown through β-Galactosidase assay (T, cultures with 4-HBA treatment; U/UT, cultures without 4-HBA treatment). ***P < 0.0001, **P < 0.001, and *P < 0.01 between promoter activities of 4-HBA treated and untreated cells in glucose minimal A media. (E) Schematic diagram of non-oxidative HAD operon along with the primer pairs (shown by arrows) used for amplifying intergenic regions IG1 and IG2. (F) Semi-quantitative PCR of intergenic regions IG1 and IG2 of non-oxidative HAD operon. Lane UT: PCR amplified cDNA template that was transcribed from RNA of cultures without 4-HBA treatment and (Lane T) with 4-HBA treatment. Amplified internal sequence of 16S rRNA transcript was taken as internal control. (G) Effect of 4-HBA on interaction of HosA with probe (U) through EMSA. Lane 1: Radiolabeled free probe (U). Lane 2: Radiolabeled probe (U) incubated with HosA. Lanes 3−8: Different molar concentrations of 4-HBA (0.5−5 mM) incubated with probe and HosA.

3. Studies on the role of Rv2989 (IclR like protein) in the physiology of *M. tuberculosis*

Summary of work done until the beginning of this reporting year (upto March 31, 2015)

In our previous studies, we characterized promoter and binding site of Rv2989 (an IclR like protein) in the intergenic region of *leuCD-Rv2989.* In order to understand physiological significance of Rv2989 in mycobacteria, we ectopically expressed *Rv2989* and observed that the constitutive expression using *hsp60p* promoter leads to toxicity. Further, a controlled expression of *Rv2989* in *M. smegmatis*, using *acep,* an acetamide inducible promoter, shows growth retardation.

Details of the work done in the current reporting period (April 1, 2015 – March 31, 2016)

In order to understand cellular events occurring with Rv2989 expression, we induced expression of Rv2989 using 0.2% acetamide and observed uninduced and induced *M. smegmatis acep-Rv2989* cells in Scanning Electron Microscope (SEM) and Transmission Electron Microscope (TEM) for morphological differences. SEM observations revealed the presence of extracellular material in induced cultures, which surround *M. smegmatis acep-Rv2989* cells (Figure 4A) an observation similar to the phenotype of non-replicating persistent mycobacteria. Observation of ultra thin sections of cells under TEM revealed the accumulation of lipid droplets in induced cultures (Figure 4B). Lipids usually get accumulated as lipid droplets in dormant mycobacteria and serves as an energy repository. As the SEM and TEM observations suggest dormant features of mycobacteria, we hypothesise Rv2989 expression possibly induce dormancy and tested for non acid fastness, a feature of dormant mycobacteria. The *M. smegmatis acep-Rv2989* after induction lost its acid fastness, while the uninduced cultures retained the property (Figure 4C), suggesting Rv2989 expression arrests growth and possibly drives *M. smegmatis* into dormancy like state. The molecular pathway involved in the initiation of this dormancy like state is yet to be elucidated.

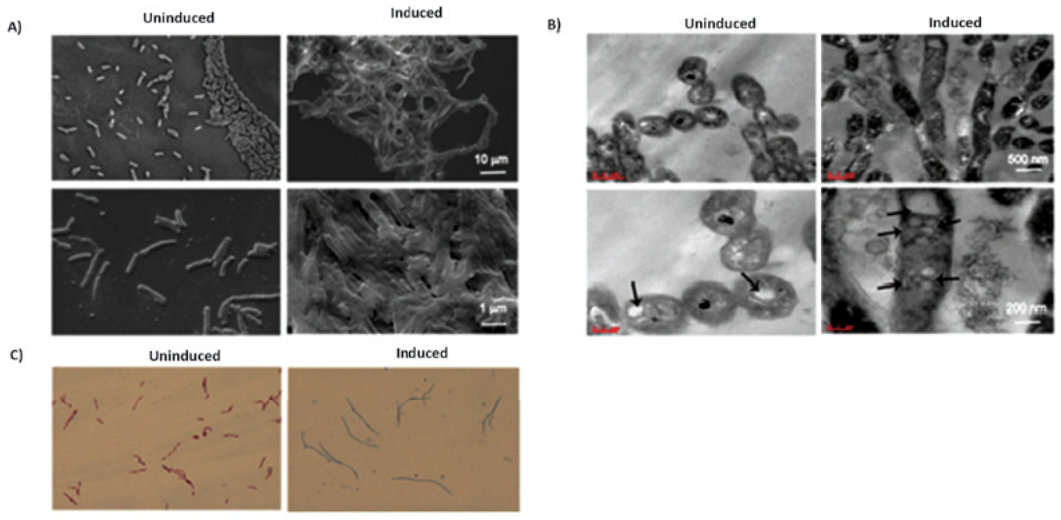


Figure 4. Effect of ectopic expression of *M. tuberculosis* Rv2989 on *M. smegmatis* (A) Scanning electron micrograph images of uninduced and induced cultures. (B) Transmission electron micrograph images of ultrathin sections of uninduced and induced cultures. Lipid droplets are shown with arrow marks. (C) Analysis of acid fastness in uninduced and induced cultures of *M. smegmatis acep-Rv2989.*

4. Characterization of structural and organizational properties of Huntingtin Interacting Protein K as intracellular aggregation sensor

Summary of work done until the beginning of this reporting year (upto March 31, 2015)

Previously, we had characterized HYPK to be an aggregation prone protein which remained in a molten globule like less densely packed conformation, which had potentiality to form lower order oligomeric seeds like dimer and trimer. These again could lead to formation of very

large aggregates, in a concentration dependent manner, both *in vitro* and *in vivo.*

Detail of the work done in the current reporting period (April 1, 2015 – March 31, 2016)

Multimerization of HYPK follows a prion like seed nucleation model

To elucidate the mechanism of HYPK multimerization, we followed the multimerization using AFM imaging along with computational modeling / docking studies. Annular assembly of HYPK by C-terminal region started with the formation of small oligomeric seed structures, which combined and coalesced among themselves. These give rise to smaller scaffold like annular structures, upon which further association of seeds made higher annular oligomeric assemblies (Figure 5).

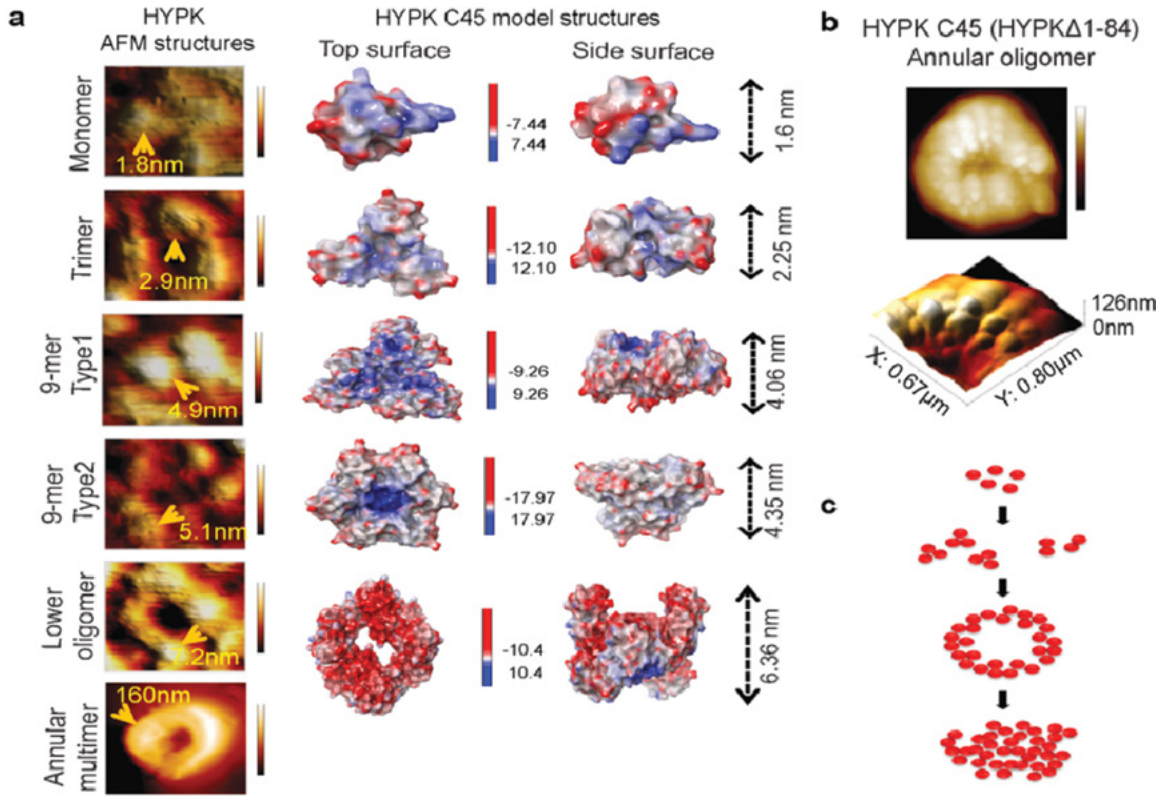


Figure 5. C-terminal dependent smaller oligomeric seed cause HYPK nucleation to form ordered annular complex. (a) Formation of HYPK annular complex. [Panel 1] Monomer. Height scale: 0-2.5nm. [Panel 2] Trimeric seed coalescence as scaffold. Height scale: 0-3.5nm. [Panel 3] Type-I 9-mer unit coalescence with each other. Height scale: 0-6nm. [Panel 4] Type-II 9-mer oligomeric nucleation scaffold. Height scale: 0-6nm. [Panel 5] Lower oligomeric nucleation scaffold complex. Height scale: 0-8nm. [Panel 6] Large annular complex. Height scale: 0-190nm. Middle and right panels show model structures of HYPK C-terminal 45 residue region monomer, trimer, Type-I 9mer, Type-II 9-mer and 18-mer. (b) AFM image of annular oligomeric complex of HYPK C45 region showed hollow scaffold and oligomeric deposition upon scaffold. Height scale: 0-129nm. (c) Schematic representation of prion like seed nucleation mediated aggregation of HYPK.

An N-terminal negative charge rich region stabilizes C-terminal LCR to prevent intracellular aggregation by HYPK

Although, the C-terminus of HYPK has high intrinsic ability to form aggregates, surprisingly, it did not form aggregates of considerably larger size in majority of cells under normal endogenous expression levels. In order to understand specific region and sequence stretches that stabilized intra-cellular HYPK and prevented aggregation, we constructed various

deletion and multiple point mutant constructs to observe the aggregation status. Binding studies of N-terminal 60 residue region or its multiple point mutant variants (ie HYPK N-60 E/A and HYPK N-60 E/D) with C-terminal 69 residue region (HYPK C-69) showed specific interactions of HYPK N-60 and HYPK N-60 E/D with HYPK C-69 but no interaction was observed between HYPK N-60 E/A with HYPK C-69. This suggests that there existed a specific charge interaction between negative charge residues in the patch of N-terminal region with (basic amino acids) of LCR, which accounted for stabilization of LCR and prevention of aggressiveness of oligomerization.

Publications

1. Roy A and Ranjan A (2016). HosA, a MarR family transcriptional regulator, represses non-oxidative hydroxyarylic acid decarboxylase operon and is modulated by 4-Hydroxybenzoic acid. ***Biochemistry*** 55(7): 1120-1134.
2. Sawhney B, Chopra K, Mishra R, Ranjan A (2015). Identification of *Plasmodium falciparum* apicoplast-targeted tRNA-guanine transglycosylase and its potential inhibitors using comparative genomics, molecular modelling, docking and simulation studies. ***Journal of Biomolecular Structure & Dynamics*** 33(11):2404-2420.

LABORATORY OF *DROSOPHILA* NEURAL DEVELOPMENT

Understanding patterning and development of Central Nervous System using *Drosophila melanogaster*

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	Neha Ghosh	Senior Research Fellow
	Raviranjan Kumar	Senior Research Fellow
	Rashmi Sipani	Senior Research Fellow
	Asif Ahmad Bakshi	Junior Research Fellow
Other Members	P Kalyani	Technical Officer
	Maheshvari C	Project Assistant (till Dec 2015)
	Sromana Mukherjee	Project Assistant (till March 2016)
	Srivatsan G	Project Assistant (till March 2016)

Objective

The key objective of the lab is to understand how neural progenitor cells attain their positional identity in developing Central Nervous System **(CNS)** of an organism and how does this translate into generation of a variety of cell types found in CNS and their respective numbers (as represented in the Fig-1). Hox family of transcription factors are known to play an important role in execution of these features along the Anterior-Posterior **(AP)** axis of the CNS during development. The molecular basis of role of Hox genes in patterning of CNS is not well investigated. Our lab is using *Drosophila melanogaster* as a model organism, to understand these phenomenons by focusing mainly on early embryonic and larval stages of development. To this end, the specific aims of our lab are as follows:

1. Understanding the molecular function of Hox gene *Abdominal-A (Abd-A)* in larval CNS patterning.

Abdominal region of the *Drosophila* larval CNS has a less number of neurons compared to its thoracic counterpart. Hox gene *Abd-A* in known to cause programmed cell death (apoptosis) of neural progenitor cells (also called Neuroblasts-**Nbs)** and therefore limit the number of neurons in abdominal region of CNS. The apoptosis is known to be mediated through activation of *reaper, hid and grim* (RHG) family of genes. The precise molecular details of how *Abd-A* cause Nb apoptosis are unknown. Genetic evidence suggests a role for a helix-loop-helix transcription factor *Grainyhead* (*Grh*) along with Abd-A in control of this apoptosis. Characterization of the molecular basis of this link is primary goal of this project. Furthermore, since Grh is involved in Nb apoptosis and is not expressed in neuronal progeny refractory to this apoptosis, it is of interest to define *grh* regulation in these cells which keeps *grh* “on” in the pNbs and “off” in the neuronal progeny of pNbs.

2. Understanding the role of Hox gene Deformed (*Dfd*) in patterning of embryonic subesophageal ganglia.

Hox genes express in CNS (in neural progenitor cells) in embryonic stages of development (as represented in Fig-1) but how does their expression patterns the embryonic nervous system is not well understood. *Deformed (Dfd)* is known to express in the cells of maxillary (Mx) and mandibular (Mn) segments of subesophageal ganglion of embryonic CNS. This project focuses on understanding auto-regulation of *Dfd* in this region and to find out how this helps in giving cells their specific positional identity. This is being done by using a 3.2kb CNS specific neural auto-regulatory enhancer for *Dfd* (NAE3.2), which recapitulates the expression of *Dfd* gene in developing embryonic CNS.

3. Investigating the role of *Abdominal-B (Abd-B)* and *Double-sex (Dsx)* in terminal CNS patterning.

There are 12 Nbs in terminal region of CNS of which 8 stop dividing in both males and females at mid L3 stage of development. The remaining 4 Nbs which we refer to as terminal Nbs

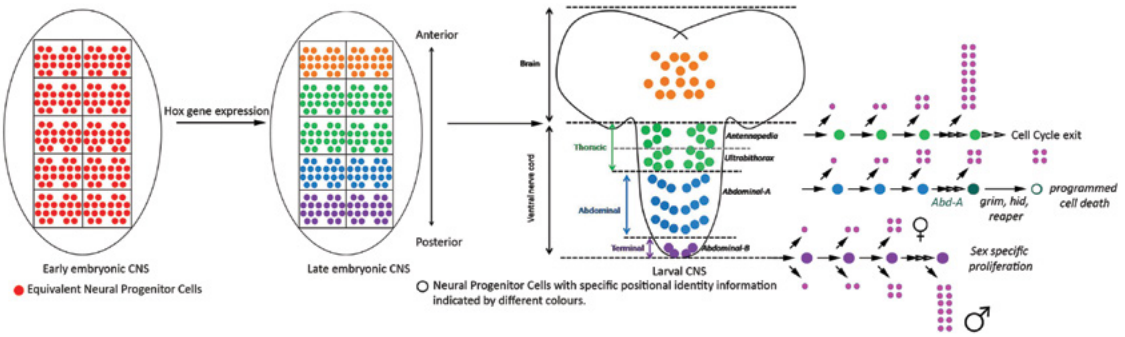

Figure 1. Precursor cells for embyo Nbs start out as equivalent cells and attain their specific positional identity by Hox gene expression. This gets reflected as specific Nbs identity and thereby determine proliferation and differentiation profile of these cells along the AP axis. In larval stages thoracic, abdominal and terminal post-embryonic Nbs (pNbs) differ in their number and proliferation profile as shown. Thoracic pNbs stop proliferation by cell cycle exit, while abdominal pNbs (in both sexes) and terminal pNbs (tNbs; in females) die as a result of apoptosis, the tNbs in males continue dividing and give rise to more neurons as shown.

(**tNbs**), behave differentially in two sexes. The hypothesis for this part of work is that Abd-B and Dsx (Double-Sex being the most downstream member of sex specification hierarchy) play a role in sex specific proliferation and apoptosis of these tNbs. Although the role of the sex determining hierarchy and Hox gene Abd-B, in growth and differentiation of *Drosophila genital* discs, is well worked out, little is known about how sex determination hierarchy and Abd-B intersects with cell proliferation and survival behavior of tNbs in the larval VNC. We intend to test the interaction between Abd-B and Dsx in gender specific proliferation and apoptosis of these cells.

Summary of work done until the beginning of this reporting year (upto March 31, 2015)

1. Understanding the molecular function of Hox gene *Abd-A* in larval CNS patterning.

The relevant enhancer for the activation of RHG family of apoptotic genes in Nbs lies within 23kb genomic region referred to as *NBRR-Neuroblast Regulatory Region.* The NBRR was divided into 5 overlapping genomic fragments (of 6-10kb). These genomic fragments were made into transgenic lines and were screened for their ability to drive pNb specific expression of lacZ reporter in late third instar larval **(LL3)** brain. The transgenic line analysis narrowed down the search to an overlapping region of 3kb fragments. A small genetic deletion generated by us when tested in trans-heterozygotic condition with a bigger deletion blocked pNb apoptosis in abdominal region, this genetically located the enhancer to the region of the genome removed in smaller deletion generated in our lab. This observation along with the deletion analysis narrowed down the enhancer search to 3kb region of the genome.

Simultaneously a 4kb enhancer of *grainyhead* responsible for its expression in CNS was sub-fragmented to narrow down the relevant enhancer for the expression of *grainyhead* in CNS to 1kb region. Currently this region is being further analysed to identify transcriptional factors that could be regulating *grainyhead* differentially in Nbs versus neurons.

2. Role of Hox gene *Deformed (Dfd)* in patterning of embryonic subesophageal ganglia.

The costaining of Dfd and Dpn (a neural progenitor specific marker) established that Dfd is expressed in neural progenitor cells (neuroblasts-Nbs). Subsequently using the *NAE3.2-lacZ* transgenic line, it was established that expression of Dfd is auto regulated in Nbs since Dpn positive cells in Mx region were LacZ positive as well. Hox genes are known to function with two other homeodomain containing transcription factors Extradenticle (Exd) and homothorax (Hth) in Drosophila (and vertebrate homologs; Pbx and Meis). A 630bp subfragment of 3.2kb genomic region of neural autoregulatory element was found to have two putative compound Hox-Exd binding sites. In vitro binding studies showed that Dfd and Exd and Hth formed a cooperative trimer

on these binding sites with different efficiencies. In vivo importance of Exd and Hth is being tested for their role in neural autoregulation.

3. Investigating the role of *Abdominal-B (Abd-B)* and *Double-sex (Dsx)* in terminal CNS patterning.

A recent report characterized the Nb lineage in terminal region. Report elucidated that female specific isoform of Dsx (DsxF) is responsible for the apoptosis of sex-specific tNbs in females while these cells continue dividing in males. The report didn't elucidate (A) the molecular mechanism behind the phenomenon of apoptosis of sex-specific tNBs in females and (B) and doesn't give any insight into how Dsx play a role in tNB proliferation and how sex specific tNbs are different from other 8 Nbs in the same region which stop dividing at mid L3 stage of development.

We started out by testing the expression of Abd-B and Dsx in tNbs in CNS of male larvae since tNbs are dead in females by late larval stages of development. We find that Abd-B and Dsx are expressed in male tNbs. Since Grh is already known to play a role in apoptosis of pNb of abdominal segments, we checked and found Grh to be expressed in tNbs of male larvae at mid L3 stage. Currently we are checking the role of Grh in female tNb apoptosis.

Simultaneously *Drosophila Cyclin E* gene is being tested to identify the mechanism behind continued sex specific proliferation of tNbs in male larval CNS. *cycE* is known to play a central role in cell cycle by promoting G1-S transition in dividing cells during cell cycle and a detailed enhancer analysis has identified a 1.9kb enhancer element which controls the expression of the gene in Nbs. This enhancer is known to have binding site for Hox gene Abd-A and Abd-B and our analysis identify potential Dsx binding sites in the enhancer. A BrDU, lacZ and Dpn staining of *cycE-1.9kb-lacZ* transgenic flies show that lacZ line marks dividing Nbs in terminal regions of CNS. The experiments are ongoing to characterize 1.9kb enhancer to understand how *cycE* integrates spatial temporal and sex specific information in tNbs.

Summary of work done from April 1, 2015-March 31, 2016

1. Understanding the molecular basis of Hox gene Abdominal-A (Abd-A) in larval CNS patterning

We narrowed down the relevant enhancer to 3kb overlapping region of two 8kb fragments (*NBRRF3 and F4*) after analysis of all 5 *enhancer-lacZ* lines of *NBRR*. We generated a smaller 2 kb *enhancer-lacZ* from this overlapping region and found that it is expressed in pNbs of abdominal and terminal region of larval central nervous system.

We have genetically isolated the apoptotic enhancer by mobilizing a transposon inserted in *NBRR* to generate a smaller deletion (*NBRR-22*). This deletion in transheterozygotic combination with already existing deletion of NBRR gives ectopic pNbs in the abdominal region of CNS at LL3 stage. The finer PCR mapping indicates that 14.5kb region of the NBRR encompassing the relevant apoptotic enhancer has been deleted in this case.

The expression of 2kb enhancer in abdominal pNb and presence of ectopic pNbs in 14.5 kb deletion suggests that we have narrowed down the relevant apoptotic enhancer from 23kb *NBRR* to 2kb region of the genome. Next the putative Hox and Grh binding sites in the 2kb region were tested for respective transcription factor binding in vitro by EMSA. We tested closely placed Hox and Grh binding sites and found that both transcription factors bind on DNA, mutant oligo analysis indicated that these bindings were specific.

An indirect way to check for activation of RHG genes by AbdA and Grh in vivo was by checking *NBRRF3-lacZ* reporter expression in abdominal pNbs, in response to Abd-A and Grh downregulation in pNbs by RNA interference. We found that *NBRRF3-lacZ* line was down regulated in surviving abdominal pNbs in response to RNA interference for AbdA and Grh. Conversely the ectopic expression of Abd-A in thoracic pNbs where Abd-A is not normally expressed resulted in ectopic expression of *NBRRF3-lacZ* in thoracic region as well, indicating the responsiveness of enhancer for Abd-A.

Considering the importance of Grh in pNbs we are trying to identify *grh* regulators in pNbs. To this end an RNA interference screen is ongoing. In this screen a battery of 465 transcription factors selected based on their spatial and temporal expression pattern in developing CNS are being knocked down in abdominal and thoracic pNbs to identify regulator of *grh* gene by scoring for downregulation of Grh protein expression.

2. Role of Hox gene *Deformed* in patterning of embryonic subesophageal ganglia.

We tested the role of Hox cofactor Exd in neural autoregulation and Dfd expression in Nbs of embryonic subesophageal ganglia by looking at Exd null mutant (exd^{1}). exd^{1} homozygous mutants showed no significant change in Dfd expression in Nbs. This is due to the fact that Exd is known to be maternally contributed. In order to circumvent the problem of maternal contribution of Exd protein, we decided to analyze hth^{P2} a strong hypomorph of *hth* gene. Since Hth is a known partner of Exd, and plays an important role in its transport into the cell nuclei, we expected that hth^{P2} will mimic a phenotype similar to *exd* complete loss of function. We found a region specific role of hth in Dfd expression. Dfd expression was completely missing in Mx Nbs, while the expression in Mn Nbs was dramatically down regulated, but low levels of Dfd could still be observed in these cells. This suggest that Hth is critical for Dfd expression in Mx Nbs but is important only for maintenance of the levels of Dfd protein in Mn Nbs, and has no role in *Dfd* neural autoregulation in Mn segments.

Our subsequent experiments with homeodomain-less (HD-less) isoform of Hth (referred to as HM-Hth); show that HM-Hth is sufficient for maintaining *Dfd* expression levels in embryonic stages, and suggest that HD of Hth is not necessary for region specific role of Hth in CNS.

Since both Exd and HM-Hth are required only for regulating levels of Dfd expression in mandibular Nbs, and neural autoregulation in these cells is independent of their roles, we propose a role for yet to be identified factor(s) in regulating core neural autoregulatory transcriptional loop. Identification of this/these factor(s) and characterization of their role in Nbs and differentiated neurons of mandibular region are ongoing.

3. Investigating the role of A*bdominal-B (Abd-B) and Double-sex (Dsx)* in terminal CNS patterning.

In order to test the role of *grh* in female tNb apoptosis, we analyzed *grh* mutant larvae. We found that many ectopic pNb were seen in the Abd-B region of *grh* mutant female larval brains compared to wild type female brains where no pNbs are reported at the same stage. Interestingly none of these cells were found to be positive for Dsx which is a conclusive marker for tNbs. This suggest tNbs apoptosis in females is independent of Grh.

A parallel analysis with *grim* mutant, a member of RHG family of apoptotic genes, showed ectopic pNbs in Abd-B region of female larval CNS. In order to conclusively test the role of grim in tNb apoptosis, we counterstained these brains for Nb marker Dpn and for tNb marker Dsx. We observed that none of the ectopic pNbs in female larval brains were Dsx positive. This suggest that *grim* doesn't play in tNb apoptosis and ectopic Nbs are embryonic in origin, and some other RHG family member(s) play a role in tNb apoptosis.

In order to locate the enhancer for the apoptotic gene activation in tNbs, we analysed a previously reported 53kb genomic deletion (*MM3*). We find that larvae which are homozygous for this deletion show ectopic pNbs in Abd-B region which are both positive for Nb marker Dpn and tNb marker Dsx. This suggest that enhancer for tNb apoptosis lies in this 53kb region. Experiments for isolation of the minimal enhancer for tNb apoptosis are ongoing.

Publications

1. Kumar R, Chotaliya M, Vuppala S, Auradkar A, Palasamudrum K, Joshi R (2015). Role of Homothorax in region specific regulation of Deformed in embryonic neuroblasts. ***Mech Dev***; 138(2); 190-197.

LABORATORY OF FUNGAL PATHOGENESIS

Understanding the pathobiology of an opportunistic human fungal pathogen *Candida glabrata*

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	Vandana Sharma	Senior Research Fellow
	Mubashshir Rasheed	Senior Research Fellow
	Priyanka Bhakt	Junior Research Fellow
	Kundan Kumar	Junior Research Fellow
	Anamika Battu	Junior Research Fellow (Since August 2015)
Other Members	Suneetha KJ	Technical Officer (Till July 2015)
	Binay Kumar Sahoo	Project JRF (During May-July 2015)
	S Surya Vamshi	Technical Officer (Since October 2015)
	Reshma Chowdary Alokam	Research Associate (Since October 2015)
	Gujjula Rahul	Project JRF (Till January 2016)
	Rajaram Purushotham	Project JRF (Since October 2015)
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Collaborators	Rajendra Prasad	JNU, New Delhi
	Naseem A Gaur	ICGEB, New Delhi
	Krishnaveni Mishra	UoH, Hyderabad
	Suman Thakur	CCMB, Hyderabad

Candida species account for 70 to 80% of bloodstream fungal infections with *Candida glabrata* being the second most frequently isolated Candida species after *C. albicans*. Despite being a successful pathogen, *C. glabrata* lacks some of the key fungal virulence attributes, and appears to rely on alternative mechanisms to survive the nutrient-poor, antimicrobial environment of the human host. Research in our laboratory is aimed at a better understanding of molecular and cellular mechanisms of *C.glabrata pathogenesis.*

Project 1: Mechanisms of iron acquisition and iron homeostasis in *C. glabrata*

Objectives

1. Identification of major iron acquisition and iron homeostasis mechanisms;
2. Identification of *C. glabrata* genes which are differentially regulated in response to iron availability; and
3. Investigation into the role of identified genes in iron homeostasis

Summary of the work done until the beginning of this reporting year (upto March 31, 2015)

The ability to acquire iron form host tissues is a major virulence factor of pathogenic organisms, and a significant correlation between host iron content and pathogenicity of an organism has been reported. This project is aimed at elucidation of the strategies that *C. glabrata* employs to acquire, transport, utilize and store iron in accordance with the iron availability. Previously, we have generated and characterized mutants disrupted for components of the high-affinity iron uptake (CgFtr1, CgFet3, CgCcc2 and CgFre6), low-affinity iron transport (CgFet4), siderophore-iron uptake (CgSit1), iron storage and utilization (CgYfh1, CgFth1 and CgFet5), host-specific iron utilization (CgHmx1, CgCcw14 and CgMam3) and transcriptional regulatory (CgAft2) systems in *C. glabrata.* We showed that the high-affinity reductive iron uptake system is required for growth under both *in vitro* iron-limiting and in vivo conditions. Further, we demonstrated for the first time that the cysteine-rich CFEM domain-containing cell wall structural protein, CgCcw14, and the putative hemolysin, CgMam3, are essential for maintenance of intracellular iron content, adherence to epithelial cells and virulence of *C. glabrata* in a murine model of systemic candidiasis.

Details of the progress made in the current reporting year (April 1, 2015 - March 31, 2016)

During the current reporting period, we investigated the role of two mitogen-activated protein kinases, CgHog1 and CgSlt2, which have recently been implicated in survival of weak acid, and cell wall, thermal and antifungal stresses, respectively, in iron homeostasis in *C. glabrata.* For this, we first examined their activation status under iron-deplete and iron-replete conditions. As shown in **Figure 1A**, we observed ~ 6-fold higher levels of phosphorylated forms of CgSlt2 and CgHog1 in iron-surplus medium-grown wild-type (*wt*) cells compared to YNB-cultured *wt* cells. Iron-limiting environment had a considerable and no effect on the activation of CgHog1 and CgSlt2 kinases, respectively (**Fig. 1A)**. Further, we generated and characterized the *C. glabrata* strain that lacked the CgHog1 kinase-encoding gene (*CAGL0M11748g*). The *Cgslt2Δ* mutant was constructed previously in our laboratory to examine the role of CgSlt2-mediated cell wall integrity pathway in survival of antifungal stress. Compared to *wt* cells, basal CgSlt2 phosphorylation was found to be ~ 9-fold higher in the *Cghog1*Δ mutant (**Fig. 1A**). However, exposure to iron-limiting and iron-surplus medium resulted in no appreciable increase in the CgSlt2 phosphorylation (**Fig. 1A**). Constitutively active CgSlt2 in the *Cghog1*Δ mutant may reflect either cell wall-related defects or cellular compensatory response to the lack of CgHog1 kinase. Further, similar to *wt* cells, a 2-fold increase in the phosphorylation of CgHog1 was observed in iron-deficient medium-grown *Cgslt2*Δ cells compared to YNB-cultured cells (Fig. 1A). However, *Cgslt2*Δ cells failed to respond appreciably to iron excess in the medium through phosphorylation of the CgHog1 kinase (Fig. 1A) which indicates a direct/indirect role of CgSlt2 in CgHog1 activation under iron-rich environmental conditions. Notably, ectopic expression of *CgHOG1* and *CgSLT2* genes restored CgSlt2 and CgHog1 phosphorylation defects of *Cghog1*Δ and *Cgslt2*Δ mutants (**Fig. 1A**).

Phenotypic characterization of *Cghog1*Δ and *Cgslt2*Δ mutants revealed growth rates similar to *wt* cells in time-course analyses. Further, *Cghog1*Δ and *Cgslt2*Δ mutants exhibited susceptibility neither to iron-limitation (caused by extracellular iron chelators BPS and ferrozine) nor to pH 7.0 condition (**Fig. 1B**). However, both mutants were found to be attenuated for growth in pH 2.0 and surplus iron-containing medium (**Fig. 1B**). An inability of *Cghog1*Δ and *Cgslt2*Δ mutants to grow in iron-rich conditions is indicative of a central role for HOG and PKC signaling pathways in survival and/or counteracting toxicity associated with excess iron. In accordance with earlier studies, the *Cgslt2*Δ mutant exhibited elevated sensitivity to the fluconazole antifungal (**Fig. 1B**). However, fluconazole had no effect on growth of the *Cghog1*Δ mutant (**Fig. 1B**). Further, the *Cghog1*Δ mutant was uniquely sensitive to thermal (42°C), detergent, salt and oxidative stress (**Fig. 1B**). Importantly, growth attenuation of *Cghog1*Δ and *Cgslt2*Δ mutants in the presence of different stressors was restored by ectopic expression of *CgHOG1* and *CgSLT2* genes in respective mutants (**Fig. 1B**). Collectively, these data indicate common roles for CgHog1 and CgSlt2 in survival of surplus iron and low pH stress, and unique functions for CgHog1 in resisting osmotic, thermal and oxidative stresses.

To delineate the functions of CgHog1 and CgSlt2 in iron homeostasis, we next measured the intracellular iron content in *Cghog1*Δ and *Cgslt2*Δ mutants, and found 2-fold higher intracellular iron levels in the *Cghog1*Δ mutant (**Fig. 1C**). Intriguingly, the *Cgslt2*Δ mutant displayed *wt*-like intracellular iron content (**Fig. 1C**). High intracellular iron levels in the *Cghog1*Δ mutant were verified by inductively coupled plasma-atomic emission spectroscopy analysis. Next, to examine if high levels of intracellular iron in the *Cghog1*Δ mutant result in constitutive downregulation of the high-affinity iron-uptake genes, we performed qPCR analyses. Compared to the *wt* cells, transcript levels of *CgAFT1*, *CgFTR1* and *CgFET3* genes, which code for an iron-responsive transcriptional activator, a high-affinity iron permease and a copper ferroxidase, respectively, were found to be ~ 2- to 3-fold lower in the *Cghog1Δ* mutant indicating that *Cghog1Δ* cells sense the intracellular environment as an iron-rich milieu (**Fig. 1D**). As expected, expression of *CgAFT1, CgFTR1* and *CgFET3* genes was similar in log-phase *wt* and *Cgslt2*Δ cells (**Fig. 1D**).

Since disrupted intracellular iron homeostasis can result in impaired iron-sulfur (Fe-S) cluster biogenesis process and activity of the Fe-S cluster-containing enzymes in the mitochondria, we quantified activity of the mitochondrial

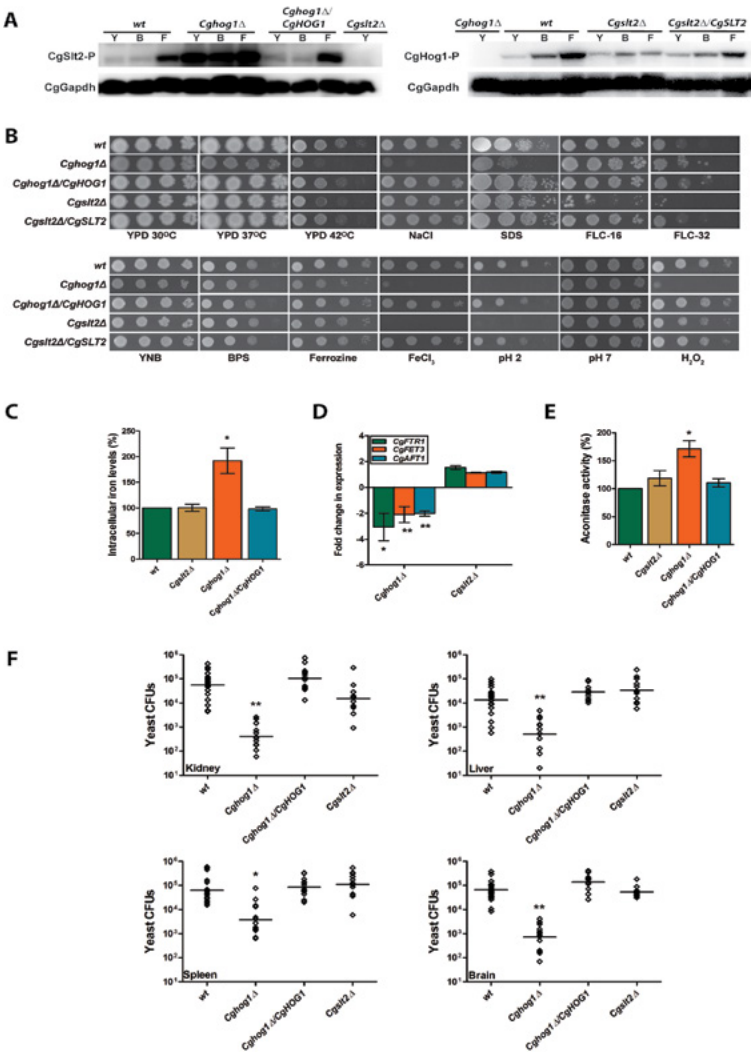

Figure 1. CgHog1 kinase is required for iron homeostasis in *C. glabrata*.

A. A western blot of CgHog1 and CgSlt2 phosphorylation in indicated *C. glabrata* cells grown in YNB medium (Y), YNB medium containing 50 μM BPS (bathophenanthroline disulfonate; B) and YNB medium supplemented with 500 μM ferric chloride (F) for 4 h at 30°C. CgGapdh was used as a loading control.

B. Serial dilution spotting assay showing sensitivity of *Cghog1*Δ and *Cgslt2*Δ mutants towards diverse stress-causing agents: sodium chloride (NaCl; 1M), sodium dodecyl sulphate (SDS; 0.05%), fluconazole (FLC; 16 and 32 μg/ml), BPS (25 μM), ferrozine (300 μM), ferric chloride ($FeCl_3$; 2.5 mM), and hydrogen peroxide (H_2O_2; 25 mM).

C. Intracellular iron levels of indicated, YPD medium-grown, log-phase *C. glabrata* cells as determined by the BPS-Fe complex absorbance. Data are presented as the percentage (mean ± SEM, n = 3-5) of the iron levels in mutants relative to *wt* cells (taken as 100%). Statistical analysis was performed using the paired, two-tailed, Student's t test (*, p≤0.05).

D. qPCR analysis of *CgAFT1*, *CgFTR1* and *CgFET3* transcript levels in log-phase, YPD medium-grown *Cghog1*Δ and *Cgslt2*Δ cells. Data (mean of 3 independent experiments ± SEM) were normalized to an internal *CgACT1* mRNA control, and represent fold change in expression in mutant cultures compared to *wt* cells. Statistical analysis was performed using the paired, two-tailed, Student's t test (*, p≤0.05; **, p≤0.01).

E. The reduced nicotinamide adenine dinucleotide-coupled assay was used to determine aconitase activity in the crude mitochondrial extracts of indicated YPD medium-grown, log-phase *C. glabrata* cells. Data represent mean ± SEM of three independent experiments. *, p≤0.05; paired two-tailed Student's t-test.

F. Assessment of the virulence potential of *Cghog1*Δ and *Cgslt2*Δ mutants in the 6-8 week-old female BALB/c mice. Diamonds represent the CFUs recovered from kidneys, liver, spleen and brain for an individual mouse. Bars represent the geometric mean (n=12-14) of CFUs per organ. Statistically significant differences in the CFUs between *wt* and the *Cghog1Δ* mutant are marked (*, p≤0.05; **, p≤0.01; two-tailed Student's unpaired t-test).

aconitase, a Fe-S enzyme, in *wt, Cghog1*Δ and *Cgslt2*Δ mutants. As shown in **Figure 1E**, compared to *wt* cells, *Cghog1*Δ cells exhibited 80% more mitochondrial aconitase activity which was brought down to *wt*-levels by ectopic expression of *CgHOG1* (**Fig. 1E**). In contrast, no appreciable change in the aconitase activity was recorded between *wt* and the *Cgslt2*Δ mutant (**Fig. 1E**). Next, to check whether cytosolic iron metabolism is also affected in the *Cghog1*Δ mutant, we measured iron present in the cytosol and found it to be 70% higher in log-phase *Cghog1*Δ cells compared to log-phase *wt* cells. As accumulation of iron in the cytosol can result in high-iron toxicity, attenuated growth of the *Cghog1*Δ mutant under iron-rich conditions could be, in part, due to higher cytoplasmic iron content. Together, these data are indicative of a role for CgHog1 in maintenance of iron homeostasis and Fe-S cluster biogenesis.

Lastly, to investigate whether the stress-responsive CgHog1 and CgSlt2 kinases are essential for survival of *C. glabrata* in a murine model of disseminated candidiasis, we examined fungal burden in four target organs in Balb/c mice infected intravenously with *wt*, *Cghog1*Δ and *Cgslt2*Δ strains. The *Cghog1*Δ mutant was found to be highly attenuated for virulence as 20- to 150-fold reduction in the organ fungal load was observed in Balb/c mice infected with the *Cghog1*Δ mutant compared to the *wt*-infected mice (**Fig. 1F**). Ectopic expression of the *CgHOG1* gene restored virulence defects of the *Cghog1*Δ mutant in kidneys, liver, spleen and brain in Balb/c mice (**Fig. 1F**). Importantly, differences in the yeast CFUs recovered between *Cgslt2*Δ- and *wt*-infected mice were not statistically significant ($p \leq 0.01$; **Fig. 1F**). Taken together, these data indicate an essential role for the CgHog1 kinase in virulence in a murine model of disseminated candidiasis which could be attributed, in part, to its role in survival of oxidative stress and maintenance of iron homeostasis. Experiments are currently underway to elucidate the molecular basis for CgHog1-mediated iron homeostasis.

Project 2: Role of SUMOylation in the pathobiology of *C. glabrata*

Objectives

1. Identification of components of SUMOylation machinery in *C. glabrata*;
2. Investigating the effects of SUMOylation disruption on the pathobiology of *C. glabrata*; and
3. Identification of factors that are SUMOylated in *C. glabrata*

This is a new activity.

Details of the progress made in the current reporting year (April 1, 2015 - March 31, 2016)

SUMOylation, the covalent reversible conjugation of SUMO (small ubiquit in-like modifier) polypeptide to lysine residuesin target proteins, is a post translational modification which plays a key regulatory role in several cellular processes including transcription and stress response. The process of SUMO attachment consists of four steps: (i) processing of the ~ 10 KDa precursor SUMO peptide by SUMO-specific proteases to reveal a carboxyl-terminal diglycine motif in the mature SUMO (ii) ATP-dependent activation of the processed SUMO through the thioester bond formation between the C-terminal glycine of SUMO and the catalytic cysteine of the E1 activating enzyme (iii) transfer of the SUMO polypeptide from the E1 enzyme to a conserved cysteine in the E2 conjugating enzyme via a thioester linkage and; (iv) E3 ligase-mediated formation of an isopeptide bond between the C-terminal glycine of the SUMO and the ε-amino group of the lysine residue within the conserved sequence on the target protein. Besides the precursor SUMO maturation, the SUMO-specific peptidases are also able to hydrolyse the isopeptide bond between SUMO and SUMO-modified proteins thereby rendering the SUMOylation process reversible.

To determine components of the SUMOylation pathway in *C. glabrata*, we performed whole proteome sequence and BLAST analyses, and identified *C. glabrata* orthologues of the proteins that are involved in SUMOylation in *Saccharomyces cerevisiae*. Of SUMO protein, SUMO-conjugating and activating enzymes and deSUMOylases identified, we were able to create deletion strains lacking CgSiz1 (a SUMO ligase), CgSiz2 (a SUMO ligase) and CgUlp2 (a deSUMOylation peptidase). Other components of the SUMOylation machinery in *C. glabrata* including the SUMO protein CgSmt3 appear to be essential for cell viability. We also constructed a double deletion strain lacking both SUMO-protein ligases CgSiz1 and CgSiz2. Phenotypic analysis of generated mutants revealed that *Cgsiz2*Δ and *Cgsiz1*Δ siz2Δ mutants displayed sensitivity to DNA damaging agents while the *Cgulp2*Δ mutant exhibited increased susceptibility to

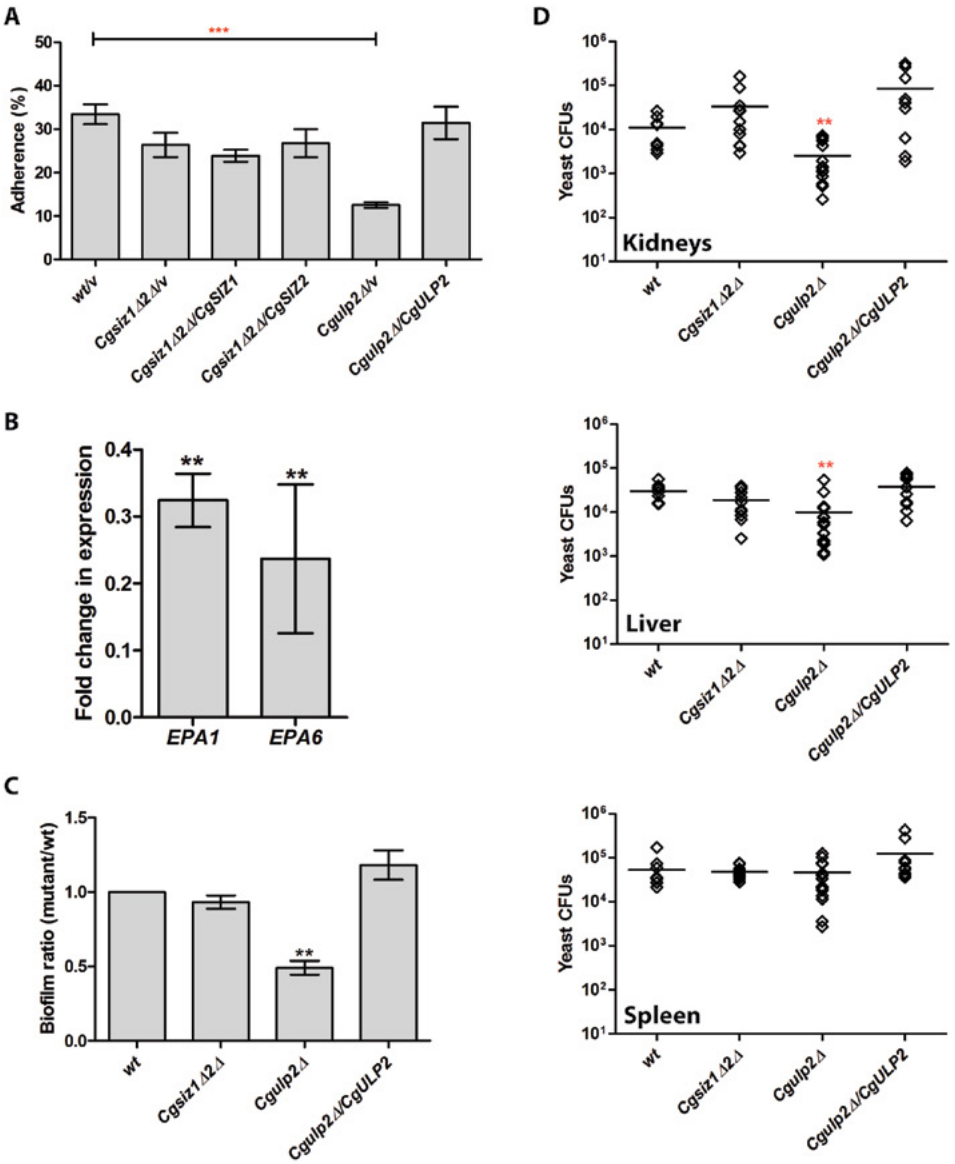

Figure 2. CgUlp2 desumoylase is required for virulence in the murine model of disseminated candidiasis.

A. Adherence of CAA medium-grown, S^{35}(Met:Cys-65:25)-labelled *C. glabrata* strains to p-formaldehyde-fixed Lec-2 ovary epithelial cells. Data represent means ± SEM of three to five independent experiments. Unpaired, two-tailed, Student's t test (***, p≤0.001).

B. Quantitative PCR analysis of *EPA1* and *EPA6* gene expression in wild-type and *Cgulp2*Δ mutant. Data (mean of 3 independent experiments ± SEM) were normalized to an internal *CgGAPDH* mRNA control, and represent fold change in expression upon *CgULP2* disruption. Paired, two-tailed, Student's t-test (**, p≤0.01).

C. Biofilm formation of indicated *C. glabrata* strains. Cells were grown in the RPMI medium containing 10% FBS for 48 h in a polystyrene 24-well plate. Cells were stained with crystal violet (0.4% in 20% (V/V) ethanol solution) for 45 minutes followed by complete destaining with 95% ethanol. Absorbance at 595 nm was recorded to measure the amount of the crystal violet stain in ethanol. Data represent mean±SEM of three independent experiments. **, p≤0.01; two-tailed paired Student's t-test.

D. 6-8 week-old, female BALB/c mice were infected intravenously with $4x10^7$ cells of indicated *C. glabrata* strains and sacrificed 7 days post infection. Diamonds represent the CFUs recovered from target organs, kidney, liver and spleen, for individual mice. Bars represent the geometric mean (n=8-14) of CFUs per organ. Statistically significant differences in the CFUs between *wt* and mutant strains are indicated (**, p≤0.01; Mann-Whitney test).

DNA damaging agents, oxidative stressors as well as to high temperature implicating CgSiz2 and CgUlp2 in survival of DNA damage, and thermal, oxidative and DNA damage stresses, respectively.

Next, we performed genome-wide transcript profiling of cells lacking the deSUMOylase using the RNA-sequencing approach, and found expression of many adhesin-encoding genes to be lower in the *Cgulp2*Δ mutant. Of note, adherence of *C. glabrata* cells to biotic and abiotic surfaces is thought to be mediated by a family of at least 23 cell wall adhesins. Further, many adhesin-encoding genes are encoded at the subtelomerc loci and subjected to the telomere position effect. To investigate the effect of reduced adhesin expression on the adherence capacity of *Cgulp2*Δ cells, we examined the ability of *Cgulp2*Δ to adhere to Lec2 ovary epithelial cells. As a control, adherence assay was also carried out with the *Cgsiz1*Δ*siz2*Δ mutant **(Fig. 2A)**. The *Cgulp2*Δ mutant displayed 2-fold less adherence to epithelial cells compared to that of the *wt* cells, which was restored back to *wt* levels in the *Cgulp2*Δ-complemented strain **(Fig. 2A)**. The hypo adherence of the *Cgulp2*Δ mutant was found to be, in part, due to a 3- to 4-fold reduced expression of two epithelial adhesin-encoding genes *EPA1* and *EPA6* in the mutant **(Fig. 2B)** indicating a role for the CgUlp2 deSUMOylase in regulated expression of adhesin-encoding genes. As Epa6 has been shown to be pivotal to biofilm formation in vitro, we next examined the effect of *EPA6* transcript levels on biofilm formation and measured the ability of *wt* and mutant strains to make biofilm on polystyrene-coated plates **(Fig. 2C)**. We observed that the *CgULP2* disruption led to a 50% reduction in the biofilm formation capacity while lack of SUMO ligases had no effect on biofilm formation in *C. glabrata* **(Fig. 2C)**.

Lastly, to investigate whether components of the SUMOylation machinery are required for virulence of *C. glabrata*, we examined fungal burden in BALB/c mice infected intravenously either with the wild-type or the *Cgsiz1*Δ*siz2*Δ and *Cgulp2*Δ mutant strains. Approximately, 10- and 8- fold lower yeast CFUs were recovered from the kidneys and liver, respectively, of the mice infected with the *Cgulp2*Δ mutant compared to CFUs retrieved from corresponding organs of the *wt*-infected mice **(Fig. 2D)**. Ectopic expression of the *CgULP2* gene restored the organ fungal burden in the *Cgulp2*Δ-infected mice **(Fig. 2D)**. Of note, no statistically significant differences in the fungal burden were seen between the spleen of *wt*- and *Cgulp2*Δ-infected mice **(Fig. 2D)**. Importantly, statistically similar yeast CFUs were obtained from all three target organs of *wt*- and *Cgsiz1*Δ*siz2*Δ-infected mice **(Fig. 2D)**. Together, these data indicate an organ-specific role for the CgUlp2 deSUMOylase and dispensability of CgSiz1 and CgSiz2 SUMO ligases in survival of *C. glabrata* in the murine model of disseminated candidiasis. Currently, we are trying to identify the SUMO proteome of *C. glabrata wt* and mutant strains.

Publications

1. Rai, M.N¶., Sharma, V¶., Balusu, S. and Kaur, R. (2015) An essential role for phosphatidylinositol 3-kinase in the inhibition of phagosomal maturation, intracellular survival and virulence in *Candida glabrata.* ***Cellular Microbiology*** 17:269-287. ¶ Equal Contribution

2. Srivastava, V.K¶., Suneetha, K.J¶. and Kaur, R. (2015) The mitogen-activated protein kinase CgHog1 is required for iron homeostasis, adherence and virulence in *Candida glabrata*. ***FEBS Journal*** 282: 2142-2166. ¶ Equal Contribution

3. Khandelwal, N.K., Kaemmer, P., Förster, T.M., Singh, A., Coste, A.T., Andes, D.R., Hube, B., Sanglard, D., Chauhan, N., Kaur, R., d'Enfert, C., Mondal, A.K. and Prasad, R. Pleiotropic effects of a vacuolar ABC transporter *MLT1 of Candida albicans* on cell function and virulence. ***Biochemical Journal*** (In press).

LABORATORY OF GENOMICS AND PROFILING APPLICATIONS

Faculty	Madhusudan Reddy Nandineni	Staff Scientist
PhD Students	Anujit Sarkar	Senior Research Fellow (till Feb. 2016)
	Soumya Rao	Senior Research Fellow
	Mugdha Singh	Senior Research Fellow
Other Members	Vineesha Oddi	Project-JRF
	Anil Kumar Challagandla	Project Assistant (till Oct. 2015)

Objectives

1. Human genetic diversity studies among various population groups in India; and
2. Plant-fungal interaction studies in the chilli-*Colletotrichum* pathosystem

Project 1: Human genetic diversity studies among various population groups in India.

Summary of work done until the beginning of this reporting year (up to March 31, 2015)

With an aim to design a single nucleotide polymorphism (SNP)-based panel for human identification (HID) in Indian populations, SNPs were shortlisted from public databases by applying various stringent filters and were genotyped using GoldenGate® Genotyping assay (Illumina, Inc, USA) in ~ 370 unrelated individuals sourced from different populations across the country to assess their performance.

In addition to the SNPs, to better understand the human genetic diversity in Indian populations and to assess the applicability of the expanded panel of autosomal and Y-chromosomal STR (short tandem repeat) loci from PowerPlex® Fusion and PowerPlex® Y23 (Promega, Madison, WI, USA) chemistries, the STR loci were genotyped in 120 male individuals from four different biogeographic regions in the country. Towards understanding the distribution and diversity of salivary microbiome in Indian populations, partial sequencing of the 16S rRNA was performed by massively parallel sequencing in 92 individuals from three biogeographic regions in the country.

Details of progress made in the current reporting year (April 1, 2015- March 31, 2016)

a) SNPs for HID purposes

In the current reporting year, 384 SNPs (which included 275 SNPs shortlisted for HID testing) were genotyped in 92 additional samples (total of 462 samples) across 12 different sampling locations and four biogeographic regions viz. North India (N=167), West India (N=87), East India (N=105) and South India (N=103). After discarding the SNPs which failed the Hardy-Weinberg equilibrium (HWE) test, those with high heterozygosity (Het ≥ 0.4) and low Wright's F-statistics (F_{st} ≤ 0.02) were retained. Among the 275 SNPs tested, 206 SNPs were found to possess the desired allelic distribution for HID purposes, from which 2-4 SNPs located distantly from each other (> 20 Mb apart) in each of the chromosomes were selected to constitute a panel of 70 SNPs. Linkage disequilibrium analyses showed no significant association between any pair of SNPs in any of the biogeographic regions. The various forensic parameters used to assess the efficiency of a panel including, random match probability (RMP, which denotes the chances that two individuals randomly selected from a population will have the same genetic profile), combined paternity index (CPI, representing the likelihood that the alleged father is the true father of the disputed child), combined probability of paternity (W, which denotes the posterior probability that the alleged father is the true father of the disputed child based on DNA evidence) and combined motherless paternity index (mPI, paternity index in the absence of the genetic profile of biological mother) were calculated using DNAView™ for these 70 SNPs. A summary of the results is shown in Table 1. The RMP based on the 70 SNPs was of the order 10^{-29} across all biogeographic regions with only minor differences among them and the probability of paternity was atleast 0.999999979, demonstrating the high power of discrimination and efficiency of these SNPs in all regions. Overall, the panel demonstrated very high forensic parameters sufficient to make unambiguous inferences in HID testing.

Table 1: Forensic statistics obtained with the SNP-based panel designed in the current study. The populations that were tested are grouped according to their biogeographic regions.

S.no.	Panel	North	West	East	South
1	Random match probability (RMP)	1e-29	9.1e-30	1.1e-29	1.1e-29
2	Combined probability of paternity (W)	0.999999983	0.999999979	0.999999981	0.99999998
3	Combined paternity index (CPI)	58600000	48600000	51300000	51200000
4	Combined motherless paternity index (mPI)	114000	96200	99300	99400

b) Human genetic variations studies in Indian populations based on expanded loci of autosomal and Y-chromosomal STRs

To study the genetic relationship among the various sub-populations from different biogeographic locations and to evaluate the applicability of the expanded STR loci in PowerPlex® Fusion (Promega, Madison, WI, USA) chemistry in Indian populations, 357 individuals from sub-populations residing in 11 different biogeographic regions of India were genotyped and the allele frequencies were calculated. A total of 275 alleles were observed for all the loci in the studied Indian populations and the STR loci were found to be highly polymorphic with an average informative index of 1.77. The combined power of discrimination (CPD; the strength of panel of markers to distinguish an individual from others) and probability of exclusion (CPE; the strength of panel of markers to exclude a particular genotype) were determined to be 0.99999999999999999999999999875 and 0.999999997200846, respectively, using PowerStats version1.2 (Promega, Madison, WI, USA). GenALEx v6.5 was used to carry out Analysis of Molecular Variance (AMOVA) and principle coordinate analysis (PCoA). AMOVA showed higher percentage of variance within individuals (97.86%) as compared to variations among individuals within populations (1.81%)

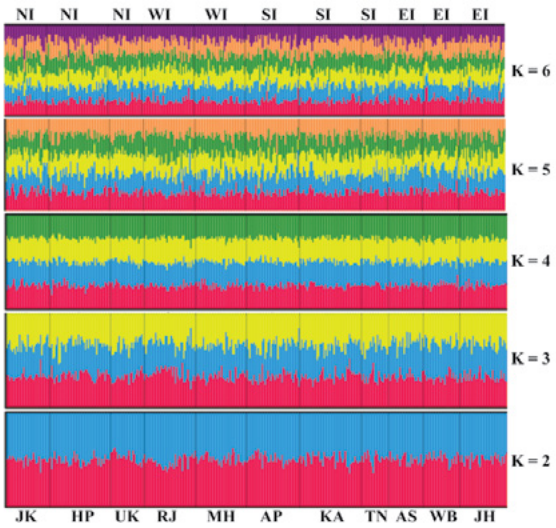

Figure 1. Clustering analysis by STRUCTURE (processed by Distruct). Clustering analysis was carried out to study the degree of similarity based on autosomal STRs in the 11 populations across different biogeographic regions of India, assuming K = 2 to 6, where K is the number of clusters. Sampling location and the major biogeographic regions are labeled below and above the plot, respectively. The abbreviations used in figure and the number of individuals from each region (N) are as following: North India (NI):Jammu and Kashmir (JK, N=31), Himachal Pradesh (HP, N=43), Uttarakhand (UK, N=24); West India (WI): Rajasthan (RJ, N=37), Maharashtra (MH, N= 36); South India (SI): Karnataka (KA, N =44), Tamil Nadu (TN, N=19), Andhra Pradesh (AP, N=38); East India (EI): West Bengal (WB, N=26), Jharkhand (JH, N=34), Assam (AS, N=25).

and amongst populations (0.33%). The PCoA suggested less genetic distance among the studied sub-populations. Further, clustering analysis performed using STRUCTURE 2.3.4, showed no significant sub-structuring in these Indian populations using the present set of markers (Figure 1). The higher values of CPD and CPE reflect the higher potential of the present panel of markers in forensic case work analysis in Indian populations. AMOVA, PCoA and clustering analysis revealed lesser genetic variation among populations, implying that this chemistry is expected to show high efficiency and similar forensic statistics throughout the Indian populations.

c) Studies on human salivary microbiome in Indian populations

The NGS data analyses of 16S rRNA sequences revealed high bacterial richness represented by 165 different bacterial genera and 785 unique OTUs in the Indian populations. Rarefaction analysis showed that the sequencing approach and depth was sufficient to ascertain the species richness in the tested saliva samples. The samples from West Bengal displayed highest number of unique genera whereas the Tamil Nadu samples showed the least. Diversity indices demonstrated that the North Indian samples displayed highest richness (alpha diversity) followed by South and East Indian samples while inter-individual diversity (beta diversity) was highest for the South Indian populations and lowest for the East Indian populations. The results indicate that overall, the samples from the South Indian populations are more dissimilar (i.e., exhibit greater population differences) than those of the North and East Indian populations.

In the current study, 79 bacterial genera, which were hitherto unreported in the Human Oral Microbiome Database (HOMD), were observed. Their abundance was observed to be significantly lower (mean abundance = 0.027%, $p = 8.07 \times 10^{-13}$) than those listed in the HOMD (mean abundance = 1.14%), indicating that sequencing depth might have helped in unraveling the rare contributors to the salivary microbiome. Statistical analyses after normalizing the current dataset for sequencing depth compared to a previous study (Li *et.al*., 2015), suggested the existence of novel bacterial genera specific to populations, indicating the role of ethnicity and/or geography in shaping the salivary microbiome.

The existence of a core salivary microbiome in the Indian populations was also investigated. The distribution of the 785 unique OTUs (obtained at 97% clustering) showed extensive sharing across all the regions as shown in Figure 2. The samples from North Indian populations shared 683 and 675 OTUs with the East and South Indian samples respectively, while the East Indian populations shared 703 OTUs with the South Indian populations. A total of 660 OTUs were found to be shared in all three geographic regions. Among these 660 OTUs, 37 OTUs were found in all individuals studied and could comprise a putative core microbiome for Indian populations. All the 37 OTUs could be assigned to 10 bacterial genera, 8 of which were part of

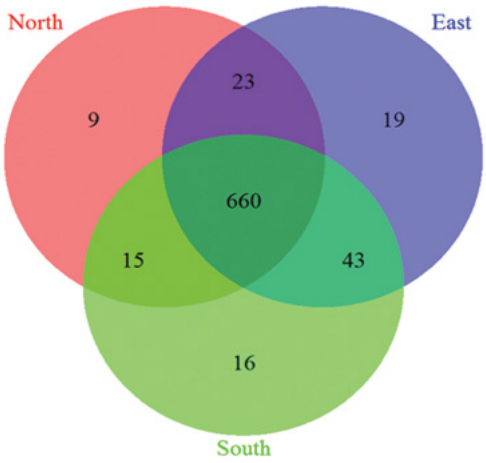

Figure 2. A core salivary microbiome as identified by OTU sharing among the studied biogeographic regions represented using Venn diagram. The distribution of 785 unique OTUs (obtained at 97% clustering) across North (orange), East (violet) and South (green) are shown.

core microbiome in several world populations observed in previous studies (Huse *et.al*., 2012, Li *et.al*., 2013), while 2 OTUs were novel although they could not be sub-classified upto the genera level. Similar to the observation based distribution of bacterial genera, analyses with OTUs also displayed high sharing of the microbiome among the Indian populations. Further analyses are under progress to understand the significance of food habits and common physical factors like latitude, longitude, altitude, etc., on the oral microbial diversity.

Project 2: Plant-Fungal interaction studies in the chilli - *Colletotrichum* pathosystem.

Summary of work done until the beginning of this reporting year (up to March 31, 2015)

Chilli (*Capsicum annuum* L.) is an important spice and a major commercial crop in India. *Colletotrichum truncatum* (formerly called as *C. capsici*) is the most predominant fungal pathogen causing chilli anthracnose leading to both pre- and post-harvest losses. With the availability of whole genome sequence for chilli and *many Colletotrichum* species, the chilli - *C. truncatum pathosystem* offers an excellent model system for studies on the infection process and molecular interactions between the host and pathogen. The present study aims to identify and characterize pathogenicity genes in *C. truncatum* to get an insight into different aspects of its biology, life-style and host specificity through whole genome and transcriptome sequencing of *C. truncatum* and random insertional mutagenesis.

We have earlier reported the *de novo* whole genome sequencing of *C. truncatum* employing Illumina HiSeq platform. The sequence assembly consisted of 81 scaffolds with a total length of 55.3 Mb (460X coverage). Preliminary annotation of the assembly using BLASTX with *C. higginsianum* genome identified 10,126 homologues in *C. truncatum*. The completeness of the draft genome assembly of *C. truncatum* was determined using Core Eukaryotic Genes Mapping Approach (CEGMA) and tBLASTn, based on coverage of orthologs of all 458 core eukaryotic genes (CEGs). In order to identify pathogenicity genes in *C. truncatum* through forward genetics approach, random insertional mutagenesis of *C. truncatum* by *Agrobacterium tumefaciens* mediated transformation (ATMT) was performed using *A. tumefaciens* strain C58C1 harboring a binary vector pBIN-GFP-hph. The resultant fungal transformants were selected on potato dextrose agar (PDA) containing hygromycin. The mitotically stable transformants were screened for partial or complete loss of pathogenicity on chilli.

Details of progress made in the current reporting year (April 1, 2015- March 31, 2016)

(a) Whole genome *de novo* sequence analysis

In order to get a consensus on the number of genes predicted by different *ab initio* gene callers and homologous genes identified through BLAST with other *Colletotrichum* spp., a gene annotation pipeline, MAKER was used. In the first run of MAKER, transcript and protein evidences from closely related spp., *C. gloeosporioides* and *C. graminicola*; as well as the proteome of *C. higginsianum* were used to identify the orthologous genes in draft assembly through BLAST. Soft-masking of repetitive elements in the genome was carried out by using repeatmasker option in MAKER with the repeat library of fungi in RepeatMasker-4.0 database and the *de novo* repeat library specific to *C. truncatum* generated by RepeatModeler – 1.0.4. *Ab initio* gene predictions were made by gene callers like SNAP and AUGUSTUS v.3.0.3 (which were trained on CEGMA output), and GENEID v.1.0 (parameters set for *Fusarium oxysporum*). The results from the run were used in subsequent runs to train SNAP and AUGUSTUS along with self-trained *ab initio* gene caller GeneMark-ES Suite 4.2. 12,776 proteins were predicted after the final MAKER run (Table 2) and were annotated by homology search with SWISS-PROT database (db) through BLASTp. The annotations for conserved protein domains (protein families or Pfam annotation) and Gene Ontology (GO) terms were obtained through interproscan-5.8-49.0 and were integrated to MAKER annotations after performing quality filter using a PERL script (kindly provided by the developers of MAKER). The functional annotation of predicted genes and secretome prediction would be carried out in future which is expected to aid in identification of effectors and pathogenicty genes in *C. truncatum.*

(b) Pathogenicity assay of fungal transformants

Around 1300 *C. truncatum* transformants generated through ATMT in the initial phase were screened for the complete or partial loss of pathogenicity on chilli. The conidial suspensions were used to inoculate *C. annuum* fruits at mature green stage for pathogenicity assay.

Table 2: Summary statistics for MAKER annotation of C. truncatum draft genome assembly	
Protein Prediction	**Number of proteins**
Total number of proteins	12,776
Proteins with Pfam domain	9,873 (77.3%)
Proteins with GO terms	6,464 (50.6%)
Proteins with homologs in SWISS-PROT db	8,627 (67.5%)

The fruits inoculated with Milli-Q water and wild type conidia were used as negative and positive controls, respectively. After secondary and tertiary screening, five transformants were found to retain the non-pathogenic phenotype, whose molecular characterization would be carried out in future. Further, additional mutants with loss of pathogenicity would be identified to understand host-pathogen interactions at the molecular level.

Publications

1. Gadipally SR, Sarkar A and Nandineni MR (2015). Selective enrichment of STRs for applications in forensic human identification. ***Electrophoresis*** 36(15): 1768-1774.

LABORATORY OF IMMUNOLOGY

Role of advanced glycation endproducts (AGE) in exerting adverse effects

Faculty	Sunil K Manna	Staff Scientist
PhD Students	S Adeel Husain Zaidi	Senior Research Fellow
	Raveendra Babu M	Senior Research Fellow
	Neeharika Verma	Senior Research Fellow
	Pankaj Gupta	Senior Research Fellow
	Shashank Saurav	Junior Research Fellow
Other Members	Nune Raviprakash	Project SRF
	T Navaneetha	Technical Assistant
Collaborators	Biswadev Bishayi	Calcutta University, Kolkata
	Tushar Basu Baul	NEHU, Shilong

Objectives

1. Understanding and regulation of inflammatory and tumorigenic responses;
2. Understanding and regulation of advanced glycation endproducts (AGE)-mediated lipogenesis and autophagy; and
3. Understanding the role of Profilin in regulation of tumorigenesis.

Summary of work done until the beginning of this reporting year (upto March 31, 2015)

The molecular mechanism of Profilin for its tumor suppressor activity is still unknown. NF-κB is known to activate many target genes involved in cell proliferation. This prompts us to profilin-stable cell (A-231) generation. Profilin overexpressing cells show low basal activity of IKK, high amount of cytoplasmic IκBα and p65, and low nuclear NF-κB DNA binding activity. Profilin did not suppress NF-κB activation when transfected with p65 or IKKβ, with or without TNF stimulation. Co-localization and *in silico* studies suggested that Profilin interacts with a protein phosphatase, PTEN and protects it from degradation. In turn, PTEN physically interacts and maintains low phosphorylated state of IKK complex and thereby suppresses NF-κB signaling. Thus, Profilin overexpressing cells show decrease in NF-κB activation mediated by most of the inducers and potentiates cell death by repressing NF-κB-dependent genes involve in cell cycle progression.

Profilin potentiates several chemotherapeutic-agents mediated cell death. Profilin overexpression suppressed migration and invasiveness of breast cancer cells. Paclitaxel and vinblastine-mediated NF-κB and NF-κB-dependent genes activation was completely inhibited in Profilin overexpressing cells. The increased p53 DNA binding activity was potentiated in Profilin overexpressing cells. The Sp1 DNA binding followed by Mdm2 expression was completely abrogated in Profilin overexpressing cells. Thus, Profilin suppress NF-κB activation and increase p53 activity by suppressing Sp1 and thereby, Mdm2 expression. Profilin synergizes with chemotherapeutic drugs to induce tumor cell death by attenuating NF-κB and upregulating p53. Thus, modulation of Profilin may be useful for effective combination therapy.

Details of progress in the current reporting year (April 1, 2015 - March 31, 2016)

1) Advanced glycation end products (AGE) potently induce autophagy through activation of RAF kinase and NF-κappa B

Advanced glycation end products (AGE) accumulate in diabetic patients and aging people due to high amounts of 3- or 4-carbon sugars derived from glucose and thereby causing multiple consequences including inflammation, apoptosis, obesity and age-related disorders. It is important to understand the mechanism of AGE-mediated signaling leading to activation of autophagy (self-eating) that might negatively assist in developing obesity and its consequences. We have detected AGE as one of the potent inducers of autophagy compared to doxorubicin and TNF (Fig.1A). AGE-mediated autophagy is inhibited by suppression of PI3 kinase (upon wortmanin

treatment) and potentiated by autophagosome maturation blocker, bafilomycin as determined by the LC3B-GFP puncta (Fig.1B). It increases autophagy in different cell types (Fig.1D) which corresponds well to the expression of RAGE (AGE receptor in these cell lines) (Fig.1C). LC3B, the marker for autophagosome is shown to increase upon AGE stimulation (Fig.1F) along with other autophagy markers (Fig.1E). AGE-mediated autophagy is suppressed partially by inhibitor of NF-κB (Fig.1G1), ERK (Fig.1G2), or PKC (Fig.1G3) alone and significantly in combination. Subsequently, *IκBα-DN* (*IκBα* dominant negative) transfected cells, even

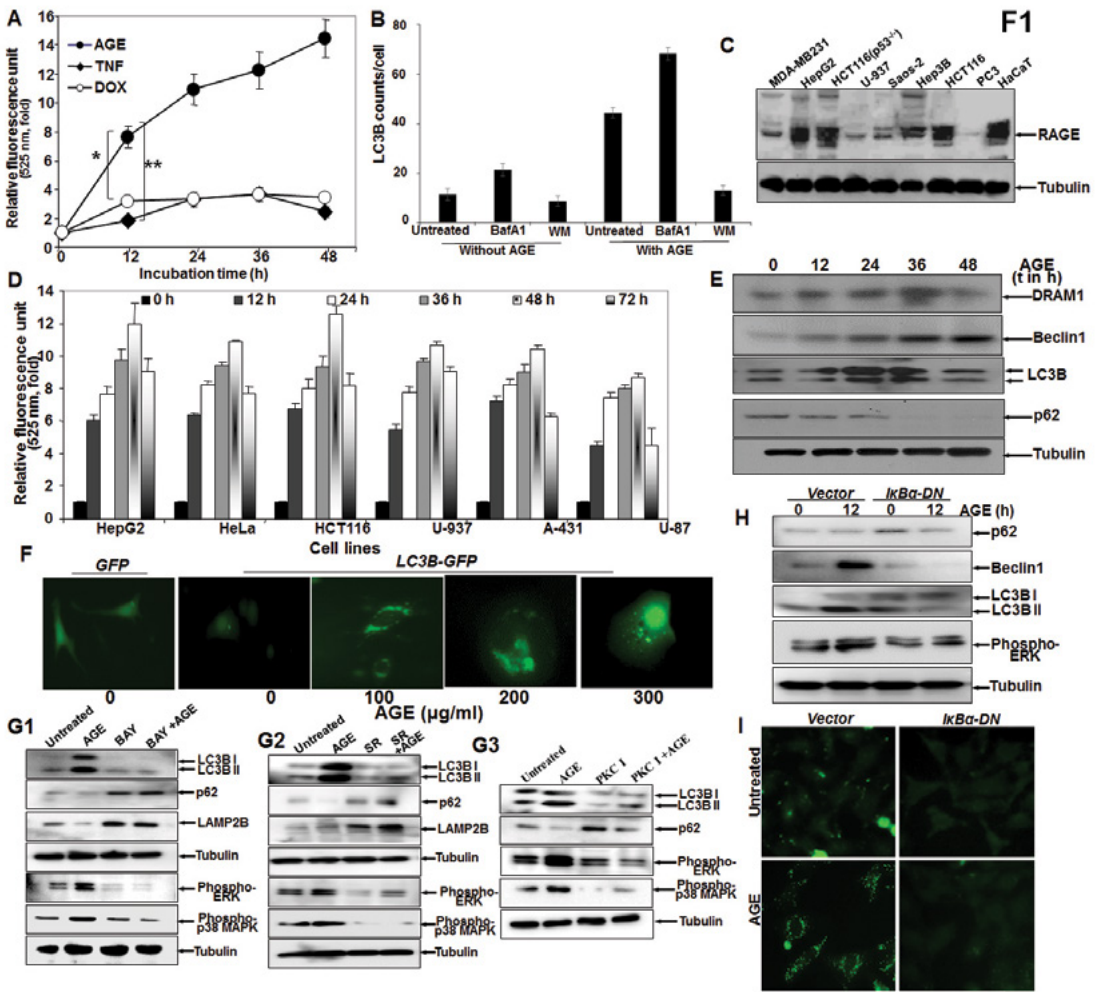

Figure 1. AGE induces autophagy. HepG2 cells were stimulated with AGE (100 μg/ml), TNF-α (1 nM) or doxorubicin (1 μM) for different times in triplicate. After treatments, cells were fixed with paraformaldehyde (4%), stained with MDC (50 μM) for 15 min, and washed thrice with PBS. Cells were collected, fluorescence was measured and indicated as fold considering unstimulated cells' value as one fold (A). Error bars represent as S.E.M., Student's t-test 'ns' indicates not significant, * $p<0.05$ and ** $p<0.01$. Immunofluorescence images were captured using ant-LC3B antibody for bafilmycin A1 (BafA1) or wortmannin (WM) treated and AGE-stimulated cells. LC3B dots are counted and plotted (B). Basal amount of RAGE was measured from whole cell extracts (WCE) of various cell lines (100 μg of protein) by Western blot (C). Different cells were incubated without or with AGE (100 μg/ml) for different times and the MDC fluorescence was determined in triplicates and data, extrapolated from three independent experiments are represented as fold of induction in mean ± S.E.M. taking unstimulated cells' value as one fold (D). WCE were prepared from AGE stimulated cells for different times and Western blot was performed to detect DRAM1, Beclin1, LC3B, and p62 (E). Representative fluorescent images were from GFP-LC3B transfected cells stimulated with different concentrations of AGE for 24 h (F). Western blot was performed from WCE, prepared from cells pre-treated with IKKβ/α inhibitor BAY11-7082 (2 μM) for 3 h (G1), sorafenib (SR) (10 μM for 3 h) (G2), or PKC inhibitor (PKC I) (2 μM for 3 h) (G3) followed by AGE (100 μg/ml) stimulation for 12 h. The amounts of LC3B, LAMP2B, p62, phospho-ERK1/2 and phospho-p38 MAPK were determined by Western blot. Cells, transfected with IκBα-DN for 6 h were incubated for 12 h. Cells were stimulated with AGE for 12 h and the amounts of Bectlin1, LC3B, p62, and phospho-ERK were determined by Western blot (H). The transfected cells, stimulated with AGE were stained with MDC and fluorescence images were represented (I).

when stimulated by AGE showed reduction in autophagy markers including Beclin1, LC3B or phosphor-ERK but p62 insignificantly (Fig.1H) suggesting the important role of NF-κB in AGE-mediated autophagy. MDC staining in these transfected cells also complemented the autophagy reduction result (Fig.1I). These data further suggest that NF-κB plays an important role in AGE-mediated autophagy.

2) AGE-mediated autophagy and lipogenesis are not mechanistically interlinked

To detect the role of AGE-mediated autophagy in lipogenesis, we determined the amount of molecular markers of autophagy. AGE stimulation increased both lipogenesis as determined by Oil Red O stained cells and autophagy as determined by MDC stained cells in time dependent manner (Fig.2A). Mangiferin was used as known inhibitor for AGE-mediated lipogenesis. To validate the probable role of autophagy in lipogenesis, Oil Red O staining was again done in presence of autophagy inhibitors and mangiferin which showed dramatic drop in lipid droplets as indicated by microscopic view (Fig.2B). AGE increased SREBP DNA binding kinetically (Fig.2C). AGE-mediated lipid accumulation as detected by Oil Red O staining was inhibited to almost 50% by PKC I or SB and PD. BAY or SR inhibited almost 80% of lipid accumulation in AGE-stimulated cells as shown by microscopic view of cells with oil red stained particles (Fig.2D). Inhibiting autophagy upon Atg7 and Atg12 shRNA transfection and subsequent

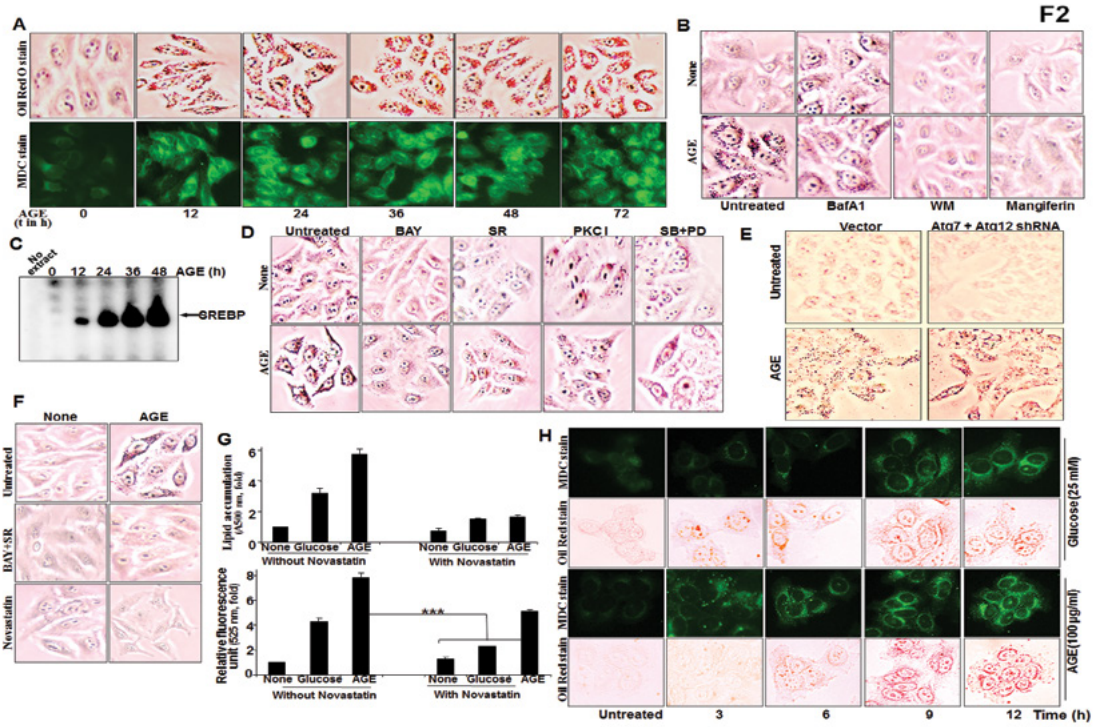

Figure 2. AGE increases lipogenesis and autophagy. MHepG2 cells were stimulated with 100 μg/ml AGE for different times. Cells were stained with MDC followed by visualized under fluorescence microscope (**A**, lower panel). Stimulated cells were incubated with Oil Red O stain and visualized under microscope (**A**, upper panel). HepG2 cells were pre-treated with mangiferin (10 μg/ml) and wortmannin (WM, 100 nM) for 3 h, or bafilomycin A1 (BafA1, 10 nM) for 5 h followed by stimulation with AGE (100 μg/ml) for 12 h. Images of Oil Red O stained cells were shown (**B**). HepG2 cells were stimulated with AGE (100 μg/ml) for different times and SREBP DNA binding was assayed (**C**). HepG2 cells were pre-treated with BAY, SR, PKC I, SB and PD, or BAY and SR for 3 h, followed by AGE stimulation of 12 h. Oil Red O stained cells were represented here (**D**). HepG2 cells, transfected with Atg7 and Atg12 shRNA were stimulated with AGE for 12 h. Cells were stained with Oil Red O and visualized under microscope (**E**). Cells were pretreated with BAY and SR or novastatin for 3 h and then stimulated with AGE for 12 h. The images were captured after Oil Red O staining (**F**). HepG2 cells were stimulated with AGE and glucose in presence or absence of Nov for 12 h and lipid accumulation as well as autophagy index were determined and represented as mean ± S.E.M. from triplicate samples of two independent experiment (**G**). HepG2 cells were treated with AGE and glucose for 0, 3, 6, 9 and 12 h. These cells were subjected to double staining. First, cells were stained with MDC followed by Oil Red O. Represented images were captured in the same view field (**H**).

stimulation with AGE resulted in the increase in accumulation of lipid droplets in cells (Fig.2E). Almost complete inhibition of lipid accumulation was observed in AGE-stimulated cells pretreated with novastatin, a known inhibitor HMG CoA pathway or SR and BAY (Fig.2F). These data suggest that NF-κB and Raf kinase pathways are involved in AGE-mediated lipid accumulation. Glucose increased lipogenesis and autophagy almost 4-fold. Compared to glucose, AGE-induced both of these to almost 8-fold. Novastatin inhibited both glucose- and AGE-mediated lipogenesis. Whereas, it inhibited glucose-, but not the AGE-mediated autophagy (Fig.2G). Cells, when incubated with 25 mM glucose or 100 μg/ml AGE for different time, showed accumulation of lipid droplets prior to autophagy induction in case of glucose, but autophagy was proceeded by accumulation of lipid droplets in case of AGE stimulation (Fig.2H). Novastatin completely inhibited AGE-mediated lipogenesis, but not the autophagy, further suggesting that AGE-mediated lipid accumulation is independent of autophagy. These data further suggested that AGE and glucose mediated autophagy and lipogenesis follow different pathways and AGE-mediated autophagy machinery initiates prior to lipogenesis which probably helps cells with supply of energy and other building blocks to assist lipogenesis and hence shifts the balance from lipolysis to lipid accumulation.

Publications

1. Sahoo BK, Zaidi AH, Gupta P, Mokhamatam RB, Raviprakash N, Mahali SK, Manna SK. (2015) A natural xanthone increases catalase activity but decreases NF-kappa B and lipid peroxidation in U-937 and HepG2 cell lines. ***European Journal of Pharmacology*** 764: 520-528.
2. Ghosh C, Raviprakash N, Manna SK, Bishayi B. (2015) Presence of Toll Like Receptor-1 in spleen, lymph node and thymus of Swiss albino mice and its modulation by Staphylococcus aureus and bacterial lipopolysaccharide. ***Indian Journal of Experimental Biology*** 53: 82-92.
3. Zaidi AH, Manna SK. (2016) Profilin-PTEN interaction suppresses NF-kappa B activation via inhibition of IKK phosphorylation. ***Biochemical Journal*** 473: 859-872.
4. Zaidi AH, Raviprakash N, Mokhamatam RB, Gupta P, Manna SK. (2016) Profilin potentiates chemotherapeutic agents mediated cell death via suppression of NF-kappa B and upregulation of p53. ***Apoptosis*** 21: 502-513.
5. Verma N, Manna SK. (2016) Advanced Glycation End Products (AGE) Potently Induce Autophagy through Activation of RAF Protein Kinase and Nuclear Factor κB (NF-κB). ***Journal of Biological Chemistry*** 291: 1461-1491.

LABORATORY OF MAMMALIAN GENETICS

Epigenetic mechanisms underlying developmental pathways

Faculty	Sanjeev Khosla	Staff Scientist
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	Imtiyaz Yaseen	Senior Research Fellow
	Thushara Thamban	Senior Research Fellow
	Ramisetti Rajeev	Junior Research Fellow
	Viplove V Agarwaal	Junior Research Fellow
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	M Srilalitha	Technical Officer
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	Vinay K. Nandicoori	NII, New Delhi
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Project 1: *DNMT3L*: Role in Development

Summary of work done until the beginning of this reporting year (up to March 31, 2015)

Previous work from our laboratory has shown the role of DNMT3L in nuclear reprogramming. HeLa cells overexpressing DNMT3L were found to have undergone nuclear reprogramming gradually and showed morphological changes only in the 20th generation post transfection of *DNMT3L* construct (Gokul et al 2009; Epigenetics 4: 322-329). Moreover, ectopic expression of *DNMT3L* caused melanotic tumors in Drosophila.

Details of progress made in the current reporting year (April 1, 2015- March 31, 2016)

We had previously shown that transgenic Drosophila that ectopically expressed DNMT3L showed melanotic tumors in some of the larvae but only when maintained for more than 5 generations. The appearance of the larvae with tumors in 5th generation progeny was not due to an abrupt change in its expression and the expression of DNMT3L remained constant in all the generations. This was true for all DNMT3L transgenic Drosophila lines as also with the use of any Gal4-drivers (*Tubulin, Actin or Daughterless*).

Ectopic *DNMT3L* expression in Drosophila caused progressive misregulation of genes. Only 205 genes were misregulated in G1 but by 5th generation a very large number of genes (3730) were aberrantly expressed. As DNMT3L is a modulator of epigenetic modifications, we examined various DNA and histone modifications in Drosophila expressing *DNMT3L*. While no change was observed in the DNA methylation levels, dramatic change was noticed in the level of histone H3 methylation especially at lysine 4 and 36. This can be seen in the representative western blot (Figure 1A) where the level of H3K4me$_3$ and H3K36me$_3$ had significantly reduced in tumor bearing G5 Drosophila larvae that were expressing DNMT3L, as compared to the control UAS-3L (G5) larvae. This observation was reinforced by immuno-staining of polytene chromosome with H3K4me$_3$ antibody where negligible H3K4me$_3$ staining was observed for the polytene chromosome in DNMT3L expressing *Tub-3L* flies (Figure 1B). Like progressive increase in transcriptional misregulation, increase in aberrant H3K4me$_3$ was also progressive. This suggested that aberrant H3K4 and K36 methylation (epimutations) were being inherited across generations. We, therefore, have uncovered a role of DNMT3L in transgenerational inheritance (Basu et al 2016).

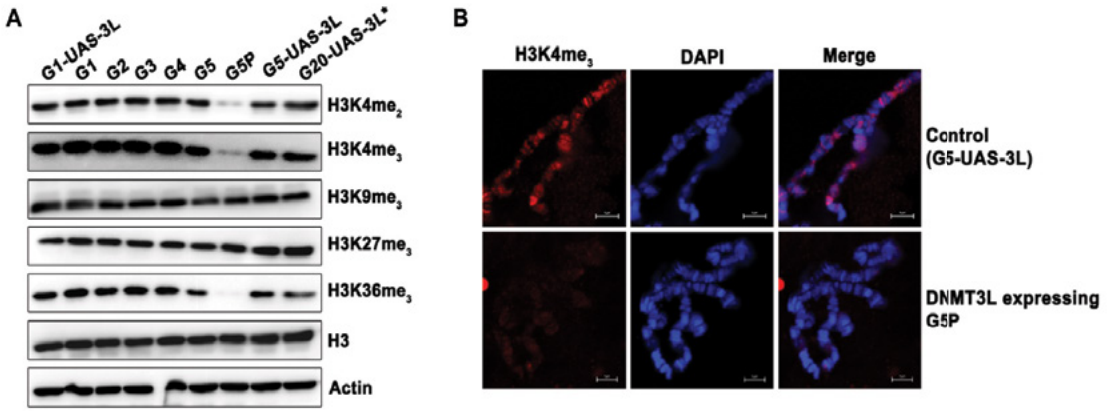

Figure 1. Accumulation of aberrant histone methylation in DNMT3L expressing *Drosophila* across successive generations. (A) Western blot analysis for the various histone modification as indicated, performed on larvae from the various generations of control and DNMT3L *expressing Drosophila* larvae. G1 and *G5-UAS-3L* are control larvae are without *GAL4* driver from G1 and G5 generation respectively. G1 to G5- *Tub-3L* larvae from the indicated generation. G5P- G5 *Tub-3L* larvae that had melanotic tumors. G20-*UAS-3L** denotes larvae from *G20* generation after crossing out of the *Tubulin-GAL4* driver. Actin was used as a loading control. (B) Immunostaining of *Drosophila* polytene chromosomes with H3K4me3 antibody.

Project 2: Host epigenetic response to infection

Summary of work done until the beginning of this reporting year (up to March 31, 2015)

We have previously identified a putative DNA methyltransferases Mtbmeth1 (Rv2966c) from mycobacteria which had the ability to methylate cytosines in the host genome in a non-CpG dinucleotide context. This methylation was correlated with change in the expression of specific host genes.

Details of progress made in the current reporting year (April 1, 2015- March 31, 2016)

In addition to a DNA methyltransferase, we have now identified and characterized a protein arginine methyltransferase from mycobacteria that can methylate histone H3 in the host cell at H3R42. The protein, Rv1988, present only in the pathogenic strains of mycobacteria including *M. tuberculosis* and *M. bovis*, has a capability to be secreted out of the mycobacterial cell, localize to the chromatin in the host nucleus and dimethylate an arginine amino acid present specifically at the 42nd position in histone H3. This arginine is quite important in the nucleosomal structure as it straddles the point where DNA enters and exits the nucleosome. Modification of this residue has the potential to profoundly affect gene transcription and indeed, Rv1988 through H3R42me$_2$ was able to repress gene expression both in *in vitro* reporter gene and in vivo infection assays.

When mice were infected with *M. smegmatis* (this mycobacterial species lacks Rv1988) ectopically expressing Rv1988, increased bacterial load (increased potential to survive in the host cell) was observed in liver, spleen and lung of infected mice. On the other hand, *M. tuberculosis* harboring a deletion for Rv1988 showed reduced survival ability during infection. Both these observations indicated that Rv1988 was a virulence factor.

Therefore, targeting of R42 by Rv1988 indicated that mycobacteria was not only utilizing a novel epigenetic mechanism to target host transcription but had chosen as a target an important residue within the nucleosome.

Our work on both Rv1988 and Rv2966c adds to a growing realization that pathogenic bacteria like *M. tuberculosis* use non-canonical mechanisms to hijack the epigenetic regulation of host transcription. Thus, Rv1988 and Rv2966c could provide *M. tuberculosis* the first line of attack during infection by dampening the action of genes involved in mounting host defense against the pathogen (Figure 2).

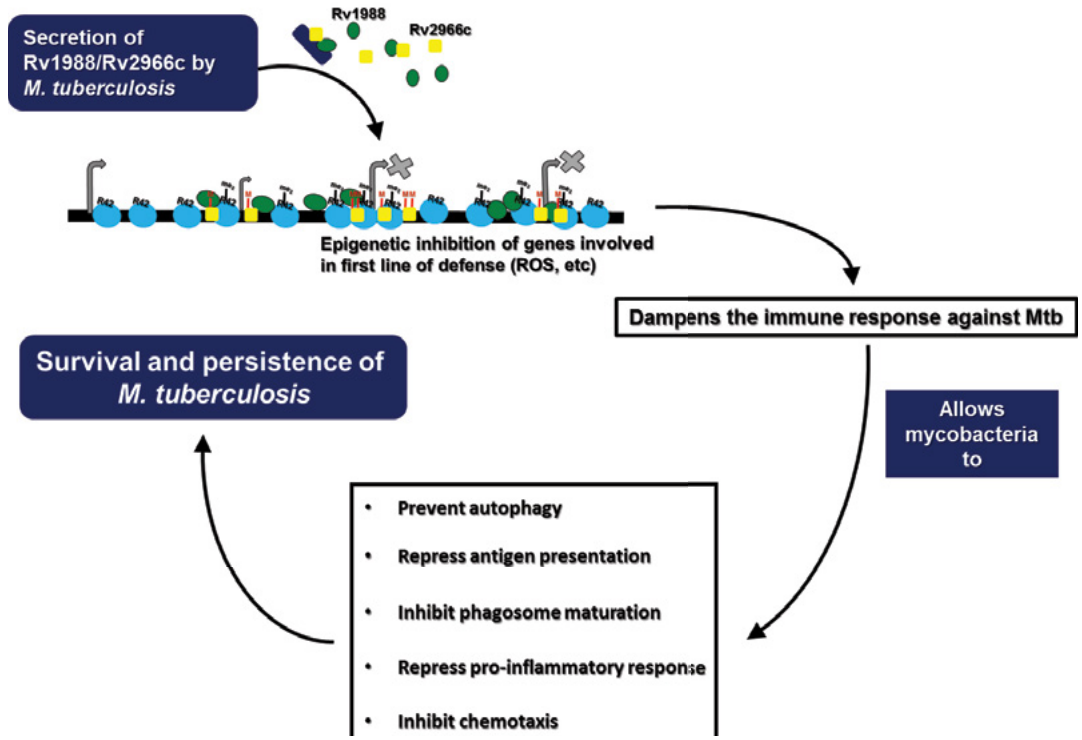

Figure 2. ***M. tuberculosis uses*** **Rv1988 and Rv2966c to hijack the host transcriptional machinery.** During infection, *M. tuberculosis* secrete proteins like Rv1988 and Rv2966c to epigenetically modulate expression of host genes involved in first line of defense including ROS activity. Dampening of the initial host defense could allow mycobacteria to utilize additional multiple factors to ensure its continued survival and persistence in the host cell. White text in blue boxes represents action by the mycobacterium. Black text in open boxes represents action in the host cell. Artwork depicts the action of Rv1988 and Rv2966c. Within the illustration, black horizontal bar depicts DNA within the host chromatin. Blue circles – Nucleosomes; green circle – Rv1988, yellow rectangles – Rv2966c; Blue rectangle – *M. tuberculosis* bacillus; me_2 – H3R42me_2; M – cytosine methylation; raised arrows – gene transcription; X – repression or inhibition of gene transcription.

Publications

1. Sharma G, Upadhyay S, Srilalitha M, Nandicoori VK, Khosla S (2015) The interaction of mycobacterial protein Rv2966c with host chromatin is mediated through non-CpG methylation and histone H3/H4 binding. ***Nucleic Acids Research*** 43:3922-3937.
2. Yaseen I, Kaur P, Nandicoori VK, Khosla S* (2015) Mycobacteria modulate host epigenetic machinery by Rv1988 methylation of a non-tail arginine of histone H3. ***Nature Communications*** 6:8922 doi: 10.1038/ncomms9922.
3. Basu, A. Tomar A, Dasari V, Mishra RK*, Khosla S* (2016) DNMT3L enables accumulation and inheritance of epimutations in transgenic Drosophila. ***Scientific Reports*** 6:19572; doi: 10.1038/srep19572. * corresponding authors

Other Publications

1. Khosla S*, Sharma G and Yaseen I (2016). Learning epigenetic regulation from mycobacteria. ***Microbial Cell*** (*in press*).

* corresponding author

LABORATORY OF MOLECULAR CELL BIOLOGY

Signal transduction pathways in macrophages and host-pathogen interaction in tuberculosis

Faculty	Sangita Mukhopadhyay	Staff Scientist - VI
PhD Students	Atul Udgata	Senior Research Fellow (till July, 2015)
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	Vishwanath Jha	Senior Research Fellow
	Komal Dolasia	Junior Research Fellow
	Shruti Srivastava	Junior Research Fellow
	KM Rohini	Junior Research Fellow
	Ravi Pal	Junior Research Fellow (from July, 2015)
Other Members	R. Nagender Rao	DST-SERB Project Investigator (from May 2015)
	Niteen Pathak	Senior Technical Officer
	Philip Abraham	ICMR Research Associate
	Nidhi Srivastava	DBT Research Associate (till December, 2015)
	Khalid Hussain Bhat	Research Associate (till June, 2015)
	Rahila Qureshi	ICMR Senior Research Fellow (from June, 2015)
	Susiharan G.S.	Project Assistant (till August, 2015)
	Faiza Nazar	Project Assistant (from November, 2015)
Collaborators	Prof. Seyed E Hasnain	Indian Institute of Technology, Delhi
	Dr. G. Narahari Sastry	IICT, Hyderabad
	Dr. Sudip Ghosh	NIN, Hyderabad
	Dr. Sanjeeva Srivastava	Indian Institute of Technology, Bombay

Objectives

Signal transduction pathways in macrophages regulating its innate-effector functions and how various candidate proteins of *Mycobacterium tuberculosis* (Mtb) interfere with macrophage signaling cascades to modulate host's protective responses against the bacilli.

Project I: Studying the TLR2 signaling pathways responsible for induction of anti- and pro-inflammatory responses in tuberculosis.

Summary of work done until the beginning of this reporting year (upto March 31, 2015)

Previous work carried out by us revealed that two PPE proteins of *Mycobacterium tuberculosis,* PPE17 and PPE18 bind to TLR2 and while interaction of PPE17 with TLR2 LRR domain 16~20 induces TNF-α and pro-inflammatory-type responses, binding of PPE18 with TLR2 LRR domain 11~15 results in generation of IL-10 and anti-inflammatory immune responses (*Nair et al.[2011], J Immunol, 186:5413; Bhat et al. [2012], J Biol Chem, 287:16930*). We demonstrated that PPE17 protein of *Mycobacterium tuberculosis* induced TLR1/2 heterodimerization, whereas PPE18 caused homodimerization of TLR2. We observed differential redistribution of IRAK3, an inactive member of the IRAK family to the cytosol during interaction of PPE17 with TLR1/2 versus PPE18 with TLR2/2, a process that is susceptible to Leptomycin B treatment. TLR1-associated signaling was indispensable for nuclear export of IRAK3 and induction of pro-inflammatory cascades in PPE17-treated macrophages as silencing of TLR1 inhibited IRAK3 export and TNF-α cytokine production upon PPE17 treatment.

Details of progress made in the current reporting year (April 1, 2015 - March 31, 2016)

a. IRAK3 regulates MAPK activity and pro-inflammatory signaling in PPE17-treated macrophages via MKP-1 (Mitogen-activated protein kinase phosphatase 1): ERK1/2 and p38MAPK have been implicated in regulation of cytokine production in response

to TLR2-triggered signaling, with ERK1/2 being responsible for TNF-α induction and p38MAPK for IL-10 production. We had observed earlier that PPE18 strongly activated p38MAPK (but not ERK1/2) that was necessary for the activation of IL-10. Since PPE17 was found to activate predominantly the pro-inflammatory cytokine like TNF-α, we expected higher activation of ERK1/2 in PPE17-treated macrophages as compared to PPE18-treated macrophages. Our data indicated that indeed the level of p38MAPK phosphorylation was lower, but ERK1/2 phosphorylation level was higher in PPE17-treated macrophages when compared with PPE18-treated macrophages. When cells were treated with PD98059, an inhibitor of ERK1/2 activity, TNF-α production in PPE17-treated macrophages was found to be inhibited. This result confirmed that TNF-α induction by PPE17 was dependent upon ERK1/2 activity. Interestingly, Leptomycin B (LMB), that prevented nuclear export of IRAK3 to the cytosol in PPE17-treated macrophages could inhibit phosphorylation of ERK1/2 and induction of TNF-α and enhance the level of phosphorylated p38MAPK in these cells and thereby able to mimic the PPE18-phenotype. These data indicate that the export of nuclear IRAK3 to the cytoplasm is necessary for inhibition of p38MAPK activation with simultaneous activation of ERK1/2 and TNF-α cytokine in PPE17-treated macrophages.

The MKP-1 is known to dephosphorylate MAPK. Evidence suggests that MKP-1 can suppress p38MAPK activation but does not affect ERK1/2 or JNK activation. Since we observed a reduction in p38MAPK activity in PPE17-treated macrophages, we examined the levels of MKP-1 in these cells and found that MKP-1 level was higher as compared to that of untreated or recombinant PPE18 (rPPE18)-treated macrophages. Interestingly, the mRNA levels of MKP-1 did not differ significantly in all the 3 groups examined (untreated, PPE17- and PPE18-treated macrophages), thus, the observed reduction in the protein levels of MKP-1 could be attributed to decreased stability of the protein in untreated and PPE18-treated macrophages. MKP-1 is known to be a labile protein and undergoes rapid turnover through proteasome mediated degradation. We therefore, next pre-treated cells with MG132, a proteasome inhibitor followed by incubation with medium alone or rPPE18 protein. MG132 was found to increase the levels of MKP-1 in both medium-treated and rPPE18-treated macrophages. We then examined if IRAK3-export mechanism was essential for MKP-1 stability in PPE17-treated macrophages. It was observed that the levels of MKP-1 decreased when LMB was used to inhibit export of nuclear IRAK3 in these macrophages. To confirm whether presence of cytosolic IRAK3 is truly important for stabilization of MKP-1, we next silenced IRAK3 expression in THP-1 macrophages using IRAK3-specific siRNA and MKP-1 levels were examined after treatment with rPPE17. It was observed that the levels of MKP-1 were poorer in THP-1 macrophages transfected with IRAK3-specific siRNA as compared to the MKP-1 levels in macrophages transfected with scrambled siRNA. These results together indicated that the export of IRAK3 to the cytoplasm in PPE17-treated macrophages was necessary to maintain MKP-1 stability resulting in reduced phosphorylation of p38MAPK with simultaneous up-regulation of phospho-ERK1/2 and TNF-α levels. The siRNA-based experiment confirms that MKP-1 has a pivotal role in influencing the MAPK pathway and TNF-α induction downstream of PPE17-induced signaling events. Our study thus indicated that PPE17 treatment led to higher export of nuclear IRAK3 to the cytoplasm resulting in increased activation of ERK1/2 and stabilization of MKP-1 which was responsible for decreased phospho-p38MAPK level. As PPE18 fails to trigger significant IRAK3 export from the nucleus to the cytosol, MKP-1 undergoes rapid degradation by the proteasomal machinery and an increased p38MAPK activity is observed in such situation resulting in poorer ERK1/2 activity.

b. IRAK3 is a target of PKCε: Since phosphorylation is often implicated in shuttling of proteins between various compartments of the cell, we speculated that IRAK3 would probably be phosphorylated during its translocation from the nucleus to the cytosol in PPE17-treated cells. *In silico* analyses of the polypeptide sequence of IRAK3 using NetPhosK and GPS revealed that IRAK3 contained four possible phosphorylation sites for PKC isoform, PKCε. PKCε is a member of the PKC family of kinases that has diverse roles in the cellular physiology and is recruited to the TLR signaling pathways *via* the MyD88 adaptor protein. We, therefore, questioned whether PKCε had a direct role in the phosphorylation and export of IRAK3 from the nucleus to the cytoplasm. In order to facilitate phosphorylation and nuclear export of IRAK3, PKCε should be localized to the nucleus. Interestingly, we found

presence of one putative NLS (^{319}RRKK^{322}) motif. To prove the fact that the nuclear translocation of PKCε was truly dependent on the NLS, we next mutated this putative NLS ^{319}RRKK^{322} motif to ^{319}GGAA^{322} and examined the localization of PKCε in THP-1 macrophages. Upon treatment of THP-1 macrophages with rPPE17 although the WT-PKCε (3X-FLAG-WT-PKCε) was able to translocate to the nucleus, the NLS mutant showed reduced nuclear translocation (Fig. 1A). Next, we speculated that PKCε translocated to the nucleus to phosphorylate nuclear IRAK3. To prove this, we co-expressed GFP-tagged IRAK3 along with WT-PKCε or Mut-PKCε [where the Lysine residue in its substrate binding domain was replaced by a Tryptophan which makes it unable to bind and phosphorylate its substrates] in HEK293 cells [both IRAK3 and PKCε are absent in HEK293 cells] and found that though both the WT-PKCε and Mut-PKCε were localized to the nucleus (Fig. 1B), nuclear IRAK3 could translocate to the cytoplasm only in cells overexpressing WT-PKCε but not Mut-PKCε (Fig. 1C). This indicated that the kinase activity of PKCε was probably essential for the nuclear export of IRAK3. We next tested if IRAK3 was truly phosphorylated by WT-PKCε in this experimental set up. When IRAK3 was pulled-down using anti-GFP Ab and subsequently probed with anti-phosphoserine Ab, we observed a prominent phosphoserine signal in IRAK3 that was co-expressed with WT-PKCε (Fig. 1D). The MS and MS/MS analysis data from TAPLIN Mass spectrometry facility (Harvard, USA) indicated that IRAK3 was phosphorylated at Ser^{110} site by PKCε. Thus, PKCε functions as an important point of signal regulation facilitating phosphorylation and translocation of IRAK3 from the nucleus to the cytosol which was important for activation of ERK1/2, stabilization of MKP-1 with concomitant downregulation of phospho-p38MAPK. Thus MAPK activity in PPE17-treated macrophages was probably influenced upstream by PKCε. In order to prove this, we next silenced PKCε expression using a shRNA construct in THP-1 macrophages (Fig. 1E). When treated with PPE17, knock-down of PKCε led to a decreased export of IRAK3 from the nucleus to the cytosol (Fig. 1F) with a concomitant reduction in phospho-ERK1/2 but increase in phospho-p38MAPK levels when compared with the control cells that received the backbone vector (Fig. 1G). These results indicate that PKCε plays a crucial role in regulating nuclear export of IRAK3 and MAPK activity downstream of TLR2 in PPE17-treated cells.

To understand how PKCε was translocated to the nucleus in PPE17-treated macrophages, we next examined the upstream signaling pathways. We observed that after the engagement of TLR2 with its ligand, adaptor molecules such as MyD88, IRAK-1, IRAK-4, and TRAF-6 were recruited at the cytosolic domain or TIR domain of the receptor. Once PPE17 interacted with the TLR1/2 heterodimer, more MyD88 and PKCε were recruited to the receptor complex and this probably allowed interaction of PKCε with IRAK1 (Fig. 1H). PKCε then translocated to the nucleus which was dependent on the IRAK1 kinase activity since pharmacological inhibitor of IRAK1/4 activity significantly abrogated nuclear translocation of PKCε. Thus, PKCε appears to be a target of IRAK1 and the close proximity of the two molecules is probably facilitated by MyD88.

Future study

We plan to design small molecule inhibitors targeting the TLR2 11~15 LRR domain to specifically inhibit anti-inflammatory signaling (known to favor *M. tuberculosis* infection) as novel therapeutics against tuberculosis.

Project II: Role of PE11 of *M. tuberculosis* in cell wall remodeling and virulence

Details of progress made in the current reporting year (April 1, 2015 - March 31, 2016)

Lipid metabolism plays an important role for the mycobacteria to survive in nutrient limited intracellular conditions and for maintenance of its lipid rich cell wall. The characteristic lipid-rich cell wall is a defining feature of Mycobacterium species. The cell wall components affect diverse *mycobacterial* phenotypes including colony morphology, biofilm formation, antibiotic resistance, and virulence. Mtb lipases/esterases play crucial roles in lipid metabolism to hydrolyse lipids and release fatty acids. The fatty acids act as precursors for the cell wall lipids and provide energy for intracellular persistence of the bacilli. Thus, it is important to study the lipases and lipid metabolism to get an insight of the molecular basis of pathogenicity of Mtb.

In silico analyses identified the presence of around 24 putative genes encoding lipolytic enzymes, including 24 lipid/ester hydrolases belonging to the so-called “Lip” family (LipC to LipZ). These have been annotated as putative

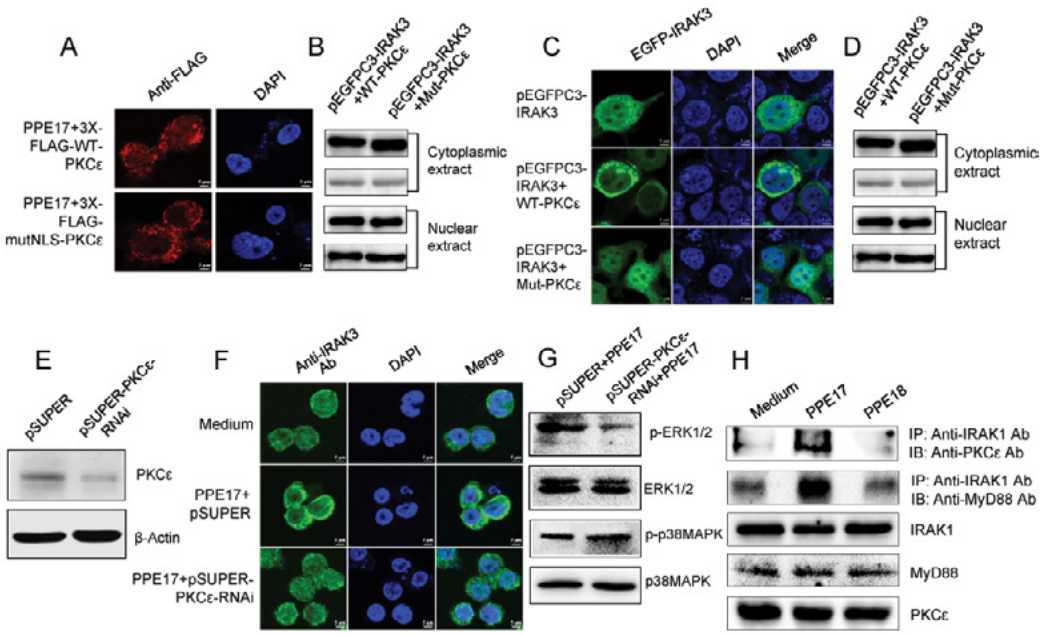

Figure 1. PKC-mediated phosphorylation is required for transport of IRAK3 from the nucleus into the cytosol. (A)THP-1 macrophages were transfected with either 3X-FLAG-WT-PKCε or 3X-FLAG-mutNLS-PKCε and were treated with 3 μg/ml of PPE17 protein for 30 min. Cells were fixed, permeabilized and stained with anti-FLAG Ab followed by anti-mouse Alexa Fluor 594 Ab. Data shown are representative of 3 independent experiments. (B) HEK293 cells were transiently co-transfected with either pcDNA-6xHis-WT-PKCε or pcDNA-6xHis-Mut-PKCε along with pEGFPC3-IRAK3. After 24 h, cells were harvested to check the level of PKCε in the cytoplasmic and nuclear extracts using anti-PKCε Ab and equal sample loading was confirmed by probing the blots with anti-β-Tubulin Ab for cytoplasmic extract and anti-Lamin B Ab for nuclear extracts. Results shown are representative of 3 independent experiments. (C-D) HEK293 cells transiently co-transfected with either pcDNA-6xHis-PKCε -WT or pcDNA-6xHis-PKCε-Mut along with pEGFPC3-IRAK3 were either analysed for IRAK3 localization by confocal microscopy (C) or lysed to check the phosphorylated IRAK3 levels using anti-GFP Ab for immunoprecipitation (IP) and anti-phosphoSerine (anti-pSerine) Ab for immunoblotting (IB) (D). About 10% of the lysates were loaded as input controls. (E-G) PMA-differentiated THP-1 macrophages were transfected with either pSUPER or pSUPER-PKCε-RNAi and at 24 h post transfection, cells were either lysed and immunoblotted with anti-PKCε Ab (E) or treated with 3 μg/ml of PPE17 for 30 min and cells were either used for confocal study for localization of IRAK3 (F) or lysed and immunoblotted to check the levels of phosphorylated ERK1/2 (p-ERK1/2) and total ERK1/2 or phosphorylated p38MAPK (p-p38MAPK) and total p38MAPK (G) in the extracts. (H) Also, THP-1 macrophages were treated with 3 μg/ml of PPE17 or PPE18 protein for 30 min. Cells were lysed and immunoprecipitated using anti-IRAK1 Ab and immunoblotted with either anti-MyD88 Ab or anti-PKCε Ab. About 10% of the lysates were loaded as input controls. Results shown are representative of three independent experiments.

esterases or lipases based on the presence of the consensus sequence GXSXG, which is a characteristic feature of members of the α/β hydrolase-fold family. One of these lipases, the LipX (also known as PE11; Rv1169c) was found to be up-regulated during starvation and palmitic acid stress conditions and infection in macrophages. Rv1169c belongs to the PE family of genes, specific to pathogenic strains like Mtb, *M. bovis* and clinical strain *CDC1551* but absent in non-pathogenic bacteria, *M. smegmatis*. Upregulation of Rv1169c in human lung granulomas and induction of B-cell response against Rv1169c in TB patients indicates that the protein is probably expressed during active TB infection and has important function *in vivo*. Interestingly, Mtb deficient in PE11 failed to grow *in vitro* indicating that the protein is essential for *in vitro* growth of the bacilli and provide clues that PE11 is probably an essential protein for Mtb growth although the detail mechanisms are not well studied.

When we expressed PE11 (Rv1169c) in M. smegmatis (PE11 is absent in *M. smegmatis*), the Scanning Electron Microscopy (SEM) data indicate that *Msmeg-Rv1169c* cells were significantly wider in diameter as compared to *Msmeg-pVV* cells. Next we examined, whether expression of Rv1169c would alter surface architecture of *M. smegmatis* using Transmission Electron Microscopy (TEM). The analysis showed a poor contrast and hyperstaining of *Msmeg-Rv1169c* compared to *Msmeg-p*VV16 bacteria.

This suggests that expression of Rv1169c in *M. smegmatis* probably alters cell wall architecture. Also, when *Msmeg-pVV16* and *Msmeg-Rv1169c* were grown on Middlebrook 7H10 agar plates containing 0.5% glycerol, 10% OADC, and 0.05% Tween 80 and incubated for 5-6 days at 37oC, we observed a distinct colony morphology in PE11 positive transformants. While the colonies of *Msmeg-pVV16* were usual irregular wrinkled acne-like structures, those of *Msmeg-Rv1169c* were found to be rounded, shiny and smooth. Further, the control colonies were dry and fragile but *Msmeg-Rv1169c* colonies were wetter and stickier. Since, Rv1169c was predicted to be a putative lipase/esterase like protein, our observations are suggestive of a role of PE11 in changing the cell wall components of *M. smegmatis.* We next characterized the enzyme activity of PE11 using esters of p-nitrophenyl (pNP), p-nitrophenylacetate (C2), p-nitrophenylbutyrate(C4),p-nitrophenyloctanoate (C8), p-nitrophenyldocanoate (C12), p-nitrophenylmyristate(C14)p-nitrophenylpalmitate (C16) and p-nitrophenylstearate (C18) as substrates and the pNP ester para-nitrophenyl-acetate containing the shortest carbon chain (C2) was found to be most efficiently hydrolyzed indicating PE11 protein is acting predominantly as an esterase rather than lipase. The turbidimetric esterase assay using a Tween 20 and Tween 80 as its substrates further confirmed the esterase activity of PE11. We found that *M. smegmatis* expressing PE11 was able to form profuse pellicles as compared to the control cells (*Msmeg-pVV*). Similarly, PE11 was found to increase the cell surface hydrophobicity causing an increased tendency of *Msmeg-PE11* to form cellular aggregates possibly due to an increase in the glycopeptidolipid content in the cell wall. We further found that *Msmeg-PE11* is more resistant to various environments stressors like SDS, lysozyme, H_2O_2, and low pH (5.5) those mimicking the hostile macrophages environments encountered by the bacilli during infection as well as against antibiotics like ethambutol, rifampicin, isoniazid, ampicillin and vancomycin. Interestingly, when we quantified the cell wall fatty acids as methyl esters (FAMEs) using a high throughput gas chromatography coupled with mass spectrometry (GC/MS), we found a similar fatty acid composition in both the strains, except an increased abundance of polar FAMEs in *Msmeg-PE11*. Mycobacterial lipids contain appreciable amounts of myristic (C14), palimitic acid (C16), and stearic (C18) and C16 - C24 monoenoic fatty acids. We found that overexpression of PE11 caused a noticeable decrease in the amount of linear C18:0 polar fatty acids, along with an increase in the branched chain polar fatty acid content (C18:10-methyl) which may increase the membrane fluidity and the ability of *Msmeg-PE11* to tolerate environmental stress. Mice infected with *Msmeg-PE11* had higher bacterial load, exacerbated organ pathology, weight loss, morbidity and mortality, indicating a potential role of this protein in mycobacterial virulence. Thus, our data suggest that PE11 is actively involved in the cell wall remodeling that may confer increased drug resistance and survival advantages to the mycobacteria inside host.

Future study

We intend to study in detail the mechanisms by which PE11 supports intracellular survival of the bacilli.

Publications

1. Singh P, Rao RN, Reddy JR, Prasad R, Kotturu SK, Ghosh S and Mukhopadhyay S (2016). PE11, a PE/PPE family protein of *Mycobacterium tuberculosis* is involved in cell wall remodeling and virulence. ***Scientific Reports*** 6: 21624.

2. Abraham PR, Udgata A, Latha GS and Mukhopadhyay S (2016). The *Mycobacterium tuberculosis* PPE protein Rv1168c induces stronger B cell response than Rv0256c in active TB patients. ***Infection, Genetics and Evolution***. 40: 339-345.

3. Ahmed A, Das A and Mukhopadhyay S (2015). Immunoregulatory functions and expression patterns of PE/PPE family members: Roles in pathogenicity and impact on anti-tuberculosis vaccine and drug design. ***IUBMB Life*** 67: 414-427.

4. Hussain BK and Mukhopadhyay S (2015). Macrophage takeover and the host-bacilli interplay during tuberculosis. ***Future Microbiology*** 10: 853-872.

Patent filed

1. Sangita Mukhopadhyay and Asma Ahmed. A novel therapeutic for treatment of sepsis. Indian Patent Application No. 201641002980. Date of filing - January 27, 2016.

LABORATORY OF MOLECULAR GENETICS

(Explanatory note: The Laboratory of Molecular Genetics, which also includes the Centre of Excellence (CoE) in Silkmoth Genetics and Genomics, was headed by CDFD's faculty member Dr. J Nagaraju who unfortunately passed away in December 2012. Subsequently, the activities of his erstwhile group have been continued by Dr K P Arun Kumar and Dr V V Satyavathi with their respective colleagues, whose individual reports are given below. The Director of CDFD is the designated coordinator of the CoE).

Centre of Excellence (CoE) for Genetics and Genomics of Silkmoths

Faculty	KP Arun Kumar	Scientist
PhD Students	Asha Minz	Senior Research Fellow (till Sep 2015)
	S Suresh Kumar	Senior Research Fellow
	G Gopinath	Senior Research Fellow
	Ch. Gangi Reddy	Junior Research Fellow
Other Members	S Annapurna Bhavani	Technical Officer
	R Lakshmi Vaishna	Technical Assistant
	Rajendra Chilukuri	Project Associate
	Sasi Bhushan S	Project Associate (till Sep 2015)
	Saikat Chakraborty	Project JRF
	Vidya T	Project JRF
	Srikakolapu Sekhar	Project JRF (till Dec 2015)

Objectives

1. Studies on the role of CCCH type zinc finger gene in Bombyx mori sex determination.
2. Role of Drosophila Noduler protein in immune response.

The progress made in the projects related to sex determination and immune response in *Bombyx mori* and *Drosophila melanogaster* respectively is reported here.

Summary of the work done until the beginning of this reporting year (upto March 31, 2015)

- Comprehensive analysis of gene expression in different embryonic stages in silkworm reveals that the onset of dosage compensation occurs at about 96h, which probably coincides with the initiation of sex specific splicing of sex determining gene doublesex, and prevails throughout. Analyses of sexed head RNA-seq data confirm the existence of complete sex chromosomal dosage compensation in B. mori.
- Studies on evolutionary dynamics of *B. mori* Z chromosome, in relation to autosomes and sex chromosomes of other animal species, indicated a strong faster-Z effect for female-biased genes, an intermediate faster-Z effect for unbiased genes, and no faster-Z effect for male-biased genes.

Details of progress made in the current reporting year (April 1, 2015 - March 31, 2016)

Objective 1: Studies on the role of (CCH) type zinc finger gene in *Bombyx mori* sex determination

Sex determination is a fundamental biological process that determines two distinct sexes. A variety of sex determination mechanisms is observed in animal species, most of which follow the chromosomal/genetic sex determination, except in some the sex is determined by environmental factors like temperature (e.g. crocodiles, alligators and few lizards). Among insects, the mechanism of sex determination is well understood in *Drosophila* and serves as a reference for all insects. In *Drosophila,* (XX is female and XY is male) the sex is determined by the dose of X-linked signalling elements (XSE) (XSE are four transcription factors *Scute, SisA, Runt and Unpaired*), which in turn is determined by the number of X chromosomes. XSE, whose expression threshold can be reached only in female embryos, confine the production of the Sex-lethal (SXL) protein to females. Thus

produced SXL directs the female specific splicing of pre-mRNA of transformer (tra¬) gene resulting in functional TRA protein. The TRA interacts with non sex-specific transformer 2 (TRA2) protein and this complex binds to the *doublesex* repeat element (d*sx*RE) in the middle of fourth exon and forces the female specific splicing of *doublesex* (*dsx*) mRNA, producing the female DSX protein. These two proteins have been shown to exhibit antagonistic functions in the process of sexual differentiation. In a few insect species like *Megaselia scalaris*, *Ceratitis capitata, Bactotocera tryoni, Lucilia cuprina and Chironomus thummi,* an epigenetic male factor from the Y chromosome decides the male development. *Culex tritaeniorhynchus* lacks the sex chromosomes and the maleness is conferred by an autosomal gene. The sex in Aedes aegypti is determined by *Nix* gene from M locus on Y chromosome like region. In hymenopteran species, the sex is maintained through haplodiploidy, where haploids develop as males and diploids develop as females. In *Nasonia vitripennis, transformer* (*Nvtra*) gene plays a crucial role in development of females, where it maintains its concentration by an autoregulatory loop through a maternally supplied TRA protein.

In lepidopterans (butterflies and moths) ZZ/ ZW or ZZ/ZO chromosomal system of sex determination is observed. The heterogametic sex (ZW and ZO) is female and the homogametic sex (ZZ) is male. It has been reported that SXL is not regulated in a sex specific fashion in *B. mori*. The orthologue of *tra* has not been identified so far in *B. mor*i, probably owing to its rapid sequence divergence in the course of evolution. The *dsx* pre-mRNA has been shown to be lacking TRA/TRA-2 binding sites. Though, the orthologues of tra2, intersex (*ix*) and fruitless (*fru*) genes have been identified in *B. mori,* their functions remain elusive. Previous studies have resulted in the identification of two RNA binding splicing inhibitors: 1) *B. mori* homolog of IGF-II mRNA binding protein (BmIMP) and 2) B. mori homolog of P-element somatic inhibitor (BmPSI), which are involved in differential splicing of Bmdsx pre-mRNA. The involvement of *Bmpsi* and *Bmimp* renders this mechanism to be unique from any other class of insects. Recently, the mechanism of *B. mori* sex determination was reported to be governed by a piRNA (*fem*) from the W-chromosome. The W-derived *fem* piRNA negatively regulates a Z-linked CCCH type zinc finger gene, *Masculinizer* (*masc*). *masc* has been shown to regulate the *Bmdsx* sex specific splicing by promoting the expression of male specific *Bmdsxm* type of splicing isoform and also dosage compensation by an unknown mechanism. Thus, this gene, *masc* is presumably non-functional in females, leading to female specific *Bmdsxf* type of splicing isoform. Further studies have shown that the over expression of *masc* gene in BmN cells has enhanced the transcription of *Bmimp* gene and most probably through this the *masc* induces the expression of male specific Bmdsxm type of splicing isoform. Thus the reported studies have shown that the sex in *B. mori* is regulated by a W encoded *fem* piRNA that negatively regulates the *masc* gene in females.

In *B. mori*, studies attempting to discover the genes involved in sex determination pathway have resulted in the identification of a female specific CCCH type znf motif encoding gene, termed as *z1* on W-chromosome and its homologous copies namely z2 and *z3* on 25th chromosome [Unpublished data]. Further, the studies of translocation of W-chromosomal fragments to autosomes have supported the existence of a strong putative epistatic female determining region called, “feminizer” on the W-chromosome. Presumably, a preliminary analysis using FISH has indicated that these *znf* genes are linked to the “feminizer” region of W-chromosome. In the current study we provide functional insights into the role of an autosomal CCCH type *znf* gene, *z2* in the *B. mori* sex determination. For the sake of simplicity and ease of understanding, we refer the gene z2 as *Bmznf-2* (NCBI acc: XP_004924549.1).

In this study, we discovered the role of *Bmznf-2* in the sex specific differential splicing of the *Bmdsx* pre mRNA. We used ovary derived BmN cells, which produce the female type of *Bmdsx* (*Bmdsxf*) splicing isoform, representing their female mode of sexual differentiation. The over-expression of *Bmznf-2* in BmN cells promoted male specific splicing isoform (*Bmdsxm*) and this correspondingly decreased *Bmdsxf* (Fig. 1A and 1B). This shift of splicing phenotype is referred as “masculinisation”. The masculinisation induced by *Bmznf-2* over-expression denotes the “gain of function” of *Bmznf-2* in BmN cells (female cells). This indirectly suggests that *Bmznf-2* may be normally inactive in female cells.

To decipher the role of *Bmznf-2* in promoting differential splicing of *Bmdsx* pre-mRNA, we

conducted RNAi based knockdown of *Bmznf-2* in BmN cells using short dsRNA. The knockdown achieved for *Bmznf-2* gene was 75 to 90%, which is considerably high and presumably enough for interfering the gene activity generally in *Bombyx*. But we found no effect on innately expressing *Bmdsxf* splicing isoform level, which indicates the null activity of *Bmznf-2* in achieving *Bmdsxf* splicing isoform in BmN cells (female).

As mentioned previously, the CDS region of *Bmznf-2* mRNA sequence could be a putative precursor of the ovarian small RNA 12564. In such a case, the over-expression experiments of *Bmznf-2* may also be treated as the over expression of the ovarian small RNA and possibly the observed masculinisation could be either by the putative BmZNF-2 protein or by some kind of gene regulation induced by the ovarian small RNA 12564. Therefore, to test which of the above two factors (BmZNF-2 protein or ovarian small RNA 12564) is actually associated in inducing masculinisation of BmN cells, we performed site directed mutagenesis of the two CCCH motifs of putative BmZNF-2 protein to unravel their role in masculinisation. By keeping the region of the ovarian small RNA 12564 intact, we generated and over-expressed two mutant pIZT constructs in BmN cells, each expressing the mutated BmZNF-2 proteins at its 1) CCCH motif 1 and 2) CCCH motif 2 respectively. The mutations resulted in the replacement of 2nd and 3rd cysteines to serines and the histidine to leucine amino acids in the CCCH motifs, which is previously demonstrated to affect the structure of the znf motif and would seriously compromise the function of CCCH znf protein. The point mutations in either the CCCH motif 1 or CCCH motif 2 has abolished the phenotype of masculinisation (Figure 1C, D), indicating the involvement of putative BmZNF-2 protein and the essentiality of znf motifs in inducing masculinisation of BmN cells. Thus our experiments in BmN cells revealed the association of BmZNF-2 protein in regulating the sex specific differential splicing of Bmdsx, and thus signifying its activity in controlling the processes of sex determination and differentiation (Figure 1A and 1B).

The above experiment suggests the role of BmZNF-2 protein in the alternative splicing of *Bmdsx* and as the mechanism of alternative splicing operates only in the nucleus of cells, we further checked the localisation of BmZNF-2 protein in BmN cells. For this, the *Bmznf-2* CDS in pIZT construct was fused with m-cherry at its C-terminal end and over-expressed in BmN cells. The fluorescent imaging clearly indicated the nuclear localisation of BmZNF-2 and m-cherry fused protein. This study implies its functional activity in the nucleus and its probable involvement (either direct or indirect) in the nuclear process like mRNA splicing.

Objective 2: Role of *Drosophila* Noduler protein in immune response

To combat infection, *Drosophila* relies on multiple innate defense reactions, which can be divided into two major categories namely cellular immune response and humoral immune response. Cellular immune response mechanisms including encapsulation, melanization and phagocytosis act as the first line of defense (Lemaitre *et al.*, 2007). Immune cells like haemocytes are involved in direct interaction with the pathogen and foreign particles to fight infection. Humoral immune response on the other hand functions by secreting a battery of effector molecules or antimicrobial peptides (AMPs), which are synthesized by fat body cells upon activation of Toll and IMD signalling cascades. Toll pathway gets upregulated by stimulus perceived by the host upon Gram-positive bacterial and fungal infection. Gram-negative bacterial infection channels the elicitation of IMD pathway. These two immune pathways play important role in clearing majority of the bacterial infections (De Gregorio *et al*., 2002). Toll pathway shares its homology with the Toll-like receptors (TLR) and Interleukin-1 receptor (IL-1R) pathways in mammals, and IMD with the Tumor Necrosis-factor receptor (TNFR) pathway. Much is known about the genes involved and mechanisms in which the immune proteins operate in the pathways. However, the factors involved in the nuclear localization and regulation of the NF-κB/Rel transcription factors in immune pathways is still unclear.

Previous studies in our laboratory on wild silkmoth, *Antheraea mylitta* immune transcriptome analysis have identified and characterized a novel immune protein that is up-regulated in hemolymph upon bacterial infection. The functional role of this protein in immune response suggested its involvement in nodule formation and therefore named as Noduler. Noduler was shown to bind a wide range of bacteria, yeast and insect haemocytes specially to the LPS, LTA and β-1,3 glucan components of microbial cell wall.

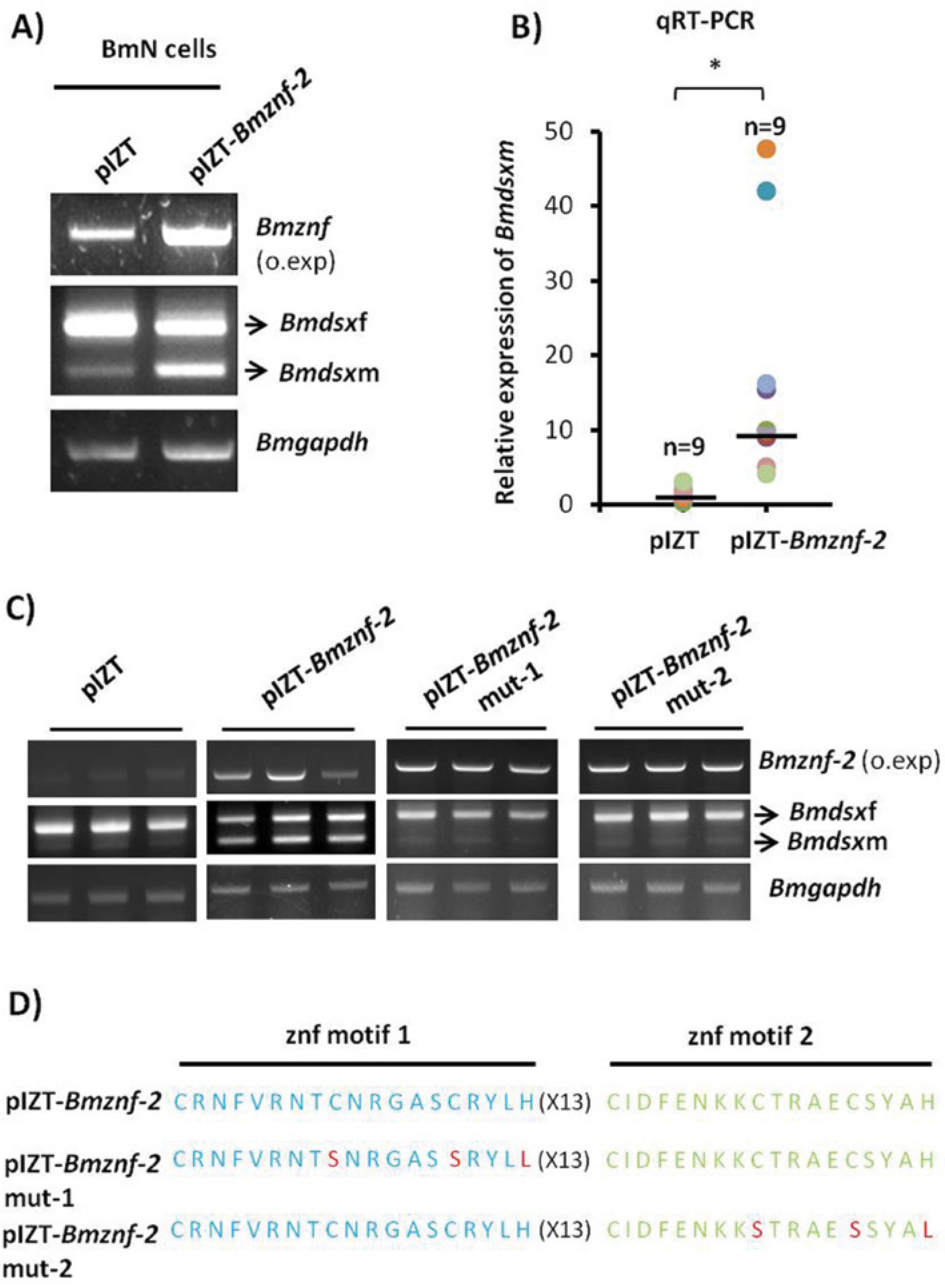

Figure 1: *Bmznf-2* favors the male specific splicing in BmN cells upon over expression. A) Increased expression of *Bmdsxm* splicing isoform (masculinisation) upon *Bmznf-2* transient over expression. B) Relative quantification of *Bmdsxm* splicing isoform, between control (pIZT) and BmZNF-2 induced (pIZT-*Bmznf-2*) samples, using real-time qRT-PCR (* indicates a significant difference, t-test, $p<0.05$). The dark lines represent the median values of the data points. C) Point mutations (two cysteines to serines and one histidine to leucine) in both the CCCH motifs has resulted in the loss of masculinisation phenotype. D) The sequences of the wild type and the mutated znf motifs of the three clones used for transfection assays.

RNA interference mediated knockdown of the noduler resulted in significant reduction in the number of nodules and consequent increase in bacterial load in larval hemolymph. These results suggested that the Noduler is involved in very early clearance of bacteria by forming nodules of haemocytes and bacterial complexes in insects. The RNAi mediated knockdown of *noduler* has also shown reduced phenoloxidase activity.

With this background, we studied the function

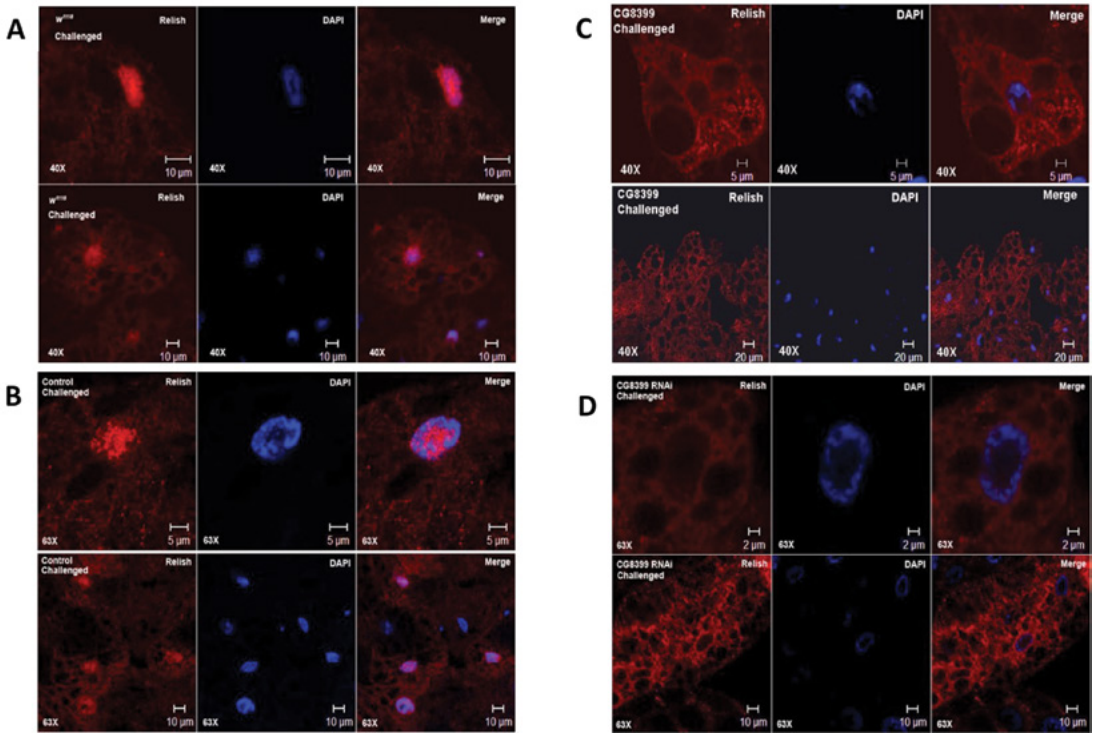

Figure 2. Relish translocates into the nuclei upon bacterial challenge in w^{1118} (**A**) and GFP expressing control (*hml-GAL4, UAS-GFP*) (**B**), whereas in Noduler mutants - CG8399 (**C**) and CG8399 RNAi (*hml-GAL4, UAS-GFP X UAS-CG8399*) (**D**) Relish fails to translocate into the nucleus. Fat bodies of third instar larvae were stained with Relish antibody (Red) two hours post *E. coli* infection. DAPI (Blue) was used to stain the nucleus. Merged images (right) are shown.

of *DmNoduler* gene (a Drosophila homolog of *Noduler* - also known as putative ferric-chelate reductase 1 homolog - DmSDR2) in immune response of *D. melanogaster*. The gene expression studies and survival assay revealed that the level of *DmNoduler* was affected by both kinds of bacterial infections, namely Gram-positive and Gram-negative. This gave us a hint of its participation in both the immune pathways and led us to explore its position in those pathways. An attempt was made to examine the association of this gene in immune response pathway by carrying out next generation sequencing (NGS) based transcriptome analysis to analyze the expression of genes that were significantly affected in the *DmNoduler* mutant flies. NGS analysis revealed that a number of antimicrobial peptides were down-regulated in infected mutant flies whereas the upstream genes in both Toll and IMD pathways were unaffected. The immunofluorescence analysis revealed *DmNoduler* to be participating at the level of NF-κB/Rel transcription factor by affecting their nuclear translocation. Here, we provide evidence for the first time that NF-κB factors Relish and Dorsal are translocated into nucleus with the aid of *DmNoduler* (Figure 2). Therefore, in the quest of addressing the immunological function of *DmNoduler* we have deciphered its vital role as a regulator of NF-κB/Rel transcription factors in both the immune pathways of *Drosophila.* With this study, we introduce a new factor to immune response cascades, which is unique as it regulates both pathways by affecting translocation of NF-κB factors.

B. Report of Dr VV Satyavathi's group

Members	VV Satyavathi	Technical Officer
	RM Pavani	Project-Junior Research Fellow (Till Jan. 2016)
	K Swetha Kumari	Project-Junior Research Fellow (Till Dec. 2015)
	K Lakshmi Prasanna	DBT-Junior Research Fellow
	Sunil Nahata	Project-Junior Research Fellow (Since Dec. 2015)
	Bajaj Deepti Madanlal	Project-Junior Research Fellow (Since Mar. 2016)
	H K Basavaraja	Breeder Consultant CoE
Collaborators	PJ Raju	APSSRDI, Hindupur
	V Sivaprasad	CSR&TI, Mysore
	Kanika Trivedy	CSR&TI, Berhampore
	Satya Prakash Sharma	CSR&TI, Pampore
	KI Basha	APSSRDI, Hindupur
	H Lakshmi	APSSRDI, Hindupur

Objectives

1. Introduction of anti-baculoviral property from transgenic silkworms to commercial silkworm strains and to conduct multilocational field trials to establish their efficacy and generate data for their regulatory approval;
2. Characterization of *Bombyx mori* nucleopolyhedrosis virus (BmNPV) resistant transgenic silkworm strains;
3. Development of baculovirus resistant silkworm strains using marker assisted selection; and
4. Identification and functional characterization of novel genes involved in immune response pathways of silkmoths.

Summary of the work done until the beginning of this reporting year (upto March 31, 2015)

The work undertaken in earlier years on each of the objectives has been summarized in the first parts of the corresponding descriptions given below.

Details of progress made in the current reporting year (April 1, 2015 - March 31, 2016)

Objective 1: Introduction of anti-baculoviral property from transgenic silkworms to commercial silkworm strains followed by multilocation contained trials

In phase I of this CoE project, transgenic silkworm lines of Nistari, expressing dsRNA for multiple essential baculoviral genes were generated using *piggyBac* transposon-based germline transgenesis. The recombinant vectors used in the study carried a portion of each of the essential baculoviral genes (*ie1, lef1, lef3* and *p74*), either in sense or antisense, or in inverted-repeat arrangement driven by silkworm cytoplasmic actin (*BmActin*) promoter; and a reporter gene encoding red fluorescent protein (*dsRed*) driven by 3XP3 promoter. The transgenic silkworms carrying the inverted repeat containing transgene showed stable protection against high doses of baculovirus infection. The anti-viral property of the baculoviral resistant transgenic lines in the Nistari genetic background was transferred to baculovirus susceptible bivoltine silkworm strain, CSR2 through transgene selection coupled with recurrent backcross strategy. For testing the efficacy of transgenic silkworms at multiple locations in India, Review Committee on Genetic Manipulation (RCGM) has permitted CDFD for the conduct of multilocational trials in contained facilities at APSSRDI, Hindupur, Andhra Pradesh and at 3 centres of Central Silk Board (CSR&TI, Mysore; CSR&TI, Berhampore, West Bengal, CSR&TI, Pampore, J&K State).

During the period under report, hybrids were generated by crossing transgenic lines of Nistari and CSR2 with various commercial local silkworm breeds. The transgenic and control lines (as per the action plan of RCGM) were tested under first trial conducted at three locations. The performance of the hybrids was assessed based on the pupation rate and cocoon traits. Under normal conditions, as expected no difference

was observed in the performance of the control and transgenic hybrids (Figure 1). The transgenic hybrids which indicated their success in inhibiting viral proliferation under laboratory trials will be assessed under multilocational contained conditions upon BmNPV infection.

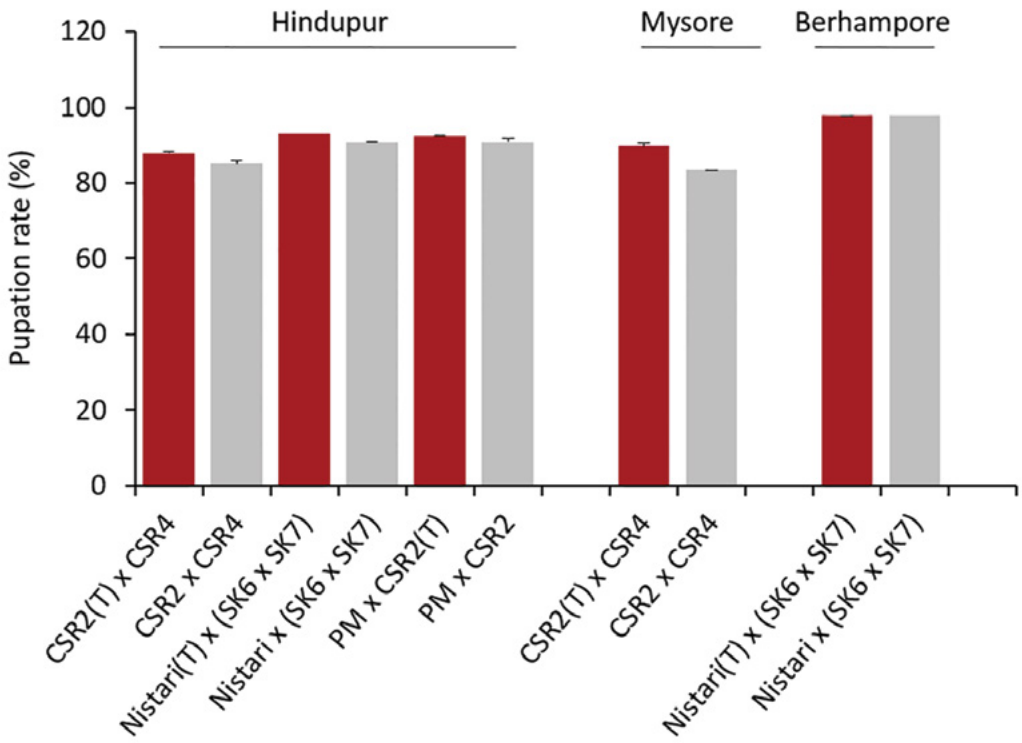

Figure 1. Performance of silkworm hybrids under normal conditions. Silkworm hybrids generated by crossing transgenic CSR2 and Nistari lines with various local commercial multivoltine (Pure Mysore - PM) and bivoltine (CSR4, SK6 x SK7) breeds were tested under 1st trial at three centres. Bars in red and grey represent transgenic and control lines, respectively; Y-axis represents pupation rate (%). Error bars represent the standard deviation.

Objective 2: Characterization and maintenance of transgenic silkworm strains

All the transgenic silkworm lines developed through RNAi approach (donor stock) are being maintained at Andhra Pradesh State Sericulture Research and Development Institute (APSSRDI), Hindupur through generations. In every cycle, the transgenic silkworm lines were monitored for transgene stability, viral load and unique traits of the strain. The batch selection of lines was performed based on visual observation of the larvae and cocoon traits. The inter-batch crossing system was meticulously performed in each cycle to realize the benefit of hybrid vigor of the lines. The transgenic CSR2 lines were advanced to BC4F34 generation. Through recurrent breeding followed by selection techniques, transgenic lines of CSR2 with cocoon weight (1.782g), shell weight (0.382g) and shell percentage (21.4%) on par with control CSR2 lines were obtained.

Objective 3: Development of baculovirus resistant silkworm strains using marker assisted selection

Second generation Illumina sequencing was performed to generate 8 pair-end libraries for the midgut and fat body tissues from baculovirus infected and control larvae of SBNP1 (resistant) and CSR2 (susceptible) strains. Based on bioinformatic pipeline, the transcript abundance was scored in the NPV infected versus control samples and the genes up/down regulated were identified. In the transcriptome analysis, *Serpin 2* is found to express differentially in the SBNP1 and CSR2 strains. Based on biochemical and RNAi assays, we found that *Serpin 2* exhibits antiviral activity and restricts viral spread by inhibiting cleavage of viral structural protein.

Objective 4: Identification and functional characterization of novel genes involved in immune response pathways of silkmoths

In a previous study, we reported functional characterization of a novel immune protein Noduler which binds specific bacterial components and hemocytes leading to nodulation response in the wild silkworm, *Antheraea mylitta*. Several genes that share sequence similarity with Noduler of *A. mylitta* have been reported from *Bombyx mori*, *Drosophila, Hyphantria cunea, Manduca sexta, Samia cynthia ricini, Lonomia obliqua,* including homosapiens. There are three Noduler homologues in *Drosophila*, two in *B. mori*, and two in homosapiens. In *A. mylitta*, Noduler is 168 amino acid (aa) with a characteristic reeler domain. The reeler domain was found to be conserved from flies to mammals. Although Noduler homologues with reeler domain are reported in mammalian system, their function in immune response is not known.

During the period under report, we attempted functional analysis of Noduler homologue, Stromal cell-derived receptor 2 upon infection in mammalian system. THP1 monocytic cell line was used for this study. Activation of macrophages was achieved by bacterial lipopolysaccharide (LPS) treatment that is required for induction of transcription of genes that encode for proinflammatory regulators of the immune response. Based on previous reports, cells were inoculated with LPS at a concentration 100 ng/ml for 2 hrs. The expression profiles of the genes in THP1 cells treated with phorbol-12-myristate-13-acetate (PMA) were observed. We found that SDR2 was up regulated upon LPS treatment. In order to understand its role in mammalian system, CRISPR Cas9 (clustered regularly interspaced short palindromic repeats) genome edititng system was used for knockout of stromal cell-derived receptor 2. The main components of this system, sgRNA and Cas9 nuclease expression clones are as shown in Figure 2. The target sequence for sgRNA synthesis used

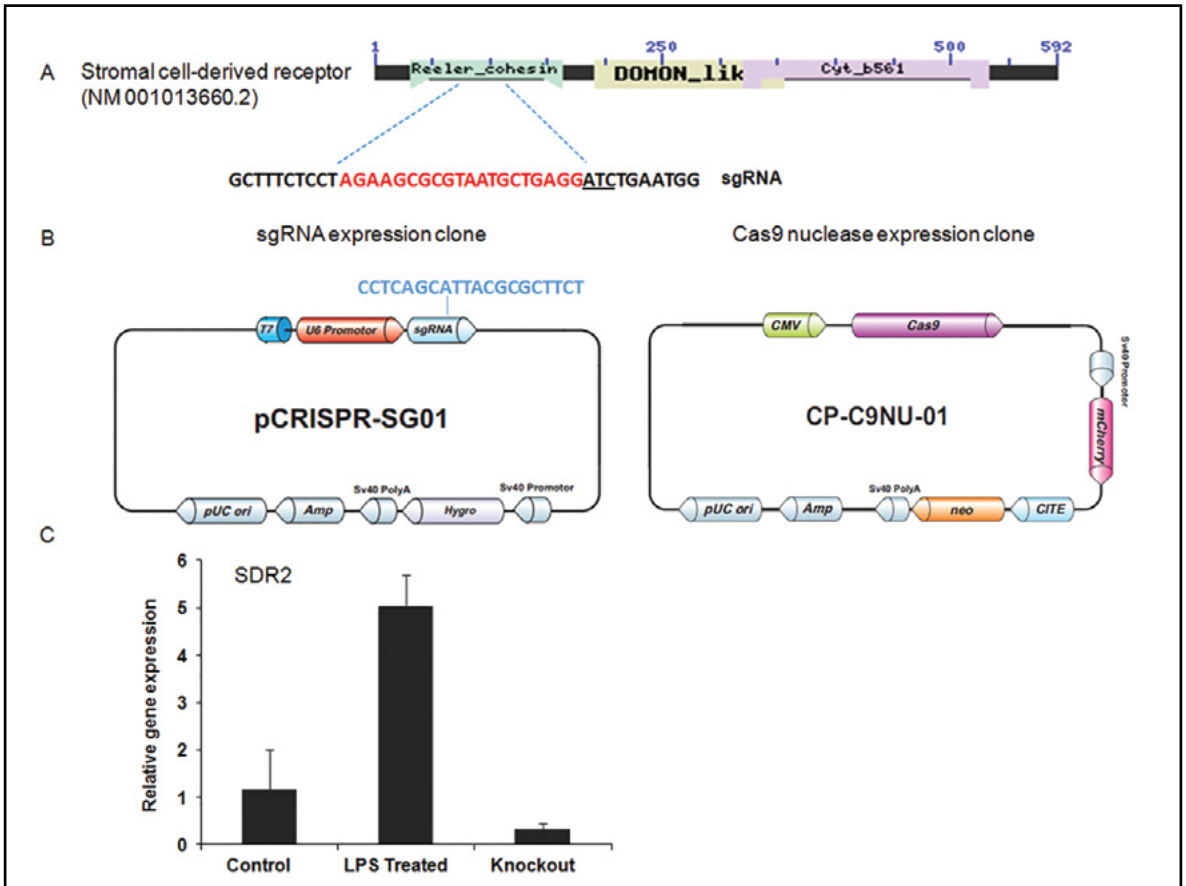

Figure 2. CRISPR/Cas9 system mediated genome editing in THP1 cells. A) Schematic representation of Reeler, Domon and Cytb domains for stromal cell-derived receptor (SDR2) gene with sgRNA target sequence at mRNA, B) Representation of sgRNA and Cas9 nuclease plasmid constructs used in the study, C) QPCR as performed for the indicated gene on RNA isolated from Control, LPS treated (2 hpi) and knocked out THP1 cells. The experiment was done in biological replicates. Error bars represent the standard deviation.

was 5'-CCTCAGCATTACGCGCTTCT-3'. The plasmids pCRISPR-SR01 and CP-C9NU-01 (custom synthesized from Genecopoiea) were cotransfected into THP1 cells and depletion of the target gene was studied by QPCR using gene specific primers. The GAPDH gene was used as a reference. Quantification of target RNA was carried out by ΔΔCT method. Around 5 fold higher level of expression of SRD2 was observed in LPS treated cells as compared to control and knocked-out cells. Future work involves further validation of results by sequencing and expression analyses.

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1. Chen Z, Nohata J, Guo H, Li S, Liu J, Guo Y, Yamamoto K, Kadono-Okuda K, Liu C, Arunkumar KP, Nagaraju J, Zhang Y, Liu S, Labropoulou V, Swevers L, Tsitoura P, Iatrou K, Gopinathan KP, Goldsmith MR, Xia Q and Mita K (2015) A comprehensive analysis of the chorion locus in silkmoth. ***Scientific Reports*** 5: 16424.
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3. Chen Z, Nohata J, Guo H, Li S, Liu J, Guo Y, Yamamoto K, Kadono-Okuda K, Liu C, Arunkumar KP, Nagaraju J, Zhang Y, Liu S, Labropoulou V, Swevers L, Tsitoura P, Iatrou K, Gopinathan KP, Goldsmith MR, Xia Q and Mita K (2015) Construction, complete sequence, and annotation of a BAC contig covering the silkworm chorion locus. ***Scientific Data*** 2: 150062.
4. Gupta AK, Mita K, Arunkumar KP and Nagaraju J (2015) Molecular architecture of silk fibroin of Indian golden silkmoth, *Antheraea assama*. ***Scientific Reports*** 5: 12706.
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6. Satyavathi VV*, Narra D and Nagaraju J (2016). Noduler an immune protein augments infection induced cell proliferation through cross-talking with p38 MAPK. ***Immunobiology*** 221: 387-397.

*Corresponding author

Other publications and Patent(s) filed/granted

1. Arunkumar KP and Sambrani N (2015) Book review of the *Annual Review of Genetics* 2014, Bonnie Bassler et al., (eds) ***Current Science*** 109: 2137-2139.

2. Satyavathi VV and Raju PJ (2016) RNAi may subserve KS-10. Opinion of Experts on KS-10, the inhibitor of diapause breed of silkworm, *Bombyx mori,* L. ***KSSRDI Technical Publications*** No. 123: 79-80.

LABORATORY OF MOLECULAR ONCOLOGY

Genomics and molecular genetics of cancer and genetic disorders

Faculty	Murali Dharan Bashyam	Staff Scientist
PhD Students	Raju Kumar	Senior Research Fellow
	A Srinivas	Senior Research Fellow
	Bala Pratyusha	Senior Research Fellow
	Ashmala Naz	Junior Research Fellow
	Sara Anisa George	Junior Research Fellow
Other Members	Mithu Raychaudhuri	DST Woman Scientist
	A Sita Rama Raju	Project Senior Research Fellow
	Ajay K Chaudhary	Technical Officer
	K Viswakalyan	Research Assistant
	K Padmavathi	Project Assistant
	Arun K Paripati	Project JRF (till July 6, 2015)
	Anupama Shirke	Project Junior Research Fellow (since July 07, 2015)
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	H A Nagarajaram	CDFD, Hyderabad
	Saumyadipta Pyne	IIPH, PHFI, Hyderabad
	G Swarnalata	Apollo Hospitals, Hyderabad
	T Subramanyeshwar Rao	BIACHRI, Hyderabad
	KVVN Raju	BIACHRI, Hyderabad
	Sujit C Patnaik	BIACHRI, Hyderabad
	M Srinivasulu	MNJ Hospital, Hyderabad
	Nagari Bheerappa	NIMS, Hyderabad
	Mohana Vamsy	Omega Hospitals, Hyderabad
	I Satish Rao	KIMS, Hyderabad
	K T Vijaya	Care hospital, Hyderabad
	Prajnya Ranganath	NIMS, Hyderabad
	Girisha KM	Kasturba Hospital, Manipal
	Sankar V Hariharan	Medical College, Trivandrum
	Sumita Danda	CMC, Vellore

Objectives

1. Identification and characterization of important deregulated genes / pathways in cancers prevalent in India; and
2. Identification and characterization of disease causing mutations in genetic disorders.

Summary of work done until the beginning of this reporting year (upto March 31, 2015)

Pancreatic Cancer (PaCa): Our previous studies revealed frequent deletion of *PAR6G*, encoding a poorly studied isoform of PAR6A that forms part of the PAR complex, in PaCa cell lines and xenografts. Similarly, *ARID1B*, encoding a SWI/SNF complex component, was shown to exhibit bi-allelic loss in MiaPaCa2 PaCa cells and single copy loss in several other PaCa cell lines. Further, evaluation of promoter methylation andexpression status in tumor samples andectopic expression in cell lines suggested a tumor suppressor role for *ARID1B* in PaCa.

Colorectal Cancer (CRC): Computational analysis of transcriptome data generated separately from Wnt- and Wnt+ rectal cancer samples revealed several differentially expressed 'gene sets'. We further extracted a differentially expressed 12 gene signature; the constituent genes were

validated in independent set of samples.

Genetic disorders: We analysed 48 Hypohidrotic ectodermal dysplasia families; mutation was detected in 40 (Ectodysplasin A1(*EDA-A1*) in 23 families, *EDAR* in 16 and ectodysplasin A receptor-associated death domain (*EDARADD*) in 1). These included one novel large ~23 Kb deletion in *EDA-A1* and the first splice site mutation ever reported in *EDARADD.*

Details of progress made in the current reporting year (April 1, 2015 - March 31, 2016)

PaCa: PAR6G was shown to co-localize with PAR3 in cell membrane of HEK293T cells (Fig. 1A). PAR6G (and not PAR6A) ectopic expression resulted in significant reduction in cell motility (as compared to vector alone) when tested in wound healing assays (Fig. 1B). In addition, PAR6G interacted with aPKC and PAR3 (Fig.1C)

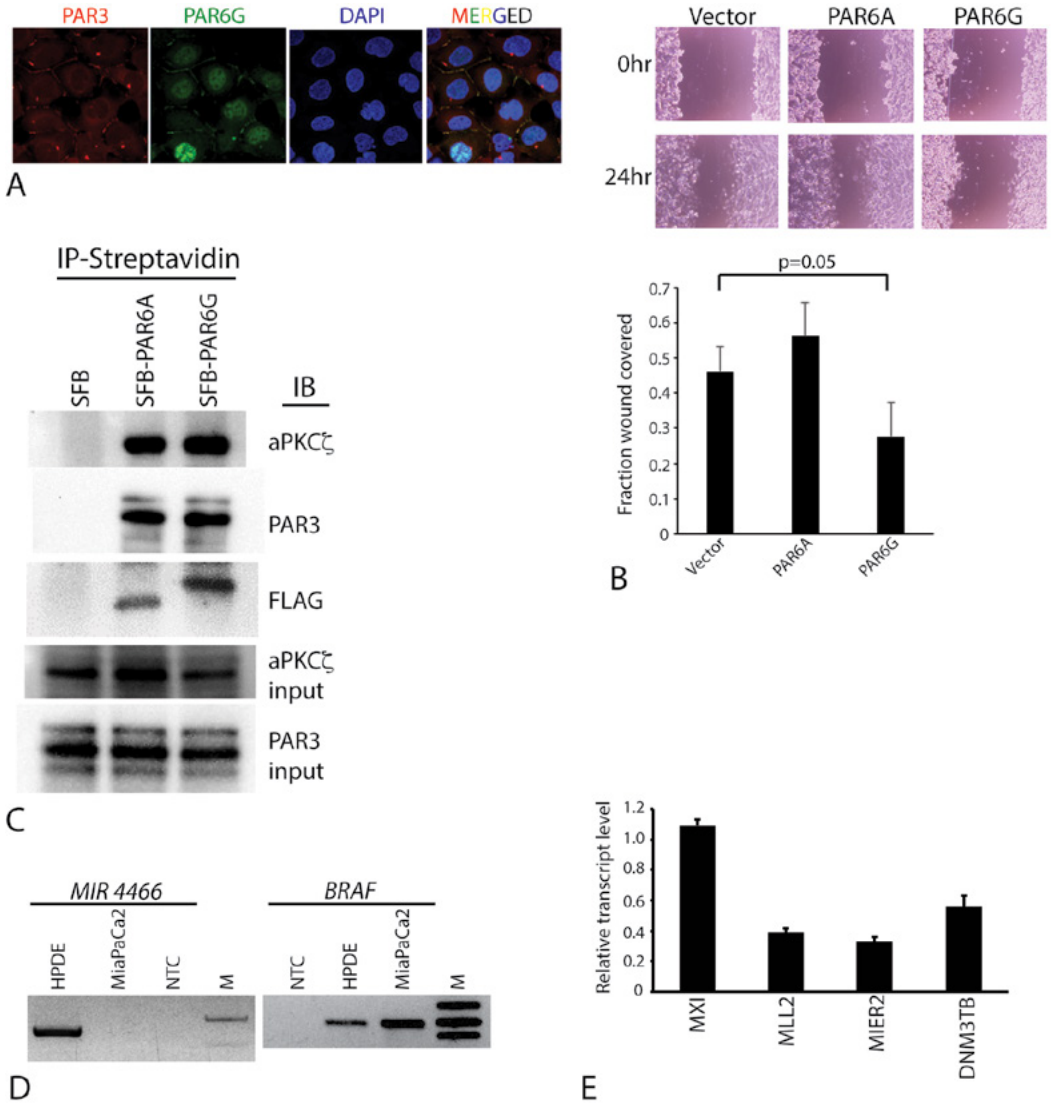

Figure 1. Studies on PAR6G (panels A-C) and novel miRNA *MIR4466* (panels D-E). Panel A, immunofluorescence staining confirms co-localization of PAR6G and PAR3 (an authentic component of the PAR complex). Panel B, PAR6G ectopic expression compromises cell motility in MCF7 cells. Panel C, immunoprecipitation followed by immunoblotting confirms PAR6G interaction with several PAR complex components. SFB; triple tag including S-protein, FLAG and Streptavidin binding peptide. Panel D, PCR analysis confirms homozygous deletion of *MIR4466* in MiaPaCa2 cells. HPDE, normal human pancreatic ductal epithelium; NTC, no template control; M, DNA ladder. BRAF amplification was used as a control. Panel E, *MIR4466* ectopic expression results in reduction in transcrip levels of *MLL2, MIER2* and *DNMT3B*; transcript level of each gene measured in *MIR4466* transfectant is represented as a fraction of the level measured in vector transfectant. *MXI* transcript levels were measured as a control.

thus confirming its role as a component of the PAR complex. A novel miRNA *MIR4466* was detected within the antisense strand of *ARID1B* first intron and its bi-allelic loss in MiaPaCa2 cells was confirmed (Fig.1D). Several putative *MIR4466* targets were identified based on bio-informatic analysis. *MIR4466* ectopic expression in MiaPaCa2 cells resulted in a significant reduction of expression of three putative targets namely *MLL2, MIER2* and *DNMT3B* (Fig. 1E).

CRC: Two-way hierarchical clustering performed on rectal cancer samples using 49 differentially expressed genes (derived from SAM analysis at q=0) distinguished Wnt- from Wnt+ rectal cancer samples (Fig. 2A). This gene cluster was validated on four independent CRC transcriptome data sets generated from the Western population. Repeating SAM at a relaxed q value (<5.0) revealed 422 differentially expressed genes that yielded Ca^{2+} signalling as the most significantly enriched biological process (Fig. 2B) and NFAT family as the most significantly enriched transcription factor (TF) when subjected to GO annotation and TF prediction online packages, respectively. Differential expression analysis of

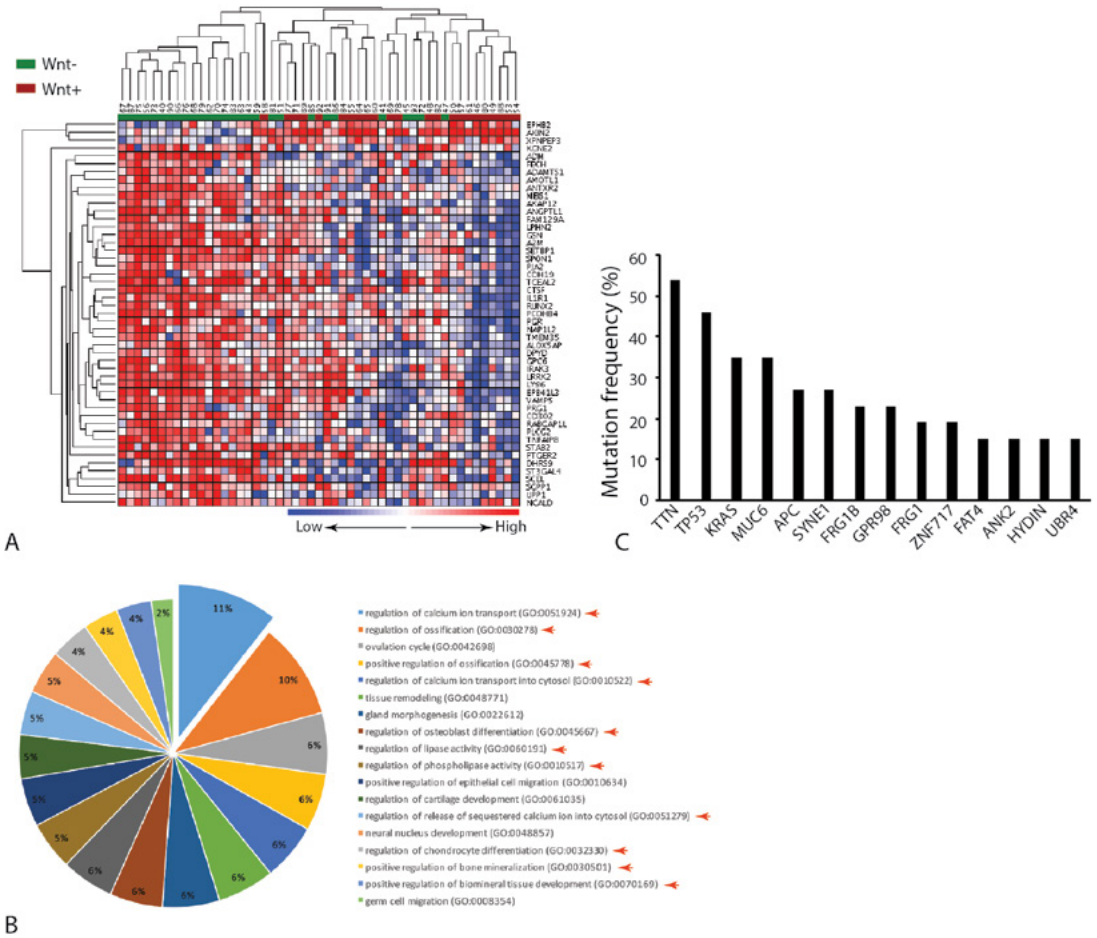

Figure 2. Characterization of Wnt- early onset sporadic rectal cancer. Panel A, a 49-gene signature differentiates Wnt- from Wnt+ rectal cancer samples. Panel B, Ca^{2+}/NFAT signalling is the most significantly enriched biological process in Wnt- rectal cancer samples. Panel C, frequently mutated genes in Wnt- rectal cancer.

next generation sequencing (NGS) based RNA Seq data generated from 18 and 8 Wnt- and Wnt+ rectal cancer RNA samples respectively also revealed Ca^{2+} signalling as one of six most significant differentially enriched pathways in Wnt- samples. NGS based exome sequencing performed on 20 Wnt- rectal cancer samples revealed mutations in well studied cancer genes

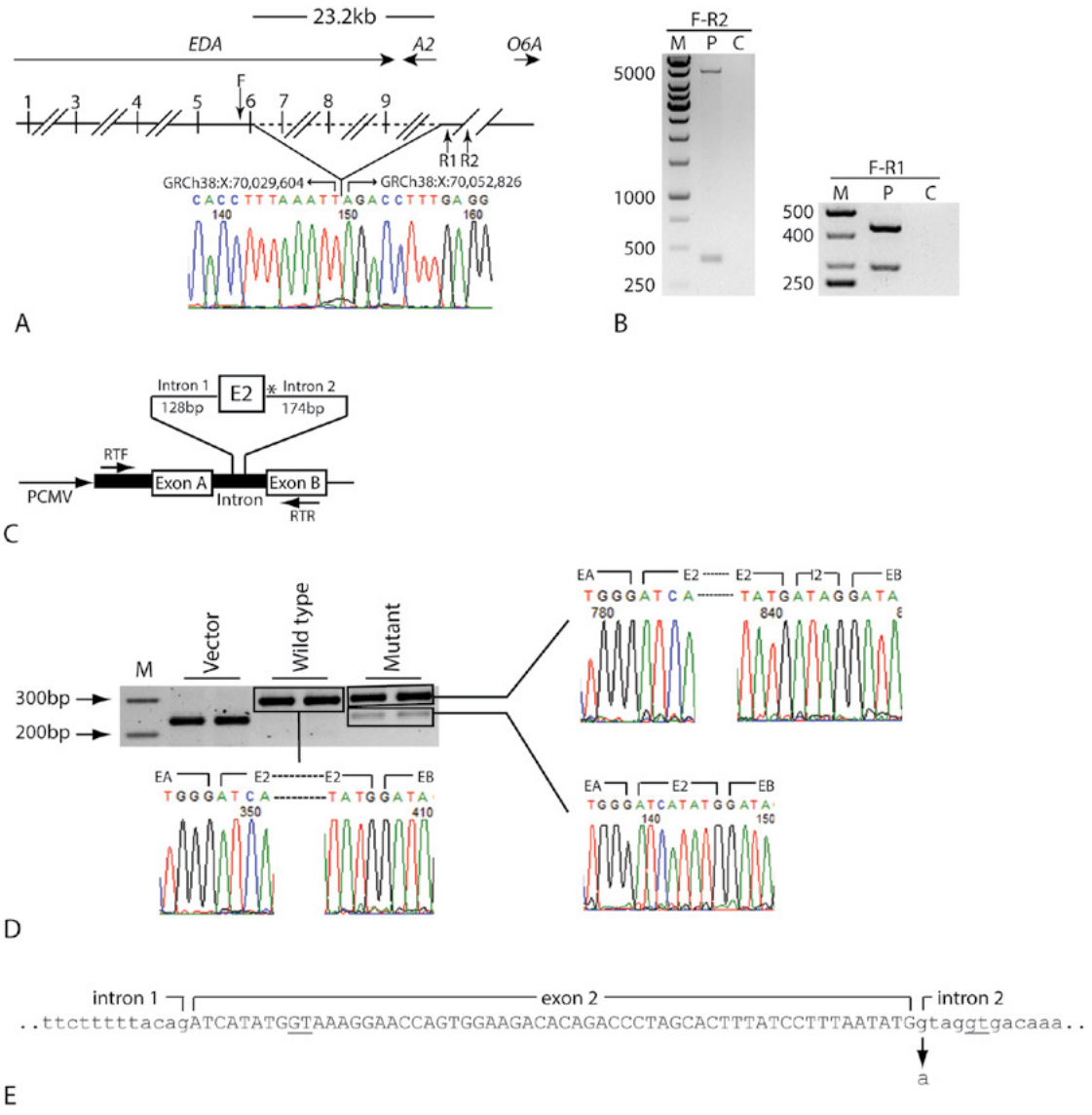

Figure 3. Panels A-B, mapping break point of HED-causing novel *EDA-A1* large deletion. Panel A shows diagrammatic representation of location of break point as well as the PCR primer pairs (common forward primer F and two reverse primers R1 and R2) used for ascertaining the exact break point. Electropherogram of sequencing reaction performed on lower band of F-R1 PCR product is also shown. *A2, AWAT2; O6A, OTUD6A.* PCR results are shown in panel B; F-R2, 5114bp and F-R1, 295bp. *Phenylalanine hydroxylase* exon 13 was amplified (428bp) as internal positive control. M, DNA ladder; P, patient DNA; C, template negative control. Panels C-E, the novel EDARADD IVS2+1G>A 5' splice site mutation disrupts exon 2 splicing. Panel C, diagrammatic representation of pCAS splicing assay vector; both *EDARADD* exon 2 PCR products (mutant and wild type; location of the 5' splice site mutation is indicated by an asterisk) were cloned into the intron separating exons A and B. Panel D, agarose gel analysis of RT-PCR performed using primer pair RTF and RTR separately for HeLa cell transfectants generated from pCAS vector and from both recombinant constructs. Sequencing electropherograms depicting exon-exon junctions generated due to activation of authentic (for wild type transfectants) and cryptic (for mutant transfectants) splice sites are also shown separately for each RT-PCR product. Lane M, 100 bp DNA ladder; EA and EB, pCAS vector exons A and B respectively; E2 and I2, *EDARADD* exon 2 and intron 2 respectively. Panel E, location of the two cryptic 5'-splice sites (underlined) identified in this study with respect to *EDARADD* exon 2. The mutation affecting the first base of intron 2 is also indicated.

APC, TP53 and *KRAS* and in additional genes including *MUC6* and *SYNE1* (Fig. 2C).

Genetic disorders: We mapped the break point of the HED-causing *EDA-A1* large deletion using PCR-DNA sequencing (Fig. 3A-B) and also characterized the novel homozygous C.120+1G>A (IVS2+1G>A) *EDARADD* IVS2+1G>A 5'-splice site mutation using ex vivo splicing assays (Fig. 3C-E). In addition, we identified an HED causing novel autosomal dominant *EDAR* p.L397H missense mutation.

Future plans and directions

1. Characterization of role of PAR6G in PAR complex.
2. Characterization of Ca^{2+}/NFAT signalling pathway driving Wnt- rectal cancer.
3. Validation of novel exonic mutations identified in Wnt- rectal cancer.
4. Characterization of selected mutations affecting transcript stability/processing identified through screening of various genetic disorders.

Publications

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3. Chaudhary AK, Girisha KM and Bashyam MD (2016). A novel EDARADD 5'-splice site mutation resulting in activation of two alternate cryptic 5'-splice sites causes autosomal recessive Hypohidrotic Ectodermal Dysplasia. ***American Journal of Medical Genetics Part A*** doi: 10.1002/ajmg.a.37607 (In press).
4. Chaudhary AK, R Mohapatra, HA Nagarajaram, P Ranganath, A Dalal, A Dutta, S Danda, KM Girisha and MD Bashyam (2015). The novel EDAR p.L397H missense mutation causes autosomal dominant hypohidrotic ectodermal dysplasia. ***Journal of the European Academy of Dermatology and Venereology*** doi: 10.1111/jdv.13587 (In press).

LABORATORY OF NEUROSPORA GENETICS

A transmission ratio distortion in crosses with hybrid Neurospora translocation strains flags a putative Bateson-Dobzhansky-Muller Incompatibility between *N. crassa* and *N. tetrasperma* genes

Faculty	DP Kasbekar	Haldane Chair
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Other Members	Sheeba A	Technical Officer
	K Sreethi Reddy	Technical Assistant
	Rekha S	Technical Assistant
	Angela Sharma	Research Assistant (till Feb. 2016)

Objectives

(1) One objective of our research is to screen for nucleus-limited genes in fungi. Nuclei bearing a null allele (*Δ*) of a nucleus-limited gene fail to be complemented by wild-type (*WT*) nuclei in a [(*WT*) + (*Δ*)] heterokaryon. No nucleus-limited gene has yet been reported in the literature, but the phenotype of some fungal mutants suggests that they may be caused by mutations in such genes.

Introgression is the transfer of genes or genomic regions from one species into another via hybridization and back-crosses. By introgressing insertional translocations from *Neurospora crassa* into the related species *N. tetrasperma* we can make hybrid translocation strains (designated as T^{Nt}) whose genome is nominally from *N. tetrasperma*, except at the *N. crassa*-derived translocation breakpoint junctions. In *T x N* crosses (*T* = translocation, *N* = normal sequence strain), the chromosomes can segregate either via alternate (ALT) or adjacent-1 (ADJ) segregation (Figure 1). In *N. crassa*, ALT produces eight viable parental-type progeny (i.e., 4*T* + 4*N*), and if the translocation is insertional, ADJ produces eight progeny with a viable duplication or its complementary inviable deficiency (i.e., 4*Dp* + 4*Df*). Since ALT and ADJ are equally likely, a *T x N* cross produces equal numbers of viable *T, N,* and *Dp* progeny. In contrast, *N. tetrasperma* T^{Nt} *x N* crosses normally produce four viable heterokaryotic [T^{Nt} + *N*] ascospores following ALT, or four viable heterokaryotic [*Dp* + *Df*] ascospores following ADJ (Figure 2). Heterokaryotic [*Dp* + *Df*] strains were never previously made in any species. The [*Dp* + *Df*] and [*T* + *N*] heterokaryons share identical genes and hence should have the same phenotype. However, if they differ in phenotype, then it could indicate that one or more 'nucleus-limited' gene is absent from the *Df nuclei*.

(2) A second objective of our research is to understand why most wild-isolated *N. crassa* strains appear to suppress meiotic silencing by unpaired DNA (MSUD) in crosses with tester strains derived in the standard laboratory Oak Ridge (OR) background. We hypothesized that sequence heterozygosity between the wild and OR genomes might cause one or more MSUD gene to become unpaired and silence itself, and douse the MSUD machinery. The wild-isolated Bichpuri-1 *a* (B) and Spurger *A* (S) strains are relatively strong suppressors of MSUD. Using these strains we constructed novel isogenic *mat-A* and *mat-a* strain pair in which new MSUD testers can be made to test for MSUD in tester-heterozygous crosses but otherwise isogenic for the B/S background.

Summary of work done until the beginning of this reporting year (upto March 31, 2015)

(1) Insertional translocations (*IT*) transfer a segment from a donor chromosome into a recipient chromosome and create three breakpoint junctions, viz, "A" on the donor chromosome, and "B" and "C" (proximal and distal) on the recipient chromosome (Figure 1). We had previously defined the breakpoint junctions of several *N. crassa ITs*. This enabled us to use PCR with breakpoint junction-specific primers to distinguish between the *T, N* and *Dp* progeny from *T x N* crosses, and allowed us to introgress four *ITs* (*EB4*, *IBj5*, *UK14-1*, and *B362i*) into *N. tetrasperma* to construct the corresponding [*T* + *N*] and [*Dp* + *Df*] heterokaryon strains. Self-crossing the heterokaryons again yielded [*T* + *N*] and [*Dp* + *Df*] progeny. The two heterokaryons types were distinguishable; [*T* + *N*] produced homokaryotic (self-sterile) conidial derivatives of

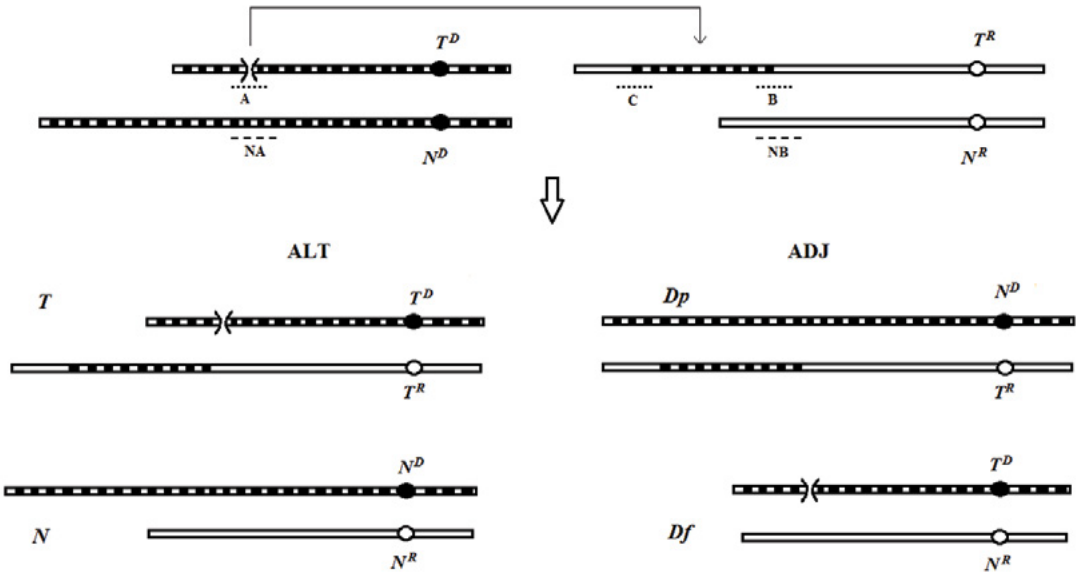

Figure 1. Alternate (ALT) and adjacent-1 (ADJ) segregation in a normal sequence (N) by insertional translocation *(T)* cross. T^D and T^R designate the translocation donor and recipient chromosomes and N^D and N^R are their *N*-derived homologues. The A, B, and C breakpoint junctions are indicated by the dotted lines, and dashed lines NA and NB indicate segments in the normal sequence homologues that are disrupted in the translocation chromosomes. In ALT, T^D and T^R segregate to one spindle pole and N^D and N^R to the other. Subsequently, meiosis II and post-meiotic mitosis generate eight parental-type nuclei, viz. 4 *T* + 4 *N*. In ADJ, N^D and T^R segregate to one pole and T^D and N^R to the other, to eventually produce eight non-parental nuclei, 4 *Dp* + 4 *Df*. The *T*, *N*, and *Dp* types are viable, whereas the *Df* type is inviable. *T* progeny contain the A, B, and C breakpoints, *Dp* contain B and C but not A, and *N* contain none.

both mating types, whereas [*Dp* + *Df*] produced viable conidial homokaryons of only the mating type of the *Dp* nucleus. To our best knowledge this was the first introgression of translocations from one species into another. Interestingly, the *Df* nuclei in the [*Dp* + *Df*] heterokaryons derived from introgression of *T(B362i)* appeared to have an apparent nucleus-limited deficit for packaging into vegetative spores (conidia). The work was published in G3 5: 1263-1272 (June 2015).

Additionally, we found that the *T(IBj5)*^{Nt}a x *E A* and *T(B362i)*^{Nt}*A x E a* crosses did not produce any asci with more than four black (viable) ascospores. We call this the “max-4 phenotype”. We hypothesized that these crosses had become homozygous for a mutation that specifically affected alternate segregation, and did not affect adjacent-1 segregation. The hypothesis was based on the fact that the *C4,T4* a strain used to construct the T^{Nt} strains and the *E* strains shared the same genetic background. Consequently, a subset of T^{Nt} x *E* crosses could have become homozygous for a mutation for the max-4 phenotype.

(2) MSUD eliminates the transcripts of any gene that is not properly paired with its homolog in meiosis, via an RNAi-mediated process. The ::*r*, ::*Bml*^{r} and ::*mei-3* tester strains contain a copy of the *r* (*Round ascospores*), Bml (*β-tubulin*) or *mei-3* gene inserted ectopically in the *his-3* locus on chromosome 1. In the cross of a tester with an OR strain of opposite mating type, the ectopic copy is unpaired in meiosis and induces the synthesis of MSUD-associated small interfering RNA (masiRNA) which silences it and its paired native homologs and results in ascus or ascospore abnormalities. Homozygous *tester A x tester* a crosses do not show MSUD, nor do crosses of the testers with the semi-dominant *Sad* suppressors of meiotic silencing, and the asci and ascospores develop normally. The suppressor alleles prevent the proper pairing of their wild-type homologues and induce them to autogenously silence themselves. We hypothesized that sequence polymorphism between the tester and wild genomes also might cause one or more gene essential for MSUD to become unpaired, silence itself, and suppress MSUD. To test this we want to make new testers

in an isogenic *mat a* and *mat A* background derived from the MSUD suppressing wild-isolates Bichpuri-1 a and Spurger *A*. A tester-heterozygous cross in this otherwise isogenic B/S background is predicted to display MSUD.

In the B/S line we mutated the *mus-51* gene needed for non-homologous end joining (NHEJ). In the *mus-51* mutant, transforming DNA can integrate only via homologous recombination, and would allow us to create well-defined reporter strains. The native r^+ gene is 3.3 kb long and located on chromosome I. A 2.3 kb 3' fragment (r^{ef}) was joined to the *hph* cassette by double-joint PCR to create the 4.1 kb r^{ef}-*hph* fusion construct. This construct is being used to transform the B/S *mus-51* mutant, and transformants selected on hygromycin medium would correspond to the ::*r2* tester made by others in the OR background. When a strain carrying ::*r2* is crossed to an OR strain of opposite mating type, most of the ascospores are round, indicating that ::*r2* is detected as unpaired in such crosses, whereas when a strain carrying ::*r2* is crossed to a strain carrying the same ::*r2*, very few round spores are produced, indicating that the ::*r2* constructs are paired in such crosses. We will mimic these crosses with our new ::*r2*-like tester in the B/S background.

Progress made in the current reporting year (April 1, 2015 - March 31, 2016)

(1) To test the hypothesis that the max-4 phenotype is due to homozygosity for a mutation common to the *T(IBj5)*^{Nt}*a*, *T(B362i)*^{Nt}*A*, *E a,* and *E A* strains, we crossed the *E* strains with wild-type *N. tetrasperma* strains of the opposite mating type, and obtained the f1 progeny. The wild type strains did not show the max-4 phenotype in crosses with *T(IBj5)*^{Nt}*a* or *T(B362i)*^{Nt}*A*, nevertheless all the f1 progeny showed the max-4 phenotype in crosses with the *T(IBj5)*^{Nt}*a* or *T(B362i)*^{Nt}*A* strain. This suggested that they all had inherited the mutation from the *E* parent, and that none had inherited the homologous wild type allele. However, examination of the f1 progeny for molecular markers polymorphic between the *E* and the wild type strains revealed independent segregation of all the seven chromosomes, rendering the hypothesis of a recessive mutation underlying the max-4 phenotype untenable. Further studies (described below) revealed that the max-4 phenotype might be caused by a Bateson-Dobzhansky-Muller incompatibility between *N. crassa* and *N. tetrasperma* genes.

Occasionally, in *N. tetrasperma* ascus development a heterokaryotic ascospore is replaced by a pair of smaller homokaryotic ascospores. Such replacement is increased in crosses with the dominant *Eight-spore* mutant, and can generate up to eight homokaryotic ascospores; either 4*T* (black) + 4*N* (black), or 4*Dp* (black) + 4*Df* (white). We found that far more *Dp* progeny were produced than *T* and *N* types in the homokaryotic progeny from crosses of some *T*^{Nt} strains with *N* type *N. tetrasperma* strains. This type of transmission ratio distortion is novel because it appears to disfavor only the homokaryotic products from ALT relative to ADJ, and it was specific to the homokaryotic progeny and did not affect the [*Dp* + *Df*] / [*T* + *N*] heterokaryon ratio. We hypothesized that a *N. crassa* gene might have triggered a Bateson-Dobzhansky-Muller incompatibility in the *N. tetrasperma* genetic background, producing insufficiency for a presumptive ascospore maturation factor. This could induce a "tragedy of the commons" in asci with >4 viable ascospores, and cause none of the ascospores to properly mature. Note that an increase in ascospore numbers because heterokaryotic ascospores are replaced by pairs of homokaryotic ascospores can happen only in [*T* + *N*] asci and not in [*Dp* + *Df*] asci. The transmission ratio distortion can potentially deplete the supply of homokaryotic *T* progeny well before the introgression crosses advance sufficiently to produce any self-fertile heterokaryons. This can undermine the introgression efforts.

The Bateson-Dobzhansky-Muller incompatibility accounts for the max-4 phenotype in the *T(IBj5)*^{Nt} *a* x *E A* and *T(B362i)*^{Nt}*A x E* a crosses, but how do we explain the apparent absence of the max-4 phenotype in crosses of *T(IBj5)*^{Nt}*a* or *T(B362i)*^{Nt} *A* with wild type *N. tetrasperma*? Crosses of *T(IBj5)*^{Nt}*a* and *T(B362i)*^{Nt}*A* with the wild type strains also showed transmission ratio distortion, in that they produced more homokaryotic *Dp* progeny than *T* and *N* types. To investigate this anomaly, we collected asci from the *T(IBj5)*^{Nt} *a* x 85*A* and *T(B362i)*^{Nt}*A* x 85a crosses onto water agar. The majority of asci were four-spored, but we could pick the rare eight-spored asci and use PCR to determine the genotype of cultures obtained following germination of their black ascospores. Unexpectedly, we found the ascospores from the 8B:0W asci had [*T* + *N*], [*Dp* + *Df*], or [*N* + *Dp*] heterokaryotic genotypes, and some were also heterokaryotic for mating type.

Ordinarily, eight-spored asci are not expected to yield any heterokaryons because each ascospore receives one of the eight nuclei generated via the post-meiotic mitosis. We suggest that in a subset of asci the nuclei must undergo additional rounds of mitosis before partitioning into the eight ascospores, which effectively masks the max-4 phenotype. This abnormality might be peculiar to crosses of the hybrid translocation strains with the wild type *N. tetrasperma*.

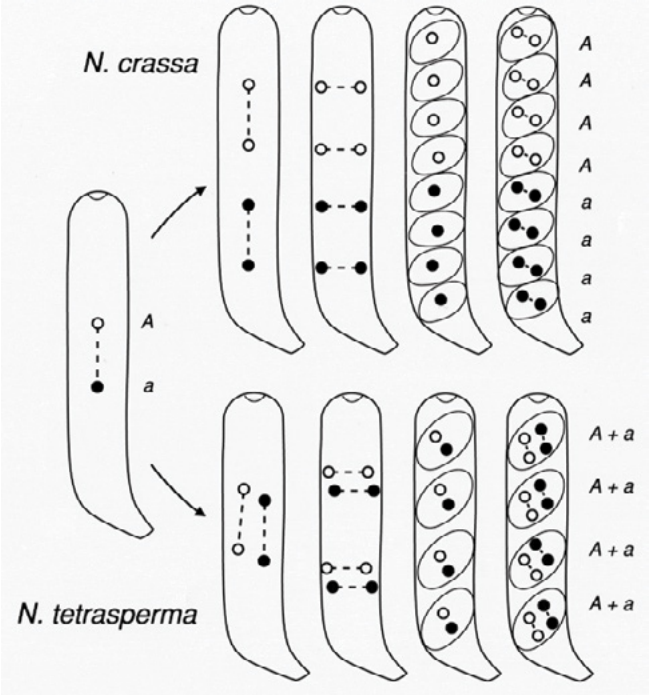

Figure 2. Ascus development in *Neurospora crassa* and *N. tetrasperma.* Fusion of the parental haploid *mat A* and *mat a* nuclei (respectively, open and filled circles) produces a diploid zygote nucleus that undergoes meiosis (leftmost panel shows meiosis I, *mat A* and *mat a* show first division segregation) and a post-meiotic mitosis (third panels from left) to generate eight haploid progeny nuclei (4 *mat A,* + 4 *mat a*). In *N. crassa* (upper panels), these nuclei are partitioned into eight initially uninucleate ascospores formed per ascus, whereas in *N. tetrasperma* (lower panels) the asci make four initially binucleate ascospores, each receiving a pair of non-sister nuclei (1 *mat A* + 1 *mat a*). *N. crassa* ascospores produce homokaryotic mycelia of *mat A* or *mat a* mating type that can mate only with mycelia derived from another ascospore of the opposite mating type. In contrast, dikaryotic [*mat A* + *mat a*] *N. tetrasperma* mycelia can undergo a self-cross. Occasionally, in *N. tetrasperma* a pair of smaller homokaryotic ascospores can replace one or more dikaryotic ascospore. The dominant *Eight-spore* (*E*) mutant increases such replacement, and can generate asci with up to eight ascospores. *N. tetrasperma* dikaryotic mycelia also produce some homokaryotic conidia (vegetative spores) by chance, and mycelia from homokaryotic conidia and ascospores can out-cross with like mycelia of the opposite mating type. (Figure adapted from N. B. Raju and D. D. Perkins, Genetics 129: 25-37, 1991.)

(2) Our attempts to transform the B/S *mus-51* mutant to hygromycin-resistance using the *r*^{ef}*-hph* fusion DNA made by double-joint PCR have not yet been successful and we are continuing with these efforts.

Publications

1. Giri, D. A., Rekha, S., and Kasbekar, D. P. (2015) Neurospora heterokaryons with complementary duplications and deficiencies in their constituent nuclei provide an approach to identify nucleus-limited genes. ***G3: Genes, Genomes, Genetics*** 5: 1263-1272.

Other Publications

1. Kasbekar, D. P. (2015) What have we learned by doing transformations in *Neurospora tetrasperma?* In : Genetic Transformation Systems in Fungi, Volume 2. Edited by M. A. van den Berg and K. Maruthachalam, Springer, Switzerland. Pages 47-52.

2. Kasbekar, D. P. (2016) Editorial. Long-drawn-out story. ***Journal of Biosciences*** 41: 1

3. Kasbekar, D. P. (2016) Obaid Siddiqi's study of the PABA1 gene of the fungus *Aspergillus nidulans*. ***INSA Special Volume on Obaid Siddiqi.***

LABORATORY OF PLANT-MICROBE INTERACTIONS

Understanding virulence mechanisms of *Xanthomonas* plant pathogens and interaction with host plants

Faculty	Subhadeep Chatterjee	Staff Scientist
PhD Students	Rikky Rai	Senior Research Fellow (Till March 2016)
	Sheo Shankar Pandey	Senior Research Fellow
	Akanksha Kakkar	Senior Research Fellow
	Raj Kumar Verma	Junior Research Fellow
	Biswajit Samal	Junior Research Fellow
	Prashantee Singh	Senior Research Fellow
	Yasobanta Padhi	Junior Research Fellow (Since 20.07.2015)
Other Members	Binod Bihari Pradhan	Technical officer
	L Santhosh Kumar	Project JRF (Till November 2015)
	Pradeep Kumar Patnana	Project JRF
	N Saraswathi	Project JRF

Objectives

1. Identification and characterization of virulence factors of *Xanthomonas;*
2. Role of cell-cell communication in *Xanthomonas* colonization and virulence;
3. Function of protein secretion system in *Xanthomonas* and role in virulence; and
4. Role of PAMP in pathogen recognition and plant defense response

Summary of work done until the beginning of this reporting year (April 1, 2014 – March 31, 2015)

Cell–cell communication mediated by diffusible signal factor (DSF) plays an important role in virulence of several *Xanthomonas* group of plant pathogens. In the bacterial pathogen of rice, *Xanthomonas oryzae pv. oryzicola,* DSF is required for virulence and in planta growth. Our results also indicate that requirement of iron uptake strategies to utilize either Fe^{3+} or Fe^{2+} form of iron for colonization may vary substantially among closely related members of the *Xanthomonas* group of plant pathogens. Apart from iron, we have identified novel role of DSF in regulating Type III secretion system which is required for pathogenicity of *Xanthomonas*. DSF deficient *rpfF* mutant are exhibit reduced Hypersensitive Response (HR) and reduced expression of Type III secretion components and effectors.

In future, we want to study the mechanism of DSF sensing which controls iron uptake and regulatory mechanisms, which are involved in DSF regulated traits such as Type III secretion, attachment and biofilm formation.

We have shown that bacteria exhibit reversible non gebnetic heterogeneity in QS. We have proposed a model based on our studies and evolutionary theory, which predicts that maintaining phenotypic heterogeneity in performing social tasks is advantageous as it can serve as a bet-hedging survival strategy. Our results have shown that bacteria maintain stochastic reversible phenotypic heterogeneity during a widely conserved QS-response that is involved in coordinating multiple social behaviors. In general, it appears that QS- mutants exhibit growth disadvantage at early log phase and compromised viability at late stationary phase. Our transcriptome analysis by microarray and translation assays indicate that QS promotes transition to stationary phase by slowing down the metabolism (transcription and translation), as an anticipation of stationary-phase stress.

Details of the progress made in the current reporting year (April 1, 2015 – March 31, 2016)

Project 1: Role of *xanthoferrin,* the α-hydroxy carboxylate type siderophore of *Xanthomonas campestris* pv. *campestris* in virulence.

Xanthomonas campestris pv. *campestris* causes black rot, a serious disease of crucifers. Xanthomonads encodes a siderophore biosynthesis and uptake gene cluster xss (*Xanthomonas*

siderophore synthesis) involved in production of a vibrioferrin type of siderophore. However, little is known about the role of siderophore in iron uptake and virulence of *X. campestris* pv. *campestris.* In this study, we show that *X. campestris* pv. *campestris* produces an α-hydroxy carboxylate type of siderophore (named xanthoferrin), which is required for growth under low-iron condition and optimum virulence (Fig. 1). A mutation in the siderophore synthesis *xssA* gene causes deficiency in siderophore production and growth under low-iron conditions. In contrast, the siderophore utilization Δ*xsuA* mutant was able to produce siderophore but exhibited a defect to utilize siderophore-iron complex. Our radiolabelled iron uptake studies confirmed that the Δ*xsuA* and Δ*xsuA* mutants exhibited defects in ferric iron uptake. The Δ*xsuA* mutant was able to utilize and transport exogenous xanthoferrin-Fe^{3}+ complex,

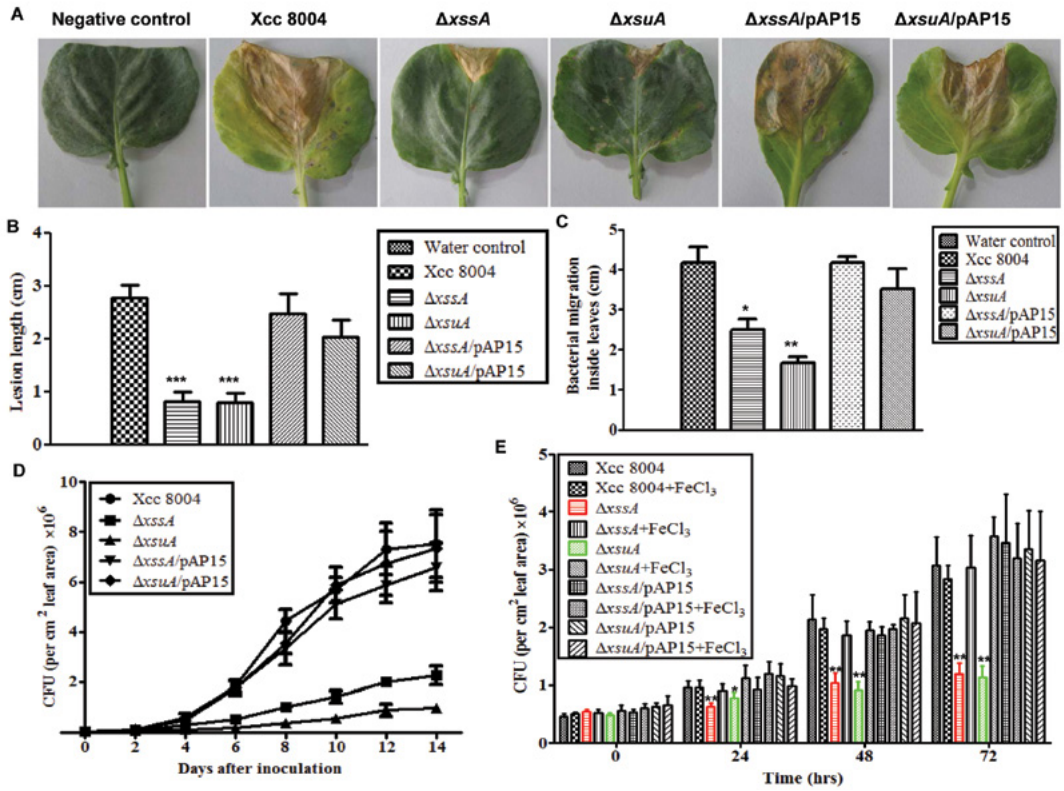

Figure 1. The *ΔxsuA* and *ΔxsuA* mutants are deficient in virulence and growth inside cabbage.

(A) Infected cabbage leaves (Indian Super Hybrid variety) with wild-type Xcc 8004, ΔxssA, ΔxsuA, ΔxssA/pAP15 and *ΔxsuA*//pAP15 strains showing lesion symptoms after 21 days post inoculation. Bacterial cultures (1 X 10^{9} cells/ml suspension) were inoculated into 30-days old plants by clip method.

(B) Quantification of lesion length at 21 days post inoculation. 25 leaves were inoculated per strain.

(C) Five days post-inoculation bacterial migration in host leaves was assayed by inoculating 1cm pieces of infected leaves, cut from base to tip, on PSA plate with respective antibiotics Migration was estimated by observing colonies formed after 1 to 3 days by the bacterial ooze from the cut ends of cabbage leaf pieces. For each experiment 6 leaves were used (three independent experiments).

(D) *In planta* growth assays of wild-type Xcc 8004, *ΔxsuA*, *ΔxsuA*, *ΔxsuA*/pAP15 and *ΔxsuA*//pAP15 strains. Bacterial populations were measured by crushing the leaves of 1cm2 areas for each and serial dilution plating at the indicated post inoculation days. For each experiment 6 leaves were used (three independent experiments).

(E) Detached leaves assay with exogenous iron supplementation. Different Xcc strains were inoculated to detached cabbage leaves by clip method. The leaves were maintained in 1 μg/ml of Benzyl amino purine (BAP; first generation synthetic cytokinin) and with or without 50 μM $FeCl_3$ supplementation. Bacterial populations were determined from 1 cm^2 leaf area at the indicated post inoculation days.

Data shown in the graphs are mean ± S.E. (n=3). * indicate p-value < 0.05; ** Indicate p-value < 0.01 and *** indicate p-value < 0.001 significance difference between the data obtained from mutants and the data obtained from wild type and complementing strains by paired student t-test.

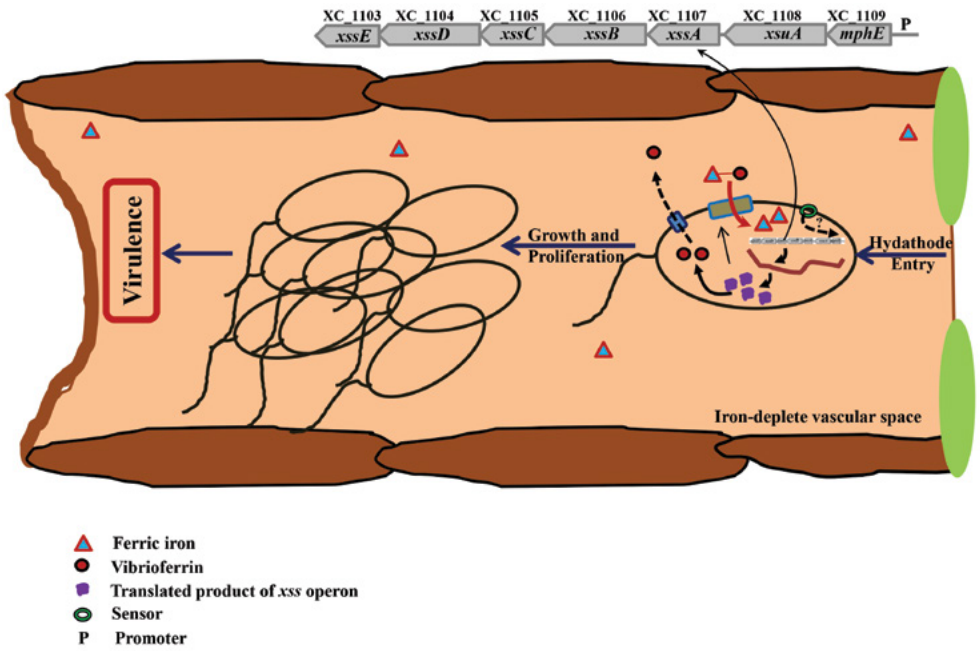

Figure 2. A model for the role of xanthoferrin mediated iron uptake *in planta* growth and virulence of Xcc. Xcc 8004 is a vascular pathogen which generally enters through the leaves hydathodes and migrates through vascular space. Inside the host, vascular spaces are iron-limiting which induce the expression of xss cluster. The xanthoferrin synthesis and release occurs in the vascular space to chelate ferric iron. The Fe^{3+} -xanthoferrin complexes are taken by bacteria through the porin made up of a membrane protein complex including TonB dependent receptor, TonB, ExbB, ExbD, ABC transporter and ATPase. Ferric irons reduce to ferrous iron by ferric reductases. Further, the iron is assimilated in various biological functions which contribute to bacterial growth, survival and subsequent disease establishment.

in contrast, the siderophore utilization or uptake mutant Δ*xsuA* exhibited defects in siderophore uptake. Expression analysis of xss operon using a chromosomal *gusA* fusion indicates that the xss operon is expressed during *in planta* growth and under low-iron conditions. Furthermore, exogenous iron supplementation in the cabbage leaves rescued the *in planta* growth deficiency of Δ*xsuA* and Δ*xsuA* mutants. Our study reveals that the siderophore xanthoferrin is an important virulence factor of *X. campestris* pv. *campestris* which promote *in planta* growth by sequestering ferric iron (Fig. 1). On the basis of our study, we have proposed a model which elucidate the role of xanthoferrin mediated iron uptake in establishing pathogenesis of Xcc under low -iron environment inside host (Fig. 2). Xcc encounters iron depleted environment inside the host, which triggers the expression of xanthoferrin synthesis and uptake genes. Xanthoferrin then released outside the bacterial cell where it starts scavenging ferric iron and eventually gets transported inside as xanthoferrin-Fe^{3+} complex through TonB dependent transporters and its auxiliary proteins ExbB and ExbD. Subsequent ferric reduction occurs inside the bacterial cell to convert Fe^{3+} to easily utilizable Fe^{2+} form, which is used by bacteria for various metabolic activities during growth and infection.

Project 2: Role of XadM , a novel adhesin of Xanthomonas oryzae pv. oryzae in virulence and biofilm formation.

We had previously identified a novel 5.241-kb open reading frame (ORF) named *xadM* that is required for optimum virulence and colonization. This ORF encodes a protein, XadM, of 1,746 amino acids that exhibits significant similarity to Rhs family proteins. The XadM protein contains several repeat domains similar to a wall-associated surface protein of *Bacillus subtilis,* which has been proposed to be involved in carbohydrate binding. We have shown that XadM is required for virulence, attachment and biofilm formation in Xoo (Fig.3). This was the first report of a role for XadM, an Rhs family protein, in adhesion and virulence of any pathogenic bacteria. In order to gain insight into the role of different domain and regions of XadM in

virulence and attachment we have made a series of N-terminal and C-terminal deletion constructs and have performed complementation analysis. The predicted XadM protein (1746 amino acid) exhibits significant similarity to RHS repeat-associated core domain (1.08e–26), RHS repeat domain (pfam 05593), and RhsA (COG3209; 8.75e–17), which is also present in the wall associated surface protein (WASP) from *Bacillus subtilis* 168. XadM protein contains at least 18 repeats with the consensus gxxvyYDxxg. Among these extensive repeat regions, three repeats with the consensus sequence motif [Gxxxx(Y or F)xYDxxG] are similar to the WAPA motif present in a WASP of *B. subtilis*. Deletion analysis indicated that both the N-terminal and central domain is required for XadM function. Further, to study the contribution of different domains of XadM, we have expressed the N terminal, RHS domain and the C-terminal domain in *E. coli* and have raised polyclonal antibody. In future, we are interested in more detail molecular characterization of XadM like Rhs family proteins and their role in virulence.

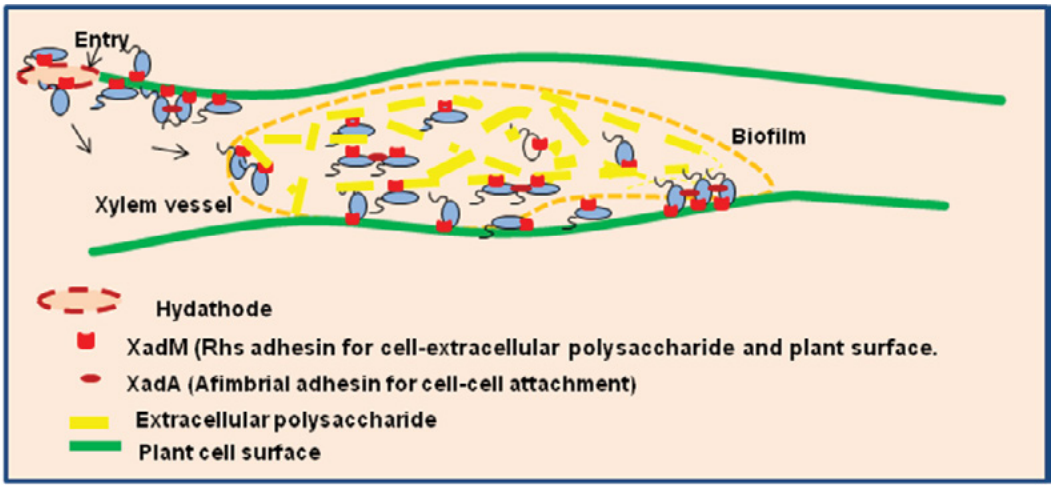

Figure 3. Proposed model for the role of XadM in biofilm formation and virulence of *Xanthomonas oryzae pv. oryzae. xadM* is expressed in the presence of plant cell wall material (cellulose, xylan) and is required for the attachment of bacteria on hydathodal openings. XadM is required for attachment of the cell with extracellular polysaccharide and with the xylem vessel and promote biofilm formation inside xylem.

Project 3: Role of DSF in inducing innate immunity in plants

Several secreted and surface associated conserved microbial molecules are recognized by host to mount the defense response. One among evolutionarily well conserved bacterial processes is the production of cell-cell signaling molecules which regulates production of multiple virulence functions by a process known as quorum sensing. In this study we have shown that a bacterial fatty acid cell-cell signaling molecule, DSF (diffusible signal factor) elicits innate immunity in plants. The DSF families of signaling molecules are highly conserved among many phytopathogenic bacteria belonging to genus *Xanthomonas* as well as in opportunistic animal pathogens. Using *Arabidopsis, Nicotiana benthamiana* and rice as model systems, we show that DSF induces hypersensitivity reaction (HR)-like response, programmed cell death, the accumulation of autofluorescent compounds, hydrogen peroxide production and induced expression of the *PATHOGENESIS-RELATED*1 (*PR-1*) gene. Furthermore, production of the DSF signaling molecule in *Pseudomonas syringae*, a non-DSF producing plant pathogen, induces the innate immune response in *Nicotiana benthamiana* host plant and also affects pathogen growth. By performing pre-and co-inoculation of DSF, we have demonstrated that the DSF induced plant defense reduces disease severity and pathogen growth in the host plant. In this study, we further demonstrate that the wild type *Xanthomonas campestris* suppress the DSF induced innate immunity by secreting xanthan, the main component of extracellular polysaccharide.

Our results indicate that plants have evolved to recognize a widely conserved bacterial communication system and may have played a role in the co-evolution of host recognition of the pathogen and the communication machinery.

We propose a model that elucidates the functional interplay between diffusible signaling factor (DSF) and extracellular polysaccharide (EPS) in *Xanthomonas*-plant interaction (Fig. 4). At the initial stage of infection and colonization (stage I), Xcc gains entry through hydathodes or stomata and colonize in the xylem vessel. At this stage (low-cell density; stage I), the production of DSF and EPS is low. At lower concentrations of DSF (presumably <10 μM), DSF may be involved in priming (sensitization) plants for cell wall-based defense mechanism, which may influence MTI (MAMP triggered immunity) mediated by MAMP's such as flagillin or LPS. MTI is further suppressed by Type III secretion system effectors. In stage II, there is increase in Xcc cell number, which is associated with increased production of DSF and EPS. Increased DSF level (20 μM or above), induces an early plant defense response (callose deposition), which is suppressed by EPS. At a late stage of infection (stage III), high level of DSF is produced (50 to 100 μM) due to further *in planta* growth of Xcc. This may lead to further increase in the production of EPS, a virulence associated factor positively regulated by DSF. High EPS level can suppress plant defense response provoked by DSF including early HR –like symptoms.

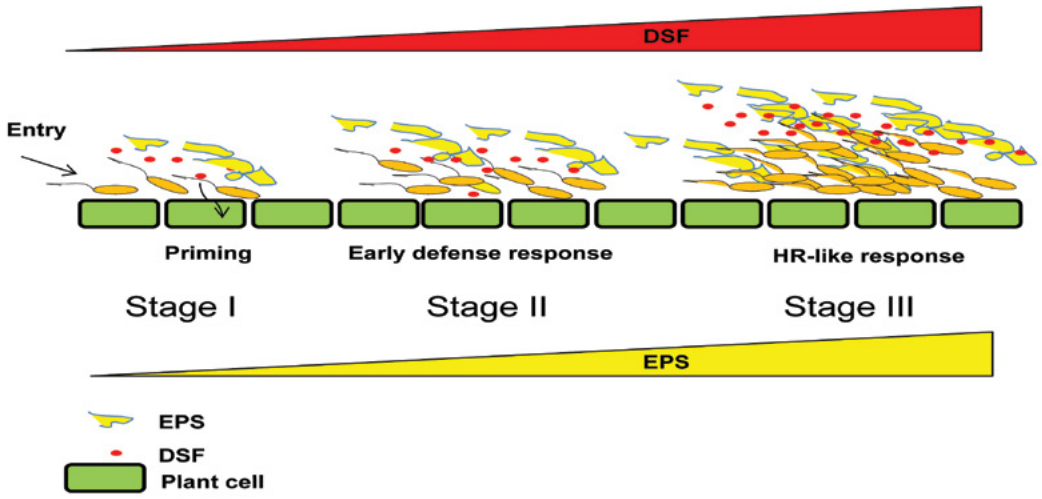

Figure 4. Proposed model for functional interplay between diffusible signaling factor (DSF) and extracellular polysaccharide (EPS) in *Xanthomonas*-plant interaction. At the initial stage of infection and colonization (stage I), Xcc gains entry through hydathodes or stomata and colonize in the xylem vessel. At this stage (at low-cell density; stage I), the production of DSF and EPS is low. At lower concentrations of DSF (10 μM or less), presumably, DSF may be involved in priming (sensitization) plants for MTI (MAMP triggered immunity) mediated by MAMP's such as flagellin or LPS (lipopolysaccharide). MTI is further suppressed by Type III secretion system effectors. At stage II, there is increase in Xcc cell number, which is associated with increased production of DSF and EPS. Increased DSF level (20 μM or above) induces an early plant defense response (callose deposition), which is suppressed by EPS. At a late stage of infection (stage III), there is a further increase in cell density due to growth of Xcc in planta. Due to high cell density, high level of DSF is produced (50 to 100 μM). This may lead to further increase in the production of EPS, a virulence associated factor positively regulated by DSF. High EPS level can suppress plant defense response provoked by DSF including early HR –like symptoms.

Publications

1. Kakkar A, Nizampatnam NR, Kondreddy A, Pradhan BB, Chatterjee S (2015) *Xanthomonas campestris* cell-cell signalling molecule DSF (diffusible signal factor) elicits innate immunity in plants and is suppressed by the exopolysaccharide xanthan. **Journal of Experimental Botany**. Vol. 66: 6697-714.
2. Rai R, Javvadi S, Chatterjee S (2015) Cell–cell signalling promotes ferric iron uptake in *Xanthomonas oryzae* pv. *oryzicola* that contribute to its virulence and growth inside rice. ***Molecular Microbiology***. Vol. 96: 708-727.

LABORATORY OF TRANSCRIPTION

Mechanism of transcription termination and antitermination in *Escherichia coli*

Faculty	Ranjan Sen	Staff Scientist
PhD Students	Sourabh Mishra	Senior Research fellow (till May, 2015)
	Mohd Zuhaib Qayyum	Senior Research fellow (till February, 2016)
	V Vishalini	Senior Research fellow
	Gairika Ghosh	Senior Research fellow
	Richa Gupta	Junior Research fellow
	Md. Hafeezunnisha	Junior Research fellow
	Chetan Amin	Junior Research fellow (Since February, 2016)
Other Members	Sudha Kalayni	Post-doctoral Fellow (Until September, 2015)
	Shweta Singh	Post-doctoral Fellow
	Pallabi Maitra	Post-doctoral Fellow (Since November, 2015)
	Sonia Agrawal	Project Assistant (Since Feb, 2015)
	Sapna Godavarthi	Technical Officer
	M Jayavardhan Reddy	Technical Assistant
Collaborators	Prof. Udayaditya Sen	SINP, Kolkata
	Dr Jayanta Mukhopadhyay	Bose Institute, Kolkata
	Prof Akira Ishihama	Hosei University, Japan.

Objectives

Fundamental questions in the area of mechanism of transcription termination and antitermination processes in bacteria is still not very clear and offers an exciting subject for study. In my laboratory, we have undertaken following studies. 1) Mechanism of action of transcription termination factor, Rho.2) Molecular basis of Rho-NusG interaction.3) Mechanism of conversion of NusA into an antiterminator by N. 4) Establishing inhibition of Rho-dependent termination by Rho proteins from different bacteria by the anti-rho factor, Psu.5) In vivo cross-talks between Rho dependent termination and other biological processes. 6) Isolating myco-bacteriocidal factors from the mycobacteriophages using metagenomics approaches.

Summary of the work done until the beginning of this reporting year (upto March 31, 2015)

- We have shown that the antiterminator protein, N, upon interacting at the RNA-exit channel of the transcription elongation complex, transforms NusA into an antiterminator by modulating NusA- RNA polymerase flap domain interactions.We proposed that in addition to affecting the RNA exit channel and the active center of the EC, β-flap domain rearrangement is also a mechanistic component in the N antitermination process (NAR, 2015).
- NusA is an essential protein that binds to RNA polymerase (RNAP) and also to the nascent RNA, and influences transcription by inducing pausing and facilitating transcription termination /antitermination. Its involvement in Rho-dependent transcription termination has been perceived, but the molecular nature of this involvement is not known. Our data strongly argued in favor of a direct competition between NusA and Rho for the access of specific sites on the nascent transcripts in different parts of the genome. We propose that this competition enables NusA to function as a global antagonist of the Rho function, which is unlike its role as a facilitator of hairpin-dependent termination (JBC, 2016).

Details of the progress in the current reporting year (April 1, 2015- March 31, 2016)

A) Molecular basis of NusG-mediated regulation of Rho-dependent transcription termination in bacteria

The bacterial transcription elongation factor NusG stimulates the Rho-dependent transcription termination through a direct interaction with Rho. The mechanistic basis of the NusG-dependency of the Rho-function is not known. Here, we describe Rho* mutants, I168V, R221C/A, P235H

that do not require NusG for their termination function. These Rho* mutants have acquired new properties, which otherwise would have been imparted by NusG. A detailed analyses revealed that they have more stable interactions at the secondary RNA binding sites of Rho, which reduced the lag in initiating its ATPase as well as the translocase activities. These more stable interactions arose from the significant spatial re-orientations of the P, Q and R structural loops of the Rho central channel. We propose that NusG imparts similar conformational changes in the central channel of Rho, yielding faster isomerization of the open to the closed hexameric states of the latter during its RNA-loading step. This acceleration stabilizes the Rho-RNA interactions at many terminators having sub-optimal rut sites, thus making Rho-NusG interactions so essential in vivo. Finally, identification of the NusG binding sites on the Rho hexamer led us to conclude that the former exerts its effect allosterically (figure 1).

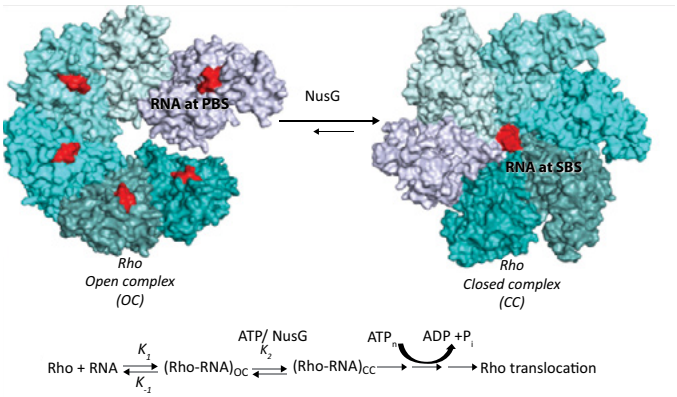

Figure 1. Cartoon showing the kinetic / equilibrium steps during the conversion of open (OC) to closed complex (CC) of the Rho hexamer. Putative step(s) those are targeted by NusG are indicated. ATP-binding and hydrolysis steps are also shown. Hexameric structures were based on the co-ordinates using the PDBs, 3ICE & 1PVO, respectively.

B) Myco-bacteriophage metagenomics technique to isolate novel myco-bactericidal factors.

Myco-bacteriophages are the phages that specifically use mycobacteria as host. They code numerous protein factors capable of modulating host machineries for their own growth advantages. Thousands of mycobacteriophages have been isolated using a single host strain, *M. smegmatis mc2155*, and about 1000 of which have been now sequenced (http://phagesdb.org). Myco-bacteriophages code for large number of novel genes that are unrelated to any known genes with unknown function. Thus these are reservoirs of new proteins as well as could be utilized to source novel myco-bacteriocidal factors.

Through phage metagenomics, we intend to identify and characterize novel protein factors from the mycobacteriophages, which are capable of killing mycobacterium upon expression in mycobacteria. These proteins may function as precursors for designing new therapeutic peptide-inhibitors of *M. tb*.

In our initial attempts, we decided to create a mixed phages genome library using few available completely sequenced phages (Bethlehem, Che9c, Che9d, Che12, D29, L5, I3 and TM4). Phages genomes were isolated and sonicated to obtain desired size of DNA fragments (≈ 1 kb or ~2 kb) to construct genome library in a pST-KT vector (an E. *coli* - *M. smegmatis*. shuttle vector) under the tetracycline inducible expression system. This library was screened in the *M. smegmatis* strain mc^{2}155. In an initial attempt about 3000 colonies were screened and re-streaked on inducible (Anhydrous tetracycline, ATC) and non-inducible (in absence of ATC) plates. Colonies those did not grow on in the presence of tetracycline were selected. Plasmids from these colonies were isolated and were sequenced to identify the phage genes, expression of which killed the *M. smegmatis* (Figure 2 and table 1). Our initial data revealed that gp89 of phage D29, gp79, gp80 of the phage Bethlehem and gp49 and gp50 of the phage Che12 are responsible for lethality. These gene products are unique to mycobacteriophages and their functions are not yet identified. Further work is in progress for characterization of these candidate genes.

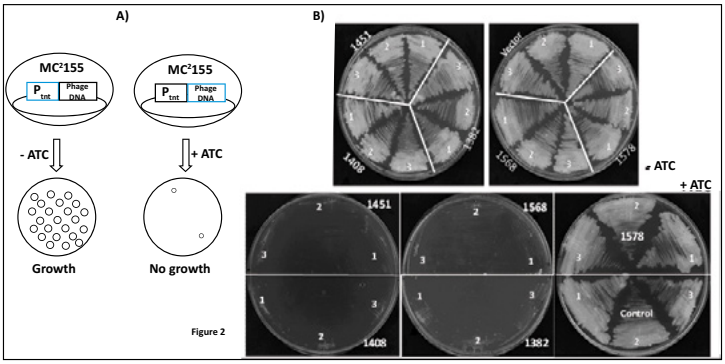

Figure 2. A) Cartoons showing the schematic representations of the genetic screen to isolate the clones that inhibit growth of *M. smegmatis* upon induction by ATC. B) Examples of few clones showing the growth inhibition.

Table1: Details of the clones that had induced lethality to *M. smegmatis* upon expression in the presence of anhydrous tetracycline (ATC) in the media:

Clones Numbers	Name of the Phages	Co-ordinate of the DNA fragments in the phage genomes	Genes (gp) present in the DNA fragment	Other remarks
1382	Che12	48331-49870	gp91(48465-48845) gp92(48842-49078) gp93(49075-49275) gp94(49283-49549) gp95(49546-49875)	Function not known
1408	Che12	41536-43207	gp73(41464-41589) gp74(41586-41861) gp75(41858-42052) gp76(42049-42288) gp77(42296-43138)	Function not known
66	Che12	30726-312205	gp49(30727-31155) gp50(31148-31360)	Function not known
1568	Che9d	37535-39202	gp61(37582-37863) gp62(38027-38224) gp63(38221-38409) gp64(38406-38750) gp65(38751-39245)	Function not known
1451	Bethlehem	49440-50222	gp78(49415-49714) gp79(49707-49886) gp80(49883-50152) gp81(50149-50352)	Function not known
934	Bethlehem	47015-47563	gp72(47038-47499) gp73(47496-47729)	Function not known
304	Bethlehem	35185-37345	gp46(35615-36106) gp47(36136-36468) gp48(36469-36651) gp49(36648-37439)	Function not known
311	Bethlehem	36173-37054	gp47(36136-36468) gp48(36469-36651) gp49(36648-37439)	Function not known
54	D29	47322-47854	gp88(46770-47492)	Function not known

Future Plans/directions

The following projects,being pursued in the lab, are in different stages of completion. 1) Involvement of Rho in transcription coupled repair process, iii) global analyses of Rho-dependent termination in different operons, iii) Testing efficacy of Psu, as an *E.coli* Rho inhibitor, iv) design of peptide-inhibitors from Psu and iv) characterization of different myco-bacteriocidal factors from mycobacteriophages.

Publications

1. Mishra S and Sen R (2015). N protein from lambdoid phages transform NusA into an antiterminator by modulating NusA-RNA polymerase flap domain interactions. ***Nucleic Acids Research.*** 43(12):5744-58.
2. Qayyum M. Z., Dey D. and Sen, R. (2016). Transcription elongation factor NusA is a negative regulator of Rho-dependent termination. ***Journal of Biological Chemistry,*** 291(15), 8090-8108.

अन्य वैज्ञानिक सेवाएँ / सुविधाएँ
Other Scientific Services / Facilities

LABORATORY ANIMAL FACILITY

Faculty Coordinators	Rashna Bhandari	Staff Scientist
	Sanjeev Khosla	Staff Scientist
Other Members	Hole Jayant Pundalik Rao	Officer In-Charge
	Sridhar Kavela	Technical Officer
	Sravani Edula	Technical Officer (Since July 2015)

Objectives

1. The main objective of the Laboratory Animal Facility (LAF) is to breed, maintain and supply laboratory animals to institutional scientists. Breeding and experimentation of all strains of mice is undertaken in individually ventilated caging systems;
2. To support research programmes that promote the health and well being of people and animals by facilitating high quality and scientifically sound research with animals;
3. To comply with regulatory government body (CPCSEA) requirements for animal experimentation and breeding; and
4. To maintain a stable and contained environment for animals and personnel working in the facility, to ensure consistent animal quality and reduce operational costs.

Summary of work done until the beginning of this reporting year (up to March 31, 2015)

The CDFD LAF started its activities on July 1, 2011, within the premises of M/s Vimta Labs Limited, located at Genome Valley, Shameerpet, Hyderabad. Infrastructure was established to house mice in individually ventilated cages (IVCs), and conduct standard experimental procedures.

All procedures conducted on animals housed in this facility are approved by the Institutional Animal Ethics Committee (IAEC) constituted by the Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA), Ministry of Environment, Forest and Climate Change (MoEF & CC), Govt. of India, at M/s Vimta Labs Ltd. Until March 2015, the facility housed approximately 900 mice of five different strains, and in 2014-15, users were supplied with 896 mice for IAEC approved experimentation.

Details of the progress made in the current reporting year (April 1,2015- March 31,2016)

During this reporting year, the CDFD LAF has housed five inbred mouse strains, including *Ip6k1*, *Nnat,* C57BL/6,*FoxNl*^{nu} and Balb/c. Mice were bred to expand the colonies and meet users' requirements. Currently this facility has approximately 629 adult and 226 newborn mice housed in 422I VC cages (Table 1). During the year, 891 mice were supplied to users for IAEC approved experimentation.

Routine IAEC approved procedures conducted on these animals include blood collection for measurement of biochemical parameters, embryo collection for the preparation of embryonic fibroblasts, tail biopsies for genotyping analysis and necropsy for histopathological analysis. Some of the experiments conducted during 2015-16 are highlighted below:

Strains	**Total** (Male+Female)	**Under Breeding** (Male+Female)	**Supplied during 2013-14**
Ip6k1	39+30	08+16	22
Nnat	76+71	06+06	11
Balb/c	66+73	09+18	634
C57BL/6	05+05	06+12	150
Foxn1^{nu}	03+01	08+16	74

Table 1. Strain-wise break up of adult mice housed at LAF as on March 31, 2016, and supplied to users during 2015-16.

- 236 Balb/c mice were injected intravenously with *Candida glabrata* for studies on comparative bio-burden of different C*andida strains.*
- 183 Balb/c mice were used to study the effect of *Mycobacterium tuberculosis* protein PPE18 on LPS-induced endotoxaemia.
- 115 C57BL/6 and 40 Balb/c mice were injected with thioglycolate by intra-peritoneal route for the generation of macrophages.
- 87 Balb/c and 35 C57BL/6 mice were injected with the non-pathogenic mycobacteria, *M. smegmatis,* expressing some candidate *Mtb* proteins, to study the in vivo immunomodulatory role of these proteins.
- 74 *FoxN1*^{nu} athymic mice were injected with oncogenic cell lines to study tumour progression and metastasis.
- 64 Balb/c mice and 4 Sprague Dawley rats were injected subcutaneously with protein antigens and polyclonal antibodies were generated successfully.
- 24 Balb/c mice were used to study vaginal bio-burden of *Candida glabrata* strains in Balb/c mice
- 22 *Ip6k1*mice were used for histopathological analysis.
- 11 *Nnat* mice were used for measurement of biochemical parameters

The IAEC approved projects in progress during this reporting year are mentioned in Table 2.

S. No.	Projects in progress
1	Functional analysis of Neuronatin's second intron by knockout strategy
2	Establishment and histopathological characterization of *Ip6k1* knockout mice - version 2
3	Signal transduction pathway in immune cells regulating their innate and effecter functions during oxidative stress
4	Protocol for comparative bio-burden study of fifteen strains of *Candida glabrata* in Balb/c mice
5	Immunization of Balb/c mice for generation of antibodies against few purified recombinant mycobacterial proteins
6	Studying the effect of PPE *I*18 (Rv1196) on LPS induced endotoxaemia in mice
7	Use of nude mice in the study of tumorigenesis
8	Protocol for generation of mouse /rat polyclonal antibodies - version 2
9	Isolation of macrophages from Balb/c mice
10	Establishment of transgenic mouse model to study the role of *Ip6k1* in tumorigenesis
11	Studying the immunomodulatory role of some candidate recombinantly purified proteins of mycobacteria
12	Studying the in vivo immunomodulatory role of some candidate PE/PPE proteins of *Mycobacterium tuberculosis* recombinantly over-expressed in the non-pathogenic mycobacterial strain of *M. smegmatis*
13	Studying the in vivo epigenetic role of some candidate proteins of *Mycobacterium tuberculosis* recombinantly over-expressed in the non-pathogenic mycobacterial strain of *M. smegmatis*
14	Protocol for testing tumorogenic and metastatic potential in nude mice
15	Investigating potential of *Mycobacterium tuberculosis* protein *PPE18* coated nano particles as therapy for microbial sepsis
16	Protocol for comparative vaginal bio-burden analysis of *Candida glabrata* strains in Balb/c mice
17	Protocol for comparative bio-burden analysis of *Candida glabrata* strains in C57BL/6 mice

Table 2. IAEC approved projects proposed by various groups at CDFD, in progress during 2015-16.

We are close to completion of CDFD's own Experimental Animal Facility which is under construction in the upcoming CDFD campus at Uppal, Hyderabad. We are working to ensure the facility's compliance with the CPCSEA preliminary inspection report received in June 2015. We look forward to the registration of this facility with CPCSEA, and the start of operations in the near future.

Figure 1

Figure 2

Figure 3

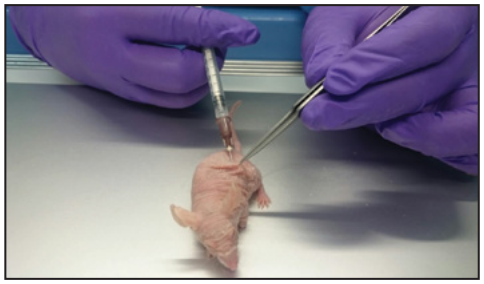

Figure 4

Figure 1. C57BL/6 female mouse with young pups. **Figure 2.** Balb/c female mouse with newborn pups. **Figure 3.** FoxN1^{nu} athymic nude mice generated at the CDFD Animal Facility. **Figure 4.** Subcutaneous injection of oncogenic cellsinto FoxN1^{nu} mice.

Future direction

Once the CDFD Experimental Animal Facility is operational, we aim to develop cryopreservation, archiving and retrieval of transgenic mouse strains for future use. Novel methods such as the CRISPR/Cas9 system will be developed to generate our own transgenic and knockout mice.

BIOINFORMATICS

Head	HA Nagarajaram	Staff Scientist
Other Members	R Chandra Mohan	Technical Officer
	Prashanthi Katta	Technical Assistant

Objectives

1. To maintain various servers, workstations, PCs, printers and other peripheral devices;
2. To maintain CDFD website, to provide web based services and e-mail services;
3. To maintain Institute-wide LAN as well as the internet connectivity;
4. To secure CDFD network from security threats;
5. To integrate Institute's network into National and International grid computing networks; and
6. To coordinate the procurement process of servers, workstations, PCs, laptops, printers, other peripheral devices and software required.

Summary of work done until the beginning of this reporting year (upto March 31, 2015)

- Activities related to installation, administration and maintenance of servers which provide various services, databases and computational jobs were undertaken.
- Internet, web, email-services were provided with enhanced functionalities.
- High-end PCs, workstations, laptops, scanners and printers were procured and installed.
- PC Annual Maintenance Contract was awarded to a new vendor M/s Accel Frontline Limited.
- Existing AMC of Zimbra email server with M/s CallippusSolutions Private Ltd. was renewed.
- Upgraded zimbra email server to the latest version.
- Coordinating the process of procurement and completed the installation setup of server with workstations and backup facility for CODIS project.
- Renewed the MoU with CDAC for availing GARUDA-grid facility.
- Procured next generation firewall and is currently getting installed.
- Upgraded the BSNL internet leased line bandwidth to 25Mbps.

Details of progress made in the current reporting year (April 1, 2015 - March 31, 2016)

- Activities related to installation, administration and maintenance of servers which provide various services, databases and computational jobs were undertaken.
- Internet, web, email-services have been provided with enhanced functionalities.
- Successfully commissioned and configured the newly procured Next-generation Firewall.
- High-end PCs, workstations, laptops, scanners and printers were procured and installed.
- Existing PC Annual Maintenance Contract with M/s Accel Frontline Limited was renewed.
- Stopped outsourcing the maintenance of Zimbra Email-Server and started in-house maintenance.
- Renewed Antivirus licenses -400 Nos. for 3 years.
- Procured Microsoft Office latest verions-2016 -100Nos. for installing/upgrading the existing versions.
- Procured two HighendSuperMicro workstationsfor Next Generation Sequencing Analysis.
- Initiated the process of procurement of servers, workstations and colour printers.
- Initiated the process of setting up of internet connection and Wi-Fi enabled local network facility at newly constructed student's hostel,Uppal.
- AMC for Dell Servers was awarded to M/s Dell International Services India Pvt. Ltd. for a period of one year.

INSTRUMENTATION

Head	Raghavendrachar J	Staff Scientist
Other Members	Members R N Mishra	Senior Technical Officer
	SD Varalaxmi	Technical Officer
	M Laxman	Technical Officer
	Satyanarayana RMK	Technical Officer
	T Ramakrishna Reddy	Tech. Assistant

Objectives

1. To maintain repair and service all the equipment in laboratory.
2. To provide pre-installation requirements for new instruments and to coordinate with the manufacturers / their agents in Installation and warranty service of the new instruments.
3. To provide the reports on the newly arrived instruments and to follow up with the suppliers for short shipped items.

Summary of work done until the beginning of this reporting year (upto March 31, 2015):

During the year 2014-15, we have installed 57 new equipments like Color Doppler Ultrasound Scanner at NIMS, Automatic Vertical Autoclaves, IP-Star Automated Robatic Work Stataion, Upright Microscopes, 2 Nos of Laser Scanning Confocal Microscopes, FLA 9500 Phosphor Imaging Sysytem, Bio-ruptors, PCR Machines, Refrigerated Centrifuges, Shaking waterbaths, -20^{0}C Freezers, Cooled Incubator, Refrigerators etc. and we have also completed 503 work orders for repair & maintenance of various laboratory equipments.

We were involved in re-organizing and installing the lab tables for the “Laboratory for Genomics and Profiling Applications” (LGPA) in the basement and install small equipments also.

We were involved in organizing the CODIS software installation and training to the DNA FP Lab at CDFD Library from 5th October 2014 to 12th October 2014.

Details of progress made in the current reporting year (April 1, 2015 – March 31, 2016)

During the year 2015-16, we have installed 59 new equipments like Automatic Vertical Autoclaves, Cytogenetics Workstation (Spectral Karyotyping system) Upright Microscopes, Inverted Fluorescence Microscope, Bio-ruptors, PCR Machines, Refrigerated Centrifuges, Shaking waterbaths, -86^{0}C Deep Freezers, -20^{0}C Freezers, Cold Cabinets, Cooled Incubator, Refrigerators etc. and we have also completed 335 work orders for repair & maintenance of various laboratory equipments.

We are involved in coordinating the operations of sophisticated instruments in CDFD through an Equipment operations outsourcing agency, M/s Sandor Life Sciences. We are also involved in coordinating with M/s Vimta Labs for CDFD animal experimentation facility in their facility at Shameerpet.

In addition, we were involved in organizing the audio & visual requirements for presentations in various seminars, lectures and workshops, CDFD Foundation day lecture at IICT auditorium, 30th DBT anniversary Lecture at IICT Auditorium, Distinguished Scientist Lectures. We have maintained most of the equipment with maximum uptime in the Laboratory. Most of the Instruments are maintained by our Instrumentation staff, thereby saving on the expensive AMCs and with very little downtime of the equipment.

प्रकाशन
Publications

RESEARCH PAPERS

* Publications of adjunct faculty of CDFD in which CDFD's affiliation is included.

** Work done elsewhere

A. Publications during the year 2015

1. Aggarwal S, Jain SJMN, Das Bhowmik A, Tandon A, and Dalal A (2015). Molecular studies on parents after autopsy identify recombinant GBA gene in a case of Gaucher disease with ichthyosis phenotype. ***American Journal of Medical Genetics Part A,*** 167: 2858-2860.

2. Aggarwal S, Kar A, Bland P, Kelsell D and Dalal A (2015). Novel ABCA12 mutations in harlequin ichthyosis: A journey from photo diagnosis to prenatal diagnosis. ***Gene*** 556: 254-256.

3. *Aggarwal S and Phadke SR (2015). Medical genetics and genomic medicine in India: current status and opportunities ahead. ***Molecular Genetics and Genomic Medicine*** 3: 160-171.

4. Ahmed A, Das A and Mukhopadhyay S (2015). Immunoregulatory functions and expression patterns of PE/PPE family members: roles in pathogenicity and impact on anti-tuberculosis vaccine and drug design. ***IUBMB Life*** 67: 414-427.

5. Uttarilli A, Ranganath P, Jain SJ, Prasad CK, Sinha A, Verma IC, Phadke SR, Puri RD, Danda S, Muranjan MN, Jevalikar G, Nagarajaram HA, Dalal AB (2015). Novel mutations of the arylsulphatase B (ARSB) gene in Indian patients with mucopolysaccharidosis type VI. ***Indian Journal of Medical Research*** 142: 414 – 425.

6. *Arora R, Aggarwal S and Deme S (2015). Ghosal hematodiaphyseal dysplasia-a concise review including an illustrative patient. ***Skeletal Radiology*** 44: 447-450.

7. Bashyam MD, Kotapalli V, Raman R, Chaudhary AK, Yadav BK, Gowrishankar S, Uppin SG, Kongara R, Sastry RA, Vamsy M, Patnaik S, Rao S, Dsouza S, Desai D and Tester A (2015). Evidence for presence of mismatch repair gene expression positive Lynch syndrome cases in India. ***Molecular Carcinogenesis*** 54: 1807-1814.

8. Bhat KH and Mukhopadhyay S (2015). Macrophage takeover and the host-bacilli interplay during tuberculosis. ***Future Microbiology*** 10: 853-872.

9. Bhavani GS, Shah H, Dalal A, Shukla A, Danda S, Aggarwal S, Phadke SR, Gupta N, Kabra M, Gowrishankar K, Gupta A, Bhat M, Puri RD, Bijarnia-Mahay S, Nampoothiri S, Mohanasundaram KM, Rajeswari S, Kulkarni AM, Kulkarni ML, Ranganath P, Radha Rama Devi A, Hariharan SV and Girisha KM (2015). Novel and recurrent mutations inWISP3and an atypical phenotype. ***American Journal of Medical Genetics Part A,*** 167: 2481-2484.

10. Bidchol AM, Dalal A, Trivedi R, Shukla A, Nampoothiri S, Sankar VH, Danda S, Gupta N, Kabra M, Hebbar SA, Bhat RY, Matta D, Ekbote AV, Puri RD, Phadke SR, Gowrishankar K, Aggarwal S, Ranganath P, Sharda S, Kamate M, Datar CA, Bhat K, Kamath N, Shah H, Krishna S, Gopinath PM, Verma IC, Nagarajaram HA, Satyamoorthy K and Girisha KM (2015). Recurrent and novel GLB1 mutations in India. ***Gene*** 567: 173-181.

11. Chakraborty S, Muthlakshmi M, Vardhini D, Jayaprakash J, Nagaraju J and Arunkumar KP (2015). Genetic analysis of Indian tasarsilkmoth (Antheraea mylitta) populations. ***Scientific Reports*** 5: 15728.

12. Chen Z, Nohata J, Guo H, Li S, Liu J, Guo Y, Yamamoto K, Kadono-Okuda K, Liu C, Arunkumar KP, Nagaraju J, Zhang Y, Liu S, Labropoulou V, Swevers L, Tsitoura P, Iatrou K, Gopinathan K, Goldsmith M, Xia Q and Mita K (2015). A comprehensive analysis of the chorion locus in silkmoth. ***Scientific Reports*** 5: 16424.

13. Chen Z, Nohata J, Guo H, Li S, Liu J, Guo Y, Yamamoto K, Kadono-Okuda K, Liu C, Arunkumar KP, Nagaraju J, Zhang Y, Liu S, Labropoulou V, Swevers L, Tsitoura P, Iatrou K, Gopinathan KP, Goldsmith MR, Xia Q and Mita K (2015). Construction, complete sequence, and annotation of a

BAC contig covering the silkworm chorion locus. ***Scientific Data*** 2: 150062.

14. Dalal A, Aneek Das Bhowmik, Divya Agrawal and Phadke SR (2015). Exome sequencing and homozygosity mapping help in identification of genetic etiology for spastic ataxia in a consanguineous family. ***Indian Journal of Medical Research.***142: 220-224.

15. Dalal AB, Ranganath P, Phadke SR, Kabra M, Danda S, Puri RD, Sankar VH, Gupta N, Patil SJ, Mandal K, Tamhankar P, Aggarwal S and Agarwal M (2015). Prenatal diagnosis in India is not limited to sex selection. ***Genetics in Medicine*** 17: 88.

16. Das Bhowmik A, and Dalal A (2015). Whole exome sequencing identifies a novel frameshift mutation in GPC3 gene in a patient with overgrowth syndrome. ***Gene,*** 572: 303-306.

17. Das Bhowmik A, Rangaswamaiah S, Srinivas G and Dalal AB (2015). Molecular genetic analysis of trinucleotide repeat disorders (TRDs) in Indian population and application of repeat primed PCR. ***European Journal of Medical Genetics*** 58: 160-167.

18 Delma CR, Somasundaram ST, Srinivasan GP, Khursheed M, Bashyam MD and Aravindan N (2015). Fucoidan from Turbinaria conoides: a multifaceted 'deliverable' to combat pancreatic cancer progression. ***International Journal of Biological Macromolecules*** 74: 447-457.

19. Dutta UR, Hansmann I and Schlote D (2015). Molecular cytogenetic characterization of a familial pericentric inversion 3 associated with short stature. ***European Journal of Medical Genetics*** 58: 154-159.

20. Gadipally SR, Sarkar A and Nandineni MR (2015). Selective enrichment of STRs for applications in forensic human identification. ***Electrophoresis*** 36: 1768-1774.

21. Ghosh C, Prakash NR, Manna SK, Bishayi B. (2015).Presence of toll like receptor-2 in spleen, lymph node and thymus of Swiss albino mice and its modulation by *Staphylococcus aureus* and bacterial lipopolysaccharide. ***Indian Journal of Experimental Biology*** 53: 82-92.

22. Giri DA, Rekha S and Kasbekar DP (2015). Neurospora heterokaryons with complementary duplications and deficiencies in their constituent nuclei provide an approach to identify nucleus-limited genes. ***G3: Genes, Genomes, Genetics*** 5: 1263-1272.

23. Gowrishankar J (2015). End of the beginning: elongation and termination features of alternative modes of chromosomal replication initiation in bacteria. ***PLoS Genetics***11: e1004909.

24. Gupta AK, Mita K, Arunkumar KP and Nagaraju J (2015). Molecular architecture of silk fibroin of Indian golden silkmoth, *Antheraea assama.* ***Nature Scientific Reports*** 5: 12706.

25. Gupta A, Uttarilli A, Dalal A and Girisha KM (2015). Hunter syndrome with late age of presentation: clinical description of a case and review of the literature. ***BMJ Case Reports*** 14: 2015pii: bcr2015209305.

26. Gupta N, Benjamin, Kar A, Munjal SD, Sarangi AN, Dalal A, and Aggarwal R (2015). Identification of Promotor and Exonic Variations, and Functional Characterization of a Splice Site Mutation in Indian Patients with Unconjugated Hyperbilirubinemia. ***PLoS One*** 10: e0145967.

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B. Publications in 2016 (Till March 31, 2016)

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C. Publications in press (as on March 31, 2016)

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80. Chaudhary AK, Mohapatra R, Nagarajaram HA, P Ranganath, Dalal A, Dutta A, Danda S, Girisha K and Bashyam MD. The novel missense EDAR p.L397H mutation causes autosomal dominant hypohidrotic ectodermal dysplasia. ***Journal of the European Academy of Dermatology & Venereology***

81. Dutta, U. The history of Human cytogenetics in India. A review. ***Gene***

82. Girisha KM, Kortüm F, Shah H, Alawi M, Dalal A, Bhavani GS and Kutsche K. A novel multiple joint dislocation syndrome associated with a homozygous nonsense variant in the EXOC6B gene. ***European Journal of Human Genetics.*** 2015

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85. **Mandal K, Ray S, Saxena D, Srivastava P, Moirangthem A, Ranganath P, Gupta M, Mukhopadhyay S, Kabra M, Phadke SR. Pycnodysostosis: mutation spectrum in five unrelated Indian children. ***Clinical Dysmorphology***

86. Nazir A, and Harinarayanan R. Inactivation of cell division protein FtsZ by SulA makes Lon indispensable for the viability of ppGpp0 strain of *Escherichia coli.* ***Journal of Bacteriology***

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89. Vimala A and Harinarayanan R. Transketolase activity modulates glycerol-3-phosphate levels in *Escherichia coli.* ***Molecular Microbiology.***

90. Qayyum M. Z., Dey D. and Sen, R. (2016). Transcription elongation factor NusA is a negative regulator of Rho-dependent termination. ***Journal of Biological Chemistry,*** 291(15), 8090-8108.

D. Other Publications

91. Aggarwal S and Dalal A (2015). Chromosomal disorders. In: Postgraduate Textbook of Pediatrics. Ed. Piyush Gupta. ***Jaypee Brothers Medical Publishers*** 36-46.

92. Arunkumar KP and Sambrani N (2015). Book review of the *Annual Review of Genetics* 2014, Bonnie Bassler et al., (eds) ***Current Science***109: 2137-2139.

93. Dalal A (2015). Niemann Pick disease. In: Postgraduate Textbook of Pediatrics. Ed. Piyush Gupta. ***Jaypee Brothers Medical Publishers*** 118 -122.

94. Gowrishankar J and Nandineni MR (2016). Why India is rooting for its DNA identification Act. ***Nature India*** doi:10.1038/nindia.2016.47.

95. *Gupta D, Gupta V, Singh V, Chawla S, Ranganath P and Phadke SR (2015). Study of polymorphisms in CFH, ARMS2 and HTRA1 genes as potential risk factors for age-related macular degeneration in Indian patients. ***International Journal of Bioassays*** 4: 3747- 3752.

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97. Maheshwar L, Ranganath P, Chilakamarri VK, Vanaja MC and Dalal AB (2015). A typical Stone Man syndrome: case report and literature review. ***Journal of Medical Science and Clinical Research*** 3: 6423-6429.

98. *Maheshwar L, Chilakamarri VK,Ranganath P, Arora AJ and Vanaja MC (2015). Clinical and Genetic Analysis of Fibrodysplasia Ossificans Progressiva: A Case Report and Literature Review. ***Journal of Clinical and Diagnostic Research*** 9: RD01-RD03.

99. *Ranganath P (2015). MicroRNA-155 and its role in malignant hematopoiesis. ***Biomarker Insights*** 10: 95-102.

100. **Ranganath P (2015). Patterns of Inheritance. In: Postgraduate Textbook of Pediatrics. Ed. Piyush Gupta. ***Jaypee Brothers Medical Publishers*** 24-31.

101. Bhavani GSL, Shah H, Shukla A, Dalal A, Girisha KM. Progressive Pseudorheumatoid Dysplasia (2016). In: Pagon RA, Adam MP, Ardinger HH, Wallace SE, Amemiya A, Bean LJH, Bird TD, Fong CT, Mefford HC, Smith RJH, Stephens K, editors. ***Gene Reviews***® [Internet]. Seattle (WA): University of Washington, Seattle; 1993-2016.

102. Haldar D (2016). Emerging epigenetic therapy of cancer. ***Spinco Biotech Cutting Edge*** 5: 9-14.

103. Kasbekar, DP (2016) Editorial. Long-drawn-out story. ***Journal of Biosciences*** 41: 1

104. Kasbekar DP (2016).Obaid Siddiqi's study of the PABA1 gene of the fungus *Aspergillus nidulans.* ***INSA Special Volume on Obaid Siddiqi.***

105. **Vijayalakshmi SR and Ranganath P (2016). An approach to genetic disorders affecting the white matter. ***Genetic Clinics*** (Official publication of Indian Academy of Medical Genetics) 9: 15-29.

106. Satyavathi VV and Raju PJ (2016). RNAi may subserve KS-10. Opinion of Experts on KS-10, the inhibitor of diapause breed of silkworm, *Bombyx mori,* L. ***KSSRDI Technical Publications*** 123: 79-80.

107. Khosla S, Sharma G and Yaseen I. Learning epigenetic regulation from mycobacteria. **Microbial Cell** (in press).

108. **Ranganath P. Thalassemia in the fetus-prenatal diagnosis. Fetal and Neonatal Hematology and Oncology. Eds. M R Lokeshwar, AnupamSachdeva. ***Jaypee Brothers Medical Publishers*** (in press).

E. Patents filed/granted

Mukhopadhyay S and Ahmed A.
A novel therapeutic to treat sepsis.
Indian Patent filed in December 2015

मानव संसाधन विकास
Human Resource Development

PhD Program

For the PhD program CDFD invites applications from highly motivated candidates willing to take up challenges in modern biology, usually in the month of March. Keeping in view the interdisciplinary nature of modern biology, the Centre especially encourages persons from different scientific disciplines to take up challenges in these areas. Those admitted as Junior Research Fellows (JRFs) are encouraged to take admission in the PhD program of Manipal University or University of Hyderabad.

The eligibility for the program is MBBS or Masters degree in any branch of Science, Technology or Agriculture from a recognized University or Institute. Candidates (other than MBBS graduates) must have cleared National Eligibility Test (NET) with valid CSIR-JRF or UGC-JRF or DBT-JRF or ICMR-JRF or ICAR-JRF or INSPIRE-PhD or JEST or GATE (All India top 50 ranks of all Chemistry, Life Sciences and Biotechnology streams). Those who have appeared for their final semester examination, but are awaiting results, are also eligible to apply. Those with independent Senior Research Fellowships (SRF) from CSIR can also apply. As the number of applicants outnumbers the seats available each year by a ratio of 1:40 or more, eligible candidates are invited for a written examination followed by interviews of short-listed candidates.

As of March 31, 2016 the Centre has 106 Research Scholars working for their doctorates in different areas of research. In the reporting year 9 of the Research Scholars have completed PhD and are pursuing careers in science elsewhere in India or abroad.

Postdoctoral Program

In addition to the JRF program, the Centre also carries out training at the post-doctoral level. The post-doctoral fellows are funded through the extramural grants that CDFD receives. Some post-doctoral fellows are also selected competitively by the DST fast track young scientist scheme, or the DBT post-doctoral fellowship program.

Summer Training Program

CDFD provides admissions to summer training program to those students who are supported either by the Indian Academy of Science, Bangalore or Jawaharlal Nehru Centre for Advanced Scientific Research, Bangalore or the Kishore Vigyanik Protsahan Yojna, New Delhi. In the reporting year 21 students received summer training at the Centre.

Training for students from BITS, Pilani

CDFD has an agreement with BITS, Pilani to provide project training to their M.Sc. students. Under this programme, the students spend 6 months to 1 year at CDFD and work on active projects being carried out here. The project work helps the students in gaining hands-on experience in modern biology. In the reporting year, 3 students were given the opportunity to avail training under this programme.

Research Scholars Conferred PhD Degree During 2015 - 2016

Scholar	Supervisor	Date of *viva voce* examination	Title of thesis
Saurabh Mishra	Dr. Ranjan Sen	29.01.2015	"Studies on the transription elongation factor NusA from EColi"
Manjari Kiran	Dr. H A Nagarajaram	03.02.2015	"Local and Global hubs in humans protein-protein interaction network"
Babul Moni Ram	Dr. Gayatri Ramakrishna	03.09.2015	"Studies on Calcineurin - NFAT Signaling in cellular proliferation and effect of its inhibitors, cyclosporine A, in Cell death response"
Swarna Gowri Thota	Dr. Rashna Bhandari	15.09.2015	"Role of inositol pyropohosphates in yeast physiology"
Suhail Yousuf	Dr. Akash Ranjan	19.10.2015	"Charcaterization and functional studies on FadR like proteins from M. tuberculosis"
Rachita H R	Dr. H A Nagarajaram	20.10.2015	"A Study on human - virus protein - protein interaction networks"
Neelam Chaudhary	Dr. M V Subba Reddy	03.03.2016	"Studies on functional interactome of WWP2: An HECT Ubiquitin E3 ligase"
Garima Sharma	Dr. Sanjeev Khosla	07.03.2016	" Host epigenetic response to *Mycobacterium tuberculosis* infection"
Rikky Rai	Dr. Subhadeep Chatterjee	08.03.2016	"Understanding the role of DSF (Diffusible Signalling Factor) in virulence of Xanthomonas plant pathogens"

पुरस्कार एवं सम्मान
Awards and Honours

AWARDS & HONOURS

FACULTY & STAFF	
Dr Arun Kumar KP	Selected as Founding Member of the Indian Young Academy of Science (INYAS) by INSA Council
Dr Rupinder Kaur	1) Wellcome Trust/DBT India Alliance Senior Fellowship 2) National Women Bioscientist award under Young Category for the year 2014 by Department of Biotechnology 3) Selected as member of Microbiology Board of Reviewers for Microbiology Society Journal, UK 4) Selected as a member of Editorial Board of the Journal of Biosciences
Dr Sangita Mukhopadhyay	1) Elected as a Fellow of the Indian National Science Academy at Annual General Meeting, New Delhi on October 14, 2015 2) Elected as Member, Guha Research Conference (GRC)
Dr Mohan Chandra Joshi	Selected for Ramalingaswami Fellowship 2014-15 by Department of Biotechnology, New Delhi
CDFD Cricket Team Dr. M Subba Reddy (Captain) Dr. Mohan Chandra Joshi, Dr. R. Nagender Rao (Man of the Match), Dr. Rajendra, Mr. Vivek Reddy, Mr. Zaffar, Mr. Mudassir, Mr. Sridhar, Mr. Mayank, Mr Dev Ashish Giri, Mr Kaushik, Mr Surya, Mr Vivek, Mr Parveen, Mr Zuhaib	Runners Up Trophy in the Cricket Tournament at CSIR- IICT (November 27-29, 2015)
Dr Subhadeep Chatterjee	Selected as an associate editor in Phytopathology an International Journal of the American Phytopathological Society (APS) for Three years (2015-2017)
Dr Rashna Bhandari	Awarded an International Research Grant by Human Frontier Science Program (HFSP) along with co-applicants Henning Jessen (Germany) and Paul Wender (USA)

AWARDS & HONOURS

PhD STUDENTS & PROJECT PERSONNEL	
Vivek Kumar Srivastava	Poster prize at the Gordon Research Conference on 'Cell Biology of Metals' held in USA in July, 2015
Shailesh Kumar Gupta	Third prize in poster presentation at World Congress on Microscopy 2015 held at Mahatma Gandhi University, Kerala from October 9-11 2015
Rajendra Kumar Angara	Third prize in poster presentation at World Congress on Microscopy 2015 held at Mahatma Gandhi University, Kerala from October 9-11 2015
Gourang Pradhan	Dr G.P. Talwar Young Scientist award - 2015 by Indian Immunology Society, Patna
Neeharika Verma	EMBO travel grant at International conference "Autophagy signalling and progression in health and disease by EMBO at Chia, Italy
Mr Aamir Ali	Travel Grant from SERB to attend American Society for Cell Biology Annual meeting at California, USA from December 12-16, 2015
Ms Shweta Singh	Appreciation award for Poster Competition in "IKMC 2015: Spreading the Innovation Spirit" Conference at HICC, Hyderabad from November 2-3, 2015
Mr Debasish Kumar Ghosh	Poster award organized by Centre for Brain Research, Indian Institute of Science, Bangalore, Bangalore from November 16-18, 2015
Mr Dev Ashish Giri Ms S Rekha Ms K Sreethi Reddy (Group Head Dr D P Kasbekar)	Second prize in the poster competition at the Asian Mycological Congress 2015, Goa (October 7-10, 2015)
Dr Aneek Das Bhowmik	Received the funding for the project under Young Scientist Scheme of Science and Engineering Research Board (SERB)

व्याख्यान, बैठक, कार्यशाला व अन्य महत्वपूर्ण कार्यक्रम

Lectures, Meetings, Workshops and Important Events

DISTINGUISHED VISITORS AND LECTURES

Visitor	Title of Lecture	Date
Dr Aprotim Mazumder TCIS, Hyderabad	Measuring the heterogeneity in DNA damage responses, from yeast to mice, with High Content and High Resolution Imaging	15.04.2015
Prof Amitabha Chattopadhyay CCMB, Uppal Road, Hyderabad	Interaction of Membrane Cholesterol with G Protein-Coupled Receptors: Novel Insights in Health & Disease	16.04.2015
Prof Avery August (2015 ASM-IUSSTF Indo-US Research Professor), Dept. of Microbiology & Immunology, Cornell University, Ithaca, New York	Tuning T Cell Behavior	28.04.2015
Dr Chitra P National Centre for Biological, TIFR, GKVK Campus, Bangalore	Designing an integrated platform for Pathogen Discovery	05.05.2015
Dr Smarajit Polley Department of Chemistry and Biochemistry, University of California San Diego, San Diego, USA	An Autocatalytic Functional Switch in IKK2/beta: A New Paradigm in Kinase Regulation	06.05.2015
Dr Punit Prasad Karolinska Institute Department of Bioscience and Nutrition NOVUM, Halsovagen 7StockholmSweden	Modulation of chromatin structure by Chromatin Remodeling Complexes: Mechanisms, Consequences and Implications	17.08.2015
Dr. Nikhil Jain Department of Molecular Virology and Microbiology Baylor College of Medicine One Baylor Plaza, Houston	Role of accessory factors in assembling ribosome	24.09.2015
Mr R Vijay Kumar ARCI, Hyderabad	New Pension Scheme	30.09.2015
Dr Vinay Tergaonkar IMCB-Singapore	Mechanism of TERT promoter reactivation in cancer	05.10.2015
Dr Mohan Chandra Joshi Laboratory of Chromosome Structure & Dynamics, CDFD	Bacterial Nucleoid revisited: (a) Twist of cohesion during chromosome segregation (b) Dynamics of chromosome organization	06.10.2015

Visitor	Title of Lecture	Date
Dr Kiran Batta Stem Cell Biology Group Cancer Research UK Manchester Institute The University of Manchester Wilmslow Road, Manchester	Making blood	12.10.2015
Dr Gopalakrishnan Aneeshkumar Arimbasseri, National Institute of Child Health and Human Development, National Institutes of Health, Bethesda, Maryland, USA	Balancing tRNA synthetic rate and modification-dependent activity for condition-appropriate translation	10.11.2015
Dr Debabrata Chakravarti Freinber Cancer Center and Nothwestern University Chicago, USA	Integrating epigenomics and nuclear hormone signaling in cancer and tissue fibrosis	23.11.2015
Dr Aashiq H Kachroo The University of Texas at AustinAustin, USA	Saccharomyces sapiens – Towards humanizing yeast	26.11.2015
Dr Sathees Raghavan Department of Biochemistry IISc, Bangalore	DNA Breaks to Repair: Insights into Oncogenesis and Cancer Therapy	30.11.2015
Dr Tim Schellberg President, Thomas Gordon Honeywell (Governmental Affairs), USA	Global update on DNA databases and legislative trends in databasing	08.12.2015
Dr Sanjeev Gupta Co-ordinator of H&D II School of Medicine, NUI Galway, Galway, Ireland	MicroRNAs in Unfolded Protein Response: Small regulators with a big impact	10.12.2015
Premas Life Sciences IMT Manesar, Gurgaon	Applications of Next Generation Sequencing in Forensic Genomics	15.12.2015
Dr Manish Jaiswal Baylor College of Medicine Houston, TX USA	Genetic dissection of neuronal maintenance and demise	08.01.2016
Dr Suvendra N Bhattacharyya Principal Scientist and Head Molecular Genetics Department CSIR-IICB, Kolkata	Regulation of miRNA activity in mammalian cells: Role of different intrinsic and extrinsic factors	11.01.2016

Visitor	Title of Lecture	Date
Mr Gopal Singh and **Mr. Ketan Shevatakar** Vikalp Social and Charitable TrustNagpur, Maharashtra	Stress management and naturopathy	12.02.2016
Dr Bama Charan Mondal University of California Los Angeles, USA	Homeostatic control mechanisms during Drosophila hematopoietic progenitor maintenance	16.02.2016
Dr Arjumand Ghazi Assistant Professor University of Pittsburg, USA	Fat, Fertility and Aging Worms	22.02.2016
Prof Toru Shimada University of Tokyo, Japan	Evolutionary genomics on host plant selection in bombycoid silkmoths	23.02.2016
Dr Srini Kaveri Director CNRS Office in India French Embassy Service for Science and Technology New Delhi	Therapeutic Antibodies : Acentury-long fascinating journey	02.03.2016
Dr Sorab Dalal Principal Investigator ACTREC Associate Professor HBNI, KS215, ACTREC, Tata Memorial Centre Kharghar Node, Navi Mumbai	14-3-3 ligand interactions as possible drug targets	03.03.2016
Dr Shivashankar Nagaraj Queensland University of Technology (QUT)Australia	A Systems Biology approach to elucidate Epithelial-Mesenchymal Transition(EMT) in cancer	22.03.2016

IMPORTANT EVENTS

Event	Date
Visit of Shri Tuhin Kanta Pandey, Joint Secretary, Cabinet Secretariat, New Delhi	16.05.2015
Anti-Terrorism Day	21.05.2015
37th Meeting of CDFD Governing Council	25.05.2015
31st meeting of the Finance Committee	25.05.2015
Celebrations of Digital India Week during 1-7 July 2015 (launched by our Hon'ble Prime Minister CDFD Quiz competition on Information Technology Awareness on 7.7.2015 in Seminar Hall, Tuljaguda)	07.07.2015
Indian Society of Developmental Biologists Biennial (InSDB-2015) meeting jointly by CDFD and CCMB	15.07.2015 to 18.07.2015
Hon'ble President of India's address to the Students and faculty members of Institutes of higher learning through Videoconference using NKN	10.08.2015
38th Meeting of CDFD Governing Council	13.08.2015
Independence Day celebrations	15.08.2015
Visit of Prof Sheel Nuna, Director, South Asia, Queensland University of Technology, Australia, Prof Peter Coaldrake, VC, Prof Ross Young, Dean, Faculty of Health and Prof Gordon Wyeth, Dean, Faculty of Science and Engineering (QUT group)	18.08.2015
Sadbhavana Diwas Pledge	20.08.2015
17th meeting of CDFD Research Area Panels-Scientific Advisory Committee (RAP-SAC)	21.08.2015 to 22.08.2015
Hindi Workshop on use Digital Tools in Rajbhasha Implementation	07.09.2015
Hindi Pakhwada Celebrations	01.09.2015 to 14.09.2015
National Sanitation Campaign	25.09.2015 to 31.10.2015

IMPORTANT EVENTS

Event Partnering Institutions	Date
Visit of students from Centre of Excellence in Biotechnology, M.P. Council of Science and Technology (MPCOST), (Deptt. Of Science & Technology, Govt. of M.P.), Vigyan Bhawan, Nehru Nagar, Bhopal	07.10.2015
Visit of Dr Harsh Vardhan, Hon'ble Minister of Science & Technology and Earth Sciences	12.10.2015
30th Year of DBT Celebration (Public lecture by Prof Ranajit Chakraborty, Department of Molecular and Medical Genetics, University of North Texas, Health Science Center, Texas)	09.11.2015
39th Meeting of CDFD Governing Council	17.11.2016
32nd Meeting of CDFD Finance Committee	17.11.2015
20th Annual General Body Meeting of Society of CDFD	28.11.2015
Premas Biotech and Illumina seminar series "Applications of NGS in Forensic Genomics" by Prof. Bruce Budowle from UNTHSC, Texas and Dr Thangaraj, CCMB, Hyderabad.	15.12.2015
"Bioinformatics for scientists" workshop conducted by Dr Ansuman Chattopadhay from University of Pittsburgh, USA	07.01.2015 to 08.01.2015
(Address by Hon'ble President of India to students / faculty of Institutes through Video-Conference using NKN on Topic-"Youth and Nation Building"	19.01.2016
Republic Day celebrations	26.01.2016
30th Year of DBT Celebration (Public lecture by Prof David Reich, Department of Genetics, Harvard Medical School, USA)	28.01.2016
CDFD Foundation Day 2016	30.01.2016
Lecture on Stress management and naturopathy by Vikalp Social and Charitable Trust, Nagpur, Maharashtra in Hindi.	12.02.2016
40th Meeting of CDFD Governing Council	18.02.2016
33rd Meeting of the CDFD Finance Committee	18.02.2016
MoU Signed with Sickle Cell Institute Chhattisgarh, Raipur	23.02.2016
Series of video-conference talks in partnership with EMBO on 'Tips on how to write a paper' by Dr Karin Dumstrei, Senior Editor, EMBO journal	15.03.2016

सी डी एफ डी कर्मचारियों की विदेशों में प्रतिनियुक्ति
Deputations Abroad of CDFD Personnel

DEPUTATIONS ABROAD - FACULTY & STAFF

Faculty/Staff	Period	Country of Visit and Purpose
Giriraj R Chandak Director (w.e.f. 27.10.2015)	12.01.2016 to 13.01.2016	**Bangladesh:** To attend the "*Genomic and lifestyle predictors of foetal outcome relevant to diabetes and obesity and their relevance to prevention strategies in South Asian people*" (GIFTS) final conference being organized by the Bangladesh University of Health Science.
J Gowrishankar	26.05.2015 to 10.06.2015	**France:** To visit the laboratories of French Principal Collaborators Dr Sylvie Rimsky at ENS, Cachan, and Dr Philippe Bouloc at Institute for integrative Biology of the Cell (12BC), CNRS, in connection with the implementation of his DST-ANR research project titled "Ünravelling new functions for the H-NS family of proteins in Gram-negative bacterial pathogens", being co-ordinated by the Indo-French Centre for the Promotion of Advanced Research (IFCPAR).
	31.07.2015 to 11.08.2015	**USA:** (i) To visit the laboratories of Profs Max Gottesman, Evgeny Nudler and Anuradha Janakiraman in New York on 31 July 2015 and 3 August 2015. (ii) To attend the "2015 Molecular Genetics of Bacteria and Phages Meeting" at of University Wisconsin, Madison, Wisconsin, USA. (iii) To visit the laboratory of Prof Andrei Kuzminov, University of Illinois at Urbana-Champaign.
Ranjan Sen	03.08.2015 to 09.08.2015	**USA:** To attend the "2015 Molecular Genetics of Bacteria and Phages Meeting" held at University of Wisonsin, Madison, USA.
Nagarajaram H A	19.07.2015 to 26.07.2015	**Portugal:** To attend the II HCV – meeting cum exchange visit as a part of New INDIGO project "An attractive and promising strategy for early cancer diagnosis through the assembly of the human cancer volatome" held at University of Madeira, Madeira Island, Protugal.
Rupinder Kaur	14.05.2015 to 24.05.2015	**France:** To participate as plenary lecturer in the 6th FEBS Advanced Lecture Course on Human Fungal Pathogens held at La Colle-sur-Loup, France.

Faculty/Staff	Period	Country of Visit and Purpose
Ashwin B Dalal	05.06.2015 to 11.07.2015	**UK:** 1. To participate and present his work in the European Society of Human Genetics, Annual Meeting held in Glasgow, UK 2. To visit the Laboratory of Dr Andrew Jackson, MRC Human Genetics Unit, MRC, IGMM, University of Edinburgh, Edinburgh, UK
	14.12.2015 to 20.12.2015	**Sri Lanka:** To attend the International Neuroscience Workshop as faculty. Meeting held at University of Sri Jayewardenepura (USJP), Colombo, Sri Lanka.
N Madhusudan Reddy	11.5.2015 to 21.06.2015	**Germany:** To conduct research as Guest Scientist in the laboratory of Prof Mark Stoneking, Department of Evolutionary Genetics, Max Planck Institute for Evolutionary Anthropology (MPI-EVA). Leipzig, Germany against his fifth visit to Prof Mark Stoneking's Laboratory as a part of the "Max Planck Partner Group Programme" (MPPGP) between CDFD and MPI-EVA awarded by the Max Planck Society, Germany.
	08.09.2015 to 17.09.2015	**UK:** To attend the short course in Forensic Genetics held at University of Central Lancashire, Preston, United Kingdom
	12.10.2015 to 18.10.2015	**USA:** 1. To attend and present recent research findings with autosomal and Y-chromosomal STR markers in Indian populations in the form of a poster at the 26th International Symposium on Human Identification (ISHI) held at Gaylord Texan Resort and Convention Center in Grapevine, Texas, USA. 2. To visit Prof Arthur Eisenberg, Director, DNA Identification Laboratory at the Department of Molecular and Medical Genetics at the University of North Texas, Health Science Centre (UNTHSC).
M V Subba Reddy	23.06.2015 to 01.07.2015	**Finland**: To attend the EMBO conference on Europhosphatase 2015:Phosphorylation switches and cellular homeostasis" held at Turku, Finland
	05.07.2015 to 10.07.2015	**Hong Kong:** To attend Gordon Research Conference on "Posttranslational modification networks" held at the Hong Kong University of Science and Technology, Hong Kong, China.

Faculty/Staff	Period	Country of Visit and Purpose
Subhadeep Chatterjee	02.08.2015 to 12.08.2015	**USA:** 1. To attend and present his work on plant-microbe interaction in the conference titled "2015 Molecular Genetics of Bacteria and Phages Meeting held at University of Wisconsin, Madison, USA. 2. To visit the Laboratory of Prof Steven E Lindow's at University of California, Berkeley (Near San Francisco, USA) for exploring future collaboration and scientific discussion.
	28.10.2015 to 04.11.2015	**China**: On the invitation of Dr Ya-Wen He, Vice Dean, Department of Microbiology, Shanghai Jiao Tong University (SJTU), Shanghai, China for academic discussion and possible collaboration and also to deliver a seminar at the Department of Microbiology
Arun Kumar K P	05.07.2015 to 11.07.2015	Austria: To attend the First Research Coordination Meeting (RCM) on "Comparing rearing efficiency and competitiveness of sterile male strains produced by genetic, transgenic or symbiont-based technologies: held at Joint FAO/IAEA Division of Nuclear Techniques in Food and Agriculture, Vienna International Centre, Vienna, Austria.
	01.10.2015 to 06.10.2015	**Japan:** 1. To participate in the special event "Dialog between Nobel Laureates and Young Leaders" and orientation and 2. To attend the 12th Annual Meeting of the Science and Technology in Society (STS) forum
	09.11.2015 to 23.11.2015	**Japan:** To visit University of Tokyo, Tokyo and National Institute of Agrobiological Sciences, Tsukuba under the joint project entitled "Collaborative studies on genomic diversity among bombycoid silkmoths in Asia" for a first exchange visit under Indo-Japan Collaborative Research Project.
Annapurna Bhavani	09.11.2015 to 23.11.2015	**Japan:** To visit University of Tokyo, Tokyo and National Institute of Agrobiological Sciences, Tsukuba under the joint project entitled "Collaborative studies on genomic diversity among bombycoid silk moths in Asia" for a first exchange visit under Indo-Japan Collaborative Research Project.
Venkata Satyavathi	18.09.2015 to 21.09.2015	**Bangladesh:** To participate in the 3rd Annual "South Asia Biosafety Conference" held at BRAC Centre Inn, Dhaka, Bangladesh.

DEPUTATIONS ABROAD - STUDENTS

Name of the Scholar	Period	Country of Visit and Purpose
Suhail Yousuf	30.05.2015 to 02.06.2015	**USA**: To attend the Conference "asm2015" 115th General Meeting of American Society for Microbiology
Ajit Roy	30.05.2015 to 02.06.2015	**USA**: To attend the Conference "asm2015" 115th General Meeting of American Society for Microbiology
Soumya Rao	08.06.2015 to 22.07.2015	**Germany:** Visiting Scholar / Guest Researcher to conduct research as a part of "Max Planck Partner Group Programme"
Swapnil Rohidas Shinde	24.06.2015 to 29.06.2015	**Finland:** To attend EMBO conference on "Europhosphatase 2015: Phosphorylation switches and cellular homeostasis"
Chanduri Venkata Lakshmi Manasa	11.07.2015 to 17.07.2015	**USA**: To attend Gordon Research Seminar and Conference on "Molecular Membrane Biology"
Vivek Kumar Srivastava	26.07.2015 to 31.07.2015	**USA**: To attend Gordon Research Conference on "Cell Biology of Metals"
Mohd. Zuhaib Qayyum	04.08.2015 to 08.08.2015	**USA:** To attend 2015 Molecular Genetics of Bacteria and Phages Meeting
Gajula Gopinath	18.08.2015 to 22.08.2015	**USA**:To attend CSHL meeting on "EUKARYOTIC mRNA PROCESSING"
Rachana Roshan Dev	18.08.2015 to 22.08.2015	**USA:** To attend CSHL meeting on "EUKARYOTIC mRNA PROCESSING"
Aushaq Bashir Malla	30.08.2015 to 04.09.2015	**UK:** To attend EMBO Meiosis conference - 2015
Neeharika Verma	09.09.2015 to 12.09.2015	**Italy:** To attend conference on Autophagy Signalling and progression in Health and disease
Raveendra Babu Mokhamatam	15.09.2015 to 19.09.2015	**USA:** To attend conference on Cell Death (CSHL 2015)
S Adeel Husain Zaidi	15.09.2015 to 19.09.2015	**USA:** To attend conference on Cell Death (CSHL 2015)
P Venkata Vivek Reddy	18.09.2015 to 22.09.2015	**Croatia:** To attend conference on Ubiquitin and Ubiquitin - like modifiers: From molecular mechanisms to human diseases
Anusha Uttarilli	24.09.2015 to 27.09.2015	**USA:** To attend CSHL meeting on "GENOME ENGINEERING: THE CRISPR / CAS REVOLUTION"
Valabhoju Vishalini	03.12.2015 to 04.12.2015	**Singapore**: To attend The 14th Asian Conference on Transcription
Aamir Ali	12.12.2015 to 16.12.2015	**USA:** To attend the American Society of Cell Biology (ASCB) annual meeting
Parul Singh	28.02.2016 to 03.03.2016	**USA:** To attend Keystone Symposia - Tuberculosis Co-morbidities and Immunopathogenesis (B6)"

सीडीएफडी के संकाय एवं अधिकारी
Faculty and Officers of CDFD

SCIENTIFIC GROUP LEADERS (FACULTY)

Dr Giriraj R Chandak
Dr J Gowrishankar
Dr D P Kasbekar
Dr Ranjan Sen
Dr Sangita Mukhopadhyay
Dr MD Bashyam
Dr Sunil Kumar Manna
Dr Nagarajaram HA
Dr Akash Ranjan
Dr Rupinder Kaur
Dr Sanjeev Khosla
Dr Ashwin B Dalal
Dr Rashna Bhandari
Dr Devyani Haldar
Dr N Madhusudan Reddy
Dr Shweta Tyagi
Dr MV Subba Reddy
Dr Subhadeep Chatterjee
Dr Sardesai Abhijit Ajit
Dr Rohit Joshi
Dr R Harinarayanan
Dr Arun Kumar KP

ADJUNCT FACULTY

Dr EA Siddiq
Prof T Ramasarma
Prof Anuradha Lohia
Dr Renu Wadhwa
Dr Prajnya Ranganath
Dr Shagun Aggarwal

OTHER GROUP LEADERS

Mr Raghavendrachar J
Ms Varsha
Ms M Kavita Rao

SENIOR ADMINISTRATIVE STAFF

Mr S Ayub Basha
Mr. J Sanjeev Rao
Mr B Jagannathacharyulu

केन्द्र की समितियाँ

(31.03.2016 तक)

Committees of the Centre

(As on 31.03.2016)

MEMBERS OF CDFD SOCIETY

Dr Harsh Vardhan Hon'ble Minister for S&T and Earth Sciences	-	President
Prof K VijayRaghavan Secretary, DBT, New Delhi	-	Member (Ex-officio)
Dr Girish Sahni Director General, CSIR, New Delhi	-	Member (Ex-officio)
Dr A K Rawat Director, DBT	-	Member (Ex-officio)
Mr J B Mohapatra FA, DBT, New Delhi	-	Member (Ex-officio)
Joint Secretary (PM) Ministry of Home Affairs, New Delhi	-	Member (Ex-officio)
Joint Secretary & Legal Adviser, Ministry of Law & Justice, New Delhi	-	Member (Ex-officio)
Director General, BPR&D, New Delhi	-	Member (Ex-officio)
Prof Partha P Majumder NIBMG, West Bengal Chairman of Scientific Advisory Committee, CDFD	-	Member (Ex-officio)
Prof VS Chauhan Visiting Scientist, ICGEB, New Delhi	-	Member
Prof Dipankar Chatterji IISc, Bangalore	-	Member
Dr Ch Mohan Rao CCMB, Hyderabad	-	Member
Dr G R Chandak Director, CDFD, Hyderabad	-	Member Secretary

MEMBERS OF CDFD GOVERNING COUNCIL

Prof K VijayRaghavan Secretary, DBT, New Delhi	-	Chairperson
Dr Girish Sahni Director General, CSIR, New Delhi	-	Member (Ex-officio)
Dr A K Rawat Director, DBT	-	Member (Ex-officio)
Ms Kusum Lata Sharma Director Finance, DBT (Nominee of FA, DBT, New Delhi)	-	Member (Ex-officio)
Mr A K Ganjoo Director, DFSS (Nominee of Joint Secretary (PM) Ministry of Home Affairs, New Delhi)	-	Member (Ex-officio)
Shri O Venkateswarlu Deputy Legal Adviser (Nominee of Joint Secretary & Legal Adviser, Ministry of Law & Justice, New Delhi)	-	Member (Ex-officio)
Dr A Radhakrishna Kini Director General, BPR&D, New Delhi	-	Member (Ex-officio)
Prof Partha P Majumder NIBMG, West Bengal Chairman of Scientific Advisory Committee, CDFD	-	Member (Ex-officio)
Prof VS Chauhan Visiting Scientist, ICGEB, New Delhi	-	Member
Prof Dipankar Chatterji IISc, Bangalore	-	Member
Dr G R Chandak Director, CDFD, Hyderabad	-	Member Secretary

MEMBERS OF CDFD RESEARCH AREA PANELS – SCIENTIFIC ADVISORY COMMITTEE (RAP-SAC)

Prof P Balaram
Director, IISc, Bangalore - Chairman

Dr Vijay Kumar
ICMR, New Delhi (ICMR Representative) - Member

Dr I Haque
DFSS, New Delhi (MHA Representative) - Member

Dr A K Rawat
DBT representative - Member

ICAR representative - Member

Dr G R Chandak
CCMB, Hyderabad (CCMB representative) - Member

Dr Veena K Parnaik
CCMB, Hyderabad - Member

Dr SK Apte
BARC, Mumbai - Member

Dr Usha Vijayraghavan
IISc, Bangalore - Member

Prof Umesh Varshney
IISc, Bangalore - Member

Dr Jaya Sivaswami Tyagi
AIIMS, New Delhi - Member

Prof MK Mathew
NCBS, Bangalore - Member

Dr Debasisa Mohanty
NII, New Delhi - Member

Dr Shubha R Phadke
SGPGI, Lucknow - Member

Dr Krishanu Ray
TIFR, Mumbai

Prof B K Thelma
University of Delhi (South Campus), New Delhi

Dr Saman Habib
CDRI, Lucknow

Prof Sriram Ramaswamy
TIFR Centre for Interdisciplinary Sciences, Hyderabad

Dr J Gowrishankar
Director, CDFD, Hyderabad - Member Secretary

MEMBERS OF CDFD ACADEMIC COMMITTEE

Prof AS Raghavendra School of Life SciencesUniversity of Hyderabad, Hyderabad	-	Chairman
Prof Anil K Tyagi University of Delhi, South Campus, New Delhi	-	Member
Dr K Satyamoorthy Manipal Life Sciences Centre, Manipal University, Manipal	-	Member
Dr DP Kasbekar CDFD, Hyderabad	-	Member
Dr Ranjan Sen CDFD, Hyderabad	-	Member
Dr Sanjeev Khosla Staff Scientist & Coordinator (Academics) CDFD, Hyderabad	-	Member Convenor

MEMBERS OF THE INSTITUTIONAL BIO-SAFETY COMMITTEE

Dr D P Kasbekar Haldane Chair, CDFD, Hyderabad (Nominee of Director, CDFD)	-	Chairman
Dr Rupinder Kaur Staff Scientist, CDFD, Hyderabad	-	Member Secretary
Dr Ashwin B Dalal Staff Scientist, CDFD, Hyderabad	-	Biosafety Officer
Dr M D Bashyam Staff Scientist, CDFD, Hyderabad	-	CDFD Expert
Dr Subhadeep Chatterjee Staff Scientist, CDFD, Hyderabad	-	CDFD Expert
Dr Ashok Khar Former Director, CMBRC, Appollo Hospitals Educational and Research Foundation	-	External Expert
Dr Manjula Reddy Senior Principal Scientist, CCMB, Hyderabad	-	DBT Nominee

MEMBERS OF THE INSTITUTIONAL BIOETHICS COMMITTEE

Prof G Manohar Rao Former Principal, PG College of Law, Osmania University, Hyderabad	-	Chairperson
Prof Sheela Prasad Associate Professor, Centre for Regional Studies, School of Social Sciences, University of Hyderabad	-	Member
Dr Mahtab S Bamji Emeritus Scientist, Dangoria Charitable Trust, Hyderabad	-	Member
Mrs Amita Kasbekar Manager, Concern India Foundation Hyderabad	-	Member
Dr M D Bashyam Staff Scientist, CDFD, Hyderabad	-	Member
Dr Ashwin B Dalal Staff Scientist, CDFD, Hyderabad	-	Member Secretary

MEMBERS OF CDFD BUILDING COMMITTEE

Prof VS Chauhan Visiting Scientist, ICGEB, New Delhi	-	Chairman
Dr J Gowrishankar Director, CDFD**,** Hyderabad	-	Member
Joint Secretary DBT, New Delhi	-	Member
Shri V H Rao Hyderabad	-	Member
Shri J Sanjeev Rao Head-Administration, CDFD, Hyderabad	-	Member
Shri BJ Acharyulu Head-F&A, CDFD, Hyderabad	-	Member
Shri S Ayub Basha Staff Scientist-V (Engg), CDFD, Hyderabad	-	Member-Convener

MEMBERS OF CDFD MANAGEMENT COMMITTEE

Director CDFD, Hyderabad	-	Chairman
Dr DP Kasbekar Haldane Chair, CDFD	-	Member
Dr M D Bashyam Staff Scientist, CDFD, Hyderabad	-	Member (for a 2 year period)
Dr Shweta Tyagi Staff Scientist, CDFD, Hyderabad	-	Member (for a 2 year period)
Shri BJ Acharyulu Head-F&A, CDFD, Hyderabad	-	Member
Shri J Sanjeev Rao Head-Administration, CDFD, Hyderabad	-	Member-Convenor

MEMBERS OF CDFD FINANCE COMMITTEE

Prof VS Chauhan Director, ICGEB, New Delhi	-	Chairman
Dr Dipankar Chatterji IISc, Bangalore	-	Member
Mr J B Mohapatra FA, DBT, New Delhi	-	Member
Dr A K Rawat Director, DBT, New Delhi	-	Member
Dr G R Chandak Director, CDFD, Hyderabad	-	Member
COFA/FAO CCMB, Hyderabad	-	Member
Mr BJ Acharyulu Head-F&A, CDFD, Hyderabad	-	Member Convenor

MEMBERS OF SEXUAL HARASSMENT COMPLAINTS COMMITTEE

Dr Sangita Mukhopadhyay Staff Scientist, CDFD, Hyderabad	-	Chairperson
Mr J Sanjeev Rao Head – Administration, CDFD, Hyderabad	-	Member
Ms V Naga Sailaja Technical Officer, CDFD, Hyderabad	-	Member
Ms MV Sukanya Technical Officer, CDFD, Hyderabad	-	Member
Mr MSA Zaman Khan Section Officer, CDFD, Hyderabad	-	Member
Ms P Jamuna Gramya Resource Centre for Women (representing an NGO)	-	Member

सूचना अधिकार अधिनियम, 2005 का परिपालन
Implementation of RTI Act, 2005

IMPLEMENTATION OF RIGHT TO INFORMATION (RTI) ACT, 2005

Appellate Authority : J Sanjeev Rao (Till 29-06-2015)
Dr D P kasbekar (From 30-06-2015)

Central Public Information Officer : M Kavita Rao

Details about the RTI applications and appeals received in CDFD

As received under the RTI Act 2005	**Opening Balance an on 01-04-2015**	**Received during the year 2015-16**			**Disposed off during the year 2015-16**				**Closing Balance as on 31-03-16**
		Received directly	Received as transfer from other Public Authorities [u/s 6(3) of Act]	Total	Decisions where applications accepted/ appeals upheld	Decisions where applications/ appeals rejected	Transferred to other Public Authorities [u/s 6(3) of Act]	Total	
Applications	0	26	6	32	30	1	0	31	1
Appeals	0	3	Not applicable	3	2	1	Not applicable	3	0

बजट एवं वित्त
Budget and Finance

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS HYDERABAD

Budget & Finance 2015-16

Sources of Funds

The Financial resources of the Centre are the Core Plan Grant-in Aid provided by the Department of Biotechnology, Government of India as against Annual Budgetary projections made by the Institute. Other resources are in the form of Research Grants provided by various National and International agencies and also from Services rendered by CDFD. The components of the core grants are Plan (Recurring) essentially for meeting expenditures on salaries, Operating expenses etc., and Plan (Non-Recurring) for meeting expenses on account of Equipments, Infrastructure and Furnishing etc.,

Receipts during the year 2015-16

Particulars	Amount in Lakhs	Percentage - %
Plan Grant in Aid	8450.00	87.92
Sponsored Projects	984.46	10.24
CDFD Services	86.41	0.90
Misc Receipts	90.24	0.94
Total	**9611.11**	**100.00**

I. Application of Funds during 2015-16 (Plan Grant in Aid)

S No	Particulars	Amount in Lakhs	Percentage- %
1	**Recurring**		
	GIA- Salaries	1284.40	15.37
	GIA-General	2016.04	24.13
	Total	**3300.44**	**39.50**
2	**Non-Recurring**		
	GIA- Capital	5055.82	60.50
	Total	**5055.82**	**60.50**
	Grand Total	**8356.26**	**100.00**

II. Application of Funds during 2015-16 (Extra Mural Projects)

S No	Particulars	Amount in Lakhs	Percentage- %
1	**Recurring**		
	Salaries	316.98	30.85
	General	562.56	54.76
	Total	**879.54**	**85.61**
2	**Non-Recurring**		
	Capital	147.89	14.39
	Total	**147.89**	**14.39**
	Grand Total	**1027.43**	**100.00**

लेखा परिक्षक की रिपोर्ट
Auditor’s Report

B Purushottam & Co.,
Chartered Accountants

AUDITOR'S REPORT

Date: 02-06-2016

The Director,
Centre for DNA Fingerprinting and Diagnostics,
Nampally,
Hyderabad – 500 001

We have audited the attached Balance Sheet of CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, Hyderabad, as at 31st March 2016 and also the Income & Expenditure Account for the year ended on that date annexed there to. These Financial Statements are the responsibility of the organization management. Our responsibility is to express an opinion on these Financial Statements based on our audit.

We report that:

1. We have obtained all the information and explanations, which to the best of our knowledge and belief were necessary for the purpose of our audit.
2. In our opinion, the organization has kept proper books of account as required by law so far, as appears from our examination of these books.
3. The Balance Sheet and Income & Expenditure account dealt with by this report is in agreement with the books of account.
4. (a) The centre has maintained accounts on accrual basis.
   (b) The Centre receives extra mural grants from various National & International agencies for specific research activities. The Centre has a policy of allocating the overheads and transfer of expenditure of CDFD to different projects at the end of the financial year after taking into account the amount of maximum permissible limit of overheads and also based on the approved budget estimates and expenditure of the respective projects during the financial year.
5. In our opinion and to the best of our information and according to the explanations given to us, the said Balance Sheet and the Income & Expenditure account read together with the notes thereon gives the required information in the manner so required and gives a true and fair view.
   a) In so far it relates to the Balance Sheet as at 31st March 2016 and
   b) In so far as it relates to the Income & Expenditure account excess of income over expenditure for the year ended on 31st March 2016.

for **for B Purushottam & Co**
Chartered Accountants
[CH SATYANARAYANA]

Place : Hyderabad
Date : 02/06/2016

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS,HYDERABAD

BALANCE SHEET AS ON 31st MARCH 2016

(Amount - Rs.)

	Schedule	Current Year	Previous Year
CORPUS/CAPITAL FUND AND LIABILITIES			
Corpus / Capital Fund	1	1686691192	1212702539
Reserves and Surplus	2	16484058	0
Earmarked / Endowment funds	3	0	0
Secured Loans & Borrowings	4	0	0
Unsecured Loans & Borrowings	5	0	0
Deffered Credit Liabilities	6	0	0
Current Liabilities and Provisions	7	85746032	70028009
TOTAL		**1788921282**	**1282730548**
ASSETS			
Fixed Assets	8	1537816689	1090185109
Investments- From Earmarked / Endowment Funds	9	71098273	35098273
Investments - Others	10	30065721	33593376
Current Assets, Loans, Advances etc.	11	149940599	123853790
Miscellaneous Expenditure		0	0
TOTAL		**1788921282**	**1282730548**
Significant Accounting Policies	24		
Contingent Liabilities and Notes on Accounts	25		

DIRECTOR
CDFD

For B. PURUSHOTTAM & CO
CHARTERED ACCOUNTANTS
(CH SATYANARAYANA)

HEAD - FINANCE & ACCOUNTS
CDFD

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS,HYDERABAD

INCOME & EXPENDITURE FOR THE YEAR ENDED 31st MARCH 2016

(Amount - Rs.)

INCOME	Schedule		Current Year		Previous Year
Income from Sales/Services	12		8641034		16481871
Grants/Subsides	13		345000000		260000000
Fees/Subscriptions	14		0		0
Income from Investments	15		18375260		27138910
Income from Royality, Publications etc.	16		0		0
Interest Earned	17		1390306		2104450
Other Income	18		7236505		3609182
Increase/(decrease) in stock of Finished goods and works-in-progress	19		0		0
TOTAL (A)			**380643105**		**309334413**
EXPENDITURE					
Establishment Expenses	20		119831151		128443061
Administrative Expenses	21		212729759		207784973
Expenditure on Grants, Subsides etc.	22		0		0
Interest	23		0		0
Depreciation (Net Total at the year-end -corresponding to Schedule 8)		70461166		81320619	
Less:Transferred to Grants-in-Aid		70461166	0	81320619	
Provision For Salaries			9780756		8395162
TOTAL (B)			**342341666**		**344623196**
Balance being excess of Income over Expenditure (A-B)			**38301440**		**-35288783**

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Transfer to Special Reserve					
Transfer to General Reserve (Lab Reserve)			8641034		
BALANCE BEING SURPLUS/(DEFLICT) CARRIED TO CORPUS/CAPITAL FUND	24		**29660405**		
SIGNIFICANT ACOUNTING POLICIES	25				
CONTINGENT LIABILITIES AND NOTES ON ACCOUNTS					

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CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS,HYDERABAD

RECEIPTS AND PAYMENTS ACCOUNT FOR THE YEAR ENDED 31st MARCH 2016

(Amount - Rs.)

RECEIPTS	Current Year	Previous Year	PAYMENTS	Current Year	Previous Year
1.Opening Balances			**1. Expenses**		
a) Cash in hand			a) Establishment Expenses (corresponding to Schedule 20)	119831151.00	128443061
b) Bank Balances			b) Administrative Expenses (corresponding to Schedule 21)	212729758.77	207784973
i) In current accounts	13313616.81	26417751	c) Schedule 22	0.00	0
ii) In deposit accounts					
iii) Savings accounts	9433617.60	4383078			
2. Grants Received			**2. Payments made against funds for various projects**		
a) From Government of India	845000000.00	410000000	(Name of the fund or project should be shown along with the particulars of		
b) From State government			payments made for each project)		
c) From other sources (details)			Projects (Annexure F)	102743689.00	96048982
(Grants for capital & revenue			CSIR(Stipend)	11956274.00	10088151
exp. To be shown seperately)			DBT(Stipend)	9595329.00	5571185
Research Associates - CSIR(Stipend)	8453559.00	11093876	DST(Stipend)	2238533.00	1340375
Research Associates - DBT(Stipend)	5344314.00	5623475	ICMR(Stipend)	3338763.00	2785432
Research Associates - DST(Stipend)	1362000.00	85239	IISC(Stipend)	265938.00	813334
Research Associates - ICMR(Stipend)	1754439.00	1589055	UGC(Stipend)	11836172.00	8242741
Research Associates - IISC(Stipend)	36400.00	1029961			
Research Associates - UGC(Stipend)	2064806.00	16736506	**3. Investments and deposits made**		
			a) Out of Earmarked/Endowement funds	420000000.00	189000000
Projects (Annexure - C)	98445681.00	108091285	b) Out of Own Funds (Investments-Others)	0.00	

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HEAD - FINANCE & ACCOUNTS
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CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS,HYDERABAD

RECEIPTS AND PAYMENTS ACCOUNT FOR THE YEAR ENDED 31st MARCH 2016

(Amount - Rs.)

RECEIPTS	Current Year	Previous Year	PAYMENTS	Current Year	Previous Year
3. Income on Investments from			**4. Expenditure on Fixed Assets & Capital Work-in-Progress**		
a) Earmarked/Endow. Funds	3168348.00	9126414	a) Purchases of Fixed Assets:		
b) Own Funds (Oth. Investment)			Books & Journals	560767.00	895458
Investments EnCashed	384000000.00	162000000	Equipment -Lab/Office/Furniture	23244176.25	74499419
			b) Expenditure on Capital Work-in-Progress:	479498388.00	119845405
4. Interest Received					
a) On Bank deposits	106041.00	0			
b) Loans, Advances etc	18012496.00		**5. Refund of surplus money/Loans**		
Interest on LC	1284265.48	2104449.88	a) To the Government of India	0.00	
Interest on Computer Advance, Conveyance Advance and HBA	19018.00	17526	b) To the State Government	0.00	
			c) To other providers of funds	0.00	
5. Other Income(Specify)					
a) Analysis Charges	8641034.00	16481871.00	**6. Finance Charges (Interest)**	0.00	
b) Lab Reserve	7843024.00	0	**7. Other Payments (Specify)**		
6. Any Other Receipts(Give Details)			Advances (Annexure-D)	158544851.00	172463825
I-Remittances (Annexure-A)	29358677.00	23453753	I-Remittances (Annexure-E)	28161879.00	23186242
			CPF A/c	7756535.00	18257438
CPF-SUB,Arrears and adv.Refund	15265679.00	10645544	New Pension Scheme	3424598.00	3136300
Sundry Receipts	7090257.00	3254256	NIMS	3376101.00	0
Application Fee	17500.00	235800			
Provident Fund Salwage	0.00	0	**8. Closing Balances**		
Free Gifts - Donations	0.00	0	a) Cash in hand		
Sale OF Tender Forms	10500.00	47000	b) Bank Balances		

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CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS,HYDERABAD

RECEIPTS AND PAYMENTS ACCOUNT FOR THE YEAR ENDED 31st MARCH 2016

(Amount - Rs.)

RECEIPTS	Current Year	Previous Year	PAYMENTS	Current Year	Previous Year
Leave Salary-Pension Contribution	44030.00	0	i) In current accounts	27660890.87	13313616.81
License Fee	55200.00	54600	ii) In deposit accounts		
Welfare Fund	0.00	0	iii) Savings accounts	11145109.00	9433617.6
NPS	3453474.00	3040743			
Advance/Refunds/Recovery/Adj(Annexure-B)	170319917.00	269637372			
NIMS	4011009.00	0			
TOTAL	**1637908902.89**	**1085149555**	**TOTAL**	**1637908902.89**	**1085149555**

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CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

BALANCE SHEET AS ON 31st MARCH 2016

(Amount - Rs.)

		Current Year		Previous Year
SCHEDULE 1 - CORPUS/CAPITAL FUND :				
Balance as at the begining of the year		1212702539.00		1169815289.00
Add : Contribution towards Corpus/Capital Fund				
CDFD Core - Plan (Non-Recurring)	500000000.00		150000000.00	
Capitalised portion of Capital Expenditure of projects	14789414.00	514789414.00	9496652.00	159496652.00
Less : Depreciation For the Year 2015-2016	70461166.00	70461166.00	81320619.00	81320619.00
Less : Excess of Expenditure over Income	29660405.00	29660405.00		0.00
		0.00	35288783.00	35288783.00
BALANCE AS AT THE YEAR - END		**1686691192.00**		**1212702539.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2016

(Amount - Rs.)

	Current Year		Previous Year	
SCHEDULE 2 -RESERVES AND SURPLUS :				
1.Capital Reserve :				
As per last Account	0.00		0.00	
Addition during the year	0.00		0.00	
Less : Deductions during the year	0.00	0.00	0.00	0.00
2.Revolution Reserve :				
As per last Account	0.00		0.00	
Addition during the year	0.00		0.00	
Less : Deductions during the year	0.00	0.00	0.00	0.00
3.Special Reserves :				
As per last Account	0.00		0.00	
Addition during the year	0.00		0.00	
Less : Deductions during the year	0.00	0.00	0.00	0.00
4.General Reserve :				
As per last Account			0.00	
Addition during the year	16484058.00	0.00		
Less : Deductions during the year	0.00	0.00	0.00	0.00
Total	**0.00**	**16484058.00**		**0.00**

DNA Fingerprinting and Diagnostics Receipts	8641034
Project Balances	7843024
Total	16484058

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2016

(Amount - Rs.)

	Current Year		Previous Year	
SCHEDULE 3 - EARMARKED/ENDOWMENT FUNDS :				
(Refer Annexures)				
(a) Opening balance of the Funds		**-13731478.00**		**-25773781.00**
(b) Additions to the Funds :				
i. Donations /grants	98445681.16		108091285.00	
ii. Income from investments made on account of funds	0.00		0.00	
iii. Other additions	0.00	98445681.16	0.00	108091285.00
TOTAL (a+b)		**84714203.16**		**82317504.00**
(c) Utilisation/Expenditure towards objective of funds				
(i) Capital Expenditure (Refer Annexures I & II)				
- Fixed Assets	14354226.00		9200996.00	
- Others	435188.00	14789414.00	295656.00	9496652.00
- Total				
(ii) Revenue Expenditure (Refer Annexures I & II)				
- Salaries, Wages and allowances etc.	31698402.00		28642978.00	
- Rent	0.00		0.00	
- Other Expenses	56255873.00	87954275.00	57909352.00	86552330.00
Total				
TOTAL (c)		102743689.00		96048982.00
NET BALANCE AS AT THE YEAR-END [(a + b)-c]		**-18029485.84**		**-13731478.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2016

(Amount - Rs.)

	Current Year		Previous Year	
SCHEDULE 4 - SCHEDULE LOANS AND BORROWINGS :				
1. Central Government		0		0
2. State Government (Specify)		0		0
3. Financial Institutions				
a) Term Loans	0		0	
b) Interest accured and due	0	0	0	0
4. Banks :				
a) Terms Loans	0	0	0	0
- Interest accured and due	0		0	
b) Other Loans	0		0	
- Interest accured and due	0	0	0	0
5. Other Institutions and Agencies		0		0
6. Debentures and Bonds		0		0
7. Others (Specify)				
TOTAL		**0**		**0**
Note: Amount due within one year				

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2016

(Amount - Rs.)

	Current Year		Previous Year	
SCHEDULE 5 - UNSECURED LOANS AND BORROWINGS :				
1. Central Government		0		0
2. State Government (Specify)		0		0
3. Financial Institutions		0		0
4. Banks :				
a) Terms Loans	0		0	
b) Other Loans	0	0	0	0
5. Other Institutions and Agencies		0		0
6. Debentures and Bonds		0		0
7. Fixed Deposits		0		0
8. Others (Specify)		0		0
TOTAL		**0**		**0**
Note: Amount due within one year				

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2016

(Amount - Rs.)

		Current Year		Previous Year
SCHEDULE 6 - DEFFERED CREDIT LIABILITIES :				
a) Acceptances secured by hypothecation of capital equipment and other assets		0		0
b) Others		0		0
TOTAL		**0**		**0**
Note: Amount due within one year				

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2016

(Amount - Rs.)

	Current Year		Previous Year	
SCHEDULE 7 - CURRENT LIABILITIES AND PROVISIONS :				
A. CURRENT LIABILITIES				
1. Acceptances				
2. Sundry Creditors				
3. Advances Received				
4. Interest accured but not due on:				
5. Statutory Liabilities:				
6. Other current Liabilities				
CDFD.CP Fund A/C(Annexure-G)	44620022.00		40638533.00	
Diagnostics Collabration With NIMS	634908.00		0.00	
EMD	1858034.00		2378534.00	
GSLI	33339.00		30785.00	
Honorarium [Advance]	0.00		0.00	
House Building Advance	129831.00		129831.00	
Income Tax	97507.00		97088.00	
Lab Security Deposit & Hostel Security Deposit	1272716.00		1242716.00	
LIC	2550.00		2550.00	
Others (I-Remittances)	296555.00		296555.00	
Out Standing Liabilities	20240618.00		11845456.00	
PPF EMPLOYER SHARE	562436.00		34566.00	
Professional Tax	98642.00		99742.00	
Public Provident Fund	406240.00		124630.00	
Royalty & Consultancy	1531642.00		1654142.00	
Security Deposit	1643475.00		1691275.00	
Service Tax	0.00		247331.00	
TA Abroad [Advance]	0.00		65249.00	
TDS	1920764.00		800515.00	
Works Tax	255858.00		253349.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2016

(Amount - Rs.)

		Current Year		Previous Year
SCHEDULE 7 - CURRENT LIABILITIES AND PROVISIONS :				
Workshop & Conference	360139.00	75965276.00	0.00	61632847.00
TOTAL (A)		**75965276.00**		**61632847.00**
B.PROVISIONS				
1. For Taxation				
2. Gratuity				
3. Superannuation/Pension				
4. Accumulated Leave Encashment				
5. Trade Warranties/Claims				
6. Others (Specify)	9780756.00	9780756.00	8395162.00	8395162.00
TOTAL (B)		**9780756.00**		**8395162.00**
TOTAL (A+B)		**85746032.00**		**70028009.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2016

(Amount - Rs.)

	GROSS BLOCK				DEPRECIATION				NET BLOCK	
SCHEDULE 8 - FIXED ASSTES	Cost/valuation As at begining of the the year	Addition during the year	Deductions during the year	Cost/valuation at the year end	As at the begining of the year	On additions during the year	On Deductions during the year	Total up to the year end	As at the Current current year end	As at the previous year end
A. FIXED ASSETS:										
1. LAND:										
a) Freehold	3900000.00	0.00	0.00	3900000.00	0.00	0.00	0.00	0.00	3900000.00	3900000.00
b) Leasehold	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
2. BUILDINGS										
a) On Freehold Land	220052369.00	0.00	0.00	220052369.00	72988620.00	14706375.00	0.00	87694995.00	132357374.00	147063749.00
b) On Leasehold Land	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
c) Ownership Flats/Premises	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
d) Superstructures on Land not belongs to the entity	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
3. PLANT MACHINERY & EQUIPMENT	674283159.05	37532003.00	0.00	711815162.05	336762469.00	53779285.00	0.00	390541754.00	321273408.05	337520690.05
4. VEHICLES	4153026.00	0.00	0.00	4153026.00	3588389.00	84696.00	0.00	3673085.00	479941.00	564637.00
5. FURNITURE, FIXTURES	16469562.00	-432166.00	0.00	16037396.00	10827570.00	542591.00	0.00	11370161.00	4667235.00	5641992.00
6. OFFICE EQUIPMENT	11651316.00	498566.00	0.00	12149882.00	9160454.00	416001.00	0.00	9576455.00	2573427.00	2490862.00
7. COMPUTER/PERIPHERALS	132023.00	0.00	0.00	132023.00	0.00	0.00	0.00	0.00	132023.00	132023.00
8. ELECTRIC INSTALLATIONS	0.00				0.00	0.00	0.00	0.00		
9. LIBRARY BOOKS	18017234.00	995955.00	0.00	19013189.00	17680649.00	846328.00	0.00	18526977.00	486212.00	336585.00
10. TUBEWELLS & WATER SUPPLY	0.00				0.00	0.00	0.00	0.00		
11. OTHER FIXED ASSETS	8857898.00	0.00	0.00	8857898.00	7998999.00	85890.00	0.00	8084889.00	773009.00	858899.00
Airconditioning works		0.00	0.00		0.00	0.00	0.00	0.00		
Aluminium partition work		0.00	0.00		0.00	0.00	0.00	0.00		
DG Set		0.00	0.00		0.00	0.00	0.00	0.00		
Paintings		0.00	0.00		0.00	0.00	0.00	0.00		
Typewriters		0.00	0.00		0.00	0.00	0.00	0.00		
Miscellaneous non consumables		0.00	0.00		0.00	0.00	0.00	0.00		
Other Assets		0.00	0.00		0.00	0.00	0.00	0.00		
EMB Net		0.00	0.00		0.00	0.00	0.00	0.00		
TOTAL	957516587.05	38594358.00	0.00	996110945.05	459007150.00	70461166.00	0.00	529468316.00	466642629.05	498509437.05
B. CAPITAL WORK-IN-PROGRESS	591675671.70	479498388.00	0.00	1071174059.70	0.00	0.00	0.00	0.00	1071174059.70	591675671.70
TOTAL	**1549192258.75**	**518092746.00**	**0.00**	**2067285004.75**	**459007150.00**	**70461166.00**	**0.00**	**529468316.00**	**1537816688.75**	**1090185108.75**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2016

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 9 - INVESTMENTS FROM EARMARKED/ENDOWMENT FUNDS :		
1. In Government Securities	0.00	0.00
2. Other approved securities	0.00	0.00
3. Shares	0.00	0.00
4. Debentures and Bonds	0.00	0.00
5. Subsidiaries and Joint Ventures	0.00	0.00
6. Others (to be specified) - STDRs (Annexure-J)	71098273.00	35098273.00
TOTAL	**71098273.00**	**35098273.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2016

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 10 - INVESTMENTS - OTHERS :		
(Annexure-K)		
1. In Government Securities	0.00	0.00
2. Other approved securities	0.00	0.00
3. Shares	0.00	0.00
4. Debentures and Bonds : UTI Bonds		
5. Subsidiaries and Joint Ventures	0.00	0.00
6. Others (to be specified) - STDRs,(CPF),CDFD CP FUND A/C	30065721.00	33593376.00
TOTAL	**30065721.00**	**33593376.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2016

(Amount - Rs.)

	Current Year		Previous Year	
SCHEDULE 11 - INVESTMENTS - OTHERS :	**Current Year**		**Previous Year**	
A. CURRENT ASSETS				
1. Inventors				
a) Stores and Spares	0.00		0.00	
b) Loose Tools	0.00		0.00	
c) Stock-in-trade				
Finished Goods	0.00		0.00	
Work-in-progress	0.00		0.00	
Raw Materials	0.00	0.00	0.00	0.00
2. Sundry Debtors:				
a) Debts Outstanding for a period exceeding six months			0.00	
b) Others-Life Membership Fees	169236.00	169236.00	165935.00	165935.00
3. Cash balances in hand (including cheques/drafts and imprest)				
4. Bank Balances:				
a) With Scheduled Banks:				
-On Current Accounts	27660889.85		13313616.81	
-On Deposit Accounts (includes margin money)	0.00		0.00	
-On Savings Accounts	11145109.42	38805999.27	9433617.60	22747234.41
b) With non-Schedules Banks:				
-On Current Accounts	0.00		0.00	
-On Deposit Accounts	0.00		0.00	
-On Savings Accounts	0.00	0.00	0.00	0.00
5. Post Office-Savings Accounts				
TOTAL (A)		**38975235.27**		**22913169.41**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2016

(Amount - Rs.)

	Current Year		Previous Year	
SCHEDULE 11 - INVESTMENTS - OTHERS :				
B. LOANS, ADVANCES AND OTHER ASSETS				
1. Loans:				
a) Staff	0.00		0.00	
b) Other Entities engaged in activities/objectives similar to that of the Entity	0.00	0.00	0.00	0.00
2. Advances and other amounts recoverable in cash or in kind or for value to be received				
a) On Capital Account (Annexure-H)	61240068.00	0.00	51994904.56	
b) Prepayments - Deposits (Annexure-I)	16488897.00		17201742.00	
c) Others	0.00	77728965.00	0.00	69196646.56
3. Income Accured:				
a) On Investments from Earmarked/Endowments Funds	0.00		0.00	
b) On Investments - Others	15206912.00		18012496.00	
c) On Loans and Advances	0.00		0.00	
d) Others	0.00	15206912.00	0.00	18012496.00
4. Claims Receivable		18029485.84		13731478.00
TOTAL (B)		110965362.84		100940620.56
TOTAL (A+B)		**149940598.11**		**123853789.97**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2016

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 12 - INCOME FROM SALES/SERVICES :		
1) Income from sales		
a) Sale of Finished Goods	0.00	0.00
b) Sale of Raw Material	0.00	0.00
c) Sale of Scraps	0.00	0.00
2) Income from Services		
a) Labour and Processing Charges	0.00	0.00
b) Professional/Consultancy Services (Analysis Charges)	8641034.00	16481871.00
c) Agency Commission and Brokerage	0.00	0.00
d) Maintenance Services (Equpiment/Property)	0.00	0.00
e) Others (Specify)	0.00	0.00
TOTAL	**8641034.00**	**16481871.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2016

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 13 - GRANTS/SUBSIDES :		
(Irrevocable Grants & Subsides Received)		
1) Central Government (DBT Plan Grant-in-Aid)	345000000.00	260000000.00
2) State Government(s)	0.00	0.00
3) Government Agencies	0.00	0.00
4) Institutions/Welfare Bodies	0.00	0.00
5) International Organisations	0.00	0.00
6) Others (Specify)	0.00	0.00
TOTAL	**345000000.00**	**260000000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2016

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 14 - FEES/SUBSCRIPTIONS :		
1) Entrance Fees	0	0
2) Annual Fees/Subscriptions	0	0
3) Seminar/Program Fees	0	0
4) Consultancy Fees	0	0
5) Others (Specify)	0	0
TOTAL	**0**	**0**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2016

(Amount - Rs.)

	Current Year		Previous Year	
SCHEDULE 15 - INCOME FROM INVESTMENTS :				
(Income on Invest from Earmarked/Endowment Funds transferred to Funds)				
1) Interest:				
a) On Govt. Securities	0.00		0.00	
b) Other Bonds/Debentures	0.00	0.00	0.00	0.00
2) Dividends:				
a) On Shares	0.00	0.00	0.00	0.00
b) On Mutual Fund Securities	0.00	0.00	0.00	0.00
3) Rents	0.00	0.00	0.00	0.00
4) Others (Specify) STDRs	18375260.00		27138910.00	0.00
TOTAL	**18375260.00**		**27138910.00**	**0.00**
TRANSFERRED TO EARMARKED/ENDOWMENT FUNDS				

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2016

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 16 - INCOME FROM ROYALITY, PUBLICATION ETC. :		
1) Income from Royality	0	0
2) Income from Publications	0	0
3) Others (Specify)	0	0
TOTAL	**0**	**0**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2016

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 17 - INTEREST EARNED :		
1) On Term Deposits		
a) With Schedule Banks	1284265.48	2104450.00
b) With Non-Scheduled Banks	0.00	0.00
c) With Institutions	0.00	0.00
d) Others	0.00	0.00
2) On Saving Accounts		
a) With Schedule Banks	106041.00	0.00
b) With Non-Scheduled Banks	0.00	0.00
c) post Office Savings Accounts	0.00	0.00
d) Others	0.00	0.00
3) On Loans		
a) Employees/Staff		
b) Others	0.00	0.00
4) Interest on Debtors and Other Receivables	0.00	0.00
TOTAL	**1390306.48**	**2104450.00**
Note :- Tax deducted at source to be indicated		

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2016 **(Amount - Rs.)**

SCHEDULE 18 - OTHER INCOME	Current Year	Previous Year
1) Profit on Sale/disposal of Assets:	0.00	0.00
a) Owned assets	0.00	0.00
b) Assets acquired out of grants, or received free of cost	0.00	0.00
2) Export Incentives realized	0.00	0.00
3) Fees for Miscellaneous Services	0.00	0.00
4) Miscellaneous Receipts		
5) Other Receipts		
Sundry Receipts	7090257.00	3254256.00
Application Fee	17500.00	235800.00
Sales of Tender Forms	10500.00	47000.00
Licence Fee	55200.00	54600.00
Interest on Computer Advance,Conveyance Advance And HBA	19018.00	17526.00
Leave Salary-Pension Contribution	44030.00	0.00
Provident Fund Salwage	0.00	0.00
Free.Gifts-Donations	0.00	0.00
TOTAL	**7236505.00**	**3609182.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2016 **(Amount - Rs.)**

SCHEDULE 19 - INCREASE/(DECREASE) IN STOCK OF FINISHED GOODS & WORK IN PROGRESS :	Current Year	Previous Year
a) Closing stock		
-Finished Goods	0	0
-Work-in-progress	0	0
Total (a)	**0**	**0**
b) Less: Opening stock		
-Finished Goods	0	0
-Work-in-progress	0	0
Total (b)	**0**	**0**
NET INCREASE/(DECREASE) [a-b]	0	0

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2016

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 20 - ESTABLISHMENT EXPENSES :		
a) Salaries and Wages	53877441.00	68828459.00
b) Allowances and Bonus	58836726.00	50691650.00
c) Contribution to Provident Fund	2247900.00	2619770.00
d) Contribution to Other Fund (NPS)	2767432.00	2358636.00
e) Staff Welfare Expenses - Medical charges	2101652.00	2136167.00
f) Expenses on Employees Retirement and Terminal Benefits	0.00	1808379.00
g) Others (specify) - Staff leased House	0.00	0.00
TOTAL	**119831151.00**	**128443061.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2016

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 21 - OTHER ADMINISTRATIVE EXPENSES :		
a) Purchases	55705243.00	77276637.00
b) Electricity and power	21498750.00	21857964.00
c) Water charges	903057.00	898347.00
d) Insurance	106035.00	90857.00
e) Repairs and maintenance	11702293.00	16452976.00
f) Rent, Rates and Taxes	30557063.00	18919374.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2016

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 21 - OTHER ADMINISTRATIVE EXPENSES :		
g) Vehicles Running and Maintenance	1176998.00	1254153.00
h) Postage, Telephone and Communication Charges	4578419.00	3037666.00
i) Printing and Stationary	1748631.00	1151024.00
j) Travelling and Conveyance Expenses	9363448.27	9897640.00
k) Expenses on Seminar/Workshops	219573.00	316177.00
l) Subscription Expenses	50894.00	38693.00
m) Expenses on Fees	34246.00	80874.00
n) Auditors Remuneration	62126.00	56180.00
o) Hospitality Expenses	952328.00	772072.00
p) Professional Charges	3686097.00	5985002.00
q) Advertisement and Publicity	472477.00	3034697.00
r) Bank Charges	26599.50	4818.00
s) Security & Cleaning Contract Charges	21601902.00	21011830.00
t) Training Course /Symposia	20600.00	-88482.00
u) Other Contingencies	9373811.00	1881362.00
v) Liveries & Blankets	127754.00	30819.00
w) Other Research Expenses	38760374.00	22011273.00
x) Office Books	1040.00	13020.00
y) Over Heads	0.00	1800000.00
TOTAL	**212729758.77**	**207784973.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2016

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 22 - EXPENDITURE ON GRANTS, SUBSIDES, ETC.		
a) Grants given to Institutions/Organisations	0.00	0.00
b) Subsidies given to Institutions/Organisations	0.00	0.00
TOTAL	**0.00**	**0.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2016

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 23 - INTEREST		
a) On Fixed Loans	0.00	0.00
b) On Other Loans (including Bank Charges)	0.00	0.00
c) Others	0.00	0.00
TOTAL	**0.00**	**0.00**

Schedule 24: Significant Accounting Policies & Schedule 25: Contingent Liabilities & Notes on Account for the period ended 31/03/2016

1. Method of Accounting:

a. The accounting system adopted by the organization is on “accrual basis”.

b. The organization has been getting plan Grant-In-Aid under the “Non-recurring” & “Recurring” heads.

2. Revenue recognition:

Income comprises of Grant-in-Aid, Internal Resources through services and interest from short term deposits. Income accounted on the basis of the Cash/DD/Cheques/Cr notes/ on line transfers received.

3. Fixed Assets:

(a) Fixed assets are stated at cost. Cost includes freight, duties, and taxes etc.,

(b) Depreciation: Depreciation Account on Fixed Assets has since been prepared at the rate prevailing to the concerned Fixed Assets as specified in the Income Tax Act, 1961 on Written Down Value Method of Depreciation.

(c) Capital work in progress has been entered to the extent of the last running account bills paid.

(d) Realization on sale of obsolete/surplus fixed assets which is not required for the purpose of research activities are adjusted against capital cost.

4. Inventories:

All purchases of chemicals, glassware and other consumables have been charged to consumption at the time of purchase.

5. Foreign Currency transactions:

Foreign Currency transactions are recognized in the books at the exchange rates prevailing on the date of transaction.

6. Investments:

Investments in STDR’s are stated at book values.

7. Advances:

It is observed from the objection book register that advances to suppliers for consumables & Equipments are to be reconciled and adjustment entries are to be passed in the books of accounts.

8. The previous year balances have been regrouped/rearranged, wherever necessary.

9. With effect from financial year 2015-16, creation of the Laboratory Reserve has been introduced as approved by the FC/GC held on 18/02/2016. Accordingly the transferable amounts as per the approved method have been transferred to Reserves and Surplus from the respective heads to the permissible limits which is reflected in Income and Expenditure Account and the Balance Sheet.

Director CDFD	Head- Finance & Accounts CDFD	for B Purushottam & Co Chartered Accountants [CH SATYANARAYANA]

Place : Hyderabad
Date : 02/06/2016

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

CLARIFICATION ON NOTES ON ACCOUNTS: 2015-16

- Notes on Accounts 1 to 2 & 4 to 6: Method of Accounting/ Revenue recognition/Fixed Asset/ Inventories/ Foreign Currency transactions/Investments:

  These are all only informatory items.

- Notes on Accounts 3: Fixed Assets:

  Depreciation has been calculated on Written down Value method and at the rates prevailing to the concerned Fixed Asset as specified on the Income Tax Act, 1961 and set off against the Grant-in-Aid (non-recurring). The details of the Depreciation on Fixed Assets are at Schedule -8 is an integral part of the financial statements.

- Notes on Accounts 7: Advances:

  The observation of the audit has been noted. The action has already been initiated to reconcile the objection book register.

B J ACHARYULU
Head Finance & Accounts
CDFD

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS Annexure-I

Details of Closing balances of various Earmarked / Endowement Funds (Refer Sch-3)

For the Year Ended 31st MARCH 2016

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
-13242813	COE1	COE1	-13755933
-13991880	COE2	COE2	-25772516
-630047	P-03	“Transgenesis and Genetic basis of Pathogen Resistance in the Silkworm, Bombyx Mori	-630047
244305	P-09	“NMITLI Project on – Latent M.Tuberculosis: New targets, Drug delivery systems, Bio enhancers & Therapeutics”	244305
-28332	P-10	“Role of upstream sequence elements in Hyper activation of transcription from Baculovirus polyhedrin gene promoter”	-28332
-576590	P-100	Effect of raective oxygen species on T-Cell immune response: An approach to understand the molecular mechanism of immunosuppression during tuberculosis - National Bioscience Award	-576590
6859801	P-101	Role of inositol pyrophosphates in cell physiology: Investigating the biochemical significance of protein pyrophosphorylation - Senior Fellowship	1
-27922	P-102	Understanding the role of Mycobacterium tuberculosis heat shockprotein 60 as Th1/Th2 immuno modular	-27922
-300000	P-103	National Bioscience Award - Regulation of mast cell signaling, apoptosis and surface receptors	-300000
-1160508	P-104	Virtual Centre of Excellence on Epigenetics	-1289897
-862685	P-105	Cloning, Characterization and analysis of chromosomal rearrangements in human genetic disorders	-862685
1036691	P-107	IYBA Project - Mechanism and role of bacterial cell-cell signaling molecules in plant defense response	366575
-454643	P-108	Establishment of EBV transformed cell lines from families with rare genetic disorders	-454643
3351336	P-109	Molecular dissection of PI3-Kinase/Akt pathway by suing proteomics based approach: A study to identify novel potential oncogenes and tumor suppressors	767943
-191391	P-110	India-Japan research project title"Identification and analysis of sex determining genes in silkmoths"	-191391
1169677	P-111	Ramalingaswami Fellowship - Refractoriness mechanism in Mosquito: cracking molecular codes at genomic scale	0
-450859	P-114	Evaluating the Calcineurin-NFAT Pathway and its regulators superoxide dismutase (SOD) AND RCAN1 (regular of Calcineurin) Down Syndrome	-450859
-1251366	P-116	DBT-India and AIST - Japan : Understanding molecular mechanisms controlling dual role of Ras, Sirtuins and CARF in relation to cellular proliferation and senescence: Novel Strategy for developing cancer therapeutics	-1251366
-2892	P-119	Analysis of DNA copy number alterations in esophaeal cancer	-2892
-769484	P-120	Effect of reactive oxygen species on macrophage signalosome: impact on antigen presentation functions and T Cell priming responses	-769484
-1130866	P-121	Identification and characterization of PTEN regulators	-1130866
388692	P-122	Understanding the role of Hox genes in anterior-posterior axis determination of the central nervous system	2951109
1402135	P-123	Establish a Max Planck Partner Group for Genetic Diversity Studies at CDFD	771699
-748411	P-124	Preparation and characterization of peroxometal compounds and studies and their biological significance in cellular signalling	-748411
442524	P-126	Rho-dependent transcription termination machinery: mechanism of action	209670
-294516	P-127	Systematic studies on the functional network of phosphatases in cell life and death	1895283
-77108	P-128	Mechanism of iron acquisition and iron homeostasis in an opportunistic human pathogen Candida glabrata	-158488
3947	P-13	“Programme to delineate gene functions in the post – genomics era by a systematic two gene knockout method”	3947
-2550050	P-130	Comparative genetic analysis of sex chromosomes and sex determining genes in silkmoths	869
398632	P-131	Structural and functional studies of Acyl CoA Binding proteins from plasmodium falciparum	398632
-640003	P-132	Characterization of tumor suppressor function of ARIDIB, a component of the human SWI/SNF chromatin remodelling complex	-12199

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS Annexure-I

Details of Closing balances of various Earmarked / Endowement Funds (Refer Sch-3)

For the Year Ended 31st MARCH 2016

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
460117	P-133	Investigating the role of Hox gene deformed in central nervous system patterning in Drosophila melanogaster	-702990
-77061	P-134	Exploration of wild silk moth biodiversity in Manipur and their genetic characterization using molecular markers	-77061
-357268	P-135	Sys TB: A Network Program for Resolving the Intracellular Dynamics of Host Phthogen Interaction in TB Infection	-336135
-292334	P-136	Raf Kinase - a key target for modem-day theraphy against tumors	-196001
759474	P-137	Signaling pathways involved in down regulation of proinflammatory responses by PPE18 protein of Mycobacterium tuberculosis: Implication of PPE18 as therapeutics	0
-1353238	P-138	Co-evaluation of Dnmt3l and Genomic imprinting	-1500300
20000	P-139	Evaluating the role of Sirtuins and epigenetic changes during cellular senescense in context of p53 status	20000
-403336	P-140	Development of baculovirus resistant silkworms strains through synthetic miRNA based knockdown of essential viral genes	-608652
-125000	P-141	Evaluating the functional role of PTEN interacting proteins in cell survival signaling and tumor suppression	-125000
-280596	P-142	Identification of H3K4 TRI Demethylase involved in erasing H3K4 trimethylation marks at E2F Responsive promoters	-81861
-534504	P-143	Microarray based characterisation of squamous cell carcinoma of the tongue occuring in non smokers	-1381684
424130	P-144	Tri-National Training Program for Psychiatric Genetics	122130
-1112243	P-145	"H3K4 HMT family regulatescell cycle progression"	3222
433858	P-146	"Role of MLL in ribosomal RNA transcription"	59533
-677839	P-147	"The Effect of Parental Education, Ethics of Research Participation and Array Comparative Genomic Hybridization in Subjects with Mental Retardation (MR) and /or Autism"	-272874
-1016335	P-149	"Role of SUMOylation in the pathobiology of Candida Glabrata "	-59917
-601366	P-151	"Human Exome Sequencing to Identify Novel Genes for Medelian Disorders "	375851
29100	P-152	"Global transcriptomics of sex specific spilicing "	-30814
641552	P-153	"An attractive and promising stragegy for early cancer diagnosis through the assembly of the human cancer volatome"	-64305
30832	P-154	"Rational design, synthetic strategies for developing organometallic anticancer compounds based on organotin and organoiron "	13510
335194	P-155	"Studies on thecellular roles of calcium signalling proteins in Neurospora crassa "	335194
-175165	P-156	"Targeting microbial quorum sensing to demonstrate potential application of cell-cell signaling molecules from Xanthomonas group of plant pathogen in diesease control "	239949
204372	P-157	"Identification of novel antifungal drug and delineation of drug resistance mechanisms in an opportunistic human fungal pathogen Candida glabrata "	-1361799
-1379658	P-158	"Modulation of host immune responses by a PPE Protein of Mycobacterium tuberculosis: Understanding its role in host - pathogen cross-talk "	-2575346
0	P-159	"Gene Targeting of microbial isolates to demonstrate potential plant growth promoting (PGP) traits by third generation sequencing "	-300000
208333	P-160	"Understanding the role of novel adhesins of Xanthomonas oryzae PV orzae in Virulence and colonization in Rice "	-41667
84656	P-161	"Analysis of co-regulation between DNA replication activity and amino acid homeostatis by transcription factor IciA/ArgP in Eschericia coli	0
-316464	P-162	Characterization and design of inhibitors of Mycobacterium tuberculosis transcription	-1021767
1052471	P-163	Unravelling new functions for the H-NS family of proteins in Gram-negative bacterial pathogens "	678659
-24671	P-164	"A Yeast based screen for discovery of novel sirtuin inhibitors as anticancer agents "	-29200
330135	P-165	"Identification and functional characterization of immune response genes in silkmoths "	1567830
2165638	P-166	"Sequencing analysis of transcriptome variants in early-onset sporadic rectal cancer "	35696
633780	P-167	"To elucidate the role of MLL complex in epigenetic specification of centromeres "	569787
788623	P-168	"A Search for nucleus -limited genes in Neurospora "	0

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS Annexure-I

Details of Closing balances of various Earmarked / Endowement Funds (Refer Sch-3)

For the Year Ended 31st MARCH 2016

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
1758108	P-169	"Implementation of 3 year DNB Program in Medical Genetics by Department of Biotechnology in colloboration with National Board of Examination ag SGHR, NIBMG&CDFD "	16915
-687887	P-17	"Studies on inosital-phosphate synthesis – a novel enzyme from Mycobacterium tuberculosis H37RV" – Transfer from IMTECH, Chandigarh "	-687887
277449	P-170	"Women Scientist Scheme "Identification and character of deregulated micro RNAs in defined sub-set of early onset sporadic rectal cancer patients using transcriptome sequencing"	-659867
1754447	P-171	"Role of vesicle-mediated transport and chromatin remodelling in the virulence of Candida glabrata "	211423
1461747	P-172	"Molecular Characterization of early onset sporadic rectal cancer "	111850
584882	P-173	"Development and application of a next generation sequencing approach for molecular genetic analysis of lysosomal storage disorders "	487953
500000	P-174	"Is non-canonical Wnt signalling a major player in early-onset sporadic rectal cancer "	520542
-509714	P-175	"Multi Centri Collaborative study of the Clinical, Biochemical and Molecular Characterization of Lysosomal storage disorders in India - The initiative for research in Lysosomal Storage Disorders"	-1432672
200103	P-176	International Atomic Energy Agency	200103
0	P-177	"Morphological and molecular taxonomy of the Phlebotomus argendtipes species complex in relation to transmission of Kala-azar in India"	-197394
0	P-179	"Quality Assurance Programme for Molecular and Prenatal Diagnosis of Hemoglobin Opathies	-50000
-274286	P-18	"Mapping of receptor binding site on the Eythrocyte binding of malaria parasyte"	-274286
0	P-180	"Collaborative studies on genomic diversity among bombycoid silkmoths in Asia "	117886
0	P-181	"To conduct multilocational field trails on transgenic BmNPV resistant silkworm strains to establish their efficacy and generate data for their regulatory approval "	1744000
0	P-182	"Ramalingaswami Fellowship	-277500
0	P-184	"Computational Approaches to Understanding Peptide- Protein Interactions involved in the Regulatory Events in the Cell"	957742
0	P-185	"Investigating potential of mycobacterium tuberculosis protein PPE18 encapsulated nanoparticle as therapy for microbial sepsis "	1632207
0	P-186	"In vivo corss-talks between Rho-dependent transcription termination and other biological processes "	2410000
0	P-187	"Understanding the mechanism of induction of innate immunity in plants by the Xanthomonas Diffusible signal factor (DSF) "	1368000
0	P-188	"Identification of Novel Genes for Intellectual Disability "	1450000
0	P-189	"Characterization of glycosylphosphatidylinositol-linked aspartyl proteases in Candida glabrata: role in pathosgenicity "	16858467
0	P-190	"Exploring mycobacteriophages to source novel factors / regulators of bacterial transcription machinery "	1100000
-1888111	P-20	"Genomic Micro array R&D Programmes on infectious diseases and Neurological Disorders"	-1888111
0.5	P-22	"Biotechnology for leather – towards cleaner processing"	0.5
-34495	P-23	"Development of PCR base assays for detection of GMO S"	-34495
-529111	P-25	"Functional studies of Human Immuno - deficiency Virus Type– 2 (HIV-2) Viral protien X (VPX)"	-529111
-79533	P-26	Occurrence of Mutations in Non dividing cells of Escherichia Coli"	-79533
-37624	P-28	Baculovirus resistance in transgenic silkworms	-37624
-310302	P-29	"Development of Hospital Surveillance system by advanced diagnostics method & Molecular DNA fingerprinting techniques"	-310302
2045696	P-30	Transcription termination and anti termination in E-coli	0
746453	P-31	Role of K-ras in Lung type II epithelial cells	0
-234000	P-33	"Molecular and Epidemiological characterisation of cryptosporidium – An enteric protozoon parasite"	-234000
26334	P-34	"Molecular analysis of lepidopteran – specific immune protiens from silkmoths"	26334
-283883	P-35	"Identification, Characterization and Physical mapping of Z-Chromosome linked genes of the silk worm, Bombyxmori"	-283883

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS Annexure-I

Details of Closing balances of various Earmarked / Endowement Funds (Refer Sch-3)

For the Year Ended 31st MARCH 2016

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
2073896	P-36	"Development of Artificial retina using Bacterio rhodospin and genetically engineered analogues "	2073896
-4058	P-40	"Antioxidants as a potential immuno adjuvant in anti tuberculosis immunotherapy"	-4058
1873605	P-41	"Construction, characterization and analysis of expressed sequences from silkworm "	1873605
-2237285	P-42	"Structural and functional studies on Mycobacterium tuberculosis heat shock proteins".	-0.36
685906.7	P-43	"A generalized mechanism of transcription termination in prokaryotes: a quest for mechanism based transcription inhibitors for microbial pathogens".	0.22
-457538	P-44	"Understanding of role of Ras and NO / iNOS signalling in promotion of hepatocellular carcinomas with persistent HBV infection"	-457538
605714	P-45	Specialized chromatin structures as epigenetic imprints to distinguish parental alleles".	0
-1586965	P-47	Research cum Training for DRDO Programme	-1586965
151826	P-48	'Molecular characterization of human liver stem cells for use in the treatment of hepatic diseases'.	151826
1041952	P-49A	International Atomic Energy Agency (IAEA)	1041952
-284065	P-51	"Understanding the mechanism of doxorubicin resistance in breast cancer celline MCF-7"	-284065
-1231118	P-52	"Nucleo Cytoplasmic transport of HIV – 1 Vpr"	-1231118
-37877	P-54	"Study of viability of Mycobacterium leprae in clinical samples and possibility of its presence in the environment using nucleic acid amplification techniques."	-37877
224	P-55	"Identification of DNA Markers for baculovirus resistance in silkworm, Bombyx mori"	224
-1231164	P-56	"Genetics of transcription-replication interplay and of stress adaptation in bacteria"	-1231164
-2215024	P-59	"An integrated Approach towards understanding the biology of Mycobacterium tuberculosis: Genetic, biochemical, immunological and structural analyses."	-2215024
482124	P-60	"National Database of Prevalent Genetic Disorders in India: Development, Curation and Services"	482124
-280000	P-61	"Dissection of a novel phenotype of lethal accumulation of potassium in Escherichia coli mutants defective in thioredoxin/thioredoxin reductase and nucleoied protein H-NS"	-280000
-278928	P-62	"HIV – 1 Pathogenesis: Role of Integrase in Reverse Transciption and Nuclear Transport of Viral Genome"	-278928
-837574	P-63	"Upgradation of the existing computing infrastructure at the Bioinformatics facility at CDFD"	-773874
-158	P-64	Biotechnology for Leather: Towards cleaner processing phase-II	-158
-582647	P-65	"Molecular, genetic and functional analysis of the chromosomal plasticity region of the gastric pathogen Helicobater pylori"	-582647
21828405	P-65A	APEDA-CDFD Centre for Basmati DNA Analysis	22811205
-681246	P-66	Human Epigenome Variation: Analysis of CpG island methylation in chromosomes 18 and Y, and in some Hox, insulin signaling and chromatin reprogramming genes	-681246
-113545	P-67	Identification of novel Esophageal Squamous cell carcinoma (ESCC) genes by using a combination of array-based CGH and gene expression micro arrays	-113545
-59874	P-68	Identification of High risk individual with pre-cancerous states of esophageal cancer.	-59874
-21336	P-70	Identification of disease causing mutations in familial hypertrophic cardiomyopathy (FHC) patients from Andhra Pradesh	-21336
-1421653	P-72	Nuances of non coding DNA near insulin-responsive genes.	-1421653
-857136	P-73	Identification and characterization of pancreatic cancer genes located within novel localized cpy number alterations	-857136
-10840	P-75	Preparing blueprint for the macromolecular crystallography beamline at Indus-II synchrotron source	-10840
-50234	P-76	A study of molecular markers in childhood autism with special references to nuclear factors - α APPA B	-50234
124277	P-77	Functional characterization of Mycobacterium tuberculosis PE/PPE proteins having SH3 binding domain : Understanding their role in modulating macrophage functions	124277
1304	P-78	Task force- IMD Newborn screening for Congenital Hypothyroidism & Congenital Adrenal Hyperplasia: A multicentric study	1304
-105086	P-79	Understanding the role of AGE proteins in inducing inflammatory responses and its regulation	-105086
-608222	P-80	Referral centre for detection of genetically modified foods employing DNA-based markets	-608222

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS Annexure-I

Details of Closing balances of various Earmarked / Endowement Funds (Refer Sch-3)

For the Year Ended 31st MARCH 2016

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
143470	P-81	Reconstructing Cellular Networks: Two-component regulatory systems	143470
62620	P-81A	Financial assistance for award of J C Bose Fellowship to Dr J Gowrishankar	2620
-369021	P-82	Functional genomic analysis of Candida Glabrata-macrophage	-369021
-1155594	P-83	Prokaryotic Transcription termination factor, Rho: Mechanism of Action and Biology	-1155594
-1150	P-84	Preparing for vaccine efficacy trials: Baseline epidemiology, improved diagnosis, markers of protection and phase I/II trials	-1150
-106479	P-84A	Human epigenetic to the rescue of human identification process: Enriching human DNA from DNA mixture employing antibodies directed against 5-methylcytosine followed by whole genome amplification	-106479
-1118755	P-85	IdeR associated gene regulatory network in mycobacteria	-1118755
-65698	P-87	Comparative genomics of wild silkmoths	-65698
-636286	P-90	Role of Yapsins in the Pathobiology of Candida Glabrata	-636286
-1098900	P-91	DMMT3L: epigenetic correlation with cancer	-1098900
268823	P-92	Swarnajayanti fellowship proj on "Designing transcription anti-terminators: a novel approach for making new inhibitors of gene expression"	268823
-611833	P-93/ A1	Virtual Centre of Excellence on multidisciplinary approaches aimed at interventions against tuberculosis	-611833
-3025061	P-93/ A2	Virtual Centre of Excellence on multidisciplinary approaches aimed at interventions against Mycobacterium tuberculosis	-3038491
1110000	P-93B2 (II)	Evaluation of peptides / small molecules targeting ESAT-6:B2M interaction and PPE18-TLR2 interaction as potent anti tuberculosis therapautics	483835
-276552	P-97	Proteome-wide Analysis of Serine pyrophosphorylation by inositol pyrophosphates	-276552
-236042	P-98	Role of cell - cell signaling mediated by Diffusible signaling factor (DSF) in Xanthomonas virulence	-236042
-567516	P-99	Role of inositol Pyrophosphates in eukaryotic cell growth, proliferation and ribosomae biogenesis	-567516
-13731478.8			**-18029486.64**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS Annexure-II

Details of Closing balances of various Earmarked / Endowement Funds (Refer Sch-3)

For the Year Ended 31st MARCH 2016

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
11713327	COE-I	COE for Genetics and Genomics of silkmoths	11713327
10156100	COE-II	DBT Centre of Excellence for Microbial Biology	12450437
600000	P-03	"Transgenesis and Genetic basis of Pathogen Resistance in the Silkworm, Bombyx Mori	600000
329289	P-07	"Collection of well characterised clinical samples and strains of Mycobacterium tuberculosis and development of molecular techniques for detection of drug resistant strains – Multi Centric Project"	329289
588400	P-09	"NMITLI Project on – Latent M.Tuberculosis: New targets, Drug delivery systems, Bio enhancers & Therapeutics"	588400
47400	P-10	"Role of upstream sequence elements in Hyper activation of transcription from Baculovirus polyhedrin gene promoter"	47400
17784	P-100	Effect of raective oxygen species on T-Cell immune response: An approach to understand the molecular mechanism of immunosuppression during tuberculosis - National Bioscience Award	17784
13084732	P-101	Role of inositol pyrophosphates in cell physiology: Investigating the biochemical significance of protein pyrophosphorylation - Senior Fellowship	14378004
698550	P-102	Understanding the role of Mycobacterium tuberculosis heat shockprotein 60 as Th1/Th2 immuno modular	698550
1000000	P-107	IYBA Project - Mechanism and role of bacterial cell-cell signaling molecules in plant defense response	1000000
915968	P-109	Molecular dissection of PI3-Kinase/Akt pathway by suing proteomics based approach: A study to identify novel potential oncogenes and tumor suppressors	3711105
206800	P-111	Ramalingaswami Fellowship - Refractoriness mechanism in Mosquito: cracking molecular codes at genomic scale	206800
0	P-112	Ramanujan Fellowship	0
670095	P-113	Clinical and molecular genetic analysis of squamous cell carcinoma of the tongue	670095
475900	P-114	Evaluating the Calcineurin-NFAT Pathway and its regulators superoxide dismutase (SOD) AND RCAN1 (regular of Calcineurin) Down Syndrome	475900
4580214	P-115	Setting up of the National Institute of Animal Biotechnology	4580214
800000	P-116	DBT-India and AIST - Japan : Understanding molecular mechanisms controlling dual role of Ras, Sirtuins and CARF in relation to cellular proliferation and senescence: Novel Strategy for developing cancer therapeutics	800000
183443	P-118	Construction of regulatory networks in Mycobacterium tuberculosis through analysis of gene expression data and transcription regulation predictions. (MOU with Russian Foundation)	183443
529750	P-12	Molecular genetics and Functional genomics of M.Tuberculosis patient isolates in India	529750
10824792	P-122	Understanding the role of Hox genes in anterior-posterior axis determination of the central nervous system	12079632
1022127	P-123	Establish a Max Planck Partner Group for Genetic Diversity Studies at CDFD	1509561
591694	P-126	Rho-dependent transcription termination machinery: mechanism of action	758900
6755620	P-127	Systematic studies on the functional network of phosphatases in cell life and death	6776327
1690360	P-128	Mechanism of iron acquisition and iron homeostasis in an opportunistic human pathogen Candida glabrata	1770000
1334600	P-13	"Programme to delineate gene functions in the post – genomics era by a systematic two gene knockout method"	1334600
81500	P-130	Comparative genetic analysis of sex chromosomes and sex determining genes in silkmoths	1008000
1018512	P-133	Investigating the role of Hox gene deformed in central nervous system patterning in Drosophila melanogaster	1054297
5500000	P-135	Sys TB: A Network Program for Resolving the Intracellular Dynamics of Host Phthogen Interaction in TB Infection	5500000
815232	P-137	Signaling pathways involved in down regulation of proinflammatory responses by PPE18 protein of Mycobacterium tuberculosis: Implication of PPE18 as therapeutics	900000
565518	P-138	Co-evaluation of Dnmt3l and Genomic imprinting	700000
500000	P-139	Evaluating the role of Sirtuins and epigenetic changes during cellular senescense in context of p53 status	500000
5163243	P-14	"Comparitive and functional genomics approaches for the identification and characterization of genes responsible for multi drug resistance of mycobacterium tuberculosis"	5163243

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS Annexure-II

Details of Closing balances of various Earmarked / Endowement Funds (Refer Sch-3)

For the Year Ended 31st MARCH 2016

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
500000	P-140	Development of baculovirus resistant silkworms strains through synthetic miRNA based knockdown of essential viral genes	500000
651933	P-142	Identification of H3K4 TRI Demethylase involved in erasing H3K4 trimethylation marks at E2F Responsive promoters	650000
1868000	P-145	"H3K4 HMT family regulatescell cycle progression "	1868000
1000000	P-146	"Role of MLL in ribosomal RNA transcription"	1000000
468720	P-149	"Role of SUMOylation in the pathobiology of Candida Glabrata "	469000
6000000	P-15	"The Helicobacter Pylori genome programme – Genome sequencing, functional analysis and comparitive genomics of the strains obtained from Indian patients"	6000000
3000000	P-153	"An attractive and promising stragegy for early cancer diagnosis through the assembly of the human cancer volatome"	3000000
132495	P-154	"Rational design, synthetic strategies for developing organometallic anticancer compounds based on organotin and organoiron "	132495
0	P-155	"Studies on thecellular roles of calcium signalling proteins in Neurospora crassa "	0
0	P-156	"Targeting microbial quorum sensing to demonstrate potential application of cell-cell signaling molecules from Xanthomonas group of plant pathogen in diesease control "	-4634
992265	P-157	"Identification of novel antifungal drug and delineation of drug resistance mechanisms in an opportunistic human fungal pathogen Candida glabrata "	992265
299941	P-158	"Modulation of host immune responses by a PPE Protein of Mycobacterium tuberculosis: Understanding its role in host - pathogen cross-talk "	343121
1814901	P-16	NMITLI Project on – Latent M.Tuberculosis: New targets, Drug delivery systems, Bio enhancers & Therapeutics	1814901
0	P-165	Identification and functional characterization of immune response genes in silkmoths	160082
0	P-166	Sequencing analysis of transcriptome variants in early-onset sporadic rectal cancer	2000000
39304	P-167	"To elucidate the role of MLL complex in epigenetic specification of centromeres "	560757
31450	P-168	"A Search for nucleus -limited genes in Neurospora "	396000
0	P-171	Role of vesicle-mediated transport and chromatin remodelling in the virulence of Candida glabrata	295560
0	P-172	Molecular Characterization of early onset sporadic rectal cancer	1388150
244400	P-17	"Studies on inositol-phosphate synthesis – a novel enzyme from Mycobacterium tuberculosis H37RV" – Transfer from IMTECH, Chandigarh	244400
344020	P-18	"Mapping of receptor binding site on the Eythrocyte binding of malaria parasyte"	344020
7246511	P-19	"Construction of Integrated RAPD, RFLP and Microsatellite linkage map of the Silkworm, Bombyx mori and its corelation with the Phenotypic linkage Map"	7246511
27331134	P-20	"Genomic Micro array R&D Programmes on infectious diseases and Neurological Disorders"	27331134
5300000	P-21	Development of Versatile, portable software for Bio-informatics	5300000
603747	P-22	"Biotechnology for leather – towards cleaner processing"	603747
375999	P-23	"Development of PCR base assays for detection of GMO S"	375999
0	P-24	Establishing a central facility on "Aerosol challenge in a containment facility"	0
600000	P-25	"Functional studies of Human Immuno - deficiency Virus Type– 2 (HIV-2) Viral protien X (VPX)"	600000
500000	P-26	Occurrence of Mutations in Non dividing cells of Escherichia Coli"	500000
260367	P-29	"Development of Hospital Surveillance system by advanced diagnostics method & Molecular DNA fingerprinting techniques"	260367
3746538	P-30	Transcription termination and anti termination in E-coli	3746538
3131006	P-31	Role of K-ras in Lung type II epithelial cells	3131006
4857938	P-36	"Development of Artificial retina using Bacterio rhodospin and genetically engineered analogues "	4857938
358470	P-39	"Computational analysis and functional characterization of mycobacterial protien(s) interacting with macrophase effector – APC functions – an approach to understand the molecular basis of pathogenesis of M. tuberculosis"	358470
49738	P-40	"Antioxidants as a potential immuno adjuvant in anti tuberculosis immunotherapy"	49738

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS Annexure-II

Details of Closing balances of various Earmarked / Endowement Funds (Refer Sch-3)

For the Year Ended 31st MARCH 2016

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
3894086	P-41	“Construction, characterization and analysis of expressed sequences from silkworm ”	3894086
9500000	P-42	“Structural and functional studies on Mycobacterium tuberculosis heat shock proteins”.	9500000
11970000	P-43	“A generalized mechanism of transcription termination in prokaryotes: a quest for mechanism based transcription inhibitors for microbial pathogens”.	11970000
3331377	P-45	Specialized chromatin structures as epigenetic imprints to distinguish parental alleles”.	3331377
416137	P-46	“Effect of reactive oxygen species (ROS) on immune response : Relevance in immunosuppression and Pathogenesis”	416137
377567	P-47	Research cum Training for DRDO Programme	377567
1413292	P-48	‘Molecular characterization of human liver stem cells for use in the treatment of hepatic diseases’.	1413292
198095	P-50	“Cervical cancer prevention by multiple strategies in rural community in Andhra pradesh”	198095
401738	P-51	“Understanding the mechanism of doxorubicin resistance in breast cancer celline MCF-7”	401738
1359129	P-52	“Nucleo Cytoplasmic transport of HIV – 1 Vpr”	1359129
1114495	P-53	Collaborative research project on molecular ecology and systematics	1114495
1163764	P-56	“Genetics of transcription-replication interplay and of stress adaptation in bacteria”	1163764
2131403	P-57	Improved genome annotation through a combination of machine learning and experimental methods: Plasmodium falciparum as a case study.	2131403
63000	P-58	“Indo-Malaysian collaboration in Bioinformatics: Development and maintenance of a web-portal hosting databases and tools of common interest”	63000
32974662	P-59	“An integrated Approach towards understanding the biology of Mycobacterium tuberculosis: Genetic, biochemical, immunological and structural analyses.”	32974662
5720800	P-60	“National Database of Prevalent Genetic Disorders in India: Development, Curation and Services”	5720800
4308314	P-62	“HIV – 1 Pathogenesis: Role of Integrase in Reverse Transciption and Nuclear Transport of Viral Genome”	4308314
9637574	P-63	“Upgradation of the existing computing infrastructure at the Bioinformatics facility at CDFD”	9637574
600585	P-64	Biotechnology for Leather: Towards cleaner processing phase-II	600585
260000	P-65	“Molecular, genetic and functional analysis of the chromosomal plasticity region of the gastric pathogen Helicobater pylori”	260000
16924622	P-65A	APEDA-CDFD Centre for Basmati DNA Analysis	16924622
264430	P-66	Human Epigenome Variation: Analysis of CpG island methylation in chromosomes 18 and Y, and in some Hox, insulin signaling and chromatin reprogramming genes	264430
622747	P-67	Identification of novel Esophageal Squamous cell carcinoma (ESCC) genes by using a combination of array-based CGH and gene expression micro arrays	622747
235593	P-69	ICMR adhoc New Scheme Understanding the role of PE/PPE family of M tuberculosis in the activation of HIV virus type I long terminal repeat (HIV-ILTP)	235593
1012807	P-70	Identification of disease causing mutations in familial hypertrophic cardiomyopathy (FHC) patients from Andhra Pradesh	1012807
1573795	P-71	Referral Centre for Genetic fidelity testing of tissue culture raised plants	1573795
45653	P-72	Nuances of non coding DNA near insulin-responsive genes.	45653
1000000	P-74	Molecular basic of insect plant interactions in rice under the national fund for basic and strategic research in agriculture	1000000
33672	P-75	Preparing blueprint for the macromolecular crystallography beamline at Indus-II synchrotron source	33672
245266	P-76	A study of molecular markers in childhood autism with special references to nuclear factors - α APPA B	245266
1543605	P-77	Functional characterization of Mycobacterium tuberculosis PE/PPE proteins having SH3 binding domain : Understanding their role in modulating macrophage functions	1543605
0	P-78	Task force- IMD Newborn screening for Congenital Hypothyroidism & Congenital Adrenal Hyperplasia: A multicentric study	0
496826	P-79	Understanding the role of AGE proteins in inducing inflammatory responses and its regulation	496826
4192480	P-80	Referral centre for detection of genetically modified foods employing DNA-based markets	4192480
205073	P-81A	Financial assistance for award of J C Bose Fellowship to Dr J Gowrishankar	205073

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS Annexure-II

Details of Closing balances of various Earmarked / Endowement Funds (Refer Sch-3)

For the Year Ended 31st MARCH 2016

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
1480220	P-82	Functional genomic analysis of Candida Glabrata-macrophage	1480220
912255	P-83	Prokaryotic Transcription termination factor, Rho: Mechanism of Action and Biology	912255
388583	P-83A	Understanding the mechanism of Azadirachtin-mediated cell signaling: role in anti-inflammation and anti-tumorigenesis	388583
44854	P-84	Preparing for vaccine efficacy trials: Baseline epidemiology, improved diagnosis, markers of protection and phase I/II trials	44854
1430573	P-84A	Human epigenetic to the rescue of human identification process: Enriching human DNA from DNA mixture employing antibodies directed against 5-methylcytosine followed by whole genome amplification	1430573
374630	P-89	Characterization of Mycobacterium tuberculosis transcription machinery and Bacteriophage metagenomics	374630
1376869	P-90	Role of Yapsins in the Pathobiology of Candida Glabrata	1376869
932151	P-91	DMMT3L: epigenetic correlation with cancer	932151
8500000	P-92	Swarnajayanti fellowship proj on "Designing transcription anti-terminators: a novel approach for making new inhibitors of gene expression"	8500000
2212534	P-93/A1	Virtual Centre of Excellence on multidisciplinary approaches aimed at interventions against tuberculosis	2212534
900000	P-93/A2	Virtual Centre of Excellence on multidisciplinary approaches aimed at interventions against Mycobacterium tuberculosis	913430
246320	P-95	Construction of regulatory networks in prokaryotes through protein: Protein interaction predictions and transcription regulation predictions. (MOU with Russian Foundation)	246320
1000000	P-97	Proteome-wide Analysis of Serine pyrophosphorylation by inositol pyrophosphates	1000000
2816418	P-98	Role of cell - cell signaling mediated by Diffusible signaling factor (DSF) in Xanthomonas virulence	2816418
2963482	P-99	Role of inositol Pyrophosphates in eukaryotic cell growth, proliferation and ribosomae biogenesis	2963482
299021303			**313375529**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2016

Annexure: A Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	I-Remittances	
5410533.00	TDS	6628892.00
7678934.00	Income Tax	9360877.00
13910.00	Works Tax	2509.00
1732202.00	LIC	1824286.00
275017.00	GSLI	208037.00
2686575.00	Public Provident Fund	2806680.00
573726.00	Professional Tax	584200.00
3453615.00	Service Tax	4374299.00
998280.00	Others (I-Remittances)	769380.00
411095.00	Health Insurance	533695.00
185300.00	ECCS	1462386.00
34566.00	PPF EMPLOYER SHARE	803436.00
23453753.00		**29358677.00**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2016

Annexure: B Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	Advance refunds/recovery/Adjst.	
478737.00	Advance for purchases by Staff	531359.00
255558.00	AMC for Equipment [Advance]	0.00
54643035.00	Chemicals [Advance]	12309522.00
70453.00	Computer Advance [Research Fellows]	97626.00
85330.00	Computer Advance [Staff]	121892.00
3123522.00	Consumables, glassware and Spares [Advance]	10273920.00
80600.00	Conveyance Advance	64360.00
168000.00	EMD	38500.00
76669827.00	Equipment [Advance]	15673247.00
132375.00	Festival Advance	171225.00
0.00	GDA [Others]	2450.00

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2016

Annexure: B Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
5915870.00	General Deposits And Advances	3357295.00
120836854.00	Inter Bank Transfer	121500000.00
174000.00	Lab Security Deposit & Hostel Security Deposit	159000.00
1358506.00	LTC [Advance]	824965.00
9166.00	Other Research Expenses [Advance]	0.00
304927.00	Others [Advances]	36264.00
440208.00	Revolving Advance	343759.00
30000.00	Security Deposit	0.00
1266313.00	TA Abroad [Advance]	206595.00
2024892.00	TA With in India [Advance]	2481663.00
12000.00	Trainee Security Deposit	12000.00
1557199.00	Workshop & Conference	2114275.00
269637372.00		**170319917.00**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2016

Annexure: C Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	Projects - Receipts	
9102000.00	COE1/CORE	8335000.00
732000.00	COE1/P-I	638000.00
459000.00	COE1/P-II	491000.00
1090000.00	COE1/P-III	1086000.00
2186000.00	COE2-II/P-1	650000.00
1093000.00	COE2-II/P-A	0.00
500000.00	COE2-II/P-B	0.00
1093000.00	COE2-II/P-C	0.00
500000.00	COE2-II/P-D	0.00
1093000.00	COE2-II/P-E	0.00
11236000.00	COE2-II-Core	0.00
463000.00	COE-I/P-IV	331000.00
9098800.00	P-101	3868930.00

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2016

Annexure: C Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
2898000.00	P-104	0.00
227909.00	P-106	0.00
1854000.00	P-107	0.00
5056000.00	P-109	2479000.00
1635000.00	P-111	0.00
828000.00	P-120	0.00
1213195.00	P-122	8005983.00
2449811.00	P-123	1413360.00
1433700.00	P-126	0.00
4990612.00	P-127	6736571.00
807800.00	P-128	0.00
0.00	P-130	4024000.00
1902500.00	P-131	0.00
3046200.00	P-132	0.00
867000.00	P-133	0.00
235000.00	P-134	0.00
2371000.00	P-135	2430700.00
570000.00	P-136	0.00
2500000.00	P-137	-464025.00
520000.00	P-139	0.00
835000.00	P-140	0.00
600000.00	P-141	0.00
935920.00	P-142	196800.00
1144199.00	P-143	0.00
424130.00	P-144	0.00
1870600.00	P-145	1200000.00
809000.00	P-146	0.00
0.00	P-147	500000.00
0.00	P-149	1420800.00
153846.00	P-150	0.00
0.00	P-151	1756400.00
2562571.00	P-152	1931400.00
621000.00	P-153	0.00
943000.00	P-154	930000.00
1076500.00	P-156	1706000.00

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2016

Annexure: C Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
1317000.00	P-157	0.00
531649.00	P-160	687200.00
0.00	P-163	1062777.00
188000.00	P-164	0.00
0.00	P-165	2858334.00
4383200.00	P-166	574700.00
1700000.00	P-167	1500000.00
1400000.00	P-168	1000000.00
1890000.00	P-169	0.00
820000.00	P-170	0.00
2415730.00	P-171	0.00
2100000.00	P-172	1200000.00
699782.00	P-173	699782.00
500000.00	P-174	500000.00
200103.00	P-176	0.00
0.00	P-177	225000.00
0.00	P-178	1000000.00
0.00	P-179	50000.00
0.00	P-180	200000.00
0.00	P-181	1744000.00
0.00	P-184	1060000.00
0.00	P-185	1648000.00
0.00	P-186	2410000.00
0.00	P-187	1368000.00
0.00	P-188	1450000.00
0.00	P-189	16858467.00
0.00	P-190	1100000.00
0.00	P-42	6869463.64
0.00	P-43	75038.52
237292.00	P-49A	0.00
1211236.00	P-65A	1338000.00
1360000.00	P-81A	1300000.00
1110000.00	P-93B2 (II)	0.00
108091285.00		**98445681.16**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2016

Annexure: D Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	Advances	
538638.00	Advance for purchases by Staff	596022.00
251855.00	AMC for Equipment [Advance]	0.00
4139900.00	Chemicals [Advance]	4716258.00
168592.00	Computer Advance [Research Fellows]	140000.00
270000.00	Computer Advance [Staff]	120000.00
0.00	Computer maintenance [Advance]	0.00
9467022.00	Consumables, glassware and Spares [Advance]	4743564.00
0.00	Conveyance [Advance]	1800.00
30000.00	Conveyance Advance	120000.00
42000.00	DG Set Maintenance [Advance]	0.00
147200.00	EMD	559000.00
28608232.00	Equipment [Advance]	17952399.00
0.00	Fellowship [Advance]	0.00
161250.00	Festival Advance	166500.00
0.00	GDA [Others]	105900.00
0.00	General Deposits And Advances	2541000.00
8000.00	Honorarium [Advance]	0.00
199000.00	Human Resource Develpment - Training of Staff - Conferences [Advance]	0.00
120836854.00	Inter Bank Transfer	121500000.00
101594.00	Lab Security Deposit & Hostel Security Deposit	129000.00
99351.00	Liveries & Blankets [Advance]	0.00
1519510.00	LTC [Advance]	698550.00
238481.00	Medical [Advance]	0.00
0.00	Membership Fee [Advance]	3301.00
6230.00	Others [Advances]	209077.00
1000.00	Others [Maintenance Advance]	0.00
1264.00	Postage-Courier [Advance]	0.00
392500.00	Revolving Advance	358000.00
600000.00	Royalty & Consultancy	122500.00
25000.00	Scientific Workshops - Symposiums - Seminars [Advance]	0.00
142500.00	Security Deposit	47800.00
743761.00	TA Abroad [Advance]	362000.00
1731760.00	TA With in India [Advance]	2215217.00
11000.00	Trainee Security Deposit	10500.00
0.00	Transport maintenance [Advance]	11510.00
1981331.00	Workshop & Conference	1114953.00
172463825.00		158544851.00

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2016

Annexure: E Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	I-Remittances	
185300.00	ECCS	1462386.00
507594.00	GSLI	205483.00
558782.00	Health Insurance	672784.00
7639801.00	Income Tax	9360458.00
1732202.00	LIC	1824286.00
970820.00	Others (I-Remittances)	769380.00
0.00	PPF EMPLOYER SHARE	275566.00
570911.00	Professional Tax	585300.00
2678290.00	Public Provident Fund	2525070.00
3128141.00	Service Tax	4972523.00
5214401.00	TDS	5508643.00
0.00	Works Tax	0.00
23186242.00		**28161879.00**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2016

Annexure: F Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	Projects - Expenditure	
8700539.00	COE1/CORE	8636177.00
637866.00	COE1/P-I	693390.00
491226.00	COE1/P-II	664953.00
1059200.00	COE1/P-III	1059200.00
4606321.00	COE2/CORE	0.00
0.00	COE2/P-1	0.00
343200.00	COE2/P-2	0.00
269100.00	COE2/P-A	0.00
269100.00	COE2/P-B	0.00
0.00	COE2/P-C	0.00
114735.00	COE2-II/P-1	2216484.00
289700.00	COE2-II/P-A	829368.00
200000.00	COE2-II/P-B	810077.00
289700.00	COE2-II/P-C	225665.00

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2016

Annexure: F Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
0.00	COE2-II/P-D	200000.00
16774.00	COE2-II/P-E	362287.00
1712677.00	COE2-II-Core	7786755.00
330839.00	COE-I/P-IV	340400.00
5966877.00	P-101	10728730.00
751285.00	P-104	129389.00
832709.00	P-107	670116.00
1762354.00	P-109	5062393.00
915739.00	P-111	1169677.00
122761.00	P-120	0.00
5201628.00	P-122	5443566.00
1560986.00	P-123	2043796.00
198495.00	P-124	0.00
172619.00	P-125	0.00
1026566.00	P-126	232854.00
5569121.00	P-127	4546772.00
275966.00	P-128	81380.00
2790.00	P-13	0.00
5415581.00	P-130	1473081.00
258529.00	P-131	0.00
1519732.00	P-132	-627804.00
941497.00	P-133	1163107.00
155624.00	P-134	0.00
2429945.00	P-135	2409567.00
875952.00	P-136	-96333.00
1784667.00	P-137	295449.00
715159.00	P-138	147062.00
520000.00	P-139	0.00
1384427.00	P-140	205316.00
501463.00	P-141	0.00
814638.00	P-142	-1935.00
927400.00	P-143	847180.00
0.00	P-144	302000.00
1918061.00	P-145	84535.00
1138581.00	P-146	374325.00
719150.00	P-147	95035.00
1287200.00	P-149	464382.00
125750.00	P-150	0.00

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2016

Annexure: F Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
1196347.00	P-151	779183.00
3647616.00	P-152	1991314.00
3593010.00	P-153	705857.00
999600.00	P-154	947322.00
2178297.00	P-156	1290886.00
2057293.00	P-157	1566171.00
2001445.00	P-158	1195688.00
300000.00	P-159	300000.00
687200.00	P-160	937200.00
265344.00	P-161	84656.00
552135.00	P-162	705303.00
953577.00	P-163	1436589.00
186000.00	P-164	4529.00
1239547.00	P-165	1620639.00
2217562.00	P-166	2704642.00
1066220.00	P-167	1563993.00
611377.00	P-168	1788623.00
131892.00	P-169	1741193.00
542551.00	P-170	937316.00
661283.00	P-171	1543024.00
638253.00	P-172	2549897.00
114900.00	P-173	796711.00
0.00	P-174	479458.00
509714.00	P-175	922958.00
0.00	P-177	422394.00
0.00	P-178	1000000.00
0.00	P-179	100000.00
0.00	P-180	82114.00
0.00	P-182	277500.00
0.00	P-184	102258.00
0.00	P-185	15793.00
0.00	P-30	2045696.00
0.00	P-31	746453.00
0.00	P-42	4632179.00
0.00	P-43	760945.00
0.00	P-45	605714.00
0.00	P-63	-63700.00
0.00	P-65A	355200.00

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2016

Annexure: F Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
1760833.00	P-81A	1360000.00
218818.00	P-88	0.00
6088.00	P-93/A1	0.00
555228.00	P-93/A2	13430.00
0.00	P-93B2 (II)	626165.00
32623.00	P-98	0.00
96048982.00		**102743689.00**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2016

Annexure: G Forming part of Balance Sheet

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	CDFD C.P.F ACCOUNT	
37788349.00	Opening Balance Add	40638533.37
5433264.00	Employee subscription/ refunds	5518714.00
0.00	Transfer from other departments	466203.00
0.00	Institute contribution (inc. Projects staff)	0.00
208230.00	Interest received	86454.00
2791310.00	Less Advances/withdrawals/Transfer/Adjst	2089882.00
40638533.00		**44620022.37**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2016

Annexure: H Forming part of Balance Sheet

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	LOANS AND ADVANCES	
206242.00	Advance for purchases by Staff	270904.50
4310.00	Advances [Previous Years]	4310.00
10553396.00	Chemicals [Advance]	2960132.00
114999.00	Computer Advance [Research Fellows]	157373.00
327270.00	Computer Advance [Staff]	325378.00
17635061.00	Consumables, glassware and Spares [Advance]	12104705.00

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2016

Annexure: H Forming part of Balance Sheet

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
0.00	Conveyance [Advance]	1800.00
127648.00	Conveyance Advance	183288.00
6638.00	DA [Advance]	6638.00
270864.00	Equipment [Advance]	2550016.00
104175.00	Festival Advance	99450.00
282172.00	Health Insurance	421261.00
130351.00	Liveries & Blankets [Advance]	130351.00
2685964.00	LTC [Advance]	2559549.00
30843.00	Miscellaneous Salary [Advance]	30843.00
95557.00	NPS Subscription	66681.00
22700.00	Office Equipment [Advance]	22700.00
5652868.00	Others [Advances]	5825681.00
53387.00	Pay of Establishment [Advance]	53387.00
304569.00	Rent [Advance]	304569.00
12343905.00	Research Fellows-Associates	32559396.00
105466.00	Revolving Advance	119707.00
0.00	Service Tax	350893.00
0.00	TA Abroad [Advance]	90156.00
270836.56	TA With in India [Advance]	4390.00
26500.00	Trainee Security Deposit	25000.00
0.00	Transport maintenance [Advance]	11510.00
639183.00	Workshop & Conference	0.00
51994904.56		**61240068.50**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2016

Annexure: I Forming part of Balance Sheet

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	DEPOSITS	
16465765.00	General Deposits And Advances	15649470.00
735977.00	GDA[Others]	839427.00
17201742.00		**16488897.00**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2016

Annexure: J Forming part of Balance Sheet

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	INVESTMENT A/C	
35098273.00	Investments	71098273.00
0.00	Other Investments	0.00
35098273.00		**71098273.00**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2016

Annexure: K Forming part of Balance Sheet

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	CDFD C.P.F INVESTMENT A/C	
33131298.00	Deposit with Banks	33593376.00
5466128.00	Employee subscription	5666653.00
5004050.00	Less Transfer To Bank A/C	9194308.00
33593376.00		**30065721.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-03: "Transgenesis and Genetic basis of Pathogen Resistance in the Silkworm, Bombyx Mori
P.I:
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	630047.00	Opening Balance	630047.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**630047.00**		**630047.00**
630047.00	Excess of Expenditure Over Income	630047.00	0.00	Closing Balance	0.00
630047.00		**630047.00**	**630047.00**		**630047.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-09: "NMITLI Project on – Latent M.Tuberculosis: New targets, Drug delivery systems, Bio enhancers & Therapeutics"
P.I: Dr Seyed E Hasnain
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
244305.00	Opening Balance	244305.00		Opening Balance	0.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
244305.00		**244305.00**	**0.00**		**0.00**
0.00	Excess of Expenditure Over Income	0.00	244305.00	Closing Balance	244305.00
244305.00		**244305.00**	**244305.00**		**244305.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-10: "Role of upstream sequence elements in Hyper activation of transcription from Baculovirus polyhedrin gene promoter"

P.I: Dr M D Bashyam

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		28332.00		Opening Balance	28332.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**28332.00**			**28332.00**	
28332.00		Excess of Expenditure Over Income	28332.00		0.00		Closing Balance	0.00	
28332.00			**28332.00**		**28332.00**			**28332.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-13: "Programme to delineate gene functions in the post – genomics era by a systematic two gene knockout method"

P.I: Dr J Gowrishankar

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
6737.00		Opening Balance	3947.00				Opening Balance	0.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		2790.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
6737.00			**3947.00**		**2790.00**			**0.00**	
0.00		Excess of Expenditure Over Income	0.00		3947.00		Closing Balance	3947.00	
6737.00			**3947.00**		**6737.00**			**3947.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-17: "Studies on inosital-phosphate synthesis – a novel enzyme from Mycobacterium tuberculosis H37RV" – Transfer from IMTECH, Chandigarh
P.I: Dr Sekhar C Mande
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	687887.00	Opening Balance	687887.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**687887.00**		**687887.00**
687887.00	Excess of Expenditure over Income	687887.00	0.00	Closing Balance	0.00
687887.00		**687887.00**	**687887.00**		**687887.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-18: "Mapping of receptor binding site on the Eythrocyte binding of malaria parasyte"
P.I: Dr Akash Ranjan
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	274286.00	Opening Balance	274286.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**274286.00**		**274286.00**
274286.00	Excess of Expenditure over Income	274286.00	0.00	Closing Balance	0.00
274286.00		**274286.00**	**274286.00**		**274286.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-20: "Genomic Micro array R&D Programmes on infectious diseases and Neurological Disorders"
P.I: Dr Hasnain & Dr Bashyam
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		1888111.00		Opening Balance	1888111.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**1888111.00**			**1888111.00**	
1888111.00		Excess of Expenditure over Income	1888111.00		0.00		Closing Balance	0.00	
1888111.00			**1888111.00**		**1888111.00**			**1888111.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-22: "Biotechnology for leather-towards cleaner processing"
P.I: Dr J Gowrishankar
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.50		Opening Balance	0.50		0.00		Opening Balance	0.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.50			**0.50**		**0.00**			**0.00**	
0.00		Excess of Expenditure over Income	0.00		0.50		Closing Balance	0.00	
0.50			**0.50**		**0.50**			**0.50**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-23: "Development of PCR base assays for detection of GMOS"
P.I: Dr Nagaraju & Dr Niyaz Ahmed
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		34495.00		Opening Balance	34495.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**34495.00**			**34495.00**	
34495.00		Excess of Expenditure over Income	34495.00		0.00		Closing Balance	0.00	
34495.00			**34495.00**		**34495.00**			**34495.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-25: "Functional studies of Human Immuno - deficiency Virus Type– 2 (HIV-2) Viral protien X (VPX)"
P.I: Dr Mahalingam & Dr Mande
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		529111.00		Opening Balance	529111.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**529111.00**			**529111.00**	
529111.00		Excess of Expenditure over Income	529111.00		0.00		Closing Balance	0.00	
529111.00			**529111.00**		**529111.00**			**529111.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-26: Occurrence of Mutations in Non dividing cells of Escherichia Coli"
P.I:
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		79533.00		Opening Balance	79533.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**79533.00**			**79533.00**	
79533.00		Excess of Expenditure over Income	79533.00		0.00		Closing Balance	0.00	
79533.00			**79533.00**		**79533.00**			**79533.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-28: Baculovirus resistance in transgenic silkworms
P.I:
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		37624.00		Opening Balance	37624.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**37624.00**			**37624.00**	
37624.00		Excess of Expenditure over Income	37624.00		0.00		Closing Balance	0.00	
37624.00			**37624.00**		**37624.00**			**37624.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-29: "Development of Hospital Surveillance system by advanced diagnostics method & Molecular DNA fingerprinting techniques"
P.I: Dr K Prashanth
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	310302.00	Opening Balance	310302.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**310302.00**		**310302.00**
310302.00	Excess of Expenditure over Income	310302.00	0.00	Closing Balance	0.00
310302.00		**310302.00**	**310302.00**		**310302.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-30: Transcription termination and anti termination in E-coli
P.I: Dr Ranjan Sen
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
2045696.00	Opening Balance	2045696.00		Opening Balance	
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
2045696.00		**2045696.00**	**0.00**		**0.00**
0.00	Excess of Expenditure over Income	0.00	2045696.00	Closing Balance	2045696.00
2045696.00		**2045696.00**	**2045696.00**		**2045696.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-31: Role of K-ras in Lung type II epithelial cells
P.I: Dr Gayatri Ramakrishna
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
746453.00		Opening Balance	746453.00				Opening Balance		
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
746453.00			**746453.00**		**0.00**			**0.00**	
0.00		Excess of Expenditure over Income	0.00		746453.00		Closing Balance	746453.00	
746453.00			**746453.00**		**746453.00**			**746453.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-33: "Molecular and Epidemiological characterisation of cryptosporidium – An enteric protozoon parasite"
P.I: Dr Radha Rama Devi
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		234000.00		Opening Balance	234000.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**234000.00**			**234000.00**	
234000.00		Excess of Expenditure over Income	234000.00		0.00		Closing Balance	0.00	
234000.00			**234000.00**		**234000.00**			**234000.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-34: "Molecular analysis of lepidopteran – specific immune protiens from silkmoths"
P.I: Dr J Nagaraju
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
26334.00		Opening Balance	26334.00						
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
26334.00			**26334.00**		**0.00**			**0.00**	
0.00		Excess of Expenditure over Income	0.00		26334.00		Closing Balance	26334.00	
26334.00			**26334.00**		**26334.00**			**26334.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-35: "Identification, Characterization and Physical mapping of Z-Chromosome linked genes of the silk worm, Bombyxmori"
P.I: Dr J Nagaraju
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		283883.00		Opening Balance	283883.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**283883.00**			**283883.00**	
283883.00		Excess of Expenditure over Income	283883.00		0.00		Closing Balance	0.00	
283883.00			**283883.00**		**283883.00**			**283883.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-36: "Development of Artificial retina using Bacterio rhodospin and genetically engineered analogues "
P.I: Dr Sekhar C Mande
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
2073896.00		Opening Balance	2073896.00				Opening Balance		
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
2073896.00			**2073896.00**		**0.00**			**0.00**	
0.00		Excess of Expenditure over Income	0.00		2073896.00		Closing Balance	2073896.00	
2073896.00			**2073896.00**		**2073896.00**			**2073896.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-40: "Antioxidants as a potential immuno adjuvant in anti tuberculosis immunotherapy"
P.I: Dr Sangita Mukhopadhyay
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		4058.00		Opening Balance	4058.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**4058.00**			**4058.00**	
4058.00		Excess of Expenditure over Income	4058.00		0.00		Closing Balance	0.00	
4058.00			**4058.00**		**4058.00**			**4058.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-41: "Construction, characterization and analysis of expressed sequences from silkworm "
P.I: Dr J Nagaraju
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
1873605.00	Opening Balance	1873605.00		Opening Balance	
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1873605.00		**1873605.00**	**0.00**		**0.00**
0.00	Excess of Expenditure over Income	0.00	1873605.00	Closing Balance	1873605.00
1873605.00		**1873605.00**	**1873605.00**		**1873605.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-42: "Structural and functional studies on Mycobacterium tuberculosis heat shock proteins".
P.I: Dr Sekhar C Mande
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	2237285.00	Opening Balance	2237285.00
0.00	Grant In Aid	6869463.64	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	4632179.00
0.00		**6869463.64**	**2237285.00**		**6869464.00**
2237285.00	Excess of Expenditure over Income	0.36	0.00	Closing Balance	0.00
2237285.00		**6869464.00**	**2237285.00**		**6869464.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-43: "A generalized mechanism of transcription termination in prokaryotes: a quest for mechanism based transcription inhibitors for microbial pathogens".

P.I: Dr Ranjan Sen

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
685906.70		Opening Balance	685906.70				Opening Balance		0.00
0.00		Grant In Aid	75038.52		0.00		Salaries - Manpower		0.00
0.00			0.00		0.00		Consumables		0.00
0.00			0.00		0.00		Contingencies		0.00
0.00			0.00		0.00		Travel		0.00
0.00			0.00		0.00		Overheads		0.00
0.00			0.00		0.00		Equipment		0.00
0.00			0.00		0.00		Books		0.00
0.00			0.00		0.00		AMC		0.00
0.00			0.00		0.00		Others		0.00
0.00			0.00		0.00		Transfer of Funds		760945.00
685906.70			**760945.22**		**0.00**				**760945.00**
0.00		Excess of Expenditure Over Income	0.00		685906.70		Closing Balance		0.22
685906.70			**760945.22**		**685906.70**				**760945.22**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-44: "Understanding of role of Ras and NO / iNOS signalling in promotion of hepatocellular carcinomas with persistent HBV infection"

P.I: Dr Gayatri Ramakrishna

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		457538.00		Opening Balance		457538.00
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower		0.00
0.00			0.00		0.00		Consumables		0.00
0.00			0.00		0.00		Contingencies		0.00
0.00			0.00		0.00		Travel		0.00
0.00			0.00		0.00		Overheads		0.00
0.00			0.00		0.00		Equipment		0.00
0.00			0.00		0.00		Books		0.00
0.00			0.00		0.00		AMC		0.00
0.00			0.00		0.00		Others		0.00
0.00			0.00		0.00		Transfer of Funds		0.00
0.00			**0.00**		**457538.00**				**457538.00**
457538.00		Excess of Expenditure over Income	457538.00		0.00		Closing Balance		0.00
457538.00			**457538.00**		**457538.00**				**457538.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-45: Specialized chromatin structures as epigenetic imprints to distinguish parental alleles".
P.I: Dr Sanjeev Khosla
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
605714.00		Opening Balance	605714.00				Opening Balance		
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
605714.00			**605714.00**		**0.00**			**0.00**	
0.00		Excess of Expenditure over Income	0.00		605714.00		Closing Balance	605714.00	
605714.00			**605714.00**		**605714.00**			**605714.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-47: Research cum Training for DRDO Programme
P.I: Dr Gowrishankar, Dr Mahalingam, Dr Mande, Dr Nagaraju, Dr Ni
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		1586965.00		Opening Balance	1586965.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**1586965.00**			**1586965.00**	
1586965.00		Excess of Expenditure over Income	1586965.00		0.00		Closing Balance	0.00	
1586965.00			**1586965.00**		**1586965.00**			**1586965.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-48: 'Molecular characterization of human liver stem cells for use in the treatment of hepatic diseases'.
P.I: Dr Sanjeev Khosla
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
151826.00		Opening Balance	151826.00						
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower		0.00
0.00			0.00		0.00		Consumables		0.00
0.00			0.00		0.00		Contingencies		0.00
0.00			0.00		0.00		Travel		0.00
0.00			0.00		0.00		Overheads		0.00
0.00			0.00		0.00		Equipment		0.00
0.00			0.00		0.00		Books		0.00
0.00			0.00		0.00		AMC		0.00
0.00			0.00		0.00		Others		0.00
0.00			0.00		0.00		Transfer of Funds		0.00
151826.00			**151826.00**		**0.00**				**0.00**
0.00		Excess of Expenditure over Income	0.00		151826.00		Closing Balance		151826.00
151826.00			**151826.00**		**151826.00**				**151826.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-49A: International Atomic Energy Agency (IAEA)
P.I: J Nagaraju
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
308361.00		Opening Balance	804660.00				Opening Balance		0.00
804660.00		Opening Balance	1041952.00				Opening Balance		0.00
237292.00		Grant In Aid	0.00		0.00		Salaries - Manpower		0.00
0.00			0.00		0.00		Consumables		0.00
0.00			0.00		0.00		Contingencies		0.00
0.00			0.00		0.00		Travel		0.00
0.00			0.00		0.00		Overheads		0.00
0.00			0.00		0.00		Equipment		0.00
0.00			0.00		0.00		Books		0.00
0.00			0.00		0.00		AMC		0.00
0.00			0.00		0.00		Others		0.00
0.00			0.00		0.00		Transfer of Funds		0.00
1041952.00			**1041952.00**		**0.00**				**0.00**
0.00		Excess of Expenditure Over Income	0.00		1041952.00		Closing Balance		1041952.00
1041952.00			**1041952.00**		**1041952.00**				**1041952.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-51: "Understanding the mechanism of doxorubicin resistance in breast cancer celline MCF-7"
P.I: Dr Sunil Kumar Manna
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		284065.00		Opening Balance	284065.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**284065.00**			**284065.00**	
284065.00		Excess of Expenditure over Income	284065.00		0.00		Closing Balance	0.00	
284065.00			**284065.00**		**284065.00**			**284065.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-52: "Nucleo Cytoplasmic transport of HIV – 1 Vpr"
P.I: Dr Mahalingam & Dr Manna
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		1231118.00		Opening Balance	1231118.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**1231118.00**			**1231118.00**	
1231118.00		Excess of Expenditure over Income	1231118.00		0.00		Closing Balance	0.00	
1231118.00			**1231118.00**		**1231118.00**			**1231118.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-54: "Study of viability of Mycobacterium leprae in clinical samples and possibility of its presence in the environment using nucleic acid amplification techniques."

P.I: Dr Niyaz Ahmed

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	37877.00	Opening Balance	37877.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**37877.00**		**37877.00**
37877.00	Excess of Expenditure over Income	37877.00	0.00	Closing Balance	0.00
37877.00		**37877.00**	**37877.00**		**37877.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-55: "Identification of DNA Markers for baculovirus resistance in silkworm, Bombyx mori"

P.I: Dr J Nagaraju

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
224.00	Opening Balance	224.00			
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
224.00		**224.00**	**0.00**		**0.00**
0.00	Excess of Expenditure over Income	0.00	224.00	Closing Balance	224.00
224.00		**224.00**	**224.00**		**224.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-56: "Genetics of transcription-replication interplay and of stress adaptation in bacteria"
P.I: Dr Gowrishankar & Dr K Anupama
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		1231164.00		Opening Balance	1231164.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**1231164.00**			**1231164.00**	
1231164.00		Excess of Expenditure over Income	1231164.00		0.00		Closing Balance	0.00	
1231164.00			**1231164.00**		**1231164.00**			**1231164.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-59: "An integrated Approach towards understanding the biology of Mycobacterium tuberculosis: Genetic, biochemical, immunological and structural analyses."
P.I: Dr Hasnain, Dr Gowrishankar, Dr Mande, Dr Ranjan Sen
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		2215024.00		Opening Balance	2215024.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**2215024.00**			**2215024.00**	
2215024.00		Excess of Expenditure over Income	2215024.00		0.00		Closing Balance	0.00	
2215024.00			**2215024.00**		**2215024.00**			**2215024.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-60: "National Database of Prevalent Genetic Disorders in India: Development, Curation and Services"
P.I: Dr H A Nagarajaram
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
482124.00		Opening Balance	482124.00						
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
482124.00			**482124.00**		**0.00**			**0.00**	
0.00		Excess of Expenditure over Income	0.00		482124.00		Closing Balance	482124.00	
482124.00			**482124.00**		**482124.00**			**482124.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-61: "Dissection of a novel phenotype of lethal accumulation of potassium in Escherichia coli mutants defective in thioredoxin/thioredoxin reductase and nucleoied protein H-NS"
P.I: Dr Abhijit A Sardesai
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		280000.00		Opening Balance	280000.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**280000.00**			**280000.00**	
280000.00		Excess of Expenditure over Income	280000.00		0.00		Closing Balance	0.00	
280000.00			**280000.00**		**280000.00**			**280000.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-62: "HIV – 1 Pathogenesis: Role of Integrase in Reverse Transciption and Nuclear Transport of Viral Genome"
P.I: Dr S Mahalingam
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		278928.00		Opening Balance	278928.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**278928.00**			**278928.00**	
278928.00		Excess of Expenditure over Income	278928.00		0.00		Closing Balance	0.00	
278928.00			**278928.00**		**278928.00**			**278928.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-63: "Upgradation of the existing computing infrastructure at the Bioinformatics facility at CDFD"
P.I: Dr Seyed E Hasnain
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		837574.00		Opening Balance	837574.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	-63700.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**837574.00**			**773874.00**	
837574.00		Excess of Expenditure Over Income	773874.00		0.00		Closing Balance	0.00	
837574.00			**773874.00**		**837574.00**			**773874.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-64: Biotechnology for Leather: Towards cleaner processing phase-II
P.I: Dr J Gowrishankar
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		158.00		Opening Balance	158.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**158.00**			**158.00**	
158.00		Excess of Expenditure over Income	158.00		0.00		Closing Balance	0.00	
158.00			**158.00**		**158.00**			**158.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-65: "Molecular, genetic and functional analysis of the chromosomal plasticity region of the gastric pathogen Helicobater pylori"
P.I: Dr Ayesha Alvi
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		582647.00		Opening Balance	582647.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**582647.00**			**582647.00**	
582647.00		Excess of Expenditure over Income	582647.00		0.00		Closing Balance	0.00	
582647.00			**582647.00**		**582647.00**			**582647.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-65A: APEDA-CDFD Centre for Basmati DNA Analysis
P.I: Dr J Nagaraju
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
20617169.00	Opening Balance	21828405.00		Opening Balance	0.00
1211236.00	Grant In Aid	0.00	0.00	Salaries - Manpower	355200.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
21828405.00		**21828405.00**	**0.00**		**355200.00**
0.00	Excess of Expenditure Over Income	0.00	21828405.00	Closing Balance	21473205.00
21828405.00		**21828405.00**	**21828405.00**		**21828405.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-66: Human Epigenome Variation: Analysis of CpG island methylation in chromosomes 18 and Y, and in some Hox, insulin signaling and chromatin reprogramming genes
P.I: Dr Sanjeev Khosla
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	681246.00	Opening Balance	681246.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**681246.00**		**681246.00**
681246.00	Excess of Expenditure over Income	681246.00	0.00	Closing Balance	0.00
681246.00		**681246.00**	**681246.00**		**681246.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-67: Identification of novel Esophageal Squamous cell carcinoma (ESCC) genes by using a combination of array-based CGH and gene expression micro arrays

P.I: Dr M D Bashyam

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		113545.00		Opening Balance	113545.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**113545.00**			**113545.00**	
113545.00		Excess of Expenditure over Income	113545.00		0.00		Closing Balance	0.00	
113545.00			**113545.00**		**113545.00**			**113545.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-68: Identification of High risk individual with pre-cancerous states of esophageal cancer.

P.I: Dr Gayatri Ramakrishna

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		59874.00		Opening Balance	59874.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**59874.00**			**59874.00**	
59874.00		Excess of Expenditure over Income	59874.00		0.00		Closing Balance	0.00	
59874.00			**59874.00**		**59874.00**			**59874.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-70: Identification of disease causing mutations in familial hypertrophic cardiomyopathy (FHC) patients from Andhra Pradesh
P.I: Dr M D Bashyam
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		21336.00		Opening Balance	21336.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**21336.00**			**21336.00**	
21336.00		Excess of Expenditure over Income	21336.00		0.00		Closing Balance	0.00	
21336.00			**21336.00**		**21336.00**			**21336.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-72: Nuances of non coding DNA near insulin-responsive genes.
P.I: Dr Nirmala Yabaluri
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		1421653.00		Opening Balance	1421653.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**1421653.00**			**1421653.00**	
1421653.00		Excess of Expenditure over Income	1421653.00		0.00		Closing Balance	0.00	
1421653.00			**1421653.00**		**1421653.00**			**1421653.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-73: Identification and characterization of pancreatic cancer genes located within novel localized cpy number alterations

P.I: Dr M D Bashyam

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		857136.00		Opening Balance	857136.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**857136.00**			**857136.00**	
857136.00		Excess of Expenditure over Income	857136.00		0.00		Closing Balance	0.00	
857136.00			**857136.00**		**857136.00**			**857136.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-75: Preparing blueprint for the macromolecular crystallography beamline at Indus-II synchrotron source

P.I: Dr Sekhar C Mande

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		10840.00		Opening Balance	10840.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**10840.00**			**10840.00**	
10840.00		Excess of Expenditure over Income	10840.00		0.00		Closing Balance	0.00	
10840.00			**10840.00**		**10840.00**			**10840.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-76: A study of molecular markers in childhood autism with special references to nuclear factors - ± APPA B

P.I: Dr S K Manna

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		50234.00		Opening Balance	50234.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**50234.00**			**50234.00**	
50234.00		Excess of Expenditure over Income	50234.00		0.00		Closing Balance	0.00	
50234.00			**50234.00**		**50234.00**			**50234.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-77: Functional characterization of Mycobacterium tuberculosis PE/PPE proteins having SH3 binding domain : Understanding their role in modulating macrophage functions

P.I: Dr Sangita Mukhopadhyay

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
124277.00		Opening Balance	124277.00						
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
124277.00			**124277.00**		**0.00**			**0.00**	
0.00		Excess of Expenditure over Income	0.00		124277.00		Closing Balance	124277.00	
124277.00			**124277.00**		**124277.00**			**124277.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-78: Task force- IMD Newborn screening for Congenital Hypothyroidism & Congenital Adrenal Hyperplasia: A multicentric study
P.I: Dr A Radha Rama Devi
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
1304.00		Opening Balance	1304.00				Opening Balance		
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
1304.00			**1304.00**		**0.00**			**0.00**	
0.00		Excess of Expenditure over Income	0.00		1304.00		Closing Balance	1304.00	
1304.00			**1304.00**		**1304.00**			**1304.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-79: Understanding the role of AGE proteins in inducing inflammatory responses and its regulation
P.I: Dr S K Manna
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		105086.00		Opening Balance	105086.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**105086.00**			**105086.00**	
105086.00		Excess of Expenditure Over Income	105086.00		0.00		Closing Balance	0.00	
105086.00			**105086.00**		**105086.00**			**105086.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-80: Referral centre for detection of genetically modified foods employing DNA-based markets
P.I: Dr Madhusudan Reddy
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		608222.00		Opening Balance	608222.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
					0.00		Consumables	0.00	
					0.00		Contingencies	0.00	
					0.00		Travel	0.00	
					0.00		Overheads	0.00	
					0.00		Equipment	0.00	
					0.00		Books	0.00	
					0.00		AMC	0.00	
					0.00		Others	0.00	
					608222.00		Transfer of Funds	0.00	
0.00			**0.00**		**608222.00**			**608222.00**	
608222.00		Excess of Expenditure over Income	608222.00		0.00		Closing Balance	0.00	
608222.00			**608222.00**		**608222.00**			**608222.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-81: Reconstructing Cellular Networks: Two-component regulatory systems
P.I: Dr Shekhar Mande
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
143470.00		Opening Balance	143470.00						
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
143470.00			**143470.00**		**0.00**			**0.00**	
0.00		Excess of Expenditure over Income	0.00		143470.00		Closing Balance	143470.00	
143470.00			**143470.00**		**143470.00**			**143470.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-81A: Financial assistance for award of J C Bose Fellowship to Dr J Gowrishankar
P.I: Dr J Gowrishankar
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
463453.00	Opening Balance	62620.00		Opening Balance	0.00
1360000.00	Grant In Aid	1300000.00	300000.00	Salaries - Manpower	300000.00
0.00		0.00	962116.50	Consumables	526318.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	429371.50	Travel	473682.00
0.00		0.00	60000.00	Overheads	60000.00
0.00		0.00	9345.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1823453.00		**1362620.00**	**1760833.00**		**1360000.00**
0.00	Excess of Expenditure Over Income	0.00	62620.00	Closing Balance	2620.00
1823453.00		**1362620.00**	**1823453.00**		**1362620.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-82: Functional genomic analysis of Candida Glabrata-macrophage
P.I: Dr Rupinder Kaur
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	36904.00	Opening Balance	369021.00
0.00	Grant In Aid	0.00	1300.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**369021.00**		**369021.00**
369021.00	Excess of Expenditure Over Income	369021.00	0.00	Closing Balance	0.00
369021.00		**369021.00**	**369021.00**		**369021.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-83: Prokaryotic Transcription termination factor, Rho: Mechanism of Action and Biology
P.I: Dr Ranjan Sen
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		1155594.00		Opening Balance	1155594.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**1155594.00**			**1155594.00**	
1155594.00		Excess of Expenditure over Income	1155594.00		0.00		Closing Balance	0.00	
1155594.00			**1155594.00**		**1155594.00**			**1155594.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-84: Preparing for vaccine efficacy trials: Baseline epidemiology, improved diagnosis, markers of protection and phase I/II trials
P.I: Dr Niyaz Ahmed
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		1150.00		Opening Balance	1150.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**1150.00**			**1150.00**	
1150.00		Excess of Expenditure over Income	1150.00		0.00		Closing Balance	0.00	
1150.00			**1150.00**		**1150.00**			**1150.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-84A: Human epigenetic to the rescue of human identification process: Enriching human DNA from DNA mixture employing antibodies directed against 5-methylcytosine followed by whole genome amplification

P.I: Dr Madhusudan Reddy

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		106479.00		Opening Balance	106479.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**106479.00**			**106479.00**	
106479.00		Excess of Expenditure over Income	106479.00		0.00		Closing Balance	0.00	
106479.00			**106479.00**		**106479.00**			**106479.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-85: IdeR associated gene regulatory network in mycobacteria

P.I: Dr Akash Ranjan

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		1118755.00		Opening Balance	1118755.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**118755.00**			**1118755.00**	
1118755.00		Excess of Expenditure over Income	1118755.00		0.00		Closing Balance	0.00	
1118755.00			**1118755.00**		**1118755.00**			**1118755.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-87: Comparative genomics of wild silkmoths
P.I: Dr J Nagaraju
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		65698.00		Opening Balance	65698.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**65698.00**			**65698.00**	
65698.00		Excess of Expenditure over Income	65698.00		0.00		Closing Balance	0.00	
65698.00			**65698.00**		**65698.00**			**65698.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-90: Role of Yapsins in the Pathobiology of Candida Glabrata
P.I: Dr Rupinder Kaur
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		636286.00		Opening Balance	636286.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**636286.00**			**636286.00**	
636286.00		Excess of Expenditure over Income	636286.00		0.00		Closing Balance	0.00	
636286.00			**636286.00**		**636286.00**			**636286.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-91: DMMT3L: epigenetic correlation with cancer
P.I: Dr Sanjeev Khosla
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		1098900.00		Opening Balance	1098900.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**1098900.00**			**1098900.00**	
1098900.00		Excess of Expenditure over Income	1098900.00		0.00		Closing Balance	0.00	
1098900.00			**1098900.00**		**1098900.00**			**1098900.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-92: Swarnajayanti fellowship proj on "Designing transcription anti-terminators: a novel approach for making new inhibitors of gene expression"
P.I: Dr Ranjan Sen
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
268823.00		Opening Balance	268823.00				Opening Balance	0.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
268823.00			**268823.00**		**0.00**			**0.00**	
0.00		Excess of Expenditure Over Income	0.00		268823.00		Closing Balance	268823.00	
268823.00			**268823.00**		**268823.00**			**268823.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-93/A1 : Virtual Centre of Excellence on multidisciplinary approaches aimed at interventions against tuberculosis
P.I.: Dr Shekar
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		605745.00		Opening Balance	611833.00	
0.00		Grant In Aid	0.00		6088.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**611833.00**			**611833.00**	
611833.00		Excess of Expenditure Over Income	611833.00		0.00		Closing Balance	0.00	
611833.00			**611833.00**		**611833.00**			**611833.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-93/A2 : Virtual Centre of Excellence on multidisciplinary approaches aimed at interventions against Mycobacterium tuberculosis
P.I.: Dr. Sangita Mukhopadhyay
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		2469833.00		Opening Balance	3025061.00	
0.00		Grant In Aid	0.00		495876.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		59352.00		Equipment	13430.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**3025061.00**			**3038491.00**	
3025061.00		Excess of Expenditure Over Income	3038491.00		0.00		Closing Balance	0.00	
3025061.00			**3038491.00**		**3025061.00**			**3038491.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-93B2 (II) : Evaluation of peptides / small molecules targeting ESAT-6:B2M interaction and PPE18-TLR2 interaction as potent anti tuberculosis therapautics

P.I.: Dr Sangita Mukhopadhyay

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	1110000.00				Opening Balance	0.00	
1110000.00		Grant In Aid	0.00		0.00		Salaries - Manpower	301209.00	
0.00			0.00		0.00		Consumables	305752.00	
0.00			0.00		0.00		Contingencies	11581.00	
0.00			0.00		0.00		Travel	7623.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
1110000.00			**1110000.00**		**0.00**			**626165.00**	
0.00		Excess of Expenditure Over Income	0.00		1110000.00		Closing Balance	483835.00	
1110000.00			**1110000.00**		**1110000.00**			**1110000.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-97: Proteome-wide Analysis of Serine pyrophosphorylation by inositol pyrophosphates

P.I: Dr Rashna Bhandari

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		276552.00		Opening Balance	276552.00	
0.00		Grant In Aid	0.00		96284.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**276552.00**			**276552.00**	
276552.00		Excess of Expenditure Over Income	276552.00		0.00		Closing Balance	0.00	
276552.00			**276552.00**		**276552.00**			**276552.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-98: Role of cell - cell signaling mediated by Diffusible signaling factor (DSF) in Xanthomonas virulence
P.I: Dr Subhadeep Chatterjee
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		203419.00		Opening Balance	236042.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		32623.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**236042.00**			**236042.00**	
236042.00		Excess of Expenditure Over Income	236042.00		0.00		Closing Balance	0.00	
236042.00			**236042.00**		**236042.00**			**236042.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-99: Role of inositol Pyrophosphates in eukaryotic cell growth, proliferation and ribosomae biogenesis
P.I: Dr Rashna Bhandari
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		567516.00		Opening Balance	567516.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**567516.00**			**567516.00**	
567516.00		Excess of Expenditure Over Income	567516.00		0.00		Closing Balance	0.00	
567516.00			**567516.00**		**567516.00**			**567516.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-100: Effect of raective oxygen species on T-Cell immune response: An approach to understand the molecular mechanism of immunosuppression during tuberculosis - National Bioscience Award

P.I: Dr Sangita Mukhopadhyay

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		576590.00		Opening Balance	576590.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**576590.00**			**576590.00**	
576590.00		Excess of Expenditure Over Income	576590.00		0.00		Closing Balance	0.00	
576590.00			**576590.00**		**576590.00**			**576590.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-101: Role of inositol pyrophosphates in cell physiology: Investigating the biochemical significance of protein pyrophosphorylation - Senior Fellowship

P.I: Dr Rashna Bhandari

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
3727878.00		Opening Balance	6859801.00				Opening Balance	0.00	
9098800.00		Grant In Aid	3868930.00		1227962.00		Salaries - Manpower	842267.00	
0.00			0.00		2883561.00		Consumables	7602852.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		268246.00		Travel	15000.00	
0.00			0.00		526687.00		Overheads	975339.00	
0.00			0.00		887105.00		Equipment	1293272.00	
0.00			0.00		173316.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
12826678.00			**10728731.00**		**5966877.00**			**10728730.00**	
0.00		Excess of Expenditure Over Income	0.00		6859801.00		Closing Balance	1.00	
12826678.00			**10728731.00**		**12826678.00**			**10728731.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-102: "Understanding the role of Mycobacterium tuberculosis heat shockprotein 60 as Th1/Th2 immuno modular"
P.I: Dr Sangita Mukhopadhyay
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		27922.00		Opening Balance	27922.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**27922.00**			**27922.00**	
27922.00		Excess of Expenditure Over Income	27922.00		0.00		Closing Balance	0.00	
27922.00			**27922.00**		**27922.00**			**27922.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-103: National Bioscience Award - Regulation of mast cell signaling, apoptosis and surface receptors
P.I: Dr Sunil Kumar Manna
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		300000.00		Opening Balance	300000.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**300000.00**			**300000.00**	
300000.00		Excess of Expenditure Over Income	300000.00		0.00		Closing Balance	0.00	
300000.00			**300000.00**		**300000.00**			**300000.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-104: Virtual Centre of Excellence on Epigenetics
P.I: Dr Sanjeev Khosla
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	3307223.00	Opening Balance	1160508.00
2898000.00	Grant In Aid	0.00	403779.00	Salaries - Manpower	125806.00
0.00		0.00	220853.00	Consumables	0.00
0.00		0.00	100000.00	Contingencies	0.00
0.00		0.00	26653.00	Travel	3583.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
2898000.00		**0.00**	**4058508.00**		**1289897.00**
1160508.00	Excess of Expenditure Over Income	1289897.00	0.00	Closing Balance	0.00
4058508.00		**1289897.00**	**4058508.00**		**1289897.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-105: Cloning, Characterization and analysis of chromosomal rearrangements in human genetic disorders
P.I: Dr Ashwin B Dalal
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	862685.00	Opening Balance	862685.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**862685.00**		**862685.00**
862685.00	Excess of Expenditure Over Income	862685.00	0.00	Closing Balance	0.00
862685.00		**862685.00**	**862685.00**		**862685.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-107: DBT IYBA Project on "Mechanism and role of bacterial cell-cell signaling molecules in plant defense response"
P.I: Dr Subhadeep Chatterjee
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
15400.00	Opening Balance	1036691.00		Opening Balance	0.00
1854000.00	Grant In Aid	0.00	232709.00	Salaries - Manpower	70116.00
0.00		0.00	600000.00	Consumables	589798.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	10202.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1869400.00		**1036691.00**	**832709.00**		**670116.00**
0.00	Excess of Expenditure Over Income	0.00	1036691.00	Closing Balance	366575.00
1869400.00		**1036691.00**	**1869400.00**		**1036691.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-108: Establishment of EBV transformed cell lines from families with rare genetic disorders
P.I: Dr Ashwin B Dalal
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	454643.00	Opening Balance	454643.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**454643.00**		**454643.00**
454643.00	Excess of Expenditure Over Income	454643.00	0.00	Closing Balance	0.00
454643.00		**454643.00**	**454643.00**		**454643.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-109: Molecular dissection of PI3-Kinase/Akt pathway by suing proteomics based approach: A study to identify novel potential oncogenes and tumor suppressors

P.I: Dr M Subba Reddy

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
57690.00	Opening Balance	3351336.00		Opening Balance	0.00
5056000.00	Grant In Aid	2479000.00	211664.00	Salaries - Manpower	739256.00
0.00		0.00	1550000.00	Consumables	1517891.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	10109.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	690.00	Equipment	2795137.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
5113690.00		**5830336.00**	**1762354.00**		**5062393.00**
0.00	Excess of Expenditure Over Income	0.00	3351336.00	Closing Balance	767943.00
5113690.00		**5830336.00**	**5113690.00**		**5830336.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-110: India-Japan research project title"Identification and analysis of sex determining genes in silkmoths"

P.I: Dr J Nagaraju

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	191391.00	Opening Balance	191391.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**191391.00**		**191391.00**
191391.00	Excess of Expenditure Over Income	191391.00	0.00	Closing Balance	0.00
191391.00		**191391.00**	**191391.00**		**191391.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-111: Ramalingaswami Fellowship - Refractoriness mechanism in Mosquito: cracking molecular codes at genomic scale

P.I: Dr Shweta Tyagi

Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
450416.00	Opening Balance	1169677.00		Opening Balance	0.00
1635000.00	Grant In Aid	0.00	247950.00	Salaries - Manpower	1175750.00
0.00		0.00	650767.00	Consumables	6073.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	17022.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
2085416.00		**1169677.00**	**915739.00**		**1169677.00**
0.00	Excess of Expenditure Over Income	0.00	1169677.00	Closing Balance	0.00
2085416.00		**1169677.00**	**2085416.00**		**1169677.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-114: Evaluating the Calcineurin-NFAT Pathway and its regulators superoxide dismutase (SOD) AND RCAN1 (regular of Calcineurin) Down Syndrome

P.I: Dr Gayatri Ramakrishna, Dr Ashwin Dalal

Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	450859.00	Opening Balance	450859.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		**0.00**	**450859.00**		**450859.00**
450859.00	Excess of Expenditure Over Income	450859.00	0.00	Closing Balance	0.00
450859.00		**450859.00**	**450859.00**		**450859.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-116: DBT-India and AIST - Japan : Understanding molecular mechanisms controlling dual role of Ras, Sirtuins and CARF in relation to cellular proliferation and senescence: Novel Strategy for developing cancer therapeutics

P.I: Dr Gayatri Ramakrishna

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance		0.00	1251366.00		Opening Balance	1251366.00	
0.00		Grant In Aid		0.00	0.00		Salaries - Manpower	0.00	
0.00				0.00	0.00		Consumables	0.00	
0.00				0.00	0.00		Contingencies	0.00	
0.00				0.00	0.00		Travel	0.00	
0.00				0.00	0.00		Overheads	0.00	
0.00				0.00	0.00		Equipment	0.00	
0.00				0.00	0.00		Books	0.00	
0.00				0.00	0.00		AMC	0.00	
0.00				0.00	0.00		Others	0.00	
0.00				0.00	0.00		Transfer of Funds	0.00	
0.00				**0.00**	**1251366.00**			**1251366.00**	
1251366.00		Excess of Expenditure Over Income	1251366.00		0.00		Closing Balance	0.00	
1251366.00			**1251366.00**		**1251366.00**			**1251366.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-119: Analysis of DNA copy number alterations in esophaeal cancer

P.I: Dr M D Bashyam

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance		0.00	2892.00		Opening Balance	2892.00	
0.00		Grant In Aid		0.00	0.00		Salaries - Manpower	0.00	
0.00				0.00	0.00		Consumables	0.00	
0.00				0.00	0.00		Contingencies	0.00	
0.00				0.00	0.00		Travel	0.00	
0.00				0.00	0.00		Overheads	0.00	
0.00				0.00	0.00		Equipment	0.00	
0.00				0.00	0.00		Books	0.00	
0.00				0.00	0.00		AMC	0.00	
0.00				0.00	0.00		Others	0.00	
0.00				0.00	0.00		Transfer of Funds	0.00	
0.00				**0.00**	**2892.00**			**2892.00**	
2892.00		Excess of Expenditure Over Income	2892.00		0.00		Closing Balance	0.00	
2892.00			**2892.00**		**2892.00**			**2892.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-120: Effect of reactive oxygen species on macrophage signalosome: impact on antigen presentation functions and T Cell priming responses
P.I: Dr Sangita Mukhopadhyay
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		1474723.00		Opening Balance	769484.00	
828000.00		Grant In Aid	0.00		92761.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		30000.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
828000.00			**0.00**		**1597484.00**			**769484.00**	
769484.00		Excess of Expenditure Over Income	769484.00		0.00		Closing Balance	0.00	
1597484.00			**769484.00**		**1597484.00**			**769484.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-121: Identification and characterization of PTEN regulators
P.I: Dr M Subba Reddy
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		1130866.00		Opening Balance	1130866.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**1130866.00**			**1130866.00**	
1130866.00		Excess of Expenditure Over Income	1130866.00		0.00		Closing Balance	0.00	
1130866.00			**1130866.00**		**1130866.00**			**1130866.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-122: Understanding the role of Hox genes in anterior-posterior axis determination of the central nervous system
P.I: Dr Rohit Joshi
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
4377125.00	Opening Balance	388692.00		Opening Balance	0.00
1213195.00	Grant In Aid	8005983.00	939806.00	Salaries - Manpower	662020.00
0.00		0.00	2798321.00	Consumables	2843518.00
0.00		0.00	30454.00	Contingencies	32463.00
0.00		0.00	24747.00	Travel	44681.00
0.00		0.00	472875.00	Overheads	483752.00
0.00		0.00	935425.00	Equipment	1254840.00
0.00		0.00	0.00	Books	122292.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
5590320.00		**8394675.00**	**5201628.00**		**5443566.00**
0.00	Excess of Expenditure Over Income	0.00	388692.00	Closing Balance	2951109.00
5590320.00		**8394675.00**	**5590320.00**		**8394675.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-123: Establish a Max Planck Partner Group for Genetic Diversity Studies at CDFD
P.I: Dr N Madhusudan Reddy
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
513310.00	Opening Balance	1402135.00		Opening Balance	0.00
2449811.00	Grant In Aid	1413360.00	339509.00	Salaries - Manpower	395200.00
0.00		0.00	480492.00	Consumables	886802.00
0.00		0.00	100000.00	Contingencies	0.00
0.00		0.00	159294.00	Travel	274360.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	481691.00	Equipment	487434.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
2963121.00		**2815495.00**	**1560986.00**		**2043796.00**
0.00	Excess of Expenditure Over Income	0.00	1402135.00	Closing Balance	771699.00
2963121.00		**2815495.00**	**2963121.00**		**2815495.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-124: Preparation and characterization of peroxometal compounds and studies and their biological significance in cellular signalling
P.I: Dr Gayatri Ramakrishna
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		549916.00		Opening Balance	748411.00	
0.00		Grant In Aid	0.00		109200.00		Salaries - Manpower	0.00	
0.00			0.00		89295.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**748411.00**			**748411.00**	
748411.00		Excess of Expenditure Over Income	748411.00		0.00		Closing Balance	0.00	
748411.00			**748411.00**		**748411.00**			**748411.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-125: Mechanistic studies on the role of protein kinase Snfilk in cell cycle and cancer
P.I: Dr M Subba Reddy
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
172619.00		Opening Balance	0.00				Opening Balance	0.00	
0.00		Grant In Aid	0.00		-10800.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		183419.00		Transfer of Funds	0.00	
172619.00			**0.00**		**172619.00**			**0.00**	
0.00		Excess of Expenditure Over Income	0.00		0.00		Closing Balance	0.00	
172619.00			**0.00**		**172619.00**			**0.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-126: Rho-dependent transcription termination machinery: mechanism of action
P.I: Dr Ranjan Sen
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
35390.00	Opening Balance	442524.00		Opening Balance	0.00
1433700.00	Grant In Aid	0.00	302538.00	Salaries - Manpower	48729.00
0.00		0.00	513978.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	16919.00
0.00		0.00	20372.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	189678.00	Equipment	167206.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1469090.00		**442524.00**	**1026566.00**		**232854.00**
0.00	Excess of Expenditure Over Income	0.00	442524.00	Closing Balance	209670.00
1469090.00		**442524.00**	**1469090.00**		**442524.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-127: Systematic studies on the functional network of phosphatases in cell life and death
P.I: Dr M Subba Reddy
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
283993.00	Opening Balance	0.00		Opening Balance	294516.00
4990612.00	Grant In Aid	6736571.00	776984.00	Salaries - Manpower	432000.00
0.00		0.00	3758682.00	Consumables	3078989.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	63992.00	Travel	317282.00
0.00		0.00	495162.00	Overheads	384898.00
0.00		0.00	351961.00	Equipment	20707.00
0.00		0.00	122340.00	Books	312896.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
5274605.00		**6736571.00**	**5569121.00**		**4841288.00**
294516.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	1895283.00
5569121.00		**6736571.00**	**5569121.00**		**6736571.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-128: Mechanism of iron acquisition and iron homeostasis in an opportunistic human pathogen Candida glabrata

P.I: Dr Rupinder Kaur

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	608942.00	Opening Balance	77108.00
807800.00	Grant In Aid	0.00	185407.00	Salaries - Manpower	1740.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	9626.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	80933.00	Equipment	79640.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
807800.00		**0.00**	**884908.00**		**158488.00**
77108.00	Excess of Expenditure Over Income	158488.00	0.00	Closing Balance	0.00
884908.00		**158488.00**	**884908.00**		**158488.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-130: Comparative genetic analysis of sex chromosomes and sex determining genes in silkmoths

P.I: Dr J Nagaraju

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
2865531.00	Opening Balance	0.00		Opening Balance	2550050.00
0.00	Grant In Aid	4024000.00	783258.00	Salaries - Manpower	546581.00
0.00		0.00	4450000.00	Consumables	0.00
0.00		0.00	50000.00	Contingencies	0.00
0.00		0.00	132323.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	926500.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
2865531.00		**4024000.00**	**5415581.00**		**4023131.00**
2550050.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	869.00
5415581.00		**4024000.00**	**5415581.00**		**4024000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-131: Structural and functional studies of Acyl CoA Binding proteins from plasmodium falciparum
P.I: Dr Akash Ranjan
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	398632.00		1245339.00		Opening Balance	0.00	
1902500.00		Grant In Aid	0.00		212529.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		46000.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
1902500.00			**398632.00**		**1503868.00**			**0.00**	
0.00		Excess of Expenditure Over Income	0.00		398632.00		Closing Balance	398632.00	
1902500.00			**398632.00**		**1902500.00**			**398632.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-132: Characterization of tumor suppressor function of ARIDIB, a component of the human SWI/SNF chromatin remodelling complex
P.I: Dr M D Bashyam, Dr Rohit Joshi
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		2166471.00		Opening Balance	640003.00	
3046200.00		Grant In Aid	0.00		429347.00		Salaries - Manpower	-21753.00	
0.00			0.00		1068571.00		Consumables	-603137.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		21814.00		Travel	-2914.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
3046200.00			**0.00**		**3686203.00**			**12199.00**	
640003.00		Excess of Expenditure Over Income	12199.00		0.00		Closing Balance	0.00	
3686203.00			**12199.00**		**3686203.00**			**12199.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-133: Investigating the role of Hox gene deformed in central nervous system patterning in Drosophila melanogaster
P.I: Dr Rohit Joshi
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
534614.00		Opening Balance	460117.00				Opening Balance	0.00	
867000.00		Grant In Aid	0.00		287200.00		Salaries - Manpower	206034.00	
0.00			0.00		567124.00		Consumables	946755.00	
0.00			0.00		8000.00		Contingencies	0.00	
0.00			0.00		24876.00		Travel	-25467.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		54297.00		Equipment	35785.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
1401614.00			**460117.00**		**941497.00**			**1163107.00**	
0.00		Excess of Expenditure Over Income	702990.00		460117.00		Closing Balance	0.00	
1401614.00			**1163107.00**		**1401614.00**			**1163107.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-134: Exploration of wild silk moth biodiversity in Manipur and their genetic characterization using molecular markers
P.I: Dr K P Arun Kumar
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		156437.00		Opening Balance	77061.00	
235000.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		119000.00		Consumables	0.00	
0.00			0.00		30000.00		Contingencies	0.00	
0.00			0.00		6624.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
235000.00			**0.00**		**312061.00**			**77061.00**	
77061.00		Excess of Expenditure Over Income	77061.00		0.00		Closing Balance	0.00	
312061.00			**77061.00**		**312061.00**			**77061.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-135: Sys TB: A Network Program for Resolving the Intracellular Dynamics of Host Phthogen Interaction in TB Infection
P.I: Dr. Sanjeev Kholsa
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	298323.00	Opening Balance	357268.00
2371000.00	Grant In Aid	2430700.00	343200.00	Salaries - Manpower	343200.00
0.00		0.00	2000000.00	Consumables	2000000.00
0.00		0.00	50000.00	Contingencies	50000.00
0.00		0.00	36745.00	Travel	16367.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
2371000.00		**2430700.00**	**2728268.00**		**2766835.00**
357268.00	Excess of Expenditure Over Income	336135.00	0.00	Closing Balance	0.00
2728268.00		**2766835.00**	**2728268.00**		**2766835.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-136: Raf Kinase - a key target for modem-day theraphy against tumors
P.I: Dr Sunil Kumar Manna
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
13618.00	Opening Balance	0.00		Opening Balance	292334.00
570000.00	Grant In Aid	0.00	187200.00	Salaries - Manpower	-43781.00
0.00		0.00	626858.00	Consumables	-20658.00
0.00		0.00	30000.00	Contingencies	0.00
0.00		0.00	31894.00	Travel	-31894.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
583618.00		**0.00**	**875952.00**		**196001.00**
292334.00	Excess of Expenditure Over Income	196001.00	0.00	Closing Balance	0.00
875952.00		**196001.00**	**875952.00**		**196001.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-137: Signaling pathways involved in down regulation of proinflammatory responses by PPE18 protein of Mycobacterium tuberculosis: Implication of PPE18 as therapeutics

P.I: Dr Sangita Mukhopadhyay

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
44141.00		Opening Balance	759474.00				Opening Balance	0.00	
2500000.00		Grant In Aid	-464025.00		224180.00		Salaries - Manpower	195478.00	
0.00			0.00		696860.00		Consumables	0.00	
0.00			0.00		34577.00		Contingencies	0.00	
0.00			0.00		44797.00		Travel	15203.00	
0.00			0.00		100000.00		Overheads	0.00	
0.00			0.00		684253.00		Equipment	84768.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
2544141.00			**295449.00**		**1784667.00**			**295449.00**	
0.00		Excess of Expenditure Over Income	0.00		759474.00		Closing Balance	0.00	
2544141.00			**295449.00**		**2544141.00**			**295449.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-138: Co-evaluation of Dnmt3l and Genomic imprinting

P.I: Dr Sanjeev Khosla

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		638079.00		Opening Balance	1353238.00	
0.00		Grant In Aid	0.00		186160.00		Salaries - Manpower	12580.00	
0.00			0.00		500000.00		Consumables	0.00	
0.00			0.00		25000.00		Contingencies	0.00	
0.00			0.00		3999.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	134482.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**1353238.00**			**1500300.00**	
1353238.00		Excess of Expenditure Over Income	1500300.00		0.00		Closing Balance	0.00	
1353238.00			**1500300.00**		**1353238.00**			**1500300.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-139: Evaluating the role of Sirtuins and epigenetic changes during cellular senescense in context of p53 status
P.I: Dr Gayatri Ramakrishna, Dr Sanjeev Khosla
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
20000.00	Opening Balance	20000.00		Opening Balance	0.00
520000.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	500000.00	Consumables	0.00
0.00		0.00	20000.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
540000.00		**20000.00**	**520000.00**		**0.00**
0.00	Excess of Expenditure Over Income	0.00	20000.00	Closing Balance	20000.00
540000.00		**20000.00**	**540000.00**		**20000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-140: Development of baculovirus resistant silkworms strains through synthetic miRNA based knockdown of essential viral genes
P.I: Dr K P Arun Kumar
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
146091.00	Opening Balance	0.00		Opening Balance	403336.00
835000.00	Grant In Aid	0.00	284427.00	Salaries - Manpower	205316.00
0.00		0.00	583701.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	16299.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	500000.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
981091.00		**0.00**	**1384427.00**		**608652.00**
403336.00	Excess of Expenditure Over Income	608652.00	0.00	Closing Balance	0.00
1384427.00		**608652.00**	**1384427.00**		**608652.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-141: Evaluating the functional role of PTEN interacting proteins in cell survival signaling and tumor suppression

P.I: Dr M Subba Reddy

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	223537.00	Opening Balance	125000.00
600000.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	431463.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	70000.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
600000.00		**0.00**	**725000.00**		**125000.00**
125000.00	Excess of Expenditure Over Income	125000.00	0.00	Closing Balance	0.00
725000.00		**125000.00**	**725000.00**		**125000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-142: Identification of H3K4 TRI Demethylase involved in erasing H3K4 trimethylation marks at E2F Responsive promoters

P.I: Dr Shweta Tyagi

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	401878.00	Opening Balance	280596.00
935920.00	Grant In Aid	196800.00	187200.00	Salaries - Manpower	0.00
0.00		0.00	600000.00	Consumables	-2.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	27438.00	Equipment	-1933.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
935920.00		**196800.00**	**1216516.00**		**278661.00**
280596.00	Excess of Expenditure Over Income	81861.00	0.00	Closing Balance	0.00
1216516.00		**278661.00**	**1216516.00**		**278661.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-143: Microarray based characterisation of squamous cell carcinoma of the tongue occuring in non smokers
P.I: Dr M D Bashyam
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance		0.00	751303.00		Opening Balance	534504.00	
1144199.00		Grant In Aid		0.00	231400.00		Salaries - Manpower	205400.00	
0.00				0.00	696000.00		Consumables	487500.00	
0.00				0.00	0.00		Contingencies	0.00	
0.00				0.00	0.00		Travel	0.00	
0.00				0.00	0.00		Overheads	154280.00	
0.00				0.00	0.00		Equipment	0.00	
0.00				0.00	0.00		Books	0.00	
0.00				0.00	0.00		AMC	0.00	
0.00				0.00	0.00		Others	0.00	
0.00				0.00	0.00		Transfer of Funds	0.00	
1144199.00				**0.00**	**1678703.00**			**1381684.00**	
534504.00		Excess of Expenditure Over Income	1381684.00		0.00		Closing Balance	0.00	
1678703.00			**1381684.00**		**1678703.00**			**1381684.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-144 : Tri-National Training Program for Psychiatric Genetics
P.I: Dr Ashwin B Dalal
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	424130.00				Opening Balance	0.00	
424130.00		Grant In Aid		0.00		0.00	Salaries - Manpower	0.00	
0.00				0.00		0.00	Consumables	302000.00	
0.00				0.00		0.00	Contingencies	0.00	
0.00				0.00		0.00	Travel	0.00	
0.00				0.00		0.00	Overheads	0.00	
0.00				0.00		0.00	Equipment	0.00	
0.00				0.00		0.00	Books	0.00	
0.00				0.00		0.00	AMC	0.00	
0.00				0.00		0.00	Others	0.00	
0.00				0.00		0.00	Transfer of Funds	0.00	
424130.00			**424130.00**			**0.00**		**302000.00**	
0.00		Excess of Expenditure Over Income		0.00	424130.00		Closing Balance	122130.00	
424130.00			**424130.00**		**424130.00**			**424130.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-145: H3K4 HMT family regulatescell cycle progression
P.I: Dr Shweta Tyagi
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	1064782.00	Opening Balance	1112243.00
1870600.00	Grant In Aid	1200000.00	171600.00	Salaries - Manpower	72713.00
0.00		0.00	1400000.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	24740.00	Travel	11822.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	321721.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1870600.00		**1200000.00**	**2982843.00**		**1196778.00**
1112243.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	3222.00
2982843.00		**1200000.00**	**2982843.00**		**1200000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-146: Role of MLL in ribosomal RNA transcription
P.I: Dr Shweta Tyagi
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
763439.00	Opening Balance	433858.00		Opening Balance	0.00
809000.00	Grant In Aid	0.00	224800.00	Salaries - Manpower	107187.00
0.00		0.00	582862.00	Consumables	267138.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	17138.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	313781.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1572439.00		**433858.00**	**1138581.00**		**374325.00**
0.00	Excess of Expenditure Over Income	0.00	433858.00	Closing Balance	59533.00
1572439.00		**433858.00**	**1572439.00**		**433858.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-147: The Effect of Parental Education, Ethics of Research Participation and Array Comparative Genomic Hybridization in Subjects with Mental Retardation (MR) and /or Autism

P.I: Dr Ashwin B Dalal

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
41311.00	Opening Balance	0.00		Opening Balance	677839.00
0.00	Grant In Aid	500000.00	187200.00	Salaries - Manpower	82026.00
0.00		0.00	400000.00	Consumables	0.00
0.00		0.00	50000.00	Contingencies	0.00
0.00		0.00	31950.00	Travel	13009.00
0.00		0.00	50000.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
41311.00		**500000.00**	**719150.00**		**772874.00**
677839.00	Excess of Expenditure Over Income	272874.00	0.00	Closing Balance	0.00
719150.00		**772874.00**	**719150.00**		**772874.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-149: Role of SUMOylation in the pathobiology of Candida Glabrata

P.I: Dr Rupinder Kaur

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
270865.00	Opening Balance	0.00		Opening Balance	1016335.00
0.00	Grant In Aid	1420800.00	187200.00	Salaries - Manpower	153920.00
0.00		0.00	900000.00	Consumables	300000.00
0.00		0.00	200000.00	Contingencies	0.00
0.00		0.00	0.00	Travel	10182.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	280.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
270865.00		**1420800.00**	**1287200.00**		**1480717.00**
1016335.00	Excess of Expenditure Over Income	59917.00	0.00	Closing Balance	0.00
1287200.00		**1480717.00**	**1287200.00**		**1480717.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-150: Genetic and genomic basis of the evolution of bombycid and sturniid silkmoths
P.I: Dr J Nagaraju
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		28096.00		Opening Balance	0.00	
153846.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		125750.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
153846.00			**0.00**		**153846.00**			**0.00**	
0.00		Excess of Expenditure Over Income	0.00		0.00		Closing Balance	0.00	
153846.00			**0.00**		**153846.00**			**0.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-151: Human Exome Sequencing to Identify Novel Genes for Medelian Disorders
P.I: Dr Ashwin B Dalal
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
594981.00		Opening Balance	0.00				Opening Balance	601366.00	
0.00		Grant In Aid	1756400.00		343200.00		Salaries - Manpower	343200.00	
0.00			0.00		800000.00		Consumables	351886.00	
0.00			0.00		25000.00		Contingencies	25000.00	
0.00			0.00		28147.00		Travel	59097.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
594981.00			**1756400.00**		**1196347.00**			**1380549.00**	
601366.00		Excess of Expenditure Over Income	0.00		0.00		Closing Balance	375851.00	
1196347.00			**1756400.00**		**1196347.00**			**1756400.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-152 : Global transcriptomics of sex specific spilicing
P.I: Dr K P Arun Kumar
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
1114145.00	Opening Balance	29100.00		Opening Balance	0.00
2562571.00	Grant In Aid	1931400.00	343200.00	Salaries - Manpower	343200.00
0.00		0.00	3026000.00	Consumables	1648114.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	278416.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
3676716.00		**1960500.00**	**3647616.00**		**1991314.00**
0.00	Excess of Expenditure Over Income	30814.00	29100.00	Closing Balance	0.00
3676716.00		**1991314.00**	**3676716.00**		**1991314.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-153: An attractive and promising stragegy for early cancer diagnosis through the assembly of the human cancer volatome"
P.I: Dr H A Nagarajaram
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
3613562.00	Opening Balance	641552.00		Opening Balance	0.00
621000.00	Grant In Aid	0.00	374400.00	Salaries - Manpower	358800.00
0.00		0.00	70000.00	Consumables	70000.00
0.00		0.00	80000.00	Contingencies	80000.00
0.00		0.00	68610.00	Travel	197057.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	3000000.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
4234562.00		**641552.00**	**3593010.00**		**705857.00**
0.00	Excess of Expenditure Over Income	64305.00	641552.00	Closing Balance	0.00
4234562.00		**705857.00**	**4234562.00**		**705857.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-154 : Rational design, synthetic strategies for developing organometallic anticancer compounds based on organotin and organoiron

P.I: Dr Sunil Kumar Manna

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
87432.00	Opening Balance	30832.00		Opening Balance	0.00
943000.00	Grant In Aid	930000.00	249600.00	Salaries - Manpower	297322.00
0.00		0.00	700000.00	Consumables	600000.00
0.00		0.00	50000.00	Contingencies	50000.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1030432.00		**960832.00**	**999600.00**		**947322.00**
0.00	Excess of Expenditure Over Income	0.00	30832.00	Closing Balance	13510.00
1030432.00		**960832.00**	**1030432.00**		**960832.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-155: Studies on thecellular roles of calcium signalling proteins in Neurospora crassa

P.I: Dr D P Kasbekar

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
335194.00	Opening Balance	335194.00		Opening Balance	0.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
335194.00		**335194.00**	**0.00**		**0.00**
0.00	Excess of Expenditure Over Income	0.00	335194.00	Closing Balance	335194.00
335194.00		**335194.00**	**335194.00**		**335194.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-156 : Targeting microbial quorum sensing to demonstrate potential application of cell-cell signaling molecules from Xanthomonas group of plant pathogen in diesease control

PI : Dr Subhadeep Chatterjee

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
926632.00	Opening Balance	0.00		Opening Balance	175165.00
1076500.00	Grant In Aid	1706000.00	363601.00	Salaries - Manpower	345520.00
0.00		0.00	1600000.00	Consumables	1000000.00
0.00		0.00	50000.00	Contingencies	-50000.00
0.00		0.00	32201.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	132495.00	Equipment	-4634.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
2003132.00		**1706000.00**	**2178297.00**		**1466051.00**
175165.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	239949.00
2178297.00		**1706000.00**	**2178297.00**		**1706000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-157 : Identification of novel antifungal drug and delineation of drug resistance mechanisms in an opportunistic human fungal pathogen Candida glabrata

PI : Dr Rupinder Kaur

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
944665.00	Opening Balance	204372.00		Opening Balance	0.00
1317000.00	Grant In Aid	0.00	195880.00	Salaries - Manpower	165813.00
0.00		0.00	1200000.00	Consumables	1402360.00
0.00		0.00	50000.00	Contingencies	-23540.00
0.00		0.00	0.00	Travel	21538.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	611413.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
2261665.00		**204372.00**	**2057293.00**		**1566171.00**
0.00	Excess of Expenditure Over Income	1361799.00	204372.00	Closing Balance	0.00
2261665.00		**1566171.00**	**2261665.00**		**1566171.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-158 : Modulation of host immune responses by a PPE Protein of Mycobacterium tuberculosis: Understanding its role in host - pathogen cross-talk

PI : Dr Sangita Mukhopadhyay

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
621787.00	Opening Balance	0.00		Opening Balance	1379658.00
0.00	Grant In Aid	0.00	342277.00	Salaries - Manpower	100100.00
0.00		0.00	1300000.00	Consumables	1011202.00
0.00		0.00	50000.00	Contingencies	23868.00
0.00		0.00	9227.00	Travel	17338.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	299941.00	Equipment	43180.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
621787.00		**0.00**	**2001445.00**		**2575346.00**
1379658.00	Excess of Expenditure Over Income	2575346.00	0.00	Closing Balance	0.00
2001445.00		**2575346.00**	**2001445.00**		**2575346.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-159 : Gene Targeting of microbial isolates to demonstrate potential plant growth promoting (PGP) traits by third generation sequencing

PI : Dr Subhadeep Chatterjee

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
300000.00	Opening Balance	0.00		Opening Balance	0.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	300000.00	Consumables	300000.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
300000.00		**0.00**	**300000.00**		**300000.00**
0.00	Excess of Expenditure Over Income	300000.00	0.00	Closing Balance	0.00
300000.00		**300000.00**	**300000.00**		**300000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-160 : Understanding the role of novel adhesins of Xanthomonas oryzae PV orzae in Virulence and colonization in Rice

PI : Dr Subhadeep Chatterjee

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
363884.00		Opening Balance	208333.00				Opening Balance	0.00	
531649.00		Grant In Aid	687200.00		187200.00		Salaries - Manpower	187200.00	
0.00			0.00		500000.00		Consumables	750000.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
895533.00			**895533.00**		**687200.00**			**937200.00**	
0.00		Excess of Expenditure Over Income	41667.00		208333.00		Closing Balance	0.00	
895533.00			**937200.00**		**895533.00**			**937200.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-161 : Analysis of co-regulation between DNA replication activity and amino acid homeostatis by transcription factor IciA/ArgP in Eschericia coli

PI : Dr J Gowrishankar

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
350000.00		Opening Balance	84656.00				Opening Balance	0.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		10000.00		Contingencies	10000.00	
0.00			0.00		255344.00		Travel	71025.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	3631.00	
350000.00			**84656.00**		**265344.00**			**84656.00**	
0.00		Excess of Expenditure Over Income	0.00		84656.00		Closing Balance	0.00	
350000.00			**84656.00**		**350000.00**			**84656.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-162 : Characterization and design of inhibitors of Mycobacterium tuberculosis transcription
PI : Dr Ranjan Sen
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
235671.00	Opening Balance	0.00		Opening Balance	316464.00
0.00	Grant In Aid	0.00	130928.00	Salaries - Manpower	247673.00
0.00		0.00	400000.00	Consumables	422026.00
0.00		0.00	0.00	Contingencies	25000.00
0.00		0.00	21207.00	Travel	10604.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
235671.00		**0.00**	**552135.00**		**1021767.00**
316464.00	Excess of Expenditure Over Income	1021767.00	0.00	Closing Balance	0.00
552135.00		**1021767.00**	**552135.00**		**1021767.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-163 : Unravelling new functions for the H-NS family of proteins in Gram-negative bacterial pathogens
PI : Dr J Gowrishankar
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
2006048.00	Opening Balance	1052471.00		Opening Balance	0.00
0.00	Grant In Aid	1062777.00	53577.00	Salaries - Manpower	194480.00
0.00		0.00	800000.00	Consumables	800000.00
0.00		0.00	40000.00	Contingencies	30000.00
0.00		0.00	0.00	Travel	342109.00
0.00		0.00	60000.00	Overheads	70000.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
2006048.00		**2115248.00**	**953577.00**		**1436589.00**
0.00	Excess of Expenditure Over Income	0.00	1052471.00	Closing Balance	678659.00
2006048.00		**2115248.00**	**2006048.00**		**2115248.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-164 : A Yeast based screen for discovery of novel sirtuin inhibitors as anticancer agents
PI : Dr Devyani Halder
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	26671.00	Opening Balance	24671.00
188000.00	Grant In Aid	0.00	156000.00	Salaries - Manpower	4529.00
0.00		0.00	30000.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
188000.00		**0.00**	**212671.00**		**29200.00**
24671.00	Excess of Expenditure Over Income	29200.00	0.00	Closing Balance	0.00
212671.00		**29200.00**	**212671.00**		**29200.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-165 : Identification and functional characterization of immune response genes in silkmoths
PI : Dr V V Satyavathi
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
1569682.00	Opening Balance	330135.00		Opening Balance	0.00
0.00	Grant In Aid	2858334.00	122400.00	Salaries - Manpower	344600.00
0.00		0.00	1000000.00	Consumables	1000000.00
0.00		0.00	50000.00	Contingencies	50000.00
0.00		0.00	17147.00	Travel	15957.00
0.00		0.00	50000.00	Overheads	50000.00
0.00		0.00	0.00	Equipment	160082.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1569682.00		**3188469.00**	**1239547.00**		**1620639.00**
0.00	Excess of Expenditure Over Income	0.00	330135.00	Closing Balance	1567830.00
1569682.00		**3188469.00**	**1569682.00**		**3188469.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-166 : Sequencing analysis of transcriptome variants in early-onset sporadic rectal cancer
PI : Dr M D Bashyam
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	2165638.00		Opening Balance	0.00
4383200.00	Grant In Aid	574700.00	196003.00	Salaries - Manpower	192400.00
0.00		0.00	2000000.00	Consumables	500000.00
0.00		0.00	20000.00	Contingencies	0.00
0.00		0.00	1559.00	Travel	12242.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	2000000.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
4383200.00		**2740338.00**	**2217562.00**		**2704642.00**
0.00	Excess of Expenditure Over Income	0.00	2165638.00	Closing Balance	35696.00
4383200.00		**2740338.00**	**4383200.00**		**2740338.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-167 : To elucidate the role of MLL complex in epigenetic specification of centromere
PI : Dr Shweta Tyagi
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	633780.00		Opening Balance	0.00
1700000.00	Grant In Aid	1500000.00	64916.00	Salaries - Manpower	137381.00
0.00		0.00	862000.00	Consumables	885797.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	19362.00
0.00		0.00	100000.00	Overheads	0.00
0.00		0.00	39304.00	Equipment	521453.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1700000.00		**2133780.00**	**1066220.00**		**1563993.00**
0.00	Excess of Expenditure Over Income	0.00	633780.00	Closing Balance	569787.00
1700000.00		**2133780.00**	**1700000.00**		**2133780.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-168 : A Search for nucleus -limited genes in Neurospora
PI : Dr D P Kasbekar
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	788623.00		Opening Balance	0.00
1400000.00	Grant In Aid	1000000.00	29187.00	Salaries - Manpower	187200.00
0.00		0.00	450000.00	Consumables	1110910.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	740.00	Travel	25963.00
0.00		0.00	100000.00	Overheads	100000.00
0.00		0.00	31450.00	Equipment	364550.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1400000.00		**1788623.00**	**611377.00**		**1788623.00**
0.00	Excess of Expenditure Over Income	0.00	788623.00	Closing Balance	0.00
1400000.00		**1788623.00**	**1400000.00**		**1788623.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-169 : Implementation of 3 year DNB Program in Medical Genetics by Department of Biotechnology in colloboration with National Board of Examination ag SGHR, NIBMG&CDFD
PI : Dr J Gowrishankar
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	1758108.00		Opening Balance	0.00
1890000.00	Grant In Aid	0.00	0.00	Salaries - Manpower	1300000.00
0.00		0.00	81892.00	Consumables	121193.00
0.00		0.00	50000.00	Contingencies	20000.00
0.00		0.00	0.00	Travel	300000.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1890000.00		**1758108.00**	**131892.00**		**1741193.00**
0.00	Excess of Expenditure Over Income	0.00	1758108.00	Closing Balance	16915.00
1890000.00		**1758108.00**	**1890000.00**		**1758108.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-170 : Women Scientist Scheme "Identification and character of deregulated micro RNAs in defined sub-set of early onset sporadic rectal cancer patients using transcriptome sequencing"

PI : Dr Mithu Ray Chaudhuri

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	277449.00				Opening Balance	0.00	
820000.00		Grant In Aid	0.00		142551.00		Salaries - Manpower	587316.00	
0.00			0.00		300000.00		Consumables	300000.00	
0.00			0.00		50000.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		50000.00		Overheads	50000.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
820000.00			**277449.00**		**542551.00**			**937316.00**	
0.00		Excess of Expenditure Over Income	659867.00		277449.00		Closing Balance	0.00	
820000.00			**937316.00**		**820000.00**			**937316.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-171 : Role of vesicle-mediated transport and chromatin remodelling in the virulence of Candida glabrata

PI : Dr Rupinder Kaur

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	1754447.00				Opening Balance	0.00	
2415730.00		Grant In Aid	0.00		0.00		Salaries - Manpower	236080.00	
0.00			0.00		600000.00		Consumables	1011064.00	
0.00			0.00		25000.00		Contingencies	320.00	
0.00			0.00		36283.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	295560.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
2415730.00			**1754447.00**		**661283.00**			**1543024.00**	
0.00		Excess of Expenditure Over Income	0.00		1754447.00		Closing Balance	211423.00	
2415730.00			**1754447.00**		**2415730.00**			**1754447.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-172 : Molecular Characterization of early onset sporadic rectal cancer

PI : Dr M D Bashyam

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	1461747.00				Opening Balance	0.00	
2100000.00		Grant In Aid	1200000.00		38253.00		Salaries - Manpower	465412.00	
0.00			0.00		600000.00		Consumables	596335.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	100000.00	
0.00			0.00		0.00		Equipment	1388150.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
2100000.00			**2661747.00**		**638253.00**			**2549897.00**	
0.00		Excess of Expenditure Over Income	0.00		1461747.00		Closing Balance	111850.00	
2100000.00			**2661747.00**		**2100000.00**			**2661747.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-173 : Development and application of a next generation sequencing approach for molecular genetic analysis of lysosomal storage disorders

PI : Dr Ashwin B Dalal

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	584882.00				Opening Balance	0.00	
699782.00		Grant In Aid	699782.00		29900.00		Salaries - Manpower	326006.00	
0.00			0.00		85000.00		Consumables	470705.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
699782.00			**1284664.00**		**114900.00**			**796711.00**	
0.00		Excess of Expenditure Over Income	0.00		584882.00		Closing Balance	487953.00	
699782.00			**1284664.00**		**699782.00**			**1284664.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-174 : Is non-canonical Wnt signalling a major player in early-onset sporadic rectal cancer

PI : Dr M D Bashyam

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	500000.00				Opening Balance	0.00	
500000.00		Grant In Aid	500000.00		0.00		Salaries - Manpower	210432.00	
0.00			0.00		0.00		Consumables	260905.00	
0.00			0.00		0.00		Contingencies	8121.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
500000.00			**1000000.00**		**0.00**			**479458.00**	
0.00		Excess of Expenditure Over Income	0.00		500000.00		Closing Balance	520542.00	
500000.00			**1000000.00**		**500000.00**			**1000000.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-175 : Multi Centri Collaborative study of the Clinical, Biochemical and Molecular Characterization of Lysosomal storage disorders in India - The initiative for research in Lysosomal Storage Disorders"

PI : Dr Ashwin B Dalal

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00				Opening Balance	509714.00	
0.00		Grant In Aid	0.00		9714.00		Salaries - Manpower	396076.00	
0.00			0.00		500000.00		Consumables	500000.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	26882.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**509714.00**			**1432672.00**	
509714.00		Excess of Expenditure Over Income	1432672.00		0.00		Closing Balance	0.00	
509714.00			**1432672.00**		**509714.00**			**1432672.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-176 : International Atomic Energy Agency

PI : Dr K P Arun Kumar
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	200103.00				Opening Balance	0.00	
200103.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
200103.00			**200103.00**		**0.00**			**0.00**	
0.00		Excess of Expenditure Over Income	0.00		200103.00		Closing Balance	200103.00	
200103.00			**200103.00**		**200103.00**			**200103.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-177 : Morphological and molecular taxonomy of the Phlebotomus argendtipes species complex in relation to transmission of Kala-azar in India"
PI : Dr J Gowrishankar
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00				Opening Balance	0.00	
0.00		Grant In Aid	225000.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	400000.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	22394.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**225000.00**		**0.00**			**422394.00**	
0.00		Excess of Expenditure Over Income	197394.00		0.00		Closing Balance	0.00	
0.00			**422394.00**		**0.00**			**422394.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-178 : Understanding differential signaling via toll like receptor-2: A proteomics approach

PI : Dr Rameshwaram Nagender Rao

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Previous Year Rs	Receipts	Current Year Amount	Current Year Rs.	Previous Year. Amount	Previous Year. Rs	Payments	Current Year Amount	Current Year Rs
0.00		Opening Balance	0.00				Opening Balance	0.00	
0.00		Grant In Aid	1000000.00		0.00		Salaries - Manpower	507419.00	
0.00			0.00		0.00		Consumables	376554.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	16027.00	
0.00			0.00		0.00		Overheads	100000.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**1000000.00**		**0.00**			**1000000.00**	
0.00		Excess of Expenditure Over Income	0.00		0.00		Closing Balance	0.00	
0.00			**1000000.00**		**0.00**			**1000000.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-179 : Quality Assurance Programme for Molecular and Prenatal Diagnosis of Hemoglobin Opathies

PI : Dr Ashwin B Dalal

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Previous Year Rs	Receipts	Current Year Amount	Current Year Rs.	Previous Year. Amount	Previous Year. Rs	Payments	Current Year Amount	Current Year Rs
0.00		Opening Balance	0.00				Opening Balance	0.00	
0.00		Grant In Aid	50000.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	100000.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**50000.00**		**0.00**			**100000.00**	
0.00		Excess of Expenditure Over Income	50000.00		0.00		Closing Balance	0.00	
0.00			**100000.00**		**0.00**			**100000.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-180 : Collaborative studies on genomic diversity among bombycoid silkmoths in Asia

PI : Dr K P Arun Kumar

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
	0.00	Opening Balance		0.00			Opening Balance		0.00
	0.00	Grant In Aid		200000.00		0.00	Salaries - Manpower		0.00
	0.00			0.00		0.00	Consumables		0.00
	0.00			0.00		0.00	Contingencies		0.00
	0.00			0.00		0.00	Travel		82114.00
	0.00			0.00		0.00	Overheads		0.00
	0.00			0.00		0.00	Equipment		0.00
	0.00			0.00		0.00	Books		0.00
	0.00			0.00		0.00	AMC		0.00
	0.00			0.00		0.00	Others		0.00
	0.00			0.00		0.00	Transfer of Funds		0.00
	0.00			**200000.00**		**0.00**			**82114.00**
	0.00	Excess of Expenditure Over Income		0.00		0.00	Closing Balance		117886.00
	0.00			**200000.00**		**0.00**			**200000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-181 : To conduct multilocational field trails on transgenic BmNPV resistant silkworm strains to establish their efficacy and generate data for their regulatory approval

PI : Dr V V Satyavathi

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
	0.00	Opening Balance		0.00			Opening Balance		0.00
	0.00	Grant In Aid		1744000.00		0.00	Salaries - Manpower		0.00
	0.00			0.00		0.00	Consumables		0.00
	0.00			0.00		0.00	Contingencies		0.00
	0.00			0.00		0.00	Travel		0.00
	0.00			0.00		0.00	Overheads		0.00
	0.00			0.00		0.00	Equipment		0.00
	0.00			0.00		0.00	Books		0.00
	0.00			0.00		0.00	AMC		0.00
	0.00			0.00		0.00	Others		0.00
	0.00			0.00		0.00	Transfer of Funds		0.00
	0.00			**1744000.00**		**0.00**			**0.00**
	0.00	Excess of Expenditure Over Income		0.00		0.00	Closing Balance		1744000.00
	0.00			**1744000.00**		**0.00**			**1744000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-182 : Ramalingaswami Fellowship

PI : Dr Mohan C Joshi
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance		0.00			Opening Balance		0.00
0.00		Grant In Aid		0.00		0.00	Salaries - Manpower		277500.00
0.00				0.00		0.00	Consumables		0.00
0.00				0.00		0.00	Contingencies		0.00
0.00				0.00		0.00	Travel		0.00
0.00				0.00		0.00	Overheads		0.00
0.00				0.00		0.00	Equipment		0.00
0.00				0.00		0.00	Books		0.00
0.00				0.00		0.00	AMC		0.00
0.00				0.00		0.00	Others		0.00
0.00				0.00		0.00	Transfer of Funds		0.00
0.00				**0.00**		**0.00**			**277500.00**
0.00		Excess of Expenditure Over Income		277500.00		0.00	Closing Balance		0.00
0.00				**277500.00**		**0.00**			**277500.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-184 : Computational Approaches to Understanding Peptide- Protein Interactions involved in the Regulatory Events in the Cell"

PI : Dr Raghavender Surya Upadhyayula
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance		0.00			Opening Balance		0.00
0.00		Grant In Aid		1060000.00		0.00	Salaries - Manpower		92258.00
0.00				0.00		0.00	Consumables		0.00
0.00				0.00		0.00	Contingencies		0.00
0.00				0.00		0.00	Travel		0.00
0.00				0.00		0.00	Overheads		10000.00
0.00				0.00		0.00	Equipment		0.00
0.00				0.00		0.00	Books		0.00
0.00				0.00		0.00	AMC		0.00
0.00				0.00		0.00	Others		0.00
0.00				0.00		0.00	Transfer of Funds		0.00
0.00				**1060000.00**		**0.00**			**102258.00**
0.00		Excess of Expenditure Over Income		0.00		0.00	Closing Balance		957742.00
0.00				**1060000.00**		**0.00**			**1060000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-185 : Investigating potential of mycobacterium tuberculosis protein PPE18 encapsulated nanoparticle as therapy for microbial sepsis

PI : Dr Sangita Mukhopadhyay

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00				Opening Balance		0.00
0.00		Grant In Aid	1648000.00		0.00		Salaries - Manpower		0.00
0.00			0.00		0.00		Consumables		15793.00
0.00			0.00		0.00		Contingencies		0.00
0.00			0.00		0.00		Travel		0.00
0.00			0.00		0.00		Overheads		0.00
0.00			0.00		0.00		Equipment		0.00
0.00			0.00		0.00		Books		0.00
0.00			0.00		0.00		AMC		0.00
0.00			0.00		0.00		Others		0.00
0.00			0.00		0.00		Transfer of Funds		0.00
0.00			**1648000.00**		**0.00**				**15793.00**
0.00		Excess of Expenditure Over Income	0.00		0.00		Closing Balance		1632207.00
0.00			**1648000.00**		**0.00**				**1648000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-186 : In vivo corss-talks between Rho-dependent transcription termination and other biological processes

PI : Dr Ranjan Sen

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00				Opening Balance		0.00
0.00		Grant In Aid	2410000.00		0.00		Salaries - Manpower		0.00
0.00			0.00		0.00		Consumables		0.00
0.00			0.00		0.00		Contingencies		0.00
0.00			0.00		0.00		Travel		0.00
0.00			0.00		0.00		Overheads		0.00
0.00			0.00		0.00		Equipment		0.00
0.00			0.00		0.00		Books		0.00
0.00			0.00		0.00		AMC		0.00
0.00			0.00		0.00		Others		0.00
0.00			0.00		0.00		Transfer of Funds		0.00
0.00			**2410000.00**		**0.00**				**0.00**
0.00		Excess of Expenditure Over Income	0.00		0.00		Closing Balance		2410000.00
0.00			**2410000.00**		**0.00**				**2410000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-187 : Understanding the mechanism of induction of innate immunity in plants by the Xanthomonas Diffusible signal factor (DSF)

PI : Dr Subhadeep Chatterjee

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00				Opening Balance		0.00
0.00		Grant In Aid	1368000.00		0.00		Salaries - Manpower		0.00
0.00			0.00		0.00		Consumables		0.00
0.00			0.00		0.00		Contingencies		0.00
0.00			0.00		0.00		Travel		0.00
0.00			0.00		0.00		Overheads		0.00
0.00			0.00		0.00		Equipment		0.00
0.00			0.00		0.00		Books		0.00
0.00			0.00		0.00		AMC		0.00
0.00			0.00		0.00		Others		0.00
0.00			0.00		0.00		Transfer of Funds		0.00
0.00			**1368000.00**		**0.00**				**0.00**
0.00		Excess of Expenditure Over Income	0.00		0.00		Closing Balance		1368000.00
0.00			**1368000.00**		**0.00**				**1368000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-188 : Identification of Novel Genes for Intellectual Disability

PI : Dr Aneek Das Bhowmik

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00				Opening Balance		0.00
0.00		Grant In Aid	1450000.00		0.00		Salaries - Manpower		0.00
0.00			0.00		0.00		Consumables		0.00
0.00			0.00		0.00		Contingencies		0.00
0.00			0.00		0.00		Travel		0.00
0.00			0.00		0.00		Overheads		0.00
0.00			0.00		0.00		Equipment		0.00
0.00			0.00		0.00		Books		0.00
0.00			0.00		0.00		AMC		0.00
0.00			0.00		0.00		Others		0.00
0.00			0.00		0.00		Transfer of Funds		0.00
0.00			**1450000.00**		**0.00**				**0.00**
0.00		Excess of Expenditure Over Income	0.00		0.00		Closing Balance		1450000.00
0.00			**1450000.00**		**0.00**				**1450000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-189 : Characterization of glycosylphosphatidylinositol-linked aspartyl proteases in Candida glabrata: role in pathosgenicity

PI : Dr Rupinder Kaur

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance		0.00			Opening Balance		0.00
0.00		Grant In Aid		16858467.00		0.00	Salaries - Manpower		0.00
0.00				0.00		0.00	Consumables		0.00
0.00				0.00		0.00	Contingencies		0.00
0.00				0.00		0.00	Travel		0.00
0.00				0.00		0.00	Overheads		0.00
0.00				0.00		0.00	Equipment		0.00
0.00				0.00		0.00	Books		0.00
0.00				0.00		0.00	AMC		0.00
0.00				0.00		0.00	Others		0.00
0.00				0.00		0.00	Transfer of Funds		0.00
0.00			**16858467.00**		**0.00**				**0.00**
0.00		Excess of Expenditure Over Income		0.00		0.00	Closing Balance		16858467.00
0.00			**16858467.00**		**0.00**				**16858467.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-190 : Exploring mycobacteriophages to source novel factors / regulators of bacterial transcription machinery

PI : Dr Shweta Singh

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance		0.00			Opening Balance		0.00
0.00		Grant In Aid		1100000.00		0.00	Salaries - Manpower		0.00
0.00				0.00		0.00	Consumables		0.00
0.00				0.00		0.00	Contingencies		0.00
0.00				0.00		0.00	Travel		0.00
0.00				0.00		0.00	Overheads		0.00
0.00				0.00		0.00	Equipment		0.00
0.00				0.00		0.00	Books		0.00
0.00				0.00		0.00	AMC		0.00
0.00				0.00		0.00	Others		0.00
0.00				0.00		0.00	Transfer of Funds		0.00
0.00			**1100000.00**		**0.00**				**0.00**
0.00		Excess of Expenditure Over Income		0.00		0.00	Closing Balance		1100000.00
0.00			**1100000.00**		**0.00**				**1100000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
COE1/CORE : COE for Genetics and Genomics of silkmoths

PI : Dr. J. Nagaraju
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		12372212.00		Opening Balance	11970751.00	
9102000.00		Grant In Aid	8335000.00		7357519.00		Salaries - Manpower	7219530.00	
0.00			0.00		1200000.00		Consumables	1200000.00	
0.00			0.00		0.00		Contingencies	103548.00	
0.00			0.00		143020.00		Travel	113099.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
9102000.00			**8335000.00**		**21072751.00**			**20606928.00**	
11970751.00		Excess of Expenditure Over Income	12271928.00		0.00		Closing Balance	0.00	
21072751.00			**20606928.00**		**21072751.00**			**20606928.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
COE1/P-I : Comparative and function genomics of silkmoths.

PI : Dr. J. Nagaraju
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		449637.00		Opening Balance	355503.00	
732000.00		Grant In Aid	638000.00		137866.00		Salaries - Manpower	193390.00	
0.00			0.00		500000.00		Consumables	500000.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
732000.00			**638000.00**		**1087503.00**			**1048893.00**	
355503.00		Excess of Expenditure Over Income	410893.00		0.00		Closing Balance	0.00	
1087503.00			**1048893.00**		**1087503.00**			**1048893.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE1/P-II : Development of RNA interference (RNAi) based nuclear polyhedrosis virus (NPV) resistant transgenic silkmoths.

PI : Dr. J. Nagaraju

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		387740.00		Opening Balance	419966.00	
459000.00		Grant In Aid	491000.00		191226.00		Salaries - Manpower	364953.00	
0.00			0.00		300000.00		Consumables	300000.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
459000.00			**491000.00**		**878966.00**			**1084919.00**	
419966.00		Excess of Expenditure Over Income	593919.00		0.00		Closing Balance	0.00	
878966.00			**1084919.00**		**878966.00**			**1084919.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE1/P-III : Identification and Characterization of micro RNAs and their targets in silkmoth genome.

PI : Dr. J. Nagaraju

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		505830.00		Opening Balance	475030.00	
1090000.00		Grant In Aid	1086000.00		709200.00		Salaries - Manpower	709200.00	
0.00			0.00		350000.00		Consumables	350000.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
1090000.00			**1086000.00**		**1565030.00**			**1534230.00**	
475030.00		Excess of Expenditure Over Income	448230.00		0.00		Closing Balance	0.00	
1565030.00			**1534230.00**		**1565030.00**			**1534230.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
COE-I/P-IV : Identification and characterization of immune response genes of silkmoths.

PI : Dr. J. Nagaraju
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		153724.00		Opening Balance	21563.00	
463000.00		Grant In Aid	331000.00		130839.00		Salaries - Manpower	140400.00	
0.00			0.00		200000.00		Consumables	200000.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
463000.00			**331000.00**		**484563.00**			**361963.00**	
21563.00		Excess of Expenditure Over Income	30963.00		0.00		Closing Balance	0.00	
484563.00			**361963.00**		**484563.00**			**361936.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
COE2/CORE : DBT Centre of Excellence for Microbial Biology

PI : Dr J Gowrishankar, Dr K Anupama, Dr Abhijit A Sardesai, Dr R
Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		19234494.00		Opening Balance	23840815.00	
0.00		Grant In Aid	0.00		4606321.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**23840815.00**			**23840815.00**	
23840815.00		Excess of Expenditure Over Income	23840815.00		0.00		Closing Balance	0.00	
23840815.00			**23840815.00**		**23840815.00**			**23840815.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE2/P-1 : Addressing functiional properties of E. coli through genome-wide protein-protein linkage analysis

PI : Dr. J Gowrishankar

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		684083.00		Opening Balance	684083.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**684083.00**			**684083.00**	
684083.00		Excess of Expenditure Over Income	684083.00		0.00		Closing Balance	0.00	
684083.00			**684083.00**		**684083.00**			**684083.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE2/P-2 : Mechanism of transcription termination and antitermination in Escherichia coli

PI : Dr. Ranjan Sen

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		1097981.00		Opening Balance	1441181.00	
0.00		Grant In Aid	0.00		343200.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**1441181.00**			**1441181.00**	
1441181.00		Excess of Expenditure Over Income	1441181.00		0.00		Closing Balance	0.00	
1441181.00			**1441181.00**		**1441181.00**			**1441181.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE2/P-A : Occurrence of R-loops (RNA-DNA hybrids) from nascent untranslated transcripts i E. Coli

PI : Dr. J Gowrishankar, Dr.K. Anupama

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		1085152.00		Opening Balance	1354252.00	
0.00		Grant In Aid	0.00		269100.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**1354252.00**			**1354252.00**	
1354252.00		Excess of Expenditure Over Income	1354252.00		0.00		Closing Balance	0.00	
1354252.00			**1354252.00**		**1354252.00**			**1354252.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE2/P-B : Molecular genetic approaches to dissect the physiology of osmoadptation in Escherichia coli

PI : Dr. J. Gowrishankar, Dr. Abhijit A Sardesasi

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		1006509.00		Opening Balance	1275609.00	
0.00		Grant In Aid	0.00		269100.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**1275609.00**			**1275609.00**	
1275609.00		Excess of Expenditure Over Income	1275609.00		0.00		Closing Balance	0.00	
1275609.00			**1275609.00**		**1275609.00**			**1275609.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE2/P-C : Functional role and mechanisms of the ArgO exporter and the transcriptional regulator ArgP in E. Coli

PI : Dr. J Gowrishankar, Dr. Ranjan Sen

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		473354.00		Opening Balance	473354.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			**0.00**		**473354.00**			**473354.00**	
473354.00		Excess of Expenditure Over Income	473354.00		0.00		Closing Balance	0.00	
473354.00			**473354.00**		**473354.00**			**473354.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE2-II/P-1 : In vivo studies on molecular mechanism of Rho-dependent transcription termination

PI : Dr Ranjan Sen

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	2071265.00				Opening Balance	0.00	
2186000.00		Grant In Aid	650000.00		114735.00		Salaries - Manpower	1056820.00	
0.00			0.00		0.00		Consumables	1000000.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	159664.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
2186000.00			**2721265.00**		**114735.00**			**2216484.00**	
0.00		Excess of Expenditure Over Income	0.00		2071265.00		Closing Balance	504781.00	
2186000.00			**2721265.00**		**2186000.00**			**2721265.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE2-II/P-A : Role of R-loops (RNA-DNA hybrids) in generatin of transcription -replication conflicts in E.Coli

PI : Dr J Gowrishankar

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	803300.00				Opening Balance	0.00	
1093000.00		Grant In Aid	0.00		89700.00		Salaries - Manpower	629368.00	
0.00			0.00		200000.00		Consumables	200000.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
1093000.00			**803300.00**		**289700.00**			**829368.00**	
0.00		Excess of Expenditure Over Income	26068.00		803300.00		Closing Balance	0.00	
1093000.00			**829368.00**		**1093000.00**			**829368.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE2-II/P-B : Role of the ArgP transcriptional regulator and metabolism of basic amino acids Arg and Lys in E.coli

PI : Dr J Gowrishankar

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	300000.00				Opening Balance	0.00	
500000.00		Grant In Aid	0.00		0.00		Salaries - Manpower	610077.00	
0.00			0.00		200000.00		Consumables	200000.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
500000.00			**300000.00**		**200000.00**			**810077.00**	
0.00		Excess of Expenditure Over Income	510077.00		300000.00		Closing Balance	0.00	
500000.00			**810077.00**		**500000.00**			**810077.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE2-II/P-C : Investigating global RNA turnover mechanisms and their interplay with Rho-dependent transcription termination in E. coli

PI : Dr K Anupaman

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	803300.00				Opening Balance	0.00	
1093000.00		Grant In Aid	0.00		89700.00		Salaries - Manpower	25665.00	
0.00			0.00		200000.00		Consumables	200000.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
1093000.00			**803300.00**		**289700.00**			**225665.00**	
0.00		Excess of Expenditure Over Income	0.00		803300.00		Closing Balance	577635.00	
1093000.00			**803300.00**		**1093000.00**			**803300.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE2-II/P-D : Molecular, genetic and biochemical studies on physiology of K+ION homeostatis and the regulatory mechanisms mediating avoidance of its imbalance in Escherichia coli

PI : Dr Abhijit A Sardesai

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	500000.00				Opening Balance	0.00	
500000.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	200000.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
500000.00			**500000.00**		**0.00**			**200000.00**	
0.00		Excess of Expenditure Over Income	0.00		500000.00		Closing Balance	300000.00	
500000.00			**500000.00**		**500000.00**			**500000.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE2-II/P-E : Understanding (p) ppGpp-mediated functions in E.Coliby deciphering the physiology of strain lacking (p)ppGpp OR altered in its metabolism

PI : Dr J Gowrishankar

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	1076226.00				Opening Balance	0.00	
1093000.00		Grant In Aid	0.00		16774.00		Salaries - Manpower	301291.00	
0.00			0.00		0.00		Consumables	60996.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
1093000.00			**1076226.00**		**16774.00**			**362287.00**	
0.00		Excess of Expenditure Over Income	0.00		1076226.00		Closing Balance	713939.00	
1093000.00			**1076226.00**		**1093000.00**			**1076226.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE2-II-Core : DBT Centre of Excellence for Microbiology - Phase II

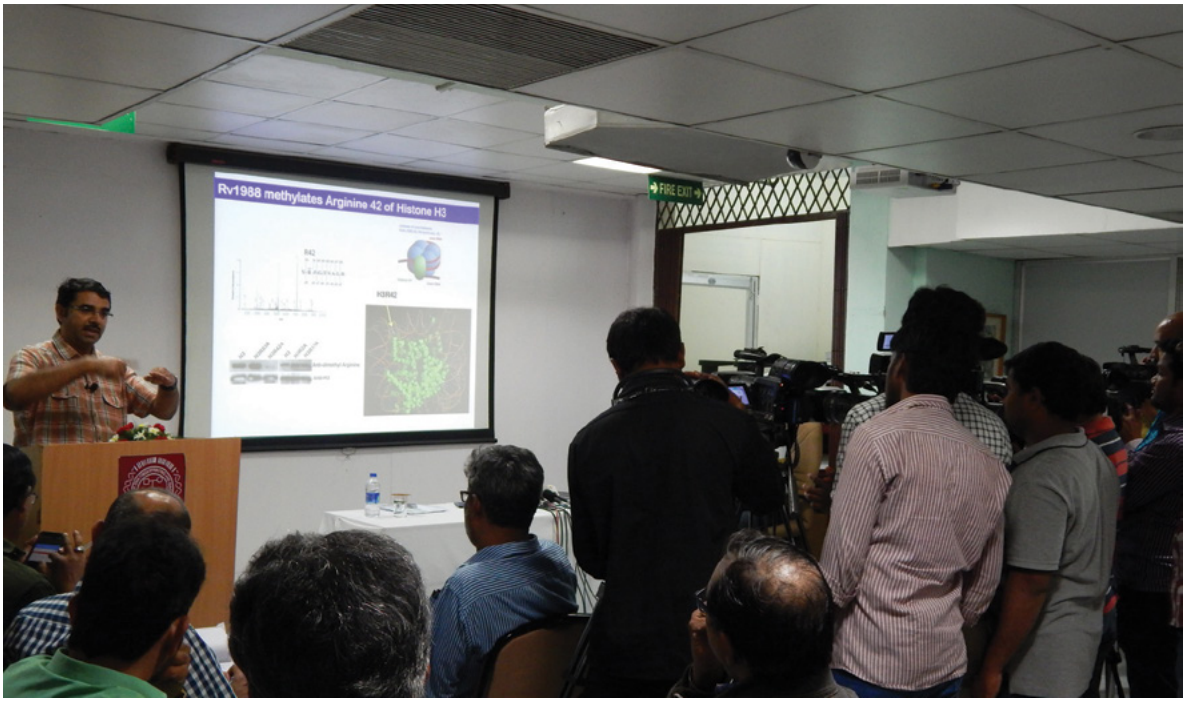
PI : Dr J Gowrishankar

Receipts and Payments Account from 01/04/2015 to 31/03/2016

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	9523323.00				Opening Balance	0.00	
11236000.00		Grant In Aid	0.00		956577.00		Salaries - Manpower	4137634.00	
0.00			0.00		600000.00		Consumables	832837.00	
0.00			0.00		0.00		Contingencies	20593.00	
0.00			0.00		0.00		Travel	11018.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		156100.00		Equipment	2134673.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	650000.00	
11236000.00			**9523323.00**		**1712677.00**			**7786755.00**	
0.00		Excess of Expenditure Over Income	0.00		9523323.00		Closing Balance	1736568.00	
11236000.00			**9523323.00**		**11236000.00**			**9523323.00**	

फोटो गैलरी
PHOTO GALLERY

Visit of Dr Harsh Vardhan, Hon'ble Minister of Science & Technology and Earth Sciences on 12.10.2015



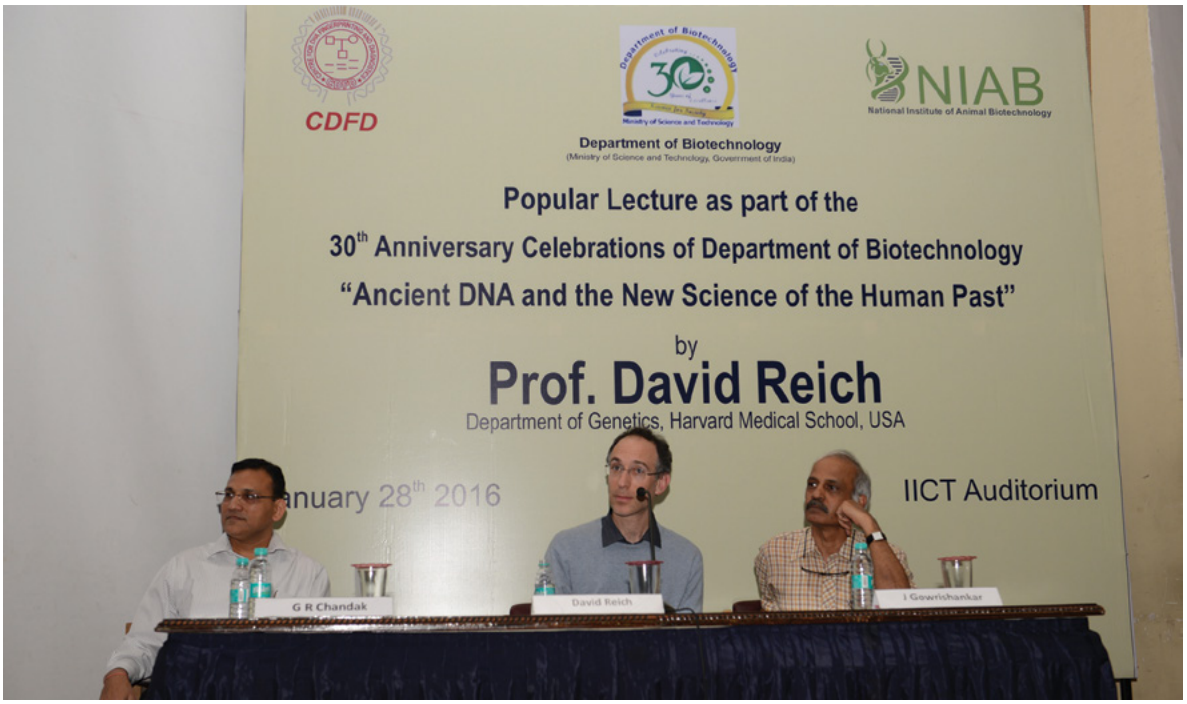
Press Conference for Dr Sanjeev Khosla's Article published in Nature Communications on 03.12.2015

Visit of Australian Delegates from University of Technology, Australia (QUT group) on 18.08.2015

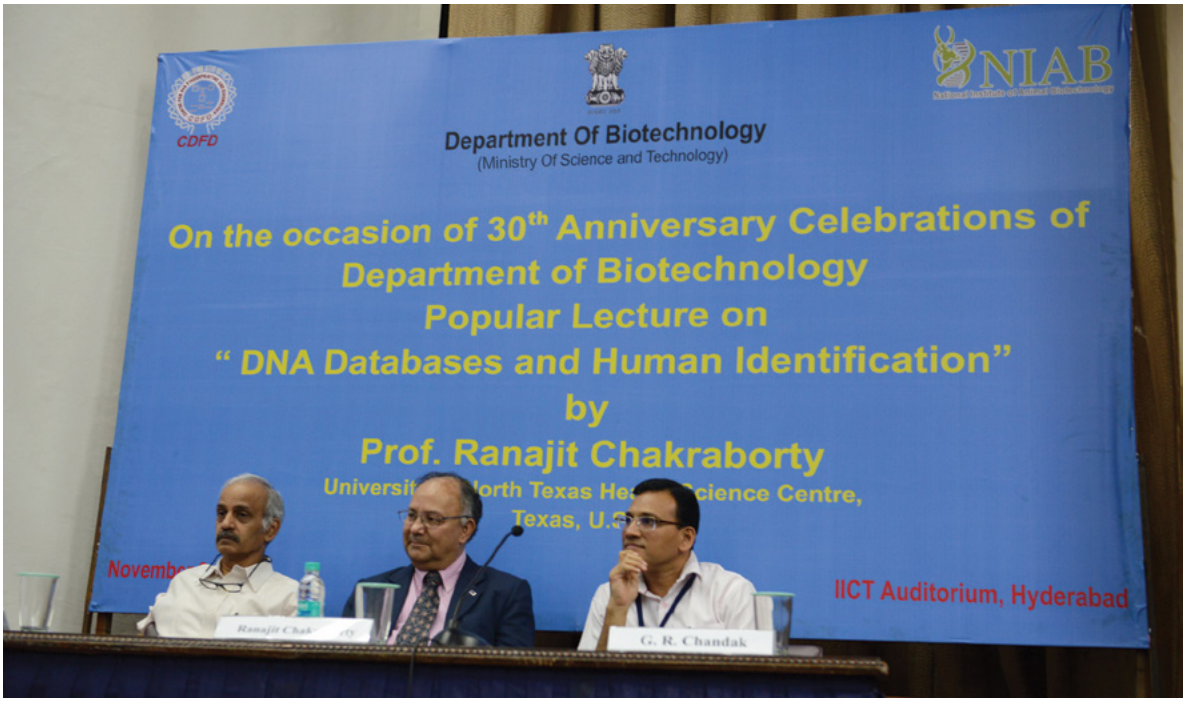
Visit of students from Centre of Excellence in Biotechnology, M.P. Council of Science and Technology (MPCOST), (Dept. of Science & Technology, Govt. of M.P.), Vigyan Bhawan, Nehru Nagar, Bhopal on 07.10.2015

Dr J Gowrishankar addressing the gathering on the Independence Day

Celebration of Digital India Week during 1-7 July 2015



Celebrations of 30th anniversary of DBT (Public lecture by Prof David Reich, Department of Genetics, Harvard Medical School, USA) on 28.01.2016.



Celebrations of 30th anniversary of DBT (Public lecture by Prof Ranajit Chakraborty, Department of Molecular and Medical Genetics, University of North Texas Health Science Center, Texas) on 09.11.2015

Glimpses of CDFD Foundation Day celebrations

Hindi Day celebrations

Workshop on Rajbhasha implementation and digital tools.

NOTES / REMARKS

NOTES / REMARKS

पीठावरण पृष्ठ का विवरण
Description of the Back Cover Page

पीठावरण पृष्ठ पर दर्शाए चित्रों का विवरण घडी की दिशानुसार नीचे से क्रमशः इस प्रकार हैं।

पहली तस्वीर में टोसिस और पॉलीडेक्टाइली दर्शाने वाले रोगियों की वंशावली और तस्वीरें हैं (ए-एफ) नियंत्रण (सामान्य) का सिंगर सिक्वेंसिंग क्रोमेटोग्राम, (जी) अभिभावक (विषमजात), (एच) और रोगी (समजात), (आई) में तीर द्वारा सी. 879जीए उत्परिवर्तन दर्शाया गया है। योजनाबद्ध एआरएमसी9 प्रोटीन सहित उत्प रिवर्तन और एआरएम डोमेन का स्थान (जे) यह तस्वीर नैदानिक प्रभाग से प्राप्त हुई है।

दूसरी तस्वीर में ड्रोसोफिला लारवा के केन्द्रीय तंत्रिका तंत्र की कंफोकल प्रक्षेपित तस्वीर, जहां हरा रंग जीएफपी मार्किंग से स्टेम कोशिकाएं और इसकी सभी संततियां दर्शाता है तथा लाल रंग ग्रेनी हैग नामक स्टैम कोशिका विशिष्ट मार्कर दर्शाते हैं। यह तस्वीर ड्रोसोफिला तंत्रिका विकास प्रयोगशाला द्वारा प्रदान की गई ।

तीसरी तस्वीर में जंतु सुविधा में नग्न चूहों पर किए जा रहे प्रयोग दर्शाए गए हैं।

चौथी तस्वीर खुले रूपांतरण (ओसी) से बंद कॉम्प्लेक्स (सीसी) तक आरएचओ हेक्सामर के काइनेटिक / साम्यता के चरणों का योजनाबद्ध प्रतिनिधित्व है। संभावित चरण जो एनयूएसजी से दर्शाए गए हैं, उन पर लक्ष्य हैं। हेक्सामेरिक संरचनाएं पीडीबी, ३आईसीई और १पीवीओ का उपयोग करते हुए निर्देशांकों पर आधारित हैं। यह तस्वीर अनुलेखन प्रयोगशाला द्वारा प्रदान की गई है।

पाँचवी तस्वीर अर्धसूत्री विभाजन में प्रोमेटाफेस में यू२ओएस कोशिका की कंफोकल तस्वीर है। अल्पा ट्यूबुलिन पीले रंग से अभिरंजित है, डीएनए लाल और सेंट्रोमियर हरा है। यह तस्वीर कोशिका चक्र नियमन प्रयोगशाला द्वारा दी गई है।

छठी तस्वीर में गोभी की पत्ती में साइडेरोफोर संश्लेषण की पादप अभिव्यक्ति दर्शाई गई है और इसमें पौधे में अल्प आयरन की दो परिस्थितियों की पुष्टि होती है जो साइडेरोफोर उदग्रहण और संश्लेषण जीनों की अभिव्ययक्ति उद्दीपित करती हैं। यह तस्वीर पादप सूक्ष्मजीव अंतःक्रिया प्रयोगशाला द्वारा दी गई है।

The figures depicted in the cover page in the clockwise order starting from the base are as follows:

The first figure shows pedigree and photographs of patients showing ptosis and polydactyly [A – F] Sanger sequencing chromatogram of Control (Normal) [G], Parent (Heterozygous)[H] and patient (homozygous)[I] showing c.879G>A mutation indicated by arrows. Schematic illustration of ARMC9 protein with location of mutation and ARM domains [J]. This image was obtained from the laboratory of human and medical genetics

The second figure is the confocal superimposed image of drosophila larval central nervous system, where green represents GFP marking the stem cells and all its progenies and red represents a stem cell specific marker called Grainyhead. This image has been provided by the Laboratory of Drosophila Neural Development.

The third photograph represents the experiment being conducted on nude mice at the animal facility.

The fourth figure is a schematic representation of the kinetic / equilibrium steps during the conversion of open (OC) to closed complex (CC) of the Rho hexamer. Putative step(s) those are targeted by NusG are indicated. Hexameric structures are based on the co-ordinates using the PDBs, 3ICE & 1PVO, respectively. This image has been provided by the Laboratory of Transcription.

The fifth image is the confocal image of a U2OS cell in prometaphase stage of mitosis, the alpha tubulin is stained in yellow, the DNA is in red and the centromere is green. This image has been given by the Laboratory of Cell Cycle Regulation.

The sixth figure represents a cabbage leaf showing the *in planta* expression of the siderophore synthesis and uptake cluster affirming the low iron condition within the plant which induces the expression of siderophore uptake and synthesis genes. This image has been provided by the Laboratory of Plant Microbe Interactions.

(पीठावरण पृष्ठ का चित्रांकन पादप रोगाणु अंतःक्रिया प्रयोगशाला की वरिष्ठ अनुसंधान अध्येता सुश्री प्रशांति सिंह द्वारा किया गया है।)

(The back cover page above has been designed by Senior Research Fellow Ms. Prashanti Singh of the Laboratory of Plant Microbe Interactions.)

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