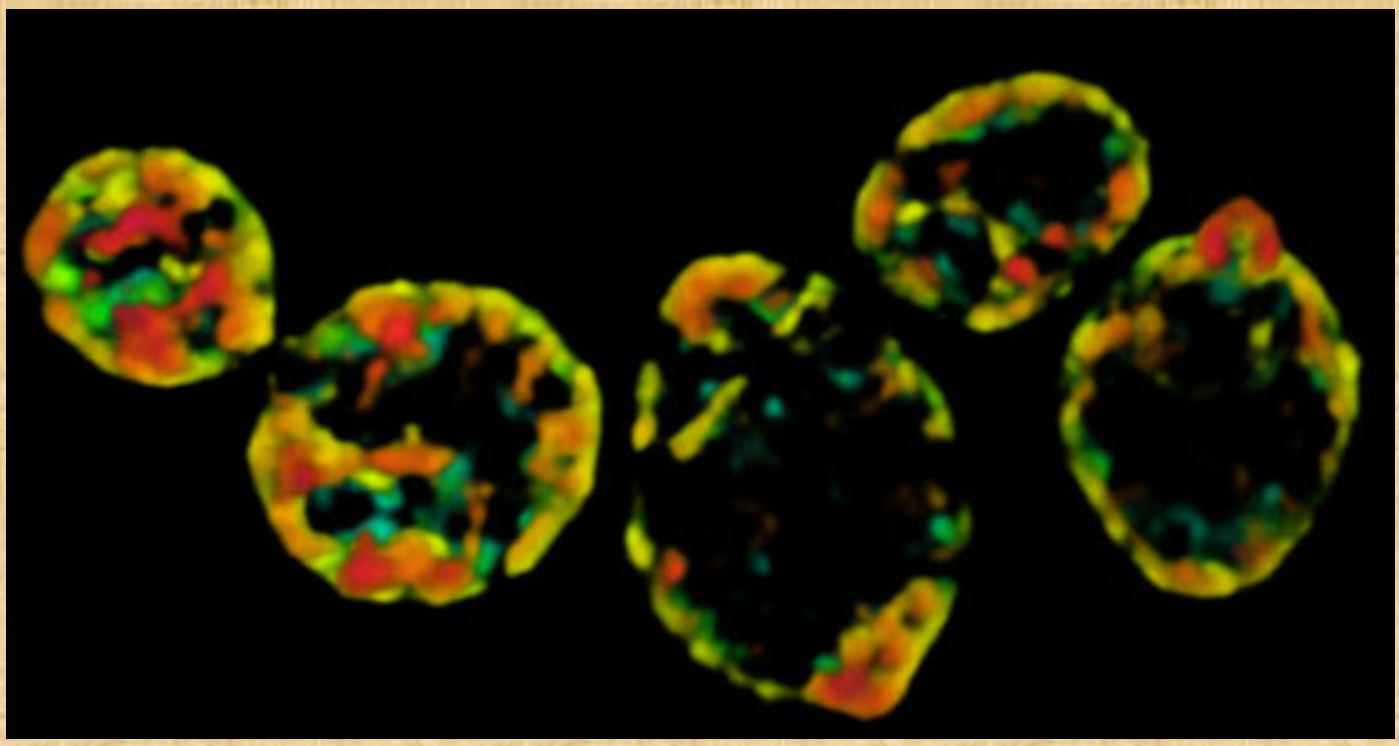
ICBYFF - 2019

XI International Conference on Biology of Yeasts and Filamentous Fungi

27 - 29 November 2019

Hyderabad, INDIA



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ICBYFF – 2019

XI International Conference on Biology of Yeasts and Filamentous Fungi

27 - 29 November 2019

Venue: School of Life Sciences
University of Hyderabad
Hyderabad, India 500 046

Programme and Abstract Book

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Scientific Programme ICBYFF-2019

XI International Conference on Biology of Yeasts and Filamentous Fungi

27th - 29th November 2019

Venue: Auditorium, School of Life Sciences, University of Hyderabad

DAY 1 - 27/11/2019		
8:00 - 9:00 AM	**Registration**	
9:00 - 9:30 AM	**Inauguration**	
Welcome and Introduction to the meeting by the organizers		
Session I: Chromatin and Transcription		
Chairperson: Jerry L. Workman		
9:30 - 9:50 AM	**Jerry L. Workman**	Phosphorylation of histone H3 threonine 11 by the Tda1 kinase under nutritional stress requires the AMPK and CK2 kinases
9:50 - 10:10 AM	**K. Natarajan**	Chromatin and transcriptional control of iron homeostasis in *Candida albicans*
10:10 - 10:30 AM	**Purnima Bhargava**	Yeast PAF1 complex counters the pol III accumulation and replication stress on the tRNA genes
10:30 - 10:50 AM	**Nishant K.T.**	Mitotic genome dynamics and instability in the baker's yeast

10:50 - 11:00 AM	**Shalini Aricthota**	DDK Hsk1 phosphorylates Sirtuin Hst4 and targets it for degradation on replication stress to stabilize stalled DNA replication forks
11:00 - 11:10 AM	**Premlata Kumari**	Mechanism of DNA-Protein crosslink repair in pathogenic yeast *Candida albicans*
11:10 - 11:40 AM	**TEA BREAK**	

Session II: Epigenetics and Gene Regulation

Chairperson: Kaustuv Sanyal

11:40 - 12:00 PM	**Kaustuv Sanyal**	The genetic switch of a major growth phase transition is fine-tuned by two residues of histone H3
12:00 - 12:20 PM	**Himanshu Sinha**	Are ribosomal protein genes buffers of phenotypic plasticity?
12:20 - 12:40 PM	**Altaf Bhat**	Understanding the mechanism of heterochromatin organization in *Schizosaccharomyces pombe*
12:40 - 1:00 PM	**Aarti Sevilimedu**	RNA-mediated regulation of heterochromatin in *S. pombe*
1:00 - 1:15 PM	**Supreet Saini**	Dynamics of public goods production and cooperation in yeast
1:15 - 1:25 PM	**Md. Hashim Reza**	Atg11, an autophagy regulator, is required for high fidelity chromosome segregation
1:25 - 2:30 PM	**LUNCH BREAK**	

Session III: RNA metabolism

Chairperson: Biswadip Das

2:30 - 2:50 PM	**Biswadip Das**	Post-transcriptional regulation of gene expression in

		Saccharomyces cerevisiae involving modulation of nuclear export and nuclear mRNA degradation
2:50 - 3:10 PM	**Shravan Kumar Mishra**	The ubiquitin-fold-activated Sde2 helps spliceosome capture distant exons
3:10 - 3:30 PM	**Purusharth I Rajyaguru**	Role of RGG-motif proteins in translation control
3:30 - 3:50 PM	**Nimisha Sharma**	Functional and structural insights into the *Schizosaccharomyces pombe* ELL-EAF transcription elongation factor complex
3:50 - 4:05 PM	**Neelam Dabas Sen**	DEAD-box RNA helicases: A family affair in governing translational control of eukaryotic gene expression
4:05 - 4:20 PM	**Flash Talks**	Arjamand Mushtaq, Deepika Trakroo, Kuladeep Das, Priyanka Sarkar, Rajlaxmi Gaine, Riddhi Banerjee, S. Sagar, Subhasish Mukherjee, Sweta Tiwari, Vasavi Bhogadi
4:20 - 6:30 PM	**TEA BREAK AND POSTER SESSION - I**	

Session IV: Fungal Pathogenesis – I		
Chairperson: D. P. Kasbekar		
6:30 - 6:50 PM	**Rajendra Prasad**	ABC superfamily transporters of an emerging pathogenic yeast *Candida auris*
6:50 - 7:10 PM	**Naweed Naqvi**	A novel morphogenetic signal in fungal pathogenesis
7:10 PM onwards	**DINNER**	

	DAY 2 - 28/11/2019	
Session V: Protein Homeostasis		
Chairperson: Michael Knop		
9:30 - 9:50 AM	**Michael Knop**	N-degron profiling: determinants of protein stability encoded in the N-terminus of proteins
9:50 - 10:10 AM	**Deepak Sharma**	Ydj1 regulates functional distinction of Ssa Hsp70s in Hsp90 chaperoning pathway
10:10 - 10:30 AM	**Parul Mishra**	Engineering ubiquitin to probe and rewire protein degradation network
10:30 - 10:50 AM	**Chandan Sahi**	The expanding world of J-domain proteins
10:50 - 11:00 AM	**Ganapathi Kandasamy**	Hsp110-mediated proteasomal degradation of Hsp70 chaperone-associated substrates in yeast
11:00 - 11:10 AM	**Darakshan Alim**	Relevance of 7-transmembrane receptor protein Rta2 in coordinating endoplasmic reticulum stress responses in *Candida albicans*
11:10 - 11:40 AM	**TEA BREAK**	
Session VI: Organelle Biology – I		
Chairperson: Dibyendu Bhattacharyya		
11:40 - 12:00 PM	**Dibyendu Bhattacharyya**	Interplay and relative positioning of functional sites on Endoplasmic Reticulum
12:00 - 12:20 PM	**Ravi Manjithaya**	Yeasts can still teach a thing or two about autophagy

12:20 - 12:40 PM	**Shirisha Nagotu**	Structural and functional analysis of disease causing mutations of Drp1 through analyses of the yeast homologue Dnm1
12:40 - 1:00 PM	**Kaustuv Datta**	Mitochondrial gene expression in response to carbon source: Control by a yeast-clade-specific putative helicase *IRC3* in *Saccharomyces cerevisiae*
1:00 - 1:10 PM	**Gurranna Male**	Nucleolar size regulates nuclear envelope shape in *Saccharomyces cerevisiae*
1:10 - 1:20 PM	**Amita Pal**	Role of eisosome proteins, Pil1 and Lsp1, mitophagy, and cell death in *Saccharomyces cerevisiae*
1:20 - 2:30 PM	**LUNCH BREAK**	

Session VII: Organelle Biology - II and Gene Expression

	Chairperson: Santanu K. Ghosh	
2:30 - 2:50 PM	**Santanu K. Ghosh**	Meiosis-specific functions of kinesin motors in maintenance of chromosome integrity
2:50 - 3:10 PM	**Mithilesh Mishra**	Acto-myosin ring and the art of fission
3:10 - 3:30 PM	**Rajan Sankaranarayanan**	Chiral proofreading and its implications for the evolution of eukaryotes
3:30 - 3:40 PM	**Mani Garg**	Scd6 and Psp2 are multicopy suppressors of clathrin deficiency
3:40 - 3:50 PM	**Sriram Varahan**	Metabolic constraints determine the self-organization of specialized heterogeneous cell groups
3:50 - 4:05 PM	**Flash Talks**	Avishek Roy, Imlitoshi Jamir, Kumari Sweta, Rajalakshmi

		Srinivasan, Rashmi K Bed, Ruchika Kumari, Shweta Kaul, Suchismita Datta, Terence Infant W.L, Yash Verma
4:05 - 6:30 PM	**TEA BREAK AND POSTER SESSION - II**	
	Session VIII: Genome Stability	
	Chairperson: Rajendra Prasad	
6:30 - 6:50 PM	**K. Muniyappa**	Mechanistic insights into meiotic chromosome synapsis and recombination in *Saccharomyces cerevisiae*
6:50 - 7:10 PM	**Narottam Acharya**	Role of CaPolη in genome stability, morphogenesis and the development of systemic candidiasis
7:10 PM onwards	**CONFERENCE DINNER**	
	DAY 3 - 29/11/2019	
	Session IX: Fungal Pathogenesis – II	
	Chairperson: Sneh Lata Panwar	
9:30 - 9:50 AM	**Sneh Lata Panwar**	The Tac1-dependent coordinated regulation of genes contributes to pathogenicity traits in *Candida albicans*
9:50 - 10:10 AM	**Ragiba Makandar**	Analyzing host responses to target *Fusarium graminearum* infection in crop plants
10:10 - 10:30 AM	**Gopaljee Jha**	Novel strategies to control fungal diseases of rice

10:30 - 10:50 AM	**Ranjan Tamuli**	The CRZ-1 transcription factor and calcium signaling genes are required for heat-shock response, normal circadian clock, and cellulose degradation in *Neurospora crassa*
10:50 - 11:05 AM	**Saif Hameed**	Natural food flavoring agent vanillin impedes CaCdr2p driven efflux and metabolic adaptability in human fungal pathogen, *Candida albicans*
11:05 - 11:15 AM	**Mubashshir Rasheed**	Glycosylphosphatidylinositol-linked aspartyl proteases regulate the secretome of the pathogenic yeast *Candida glabrata*
11:15 - 11:45 AM	**TEA BREAK**	

Session X: Protein Structure and Evolution

Chairperson: Paike Jayadeva Bhat

11:45 - 12:05 PM	**Paike Jayadeva Bhat**	Putting bits and pieces together: The saga of arrestin-related trafficking protein in *Saccharomyces cerevisiae*
12:05 - 12:25 PM	**Sreenivas Chavali**	Repeat to Reap it: Systems-level understanding of the role of amino acid repeats
12:25 - 12:40 PM	**Narendra K Bairwa**	Loss of F-box motif encoding gene *SAF1* and *RRM3* together leads to synthetic growth defect and sensitivity to hydroxyurea and methyl methane sulfonate in *S. cerevisiae*
12:40 - 12:50 PM	**Soumita Paul**	Investigating the mechanism of regulation of cellular repertoire of SKS1 mRNA in *Saccharomyces cerevisiae*

12:50 - 1:00 PM	**Anjali Mahilkar**	Laboratory evolution of allopatric speciation in *Saccharomyces cerevisiae*
1:00 - 1:10 PM	**V. Anand Parnandi**	Understanding the pathobiology of mixed-species candidiasis
1:10 - 2:00 PM	**LUNCH BREAK**	
Session XI: Cell Division		
Chairperson: Geetanjali Sundaram		
2:00 - 2:20 PM	**Geetanjali Sundaram**	Regulation of mitotic timing: Communication between CDK and MAPK
2:20 - 2:40 PM	**Pankaj V. Alone**	Alteration in the fidelity of *HIS4* start codon selection influences 3-amino-1,2,4-triazole sensitivity in hyper-GTPase defective eIF5 protein
2:40 - 2:55 PM	**Tania Bose**	Cohesin proteins control protein quality through misfolding, aggregation, and accumulation
2:55 - 4:00 PM	**Valedictory/Prize Distribution/Next Meeting Venue Discussion**	
4:00 PM	**TEA**	

Posters at a Glance

List of Posters

Poster Session I: Odd numbered posters
November 27, 2019, 4:20 - 6:30 PM

Poster Session II: Even numbered posters
November 28, 2019, 4:05 - 6:30 PM

Poster No	Author	Title
P1	**Aditya Shukla**	Deciphering the role of multicopy suppressor IZH2 in oxidative stress
P2	**Anamika Battu, Rajaram Purushotham, Partha Dey, S.Surya Vamshi**	Role for the aspartyl protease, CgYps1, in regulation of the oxido-reductive status in *Candida glabrata*
FT1/P3	**Arjamand Mushtaq**	Vigilin protein Vgl1 is required for heterochromatin mediated gene silencing in *Schizosaccharomyces pombe*
P4	**Aroni Mitra**	*Ustilago maydis* small heat shock protein UmHSP12 is a stress responsive protein
P5	**Priyanka Bhakt, Deepak Choudhary**	The phosphatidylinositol 3-phosphate 5-kinase, CgFab1, is essential for antifungal tolerance in the pathogenic yeast *Candida glabrata*
P6	**Christy Noche K. Marak**	Insights on the mechanism of calmodulin and calcium/calmodulin-dependent kinases in regulating stress tolerance and sexual development in *Neurospora crassa*

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FT2/P7	**Deepika Trakroo**	Role of kinetochore in maintaining chromosome condensation in meiosis
P8	**Doris Abhirupa Pradhan**	Screening *Trichoderma* strains for rhizosphere competence and induction of biotic stress tolerance in castor (*Ricinus communis L*.)
P9	**Fizza Askari**	Role for the lipid kinase, CgVps34, in trafficking of the membrane transporters in *Candida glabrata*
P10	**Gajanan B. Zore**	Low hydrostatic pressure inhibit morphogenesis in *Candida albicans*
P11	**Heena Shoket**	Deletion of F-box motif encoding YDR131C and retrograde signaling related RTG1 gene together leads to cell size enlargement and stress tolerance phenotype in *Saccharomyces cerevisiae*
ST6/P12	**Gurranna Male**	Nucleolar size regulates nuclear envelope shape in *Saccahromyces cerevisiae*
P13	**Kirpa Yadav**	Functional specificity and evolution of Cwc23: A ubiquitous and non-canonical J-domain protein essential for pre-mRNA splicing
P14	**Komalapriya Chandrasekaran**	Systems biology of pathogenesis mechanisms of fungal pathogen *Candida albicans*
FT3/P15	**Kuladeep Das**	D-amino acids poisoning in an opportunistic fungal pathogen *Candida albicans*

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ST8/P16	**Mani Garg**	Scd6 and Psp2 are multicopy suppressors of clathrin deficiency
ST3/P17	**Md. Hashim Reza**	Atg11, an autophagy regulator, is required for high fidelity chromosome segregation
P18	**Monika Pandita**	Loss of Glyoxylate metabolic switch regulator *UCC1* and La-motif encoding *SRO9* genes together leads to MMS and H_2O_2 stress tolerance in *Saccharomyces cerevisiae*
ST11/P19	**Premlata Kumari**	Mechanism of DNA-Protein crosslink repair in pathogenic yeast *Candida albicans*
FT19/P20	**Nayan Moni Deori, Terence Infant W.L.**	Characterizing the dual targeting and function of the peroxisomal protein Pex30
P21	**Prerna Pathak**	Structural and functional analysis of seven transmembrane receptor protein RTA3 in *Candida albicans*
P22	**Sohini Basu**	Cyclin synthesis and degradation in mitosis: Understanding new regulators
P23	**R.P. Vivek-Ananth**	Prediction and analysis of the secretome of an opportunistic fungal pathogen
FT14/P24	**Rajalakshmi Srinivasan**	Gcn4 mediates a methionine-dependent anabolic program by controlling amino acid supply
FT5/P25	**Rajlaxmi Gaine**	eIF4G(Tif4631p): A major player in nucleating the CTEXT proteome
FT11/P26	**Avishek Roy**	Identification of critical amino acid residues of calcineurin regulatory subunit (cnb-1) involved in stress response and cross talk with heat shock stress

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		pathway via its target calcineurin responsive zinc finger protein (CRZ-1) in *Neurospora crassa*
FT7/P27	**Sagar S.**	The bakers's yeast Msh4-Msh5 associates with double strand break hotspots and chromosome axis during meiosis to promote crossovers
P28	**Sajad Ahmad Padder**	Understanding the role of novel actors of drug resistance in clinical isolates of *Candida* species
ST1/P29	**Shalini Aricthota**	DDK Hsk1 phosphorylates Sirtuin Hst4 and target it for degradation on replication stress to stabilize stalled DNA replication forks
P30	**Santhosh Kumar Sariki**	Flocculation of *Saccharomyces cerevisiae* is dependent on activation of Slt2 and Rlm1 regulated by the Cell Wall Integrity pathway
P31	**Shraddheya Kumar Patel**	Deciphering the role of Pol32, the non-essential subunit of DNA polymerase delta in pathogenesis of *C. albicans*
P32	**Shreosi Chatterjee**	Diversity and ecology of manglicolous filamentous fungi isolated from Sundarban mangrove ecosystem, India
FT4/P33	**Priyanka Sarkar**	Stress : MAPK Spc1: Mitotic entry decisions
P34	**Soumitra Sau**	A role for the yeast PCNA unloader Elg1 in eliciting the DNA damage checkpoint
FT8/P35	**Subhasish Mukherjee**	Virulence function for ExAsp1, a secreted aspartyl protease from *Ustilago maydis*

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P36	**Suman Dash**	Intra-specific variation in LOH and mutation rates among *S. cerevisiae* strains
FT9/P37	**Sweta Tiwari**	Identifying the mRNA targets of eIF4G-binding translation repressor protein, Scd6
FT13/P38	**Kumari Sweta**	Screening and identification of multicopy suppressors of the genotoxic sensitivity associated with absence of ELL in *Schizosaccharomyces pombe*
FT10/P39	**Vasavi Bhogadi**	Novel function of fission yeast sirtuin Hst4 in regulation of DNA replication
ST9/P40	**Sriram Varahan**	Metabolic constraints determine the self-organization of specialized, heterogeneous cell groups
P41	**Agamani Ghosal**	Understanding the role of CDK hyperactivation as a novel trigger for MAPK activity in Schizosaccharomyces pombe
ST7/P42	**Amita Pal**	Role of eisosome proteins, Pil1 and Lsp1, mitophagy and cell death in *Saccharomyces cerevisiae*
ST13/P43	**Anand Parnandi V.**	Understanding the pathobiology of mixed-species candidiasis
ST12/P44	**Anjali Mahilkar**	Laboratory evolution of allopatric speciation in *Saccharomyces cerevisiae*
P45	**Arindam Chakraborty**	Investigating the role of Nrd1p-Nab3p-Sen1p (NNS) complex in the function of the nuclear exosome in *Saccharomyces cerevisiae*
P46	**Aswathy Narayanan**	Identification of centromeres in the rapidly emerging multidrug resistant pathogen *Candida*

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		auris reveals centromere inactivation during speciation
FT6/P47	**Riddhi Banerjee**	To gain insights into the Drp1 related disorders through analyses of equivalent orthologous mutations in budding yeast Dnm1
ST5/P48	**Darakshan Alim**	Relevance of 7-transmembrane receptor protein Rta2 in coordinating endoplasmic reticulum stress responses in *Candida albicans*
P49	**Evanjalee Albert Arokiyaraj**	A blueprint of the protein secretion machinery in *Neurospora crassa*
ST4/P50	**Ganapathi Kandasamy**	Hsp110-mediated proteasomal degradation of Hsp70 chaperone associated substrates in yeast
P51	**Khem Raj**	Transcriptome-wide alternative splicing and isoform usage during the biofilm growth phase in *Candida glabrata*
FT12/P52	**Imlitoshi J., Pallavi D.**	A genome-wide screen identifies several genes essential for maintaining nuclear architecture and uncovers novel pathways in *S. cerevisiae*
P53	**Krishnendu Guin**	Proximity and homology guided complex-translocations among the centromeres drive karyotype evolution and centromere type transition in *Candida* species complex
P54	**Kundan Kumar, Romila Moirangthem**	Histone H4 dosage modulates DNA damage response in the pathogenic yeast *Candida*

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		glabrata via homologous recombination pathway
P55	**Vaibhav Bhardwaj**	Deletion of the non-essential Rpb9 subunit of RNA polymerase II results in pleiotropic phenotypes in *Schizosaccharomyces pombe*
P56	**Lalita Panigrahi**	Understanding the role of Wat1 in TOR pathway and in cellular homeostasis
P57	**Kathirvel Ramalingam**	Siz2 promotes rDNA stability by modulating levels of Tof2 in *Saccharomyces cerevisiae*
P58	**Madhuri Shinde, Sujata Ingle, Asha Bhadeker**	Protein profiling of *Aspergillus parasiticus* var. *globusus* on exposure to Amphotericin B
ST10/P59	**Mubashshir Rasheed, Naveen Kumar**	Glycosylphosphatidylinositol-linked aspartyl proteases regulate the secretome of the pathogenic yeast *Candida glabrata*
FT15/P60	**Rashmi K Bed**	*Aspergillus terreus* strain improvement for Biodiesel Production Using Agro-Waste Substrate
P61	**Prabhat Kumar**	*Saccharomyces boulardii*: A systematic review on current scenario on metabolic engineering of probiotic yeast strain
P62	**Rachayeeta Deb**	Investigating the role of PEX25 in peroxisome biogenesis in yeast
P63	**Neha Joshi**	Investigating the role of peroxisomes in Parkinson's disease

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FT16/P64	**Ruchika Kumari**	Understanding the role of Exocyst: a Tethering Complex in Autophagy
P65	**S M Udaya Prakash**	Identification of novel inhibitors targeting 14-alpha demethylase (Erg11p) of *Candida albicans* using high throughput virtual screening combined molecular dynamics simulations (HTVS-MDS) approach
P66	**Sahu M.S.**	The high-osmolarity glycerol (HOG) MAPK pathway regulates iron homeostasis and virulence in *Candida glabrata*
P67	**Satya Ranjan Sahu**	Evaluation of pathogenic potential of various DNA polymerase knock-outstrains of *Candida albicans* and its implication in the development of live attenuated antifungal vaccine
P68	**Serena Ngiimei D.**	Understanding the molecular mechanism of zinc transporter genes in *Neurospora crassa*
P69	**Shubhangi Jagtap**	Direct transesterification for biodiesel production using wet biomass of *Yarrowia lipolytica* NCIM 3589 and its mutant grown on waste cooking oil
FT17/P70	**Shweta Kaul**	Ubiquitin mediated clearance of prion protein *in vivo*
P71	**Sonam Kumari**	CgYOR1 mediated azole resistance in *Candida glabrata* via TOR and calcineurin cascade
ST2/P72	**Soumita Paul**	Investigating the mechanism of regulation of cellular repertoire of *SKS1* mRNA in *Saccharomyces cerevisiae*

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P73	**Vineeth V.**	The E3 ubiquitin ligase Pib1 regulates effective gluconeogenic shutdown upon glucose availablility
FT18/P74	**Suchismita Datta**	Understanding the functional relationship between the bZIP transcription factors Atf1 and Pcr1 in regulation of cell division in *Schizosaccharomyces pombe*
P75	**Sunirmal Paira**	Studies on the regulation of UPR signaling by nuclear exosome/DRN in *Saccharomyces cerevisiae*
P76	**Sandip Patra**	An analysis of *Candida glabrata*-epithelial cell interaction
P77	**Viji V. Subramanian**	Chromosome end-adjacent regions (EARs) promote high density of DNA breaks on short chromosomes during sexual reproduction
FT20/P78	**Yash Verma**	MRX8 is essential for Cox1p translation in *Saccharomyces cerevisiae*
P79	**Zeenat Rashida**	Raptor/Kog1 regulates AMPK/Snf1 activation for metabolic rewiring under nutrient limitation
P80	**Darshana Baruah**	Understanding the role of phospholipase C-1 and secretory phospholipase A2 in circadian clock and biomass degradation in Neurospora crassa

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Speaker Abstracts
ICBYFF-2019

Phosphorylation of histone H3 threonine 11 by the Tda1 kinase under nutritional stress requires the AMPK and CK2 kinases.

Seunghee Oh, Tamaki Suganuma, Selene K Swanson, Michael P Washburn, Jerry L. Workman

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Email: dbalukas@stowers.org

Nutritional stress signaling in budding yeast includes phosphorylation of histone H3T11 (H3pT11). To understand upstream signaling of H3pT11, we dissected kinases involved in this process. We found that H3pT11 requires both AMPK and CK2 kinases. However, these kinases do not directly phosphorylate H3T11 but instead directly phosphorylate the Tda1 kinase. Tda1 is a yeast AMPK interacting kinase that phosphorylates H3T11 in vitro and in vivo. Phosphorylation of Tda1 is required for its proper histone kinase activity and its protein stability. The Tda1 kinase thus integrates signals of multiple upstream kinases to regulate H3T11 phosphorylation upon nutrient stress.

Chromatin and transcriptional control of iron homeostasis in *Candida albicans*

Krishnamurthy Natarajan

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How pathogens sense nutrient levels and reconfigure growth is central to the pathogens' survival strategies in host. *Candida albicans*, the major human fungal pathogen, switches from a commensal to a pathogenic form in immunocompromised hosts. Nutrient availability, in particular, that of iron level is essential for growth and virulence in host. *C. albicans* employs an elaborate set of pathways to mobilize iron from intracellular and extracellular sources. When iron levels are altered in vitro, however, *C. albicans* responds by transcriptional induction of iron uptake genes and repression of genes that utilize iron as cofactors, thereby engendering iron from external sources and conserving internal iron pools. Here, we have delineated the relative roles of key transcription factors that modulate iron homeostasis and robust virulence in *C. albicans*. We further identified the crucial requirement of the SAGA transcriptional coregulatory complex in mediating iron homeostasis gene regulation and filamentation through acetylation of histone H3-Lys9, and RNA polymerase II recruitment at target promoters in vivo.

Yeast PAF1 complex counters the pol III accumulation and replication stress on the tRNA genes

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The PAF1C (pol II-associated factor 1 complex), one of the pol II-associated factors, is reported to associate with the RNA polymerase (pol) I and pol II and influence their transcription. In comparison to pol II, only a few regulatory proteins are known for pol III, which transcribes mostly house-keeping and non-coding genes. The transcriptome of pol III is constituted mainly by tRNA genes, the known naturally occurring replication fork barriers in vivo. We found the Paf1 complex (PAF1C) interacts with the pol III transcription complex components pol III, TFIIIC and TFIIIB. Its occupancy on the pol III-transcribed genes is low and not correlated with nucleosome positions, pol III occupancy and transcription. Genotoxin exposure causes pol III but not Paf1 loss from the genes. In comparison, Paf1 deletion leads to increased DNA damage, higher occupancy of pol III, g-H2A and DNA pol2 in gene-specific manner on all the tRNA genes. The gene-specific effects of Paf1 are not due to spatial locations of tRNA genes, which are found scattered on all chromosomes. PAF1C restricts the accumulation of pol III on the gene body by influencing the pol III pause on the genes.

This reduces the replication stress caused by the pol III barrier and transcription-replication conflict on these highly transcribed genes.

Mitotic genome dynamics and instability in the baker's yeast

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The baker's yeast *Saccharomyces cerevisiae* is a useful model for understanding mutational processes due to its small genome size and generation time. We estimated genome wide intra-specific variation in mutation rates and spectrum between three homozygous and two hybrid strains of *S. cerevisiae*. We observed up to 8 fold difference in single nucleotide mutation rates among these *S. cerevisiae* genetic backgrounds. The two hybrid strains of *S. cerevisiae* also showed differences in rates of loss of heterozygosity (LOH). These results suggest that LOH rates can vary between *S. cerevisiae* hybrids based on genetic background. Further our results suggest LOH can contribute significantly to genetic changes in *S. cerevisiae*. In collaborative work (Lucas Argueso, Colorado State University), we observe that LOH is responsible for a switch in the colony morphology of a *S. cerevisiae* hybrid strain commonly used in bioethanol production causing cells to aggregate. Further, the number of secondary LOH in these hybrid strains are higher in the presence of pre-existing LOH suggestive of systemic genomic instability. Our results provide new insights into the

scale of intra-specific variation in mutation rates and also into the role of LOH in genome dynamics and instability in *S. cerevisiae*.

ST1/P29

DDK Hsk1 phosphorylates Sirtuin Hst4 and target it for degradation on replication stress to stabilize stalled DNA replication forks

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Sirtuins are highly conserved NAD+ dependent class III histone deacetylases which function in cellular processes including cell survival, DNA damage and repair. Our previous reports show that fission yeast *hst4*Δ deficient cells are sensitive to replication stress and Hst4p is downregulated on methyl methanesulfonate (MMS) treatment. The aim of the current study is to decipher the molecular mechanism of regulation of Hst4. DDK is a highly conserved kinase involved in the regulation of DNA replication. The replication dependent roles of DDK are very well studied across all model systems. However, whether DDK has any positive role in the replication stress response is a matter of debate. We found that Hst4 is targeted for degradation in a replication dependent manner upon HU and MMS stress. Next, we found out for the first time that DDK phosphorylates Hst4 at serine residues to target it for proteolysis via SCF ubiquitin ligase. This process is independent of intra-S phase checkpoint activation. The non-

degradable mutant of Hst4 (4SA-Hst4) have persisting DNA damage foci and show defects in stalled fork recovery and proper DNA damage bypass.

We also show that fork protection complex stability at the chromatin is affected in 4SA-Hst4 mutant leading to these defects. Overall, this study identifies a novel role of DDK in replication fork stabilisation independent of its role in intra-S phase checkpoint pathway. Since, sirtuins are deregulated in cancer, this study helps in identifying similar mechanistic roles in higher eukaryotes.

ST2/P19

Mechanism of DNA-Protein crosslink repair in pathogenic yeast *Candida albicans*

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Candida albicans is an opportunistic fungal pathogen exists as an integral part of the massive human microbial flora. It remains under the constant threat of DNA damages due to oxidative burst generated by innate immune cells and therapeutics such as radiation, cisplatin, AZT, etc. administered to immune-suppressed individuals. DNA-Protein Crosslinks (DPC) are one such damages where specific DNA binding proteins are covalently linked to DNA and form irreversible bulky adducts to induce genome instability and cell death. Underlying repair mechanisms of DPCs are elusive in *Candida sp*.. Currently, we are investigating involvement of three pathways in *C. albicans* namely NER, HR and DPCR. Unlike NER and PRR that target damaged portion of DNA segment, DPCR targets the protein part of the DNA-protein complex. In budding yeast and mammals, metalloproteases like Wss1 *(alias* Sprtn*)* and Tdp*1* play essential role in DPCR. The gene encoding *TDP1*, is

absent in *C. albicans*. Thus, the involvement of Wss1 dependent DPCR would become essential for *C. albicans* survival, colonization, and in the development of systemic candidiasis. Contrary to *S. cerevisiae*, homozygous deletion of *WSS1* strain alone is hypersensitive to both DNA replication inhibitor and DPC inducing agents but not to MMS, H_2O_2 etc. Detail characterisation of CaWss1 and its role in genome stability, filamentation, drug resistance and pathogenesis will be discussed.

The genetic switch of a major growth phase transition is fine-tuned by two residues of histone H3

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Histone H3 and its variants regulate gene expression but the latter are absent in most ascomycetous fungi. Here, we report the identification of a variant histone H3, which we have designated $H3V^{CTG}$ because of its exclusive presence in the CTG clade of ascomycetes, including *Candida albicans*, a human pathogen. *C. albicans* grows both as single yeast cells and hyphal filaments in the planktonic mode of growth. It also forms a three-dimensional biofilm structure in the host as well as on human catheter materials under suitable conditions. $H3V^{CTG}$ null (*hht1/hht1*) cells of *C. albicans* are viable but produce more robust biofilms than wild-type cells in both in vitro and in vivo conditions. Indeed, a comparative transcriptome analysis of planktonic and biofilm cells reveals that the biofilm circuitry is significantly altered in $H3V^{CTG}$ null cells. $H3V^{CTG}$ binds more efficiently to the promoters of many biofilm-related genes in the planktonic cells than during biofilm growth, whereas the binding of the core canonical histone H3 on the corresponding promoters largely remains unchanged. Furthermore, biofilm defects associated with master regulators,

namely, Bcr1, Tec1, and Ndt80, are significantly rescued in cells lacking $H3V^{CTG}$. The occupancy of the transcription factor Bcr1 at its cognate promoter binding sites was found to be enhanced in the absence of $H3V^{CTG}$ in the planktonic form of growth resulting in enhanced transcription of biofilm-specific genes. Further, we demonstrate that co-occurrence of valine and serine at the 31st and 32nd positions in $H3V^{CTG}$, respectively, is essential for its function. Taken together, we show that even in a unicellular organism, differential gene expression patterns are modulated by the relative occupancy of the specific histone H3 type at the chromatin level.

Are ribosomal protein genes buffers of phenotypic plasticity?

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Highly conserved across all the three domains of life, ribosomal proteins (RP), along with the rRNA, constitute the protein-making machinery of the cell and their sequences have slow evolution rates. However, they structurally differ between prokaryotes and eukaryotes and many of their functions cannot be substituted. Recent studies on the RPs provide evidence that their role is not inert in just performing translation of mRNA to protein. RPs allow bias in protein synthesis by varying the composition of the ribosomes itself within or between cells, as observed in several ribosomopathies, gene expression variation in human and mouse tissues, and between normal and cancerous cells1,2. A previous analysis from my laboratory of the growth of a deletion collection of S. cerevisiae showed that deletion strains of RP genes have the highest phenotypic variability in growth in several stress environments compared to growth in standard rich medium3.

Such studies along with the presence of several naturally occurring polymorphisms in genetically diverse *S. cerevisiae* strains makes us ask the following questions: Do these non-synonymous allelic variants in RP genes contribute to changes in growth and help in adapting to changing environments by changing the fitness of a population? To

address these questions, we selected a few pairs of genetically divergent strains in which RP genes have non-synonymous polymorphisms and generated allele-specific populations. These allelic populations are being phenotyped in different environments, and the relative fitness specific to the two alleles will be compared. This result, along with allele-specific genome-wide protein-protein interaction network experiments will provide evidence if ribosomes can act as potential buffers of phenotypic plasticity with a role in adaptation to various selective pressures.

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Understanding the mechanism of heterochromatin organization in *Schizosaccharomyces pombe*

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Heterochromatin in the fission yeast *Schizosaccharomyces pombe* is clustered at the nuclear periphery and interacts with a number of nuclear membrane proteins. However, the significance, and the factors that sequester heterochromatin at the nuclear periphery are not fully known. We have recently identified an inner nuclear membrane protein complex Lem2-Nur1, essential for heterochromatin-mediated gene silencing. We will discuss the mechanistic details of how Lem2-Nur1 complex and other factors that associate with Lem2-Nur1 in regulating heterochromatin mediated gene silencing.

RNA mediated regulation of heterochromatin in *S. pombe*

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In the fission yeast *Schizosaccharomyces pombe* (S. *pombe*), heterochromatin domains are established and maintained by protein complexes that contain numerous RNA binding domains among their components. The fission yeast HP1 protein Swi6 is one such component and contains an unstructured RNA-binding hinge, which is important for the integrity and silencing of heterochromatin. We have used an RNA aptamer that likely binds to the Swi6 hinge with high affinity, as a tool to perturb the natural interactions mediated by this domain. When the hinge is blocked by the aptamer RNA, Swi6 appears to become less restricted to the pericentromeres and is enriched at specific euchromatic loci. This suggests a role for the Swi6 hinge, along with the chromoshadow domain (previously shown) in controlling the spread of heterochromatin in *S. pombe*. Our results highlight the potential of using a synthetic aptamer RNA as a tool to perturb nucleic acid – protein interaction in vivo with the objective of understanding the functional relevance of such an interaction. As an extension of this work, we are exploring the role of the Swi6 hinge in

euchromatin as well as facultative heterochromatin. We are also interested in identifying synthetic or natural RNA regulators of heterochromatin and are setting up a screen for the same in *S. pombe*.

Dynamics of public goods production and cooperation in yeast

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Cooperation is a ubiquitous phenomenon in biology. In populations, individuals cooperating often work towards maximizing the fitness of the population, rather than their individual fitness. This places cooperators at a risk of being invaded by cheaters. We study implications of this invasion by cheaters into a cooperating population by using yeast S. cerevisiae as a model organism. The yeast produces and secretes enzymes to hydrolyze disaccharides, sucrose and melibiose. The resulting monosaccharides are then used by the cell to support growth and energy. In this talk, using concepts from game theory and mathematical modeling, I will discuss how cooperators ensure their stable existance, and prevent being eliminated by cheater cells.

T3/P17

Atg11, an autophagy regulator, is required for high fidelity chromosome segregation

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Autophagy is an evolutionarily conserved process that maintains the turnover of cytosolic components. Recently autophagy related (Atg) proteins are shown to play unconventional roles including maintenance of genomic stability. Using data mining, we find several Atg proteins physically or genetically interact with proteins of chromosome segregation machinery in *Saccharomyces cerevisiae*. In this work, we sought to determine the role of Atg proteins in chromosome segregation in *S. cerevisiae*. By screening a large collection of *atg* mutants, we identified *atg11* to be the most sensitive to thiabendazole. *atg11* displayed increased minichromosome loss, and a delay at G2/M stage in the

presence of a microtubule poison thiabendazole. Importantly, loss of Atg11 protein function resulted in shorter spindles in large-budded cells suggesting its possible role in regulating spindle dynamics. By analyzing a series of double deletion mutants of *atg11* and motor proteins, we report previously unknown genetic interactions between Atg11 and Kar3. We show that *atg11 kar3* displays slow growth and increased percentage of mitotically arrested large-budded cells having shorter spindle (1-4 µm) at 37°C. Thus, we report a previously unknown moonlighting function of Atg11 together with Kar3 in regulating the spindle length dynamics in *S. cerevisiae* during vegetative growth under nutrient-rich conditions.

Post-transcriptional regulation of gene expression in *Saccharomyces cerevisiae* involving modulation of nuclear export and nuclear mRNA degradation

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In *Saccharomyces cerevisiae*, a special class of mRNAs representing a subset of otherwise normal transcripts display very slow export and an unusually long intra-nuclear dwell time. This prolonged nuclear retention leads to their rapid degradation in the nucleus by the nuclear exosome and DRN (**D**ecay of **R**NA in the **N**ucleus) apparatus. We previously attributed their slow export to one or more hypothetical *cis*-acting, export-retarding element(s). Here, we identified such a *cis*-element (hereafter referred to as "nuclear zip code") in *SKS1* mRNA, a representative of this class of transcripts. Deletion analysis of *SKS1* mRNA identified a 202-nt RNA segment within the*SKS1* ORF, which harbors the nuclear zip code. Removal of this segment (i) abolished slow export of the transcripts, as revealed by *in situ* confocal microscopy–based localization experiments, and (ii) abrogated the susceptibility of the transcripts to degradation by the nuclear exosome/DRN.

Remarkably, fusing the *SKS1* mRNA 202-nt nuclear zip code to the 5′-segment of *CYC1* mRNA resulted in inefficient export, and susceptibility of the chimeric transcript to the nuclear exosome/DRN. These findings identify a *cis*-acting zip code element that is necessary and sufficient to impede nuclear export and results in its preferential nuclear retention, thereby impacting its abundance and cellular repertoire. We conclude that this element posttranscriptionally regulates *SKS1* gene expression levels.

The ubiquitin-fold-activated Sde2 helps spliceosome capture distant exons

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Intron-containing gene expression in eukaryotes proceeds through the process of RNA splicing to generate protein-coding messenger RNAs (mRNAs). The spliceosome removes non-coding introns from pre-mRNAs and joins exons. Spliceosomes must ensure accurate and timely excision of highly diverse introns. Sde2 is a ubiquitin fold containing splicing regulator required for splicing of selected pre-mRNAs in an intron-specific manner in *Schizosaccharomyces pombe*. *S. pombe* and human Sde2 is translated as an inactive precursor harbouring the ubiquitin fold, an invariant GGKGG motif, and a C-terminal domain (referred to as Sde2-C). The precursor gets processed at GG~K by the ubiquitin specific proteases Ubp5 and Ubp15 to separate the two polypeptides. Activated Sde2-C with N-terminal lysine gets incorporated into the spliceosome where is functions with Cactin/Cay1 and Tls1. These regulators help spliceosome excise introns with branchpoint-distant 3'-splicesite. This class of intron-specific splicing is required for optimal gene expression and alternative splicing of heterochromatin factors.

Role of RGG-motif proteins in translation control

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As a group we are focused on understanding how does a cell decide between the choices of storing an mRNA, degrading it and using it to make a polypeptide? It is believed that RNA binding proteins that associate with mRNA determine which of the above fates a certain mRNA will undergo. One such class of RNA-binding proteins that excite us contains RGG-motifs (repeats of arginine-glycine-glycine). A subset of these proteins regulates translation by targeting eIF4G1, a conserved translation repressor. The RGG-motif can be sites of arginine methylation and RNA-binding proteins are the largest class of arginine-methylated proteins. Over the past six years we have tried to address the following questions in our laboratory: 1) What is the role of arginine methylation in mRNA fate decisions? 2) How is mRNA specificity determined during translation control? 3) Is cytoplasmic mRNA fate connected to endocytosis/protein sorting?Results pertaining to some of above questions will be discussed at the meeting.

Functional and structural insights into the *Schizosaccharomyces pombe* ELL-EAF transcription elongation factor complex

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RNA polymerase II mediated expression of protein-coding genes is regulated at the level of both initiation and elongation of transcription. There has recently been an exponential increase in the number of genes whose expression is regulated at the elongation stage of transcription, and different transcription elongation factors have been shown to play a critical role in this process. ELL-EAF family of transcription elongation factors have been identified across organisms, and their role in *Schizosaccharomyces pombe* is only beginning to be understood. We have employed a variety of genetic, molecular, biochemical and biophysical approaches to elucidate their role(s) in *S. pombe*. Our work has demonstrated that these proteins regulate different phenotypes in *S. pombe*. Genome-wide transcriptomic analysis shows that these proteins affect the expression of only specific subsets of genes. Deletion mapping experiments have identified the domains of these proteins critical for their in vivo functions. Computational and biophysical techniques revealed that both ELL and EAF

have disordered regions. Our recent multicopy suppressor screen has also identified different suppressors of the *ell* deletion phenotypes. Taken together, our results provide insights into the functions of the ELL-EAF proteins, and the molecular mechanism underlying these function(s) in *S. pombe*.

DEAD-box RNA helicases: A family affair in governing translational control of eukaryotic gene expression

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Translational control is a critical step of regulating gene expression and determines cellular fate. Initiation is the most regulated and rate-limiting stage of translation that dictates both the quality and quantity of proteins being synthesized in cells. In eukaryotic translation initiation, the 43S pre-initiation complex (PIC, comprised of the 40S ribosomal subunit, eukaryotic initiation factors (eIFs) and methionyl initiator tRNA) typically attaches to the 5'end of mRNA and scans the 5' untranslated region (UTR) for the start codon. RNA structures in 5'UTRs of mRNAs can reduce initiation efficiency by impending 43S PIC attachment or subsequent scanning. DEAD-box RNA helicases eIF4A, Ded1 and Dbp1 are believed to promote translation initiation by resolving such 5'UTR structures, but the underlying mechanisms and their exact roles remain unclear. Using classical molecular genetics tools along with a recently developed high-throughput sequencing technique of ribosome profiling, we have shown the division of labor between eIF4A and Ded1 in regulating translation at genome-wide level. We also showed that Ded1 is critically required for scanning through structured 5' leaders of mRNAs, while eIF4A assists Ded1 in this step; it also promotes a step common to virtually all yeast mRNAs. Furthermore, our work also identified eIF4A-independent role for eIF4B in addition to its function as eIF4A cofactor in promoting

translation of structured mRNAs. Strikingly, we discovered that Ded1-paralog Dbp1 can substitute for Ded1's function in stimulating translation of mRNAs with long, structure-prone 5'UTRs. For many such mRNAs, Dbp1 masks the involvement of Ded1, and Dbp1 overexpression mitigates loss of Ded1 function, in stimulating translation. Using Translation Complex Profile sequencing (TCP-seq) we provided first direct evidence that Ded1& Dbp1 functionally cooperates in promoting both PIC attachment and scanning to stimulate translation of mRNAs *in vivo*. Taken together, these recent findings provide new insights into the interplay of RNA helicases and their co-factors in determining cellular protein synthesis.

ABC superfamily transporters of an emerging pathogenic yeast *Candida auris*

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Considering the recent outbreaks of drug resistant *Candida auris* infections in hospitals around the globe, this study has analyzed the entire ATP Binding Cassette (ABC) superfamily members. We subjected *C. auris* CBS 109131T to WGS and after annotation, examined the landscape of ABC proteins. Our analysis identified 28 putative ABC proteins encoded by *C. auris*. On the basis of phylogenetic analysis, domain organization, these proteins were categorized into six subfamilies. Among these, only 20 ABC proteins contained transmembrane domains and were grouped as membrane proteins. qRT-PCR results confirmed the presence of all 20 ABC proteins encoding genes as true ORFs. Further, the change in transcript levels after short-term exposure of drugs and in drug resistant *C. auris* clinical isolates underscored their relevance in drug resistance. Coinciding with the well-established role of *CDR1* as a major multidrug exporter in other yeasts, a homologue of *CDR1*, showed consistently higher expression in MDR clinical strains of *C. auris*. Together, our analysis presents the first comprehensive landscape of ABC

superfamily proteins of an important human fungal pathogen *C. auris*, which underline their role in MDR and provides an important platform for in depth analysis of their physiological relevance.

A novel morphogenetic signal in fungal pathogenesis

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Jasmonic acid is a well-known plant hormone that participates in many aspects of development, especially in the immune response against necrotrophic fungal pathogens. Interestingly, several fungi have been shown to produce mimics of jasmonic acid, but the function(s) of such fungal oxylipin derivatives has remained elusive. Recently, we showed that the rice-blast fungus, *Magnaporthe oryzae,* produces jasmonic acid, and utilizes a novel Antibiotic Biosynthesis Monooxygenase (ABM), to catalyze such intrinsic jasmonate into 12OH-JA to suppress the host defense response during invasive growth. Loss of ABM and 12OH-JA in *M. oryzae* leads to complete loss of pathogenicity. This breakthrough finding provided a novel insight into the role of jasmonate mimics in fungi. Here, we explored the function(s) of intrinsic jasmonic acid and its derivate(s) in the growth and development of *M. oryzae*. *OPR1* encodes a 12-Oxo-phytodienoic Acid Reductase essential for the formation of an intermediate product of OPDA during biogenesis of jasmonic acid. We found that loss of *OPR1* leads to strong defects in appressorium formation and pathogenesis in *M. oryzae*. Such defects were, surprisingly, reminiscent of phenotypes observed in mutants (eg *pth11*Δ and *cpka*Δ) that are compromised for cyclic AMP signaling. Genetic complementation with WT *OPR1* completely restored the pathogenesis in *opr1*Δ. Liquid chromatography-mass spectrometry helped detect and quantitate the levels of jasmonates (OPDA, JA, 12OH-JA) produced by *M. oryzae*

during infection-related development. Interestingly, exogenous JA restored the appressorium formation in the *pth11*Δ. Based on mutant analyses, and quantification of jasmonate levels during the pathogenicity cycle, we propose that JA signaling participates in the infection-related morphogenesis via a regulatory interaction with the cyclic AMP signaling cascade in *M. oryzae.* Preliminary results implicate the late endosomal compartments as scaffolds for such signaling across the fungus-plant interface.

N-degron profiling: determinants of protein stability encoded in the N-terminus of proteins

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Most eukaryotic proteins are N-terminally acetylated. This modification can be part of a degradation signals (degrons) that may encompass additional amino acid residues near the N-terminus of proteins. To investigate the occurrence and specificity of such degrons we developed N-degron profiling for yeast. We performed a comprehensive survey of degrons in the yeast N-terminome and their dependence on different degradation pathways. We find that approximately 26% of N-termini encode cryptic degrons, which exhibit high overall hydrophobicity and are frequently recognized by the E3 ubiquitin ligase Doa10. In contrast, N-terminal acetylation is rarely recognized as a degron. This screen furthermore identified several pathways where N-terminal acetylation has the opposite role and blocks degradation. In these pathways conserved N-acetyltransferases prevent N-terminal processing that would otherwise lead to protein degradation by an N-end rule pathway. We furthermore identify a master regulator of ubiquitin dependent degradation as being regulated by N-acetylation, with implications in the overall activity of the

ubiquitin proteasome system. Our analysis highlights the complex role of N-terminal acetylation on protein stability and argues that hydrophobicity of the N-terminus, not N-terminal acetylation, is a key degron determinant in nascent proteins. Our results furthermore point to a novel previously unanticipated role of the major protein modification.

Ydj1 regulates functional distinction of Ssa Hsp70s in Hsp90 chaperoning pathway

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Chaperones are integral part of cellular quality control system, that interact with partially unfolded proteins, promote their folding and if folding fails, target them for degradation. Each cell contains multiple chaperone families that are networked together to protect cells from various stresses. Though they work in collaboration with each other, different family members possess specificity for their substrate and thus function distinctly. Hsp70 and Hsp90 are two of the major cellular chaperones. Hsp70 interacts with variety of cellular proteins however Hsp90 substrates primarily belongs to kinases, transcription factors and growth hormone receptors. Hsp70 plays a major role in many of Hsp90 functions by interacting and modulating conformation of its substrates before being transferred to Hsp90s for final maturation. The basis of Hsp70 requirement in Hsp90 function is still not clear. In the present study, using v-Src as an Hsp90 substrate, we have examined the role of each of the four yeast cytosolic Ssa Hsp70 in regulating Hsp90 functions. We show that strains expressing

stress inducible Ssa3 or Ssa4, and not constitutively expressed Ssa1 or Ssa2, as sole Ssa Hsp70 isoform reduces v-Src mediated growth defect. Further study has revealed the mechanism of this functional specificity among Hsp70 isoforms in Hsp90 pathway. The study reveals a novel role of Hsp70 co-chaperone Ydj1 in determining the functional distinction among Hsp70 isoforms with regard to Hsp90 chaperoning action.

Engineering ubiquitin to probe and rewire protein degradation network

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Ubiquitin is a highly conserved protein in eukaryotic organisms exhibiting distinct conformations and linkages that mediate diverse functional roles including protein degradation and signaling. Ubiquitination occurs through an enzymatic cascade involving ubiquitin activating enzyme (E1), ubiquitin conjugating enzyme (E2) and ubiquitin ligase (E3). Proteins that play key roles in a number of human diseases such as cancer and neurodegenerative diseases, including huntingtin, ataxin-1, tau, α-synuclein, and superoxide dismutase 1 (SOD1), are targets of ubiquitin modification and often mis-regulation of the ubiquitination process is central to these diseases. Despite the central role of ubiquitin for the functioning of a cell, it is still not resolved as to how ubiquitination regulates such a wide variety of proteins and biological processes. *S. cerevisiae* ubiquitin protein has >90% sequence identity to mammalian ubiquitin, highlighting its function in conserved core cellular processes.

We attempt to dissect the known and new distinct biological processes affected by ubiquitin at the level of individual

interactions and determine the *in vivo* role of ubiquitin at a new level of structural and functional resolution. Our approach allows us to ask whether or not quantitative parameters defining ubiquitin interactions correlate with their effect on biological processes. Open questions that we attempt to answer include: Are there different classes of processes mediated by ubiquitin that can be distinguished by genetic analysis, and do they reveal distinction between various ubiquitin functions? How do the various ubiquitin mutations map to the different biological processes ubiquitin is implicated in?

The expanding world of J-domain proteins

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J-domain proteins (JDPs), originally referred to as Hsp40s, are one of the most versatile and ubiquitous group of molecular chaperones. They are obligate co-chaperones of Hsp70s. Often multiple JDPs work with a single Hsp70 to perform a myriad of cellular functions. The numbers of JDP encoding genes have significantly increased in more complex eukaryotes, suggesting towards emergence of highly complex Hsp70-JDP networks. However, studies have shown that the observed functional diversity of JDP functions in more complex eukaryotes cannot be explained by their increased number alone. Instead, regulatory differences, sub-functionalization, neo-functionalization as well as combinatorial inter and intra-class interactions between JDPs and Hsp70s is further expanding the Hsp70-JDP networks in more complex eukaryotes.

ST4/P50

Hsp110 mediated proteasomal degradation of Hsp70 chaperone associated substrates in yeast

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Proteins are essential bio-molecules that exist in all living cells and are critical to all physiological processes. However, many cellular events such as molecular crowding, translational errors, thermal stress, and oxidative damage subject the newly synthesized and the natively folded proteins to off-pathway reactions, which produce misfolded proteins and create proteotoxicity. Cells are equipped with several classes of regulatory components to buffer proteotoxic stress produced by misfolded protein species. The major regulatory components include, molecular chaperones, the ubiquitin proteasome system (UPS), and the autophagy system. Molecular chaperones and UPS plays essential role in the selective degradation pathways including both native and misfolded cellular proteins. Proteins belonging to Hsp70 chaperone and Hsp110 families (Sse1 and Sse2 proteins in yeast Saccharomyces cerevisiae) functions in the UPS mediated protein quality control pathway. However, the molecular link between Hsp70-Hsp110 chaperone complex and UPS is a poorly understood mechanism. In the current study we report that Hsp110 protein function in the turnover of aggregation prone proteins involving both ubiquitin dependent and –independent proteasome substrates. Hsp110 is essential to keep Hsp70 associated substrates soluble and

interacts with 19S regulatory particle of the proteasome, suggesting coordinated recruitment of Hsp70-substrate complexes to 26S proteasome for proteolysis. By using a highly defined ubiquitin independent proteasome substrate, we found that introduction of a single Hsp70 binding site render its degradation dependent on Hsp110. The findings define how cellular proteins use Hsp110 to get to the proteasome and to get rid of Hsp70 chaperones that sterically block degradation with profound implication for understanding cellular protein quality control and stress management.

ST5/P48

Relevance of 7-transmembrane receptor protein Rta2 in coordinating endoplasmic reticulum stress responses in *Candida albicans*

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C. albicans genome has three Rta1-like genes– *RTA2*, *RTA3* and *RTA4*. This fungal Rta1-like family of proteins may function as novel potential antifungal targets as they are unique to the fungal kingdom and do not have a murine or human homolog. Both Rta2 and Rta4, the downstream effector molecules of calcineurin pathway, are upregulated upon tunicamycin (TM) exposure. Rta2 is requisite to cope with tunicamycin induced ER stress in a Hac1-independent manner. Additionally, it also helps the cells to regain ER homeostasis after ER stress by attenuating the unfolded protein response. Transcriptional profiling of *rta2*Δ/Δ cells revealed genes enriched for the gene ontology (GO) processes related to biofilm formation, ribosomal biogenesis, cell wall and mitochondrial function. Consistent with the differential regulation of biofilm associated genes in the transcriptional profiling data, *rta2*Δ/Δ showed in vivo biofilm

defect. Sequence homology of these Rta proteins revealed the presence of signature sequence, evolutionarily conserved in the extracellular loop of these 7-transmembrane receptor proteins and the least conserved cytosolic C-terminal region. We show that the signature sequence of Rta2 do have a key role in providing tolerance to tunicamycin and promotes the cells to regain ER homeostasis, thus serving as a potential target for the antifungal drug development. This study for the first time shows the relevance of a 7-transmembrane receptor protein Rta2 during ER stress in *C. albicans*, with implications in future antifungal therapy.

Interplay and relative positioning of functional sites on Endoplasmic Reticulum

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COPI vesicles mediate Golgi-to-ER recycling, but COPI vesicle arrival sites at the ER have been poorly defined. We explored this issue using the yeast *Pichia pastoris*. ER arrival sites (ERAS) can be visualized by labeling COPI vesicle tethers such as Tip20. Our results indicate that ring-shaped ERAS surround COPII-labeled ER export sites (ERES). The dynamics of ERES and ERAS are nearly identical, indicating that these structures are tightly coupled. Displacement or degradation of Tip20 does not alter ERES organization, whereas displacement or degradation of either COPII or COPI components disrupts ERAS organization. We infer that Golgi compartments form at ERES and then bud COPI vesicles to create ERAS. As a result, ERES and ERAS are functionally linked to create bidirectional transport portals at the ER-Golgi interface. COPI vesicles likely become tethered while they bud, thereby facilitating efficient retrograde transport. In mammalian cells, the Tip20 homolog RINT1 associates with ERES, indicating possible conservation of the link between ERES and ERAS.

Inter-organelle communications occur at least by two known mechanisms – by vesicular trafficking and via direct membrane contact sites formed between adjacent organelles. The potential interplay between these two modes of communications is poorly understood. Endoplasmic reticulum (ER) participate both in vesicular trafficking as well as via direct contact-mediated transport. 'ER-Mitochondria Encounter Structures (ERMES) are membrane contact junction formed between ER and mitochondria and plays a significant

role in lipid transport and calcium exchange and homeostasis. On the other hand, early secretory pathway originates at ER exits sites (ERES). We wanted to investigate potential structural or functional correlation between ERES and ERMES. For this purpose, we have used budding yeast *Pichia pastoris* which have distinct and well-studied ERES; however, not much is known about the existence of ERMES and their role in *Pichia pastoris*. Using fluorescently labeled Pichia homologs of known ERMES resident proteins, we have established that ERMES sites do exist in *Pichia pastoris*. Surprisingly, when ERMES resident proteins are fluorescently labeled and co-expressed along with ERES markers, a juxtaposition localization between the two types of sites are often observed. Disruption of ERES cause disruption of ERMES, alter mitochondrial morphology, and de-couple ERMES-ERES proximity. Functional significance of such ERMES-ERES proximity needs to be further explored. Our preliminary data suggests such ERMES-ERES proximity may be beneficial for mitochondrial protein import.

Yeasts can still teach a thing or two about autophagy

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One of the hallmarks of (macro) autophagy is the *de novo* formation of double-membrane vesicles known as autophagosomes. Autophagosome biogenesis involves membrane contribution to several organelles including ER, Golgi and endosomes. While trafficking of Atg9 vesicles to and fro from the membrane sources to the budding autophagosomes is known to be critical for autophagosome formation, the mechanisms involved in this process remain elusive. By screening a subset of essential genes (temperature sensitive mutants), we identified several genes that are important for autophagy flux. I will talk about two sets of genes that seemingly work at the early steps of autophagosome biogenesis and are involved in the trafficking of Atg9 vesicles.

Structural and functional analysis of disease causing mutations of Drp1 through analyses of the yeast homologue Dnm1

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Evolutionarily conserved GTPase family member, dynamin-related protein 1 (Drp1) together with it's accessory proteins forms a complex regulatory network that is essential to maintain the dynamic behaviour of organelles like mitochondria and peroxisomes. Mutations in Drp1 that contribute to the pathophysiology of several human diseases are reported in the literature. However, the molecular details related to these diseases are unknown. In this study, we used the budding yeast homologue Dnm1 as a model to gain insights into the mechanistic details caused by specific Drp1 related mutations associated with diseases like infantile Parkinson's, childhood epileptic encephalopathy, refractory epilepsy, and horizontal nystagmus. Orthologous disease mutations of Dnm1 were characterized for organelle morphology, intracellular localization, and dynamics of the protein. Furthermore, the effect of the mutations on the structure of the protein using molecular dynamic simulations provided insight into the structure-function alterations caused by the mutations.

Mitochondrial gene expression in response to carbon source: control by a yeast clade specific putative helicase *IRC3* in *Saccharomyces cerevisiae*

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Alteration of OXPHOS activity as well as mitochondrial proteome heterogeneity in response to carbon source is well documented. However, numerous questions regarding its establishment at the level of mitochondrial gene expression remain. We have shown that cells deleted for *IRC3,* a DEAD/H box protein, rapidly lose respiration ability when cultured in glucose or galactose. Consistent with a role in mitochondrial translation, glucose grown *Δirc3ρ*$^{+}$ cells and *irct3ts* cells at restrictive temperature have reduced rates of translation, without any consequence on transcriptor assembled mitochondrial ribosomal subunit levels. Interestingly, when *Δirc3ρ*$^{+}$ and *irct3ts* cells were grown in galactose reduction in mitochondrial translation was observed in *Δirc3ρ*$^{+}$ but not in *irc3ts* cells at the restrictive temperature. These observations indicate Irc3p to be a mitochondrial translation regulator, albeit with a different mechanism under conditions that maintain mitochondrial function at a basal level versus those that have higher mitochondrial function. Consistent with these findings

we find that Irc3p associates with mitochondrial ribosome to different extents in cells at log phase vs post diauxic, without significant changes in protein levels. These findings together indicate that *IRC3* to be a central player in establishment and maintenance of mitochondrial proteome heterogeneity in response to carbon source.

ST6/P12

Nucleolar size regulates nuclear envelope shape in *Saccahromyces cerevisiae*

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Nuclear shape and size is cell type-specific. Change in nuclear shape is seen during cell division, development, and pathology. The nucleus of *S. cerevisiae* is spherical in interphase and becomes dumb-bell shaped during mitotic division to facilitate the transfer of one nucleus to the daughter cell. As yeast cells undergo closed mitosis, the nuclear envelope remains intact throughout the cell cycle. The pathways that regulate nuclear shape are not well characterized. The nucleus is organized into various sub-compartments, with the nucleolus being the most prominent. We have initiated a focused genetic screen for nuclear shape defects in *S. cerevisiae* to ask if the nucleolus influences the nuclear shape. We find that increasing nucleolar volume triggers non-isometric nuclear envelope expansion resulting in an abnormal nuclear envelope shape. We further show that the tethering of rDNA to the nuclear envelope is required for the appearance of these extensions.

ST7/P42

Role of eisosome proteins, Pil1 and Lsp1, mitophagy and cell death in *Saccharomyces cerevisiae*

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Plasma membrane of yeast requires high degree of organization as it carries out the diverse array of functions apart from forming the protective barrier around the cell. Eisosomes are a complex of cytoplasmic proteins which mark the sites of endocytosis at the plasma membrane. In addition, they are also known to regulate the cellular levels of phosphatidylinositol(4,5)bisphosphate[PI(4,5)P2], sphingolipid homeostatsis, and maintain membrane reservoirs for plasma membrane expansion. Pil1 and Lsp1 are the major components of eisosomes. Though eisosomes are static structures, recent study has shown that Pil1 appears to come on and off these structures. Our experiments show that Pil1 and Lsp1 localize to mitochondria. Also, we demonstrate that Pil1 overexpression induces cell death which can be rescued by human anti-apoptotic protein bcl-xl. Interestingly, we find that Pil1 is also required for autophagy as well as mitophagy. Hence, overall study shows that Pil1 plays various non-canonical functions in cell apart from its function as eisosome component.

Meiosis-specific functions of kinesin motors in maintenance of chromosome integrity

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Faithful chromosome segregation relies on assembly of the kinetochore complex that acts as a bridge between the centromere and the spindle microtubule. Kinesin motors provide forces at the kinetochore-microtubule interface for capturing/orientation/alignment of the sister chromatids (or homologs in meiosis) in prophase/metaphase and for poleward movement of the chromosomes during anaphase. Despite elucidation of the functions of kinesins in mitosis, their roles in meiosis are poorly explored. Given the temporal and mechanistic differences between mitosis and meiosis in spindle assembly/disassembly, kinetochore maturation and sister chromatid alignment and disjunction, we hypothesize that the kinesin motors might have some functions in meiosis not observed in mitosis. Interestingly, we found, unlike mitosis, assembly of mature kinetochore depends on the Cin8 (kinesin 5) motor in meiosis. We further observed that in contrast to mitosis, Cin8 (kinesin 5) and Kip3 (kinesin 8) together are indispensable in meiosis. Unexpectedly, examining the meiosis

in *cin8*Δ *kip3*Δ cells, we detected chromosome breakage in the meiosis II cells. To address the reason for the breakage, we found that the double mutant exhibits delay in the cohesin removal and spindle elongation during anaphase I. Consequently, some cells abrogate meiosis II and form dyads while some, as they progress through meiosis II, cause defect in chromosome integrity. We believe that in the latter cells, an imbalance of spindle mediated force and simultaneous persistent cohesin on the chromosomes cause their breakage. We provide evidence that tension generated by Cin8 and Kip3 through microtubule cross-linking is essential for signaling efficient cohesin removal and maintenance of chromosome integrity during meiosis.

Acto-myosin ring and the art of fission

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Cytokinesis in many eukaryotes involves the contraction of a tension producing actomyosin-based contractile ring that constricts the membrane to divide the cell. However, the detailed mechanism of contractile ring organization and contraction is not fully understood. A sliding filament mechanism where myosin pulls actin filaments like those in the sarcomere has been proposed decades ago, however, contractile rings are far more disordered and the components of the ring are highly dynamic. We have recently established, for the first time, an experimental system to study contraction of the ring to completion in vitro. We show that contractile rings undergo rapid contraction in an adenosine triphosphate (ATP) and myosin-II dependent manner in the absence of other cytoplasmic constituents. Surprisingly, neither actin polymerization nor its disassembly is required for contraction of the ring although addition of exogenous actin cross-linking proteins blocks ring contraction. Furthermore, we use cryosectioning and cryofocused ion beam milling to gain access to natively-preserved actomyosin rings for direct three-

dimensional imaging by electron cryotomography. Our results show that the ring is composed of straight, overlapping actin filaments that “saddle” the septal membrane, but they do not make contact with the membrane or gather at nodes. Correlative cryo-fluorescence light microscopy and electron cryotomography on vitreous cryosections further reveal that myosin does not form thick oligomeric filaments in the ring as it does in vitro. Finally, by tuning parameters and properties of the ring’s components to match data from electron cryotomography, we show that coarse-grained simulations could help reveal the mechanism of ring constriction.

Chiral proofreading and its implications for the evolution of eukaryotes

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D-aminoacyl-tRNA deacylase (DTD) removes D-amino acids mischarged on tRNA, thus reinforcing the 'Chiral Checkpoints' in the cell and therefore is implicated in enforcing homochirality in proteins. We elucidated the 'Chiral Proofreading' mechanism of DTD by which D-amino acids are prevented from infiltrating the translational machinery. We further showed that DTD's mechanistic design principle is based only on L-amino acid rejection and hence, besides acting on D-aminoacyl-tRNA, it also hydrolyses achiral glycine attached to tRNAs. The work thus led us to unfold a key tRNA element, called the 'Discriminator code', responsible for protecting Gly-tRNA (Gly) from DTD thus enabling protein synthesis in bacteria. However, this important discriminator element is switched in the case of opisthokonts, including yeast. Our recent experimental data using yeast as a model system suggests that such a switch is essential for the evolution of opisthokonts. The mechanistic, functional and evolutionary implications of the 'Chiral Proofreading' system beyond the 'Discriminator code' and in the emergence of eukaryotes will be discussed.

ST8/P16

Scd6 and Psp2 are multicopy suppressors of clathrin deficiency

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Clathrin, one of the major vesicular coat proteins, plays a major role in endocytosis, sorting, and retention of proteins at the trans-Golgi network. Upon deletion of Chc1 (Clathrin Heavy Chain), *Saccharomyces cerevisiae* can either be viable or inviable, depending upon the genetic background.

Scd's (suppressors of clathrin deficiency) are the plasmids that were identified to rescue the growth defect associated with Chc1 depletion in *scd*1-i state. Scd6 is one of the multi-gene suppressors identified, having a total of 8 genes. We report here that YPR129W, one of the genes on that construct is necessary and sufficient to suppress the growth defect of Chc1 depleted cells. We have also identified another novel suppressor, Psp2 (Polymerase Suppressor), which upon overexpression can also rescue the growth defect of Chc1 depleted cells.

Scd6 and Psp2 both are known to localize to RNA granules, and both have C-terminal RGG motif. Interestingly, for both

Scd6 and Psp2, RGG motif was found to be the most important domain for the suppression activity. Here, we hypothesize that these proteins might be working by reducing the protein load upon vesicular trafficking pathway somehow to balance the effect of CHC1 depletion leading to growth rescue.

ST9/P40

Metabolic constraints determine the self-organization of specialized, heterogeneous cell groups

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How phenotypically distinct states in clonal cell populations emerge and stably co-exist is an open question. We find that within a mature, clonal yeast colony growing in glucose limited conditions, cells organize into phenotypically distinct groups exhibiting opposite metabolic states. Beginning in a uniformly gluconeogenic state, cells exhibiting a contrary, high pentose phosphate pathway (PPP) activity state, spontaneously appear and proliferate, in a spatially constrained manner. Gluconeogenic cells in the colony produce and provide a resource, which we identify as trehalose. Above threshold concentrations of trehalose, cells switch to the new metabolic state and proliferate. This creates a self-organized system, where cells in this new state are sustained by trehalose consumption. In ongoing studies, we investigate how such a

state-controlling resource is itself modulated. We find that metabolite plasticity of an amino acid, aspartate, is a key determinant. Aspartate acts as a carbon substrate for trehalose production in gluconeogenic cells. Contrastingly, aspartate is differentially utilized by cells that show high PPP activity, as nitrogen donor for nucleotide synthesis. This metabolite plasticity enables the cell community as a whole to bet-hedge, with distinct advantages of each phenotypic state, allowing overall survival and growth of the colony

Mechanistic insights into meiotic chromosome synapsis and recombination in *Saccharomyces cerevisiae*

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Meiosis is a specialized form of cell division during which the diploid cells undergo a single round of DNA replication followed by two rounds of chromosome segregation to produce haploid gametes. At the center of this process is the synaptonemal complex (SC), a dense, evolutionarily conserved, proteinaceous superstructure that functions as a "molecular zipper" to juxtapose homologous chromosomes, one from each parent, to pair up and exchange genetic material by crossing over. A growing body of evidence suggests that defects in the SC leads to infertility, recurrent miscarriages and chromosome aneuploids such as the Down's syndrome, in addition to germline cancers. How the components of SC exert their function has been hotly debated from multiple perspectives. Our research has focused on delineating the structure-function relationships of *S. cerevisiae* SC proteins, the functional relationships among them, and the underlying molecular mechanism(s) by which these proteins may orchestrate meiotic chromosome synapsis and recombination. We have employed

an ensemble of molecular biological and single molecular approaches to characterize the activities of Hop1 and Red1, the two meiosis-specific proteins of the SC axial element of *S. cerevisiae*. I will present our results on how these proteins may contribute to chromosome synapsis and discuss their implications for meiosis.

Role of CaPolη in genome stability, morphogenesis and the development of systemic candidiasis

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DNA polymerase eta (Rad30/Polη), a unique translesion DNA synthesis (TLS) polymerase, promotes efficient bypass of UV-induced cyclopyrimidine dimers and cisplatin adducts. Recently, we have characterized this polymerase from pathogenic yeast *C. albicans* and showed that CaPolη plays a vital role in protecting *Candida* genome from diverse array of DNA damaging agents, not limited to UV and cisplatin. Polη deficient strain did not exhibit any hyphal development in the presence of UV and cisplatin while the wild type strain profusely developed DNA damage induced filamentation. Interestingly, serum that does not inflict any DNA damage also induces hyphal growth in *C. albicans* but requires a functionally active Polη. Genetic and biochemical studies in both yeast and humans have indicated that proliferating cell nuclear antigen (PCNA) plays a vital role in the Pol exchange process. Polη gains access to the replication fork by physically and functionally interacting with PCNA, which is mediated by highly conserved PCNA-Interaction-protein (pip) motif(s) present mostly in the non-catalytic region of pols. Interestingly, CaPolη does not harbor any pip motif. Our mutational analyses suggest that a class of Polηs those intrinsically lack pip motif, a conserved ubiquitin binding motif present in these pols interact with PCNA and become

indispensable for TLS activity. Since non-filamentous *C. albicans* is widely accepted as avirulent form, further we explored the virulence and pathogenicity of a *rad30Δ* strain in animal systems. Despite the morphological differences, both wild type and *rad30Δ C. albicans* were virulent with a varying degree of pathogenicity in mice models. Notably, mice with Th1-immunity were comparatively less susceptible to systemic fungal infection than Th2-type. Thus, our study clearly suggests that the modes of interaction of morphologically different *C. albicans* strains with the host immune cells are diverged, and host genetic background and several other attributing factors of the fungus could additionally determine their virulence.

The Tac1-dependent coordinated regulation of genes contribute to pathogenicity-traits in *Candida albicans*

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In *Candida albicans*, up regulation of genes encoding the Tac1-regulated ABC drug transporters (*CDR1* and *CDR2*) is a predominant cause for the development of antifungal resistance. Hyperactive Tac1 causes simultaneous induction of *CDR1* and *CDR2* along with *RTA3*, *IFU5* and *HSP12* in azole-resistant isolates. The significance of these co-regulated genes remain largely unexplored in *C. albicans*. Herein, we describe the role of Ifu5 (WW domain-containing protein) in coordinating normoxic and hypoxic responses by allying with the hypoxic regulator Efg1 in *C. albicans*. This alliance allows the regulation of filamentation and biofilm formation under hypoxia, thus facilitating the survival of this pathogenic fungus in oxygen-limiting environments in the mammalian host. Rta3, a 7-transmembrane receptor protein regulates the asymmetric distribution of phosphatidylcholine across the plasma membrane and biofilm formation in vivo. We propose that the purpose of co-regulation of Rta3 and Ifu5 with drug transporters may be to enable *C. albicans* to efficiently switch to the drug-

resistant biofilm mode of growth under varied oxygen conditions; empowering this fungus to resist the onslaught of antifungals in planktonic and biofilm modes of growth in the host environment. Thus the relationship between Rta3, Ifu5 and biofilm formation can be exploited for antifungal therapy.

Analyzing host responses to target *Fusarium graminearum* infection in crop plants

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The ascomycetous fungal member, *Fusarium graminearum* is a devastative pathogen of cereal crops which include wheat, barley, maize, oat, rye and triticale. The pathogen infects spikes at anthesis causing Fusarium head blight/ scab disease in infected plants and in maize it causes 'stalk or ear rot' disease. The pathogen is able to infect roots and crown region and in combination with other soil borne pathogens causes seedling blight disease also. *F. graminearum* is a hemibiotroph, capable of deriving nourishment from living plant cells initially as a biotroph and at later stages. resumes necrotrophic mode of living, by killing and drawing nutrients from dead tissues. Moreover, it is a facultative parasite, naturally existing as a saprophyte but can live as a parasite on plants. The ubiquitous nature of the pathogen ensures inoculum throughout crop growth period thereby making it difficult to combat the disease. Further, grains contaminated with mycotoxins like deoxy-

nivelanol or nivalenol are considered unfit for consumption as food, fodder or feed, since mycotoxins have been reported as potential carcinogenic agents. Considering the significance of the pathogen, it necessitates analyzing host responses to understand disease signaling of the pathogen in order to confer resistance and the findings are presented.

Novel strategies to control fungal diseases of rice

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Many filamentous fungi cause important plant diseases. One of them, namely *Rhizoctonia solani* causes devastating sheath blight disease in rice and many other plants. There is no source of complete disease resistance and the disease is mostly managed by use of fungicides. The pathogen has evolved resistance against commonly used fungicides, rendering them ineffective. Thus controlling *R. solani* is a challenge for sustainable agriculture. Through multipronged approach, involving transcriptomics, genome sequence and comparative genome analysis, we have identified key pathogenicity determinants of *R. solani*. Our data suggests that downregulation of some of these pathogen genes do impart disease tolerance. Further, we have identified a novel rice bacterium that can eat various fungi including *R. solani*. Our effort to characterize the fungal eating ability of the bacterium and utilizing thereof in controlling sheath blight disease will be discussed during my presentation.

The CRZ-1 transcription factor and calcium signaling genes are required for heat-shock response, normal circadian clock, and cellulose degradation in *Neurospora crassa*

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We previously showed that the CRZ-1 transcription factor binds to an 8-bp DNA sequences to upregulate the expression of the neuronal calcium sensor-1 (NCS-1) for the tolerance to calcium (Ca^{2+}) stress in *Neurospora crassa*. In this study, we established CRZ-1 binding to the promoter of the *heat-shock protein* (*hsp*)*-80* gene, which play a major role in heat-shock response pathway in *N. crassa*. We also found that several Ca^{2+} signaling genes *ncs-1*, *camk-2*, *plc-1*, *cmd*, *cna-1*, *cnb-1*, *splA2*, and *crz-1* play an important role in the maintenance of normal circadian clock in *N. crassa*. These genes modulate the expression of the *frq-1* and *wc-1*, which are key genes playing an essential role in normal circadian rhythm in *N. crassa*. We also identified a novel role of the Ca^{2+} signaling protein sPLA2 in cellulose degradation, and collaborative work is in progress to determine the NMR structure of sPLA2. In addition, we identified cellular role of *NcZrg-17*, a zinc transporter, in

cellulose degradation and endoplasmic reticulum stress tolerance in *N. crassa*. Therefore, Ca^{2+}-signaling are required for heat-shock response, normal circadian clock, and cellulose degradation in *N. crassa*.

Natural food flavoring agent vanillin impedes CaCdr2p driven efflux and metabolic adaptability in human fungal pathogen, *Candida albicans*

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The development of resistance against antifungal drugs intensifies the need to search for novel compounds from natural resources. Vanillin (Van) or 4-hydroxy-3-methoxybenzaldehyde produced in vanilla beans is widely used as flavoring agent and in fragrance industry. In this study antifungal potential of Van was elucidated against prevalent human fungal pathogen, *Candida albicans*. Herein, we observed that the repertoire of Van antifungal activity was not only limited to *C. albicans* and its clinical isolates but also against non-*albicans* species of *Candida*. Van synergizes with membrane targeting drugs fluconazole and amphotericin B. Mechanistic insights into the mode of action reveals membrane disruption, depleted ergosterol levels and altered plasma membrane ATPase activity. Interestingly, Van specifically inhibits *Candida* drug resistance protein 2 (CaCdr2p) activity belonging to ATP Binding Cassette (ABC) superfamily as revealed by abrogated Rhodamine 6G efflux and nile red accumulation assay with CaCdr2p over expressing strain. Insight studies into the mechanisms suggested that abrogated efflux of CaCdr2p is due to competitive mode of inhibition. RT-PCR, western blot and confocal microscopy also revealed that Van leads to reduced expressions of *CDR2*, CaCdr2p and mislocalization respectively. The metabolic adaptability under low carbon conditions and expression of

functional virulence traits marks the success of pathogen ability to cause infection. Additionally, we explored Van as a potent glyoxylate cycle inhibitor as Van phenocopied*ICL1* deletion mutant conferring hypersensitivity under low carbon utilizing conditions. Molecular docking analyses revealed the in-silico binding affinity of Van with Icl1p and Mls1p and confirmed that Van binds to the active sites of both proteins with better binding energy in comparison to their known inhibitors. Furthermore, we also explored inhibited hyphal morphogenesis, biofilm formation and cell adherence. Lastly, antifungal efficacy of Van was demonstrated by enhanced survival of *Caenorhabditis elegans* model and negligible hemolytic activity. Taken together, our results suggest that Van is potential antifungal compound that warrants further investigation in clinical applications so that it could be competently employed in therapeutic strategies to treat *Candida* infections.

Key words: *Candida*; natural compounds; membrane; efflux pump; biofilm; glyoxylate cycle

ST10/P59

Glycosylphosphatidylinositol-linked aspartyl proteases regulate the secretome of the pathogenic yeast *Candida glabrata*

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Candida glabrata is an opportunistic human fungal pathogen, which accounts for upto 30% of total *Candida* bloodstream infections. A family of eleven putative GPI (glycosylphosphatidylinositol)-anchored aspartyl proteases, also referred to as CgYapsins, is essential for intracellular survival and pathogenesis of *C. glabrata*. Using an approach of molecular, transcriptomic and proteomic analysis, we elucidated the mechanism underlying CgYapsin-mediated regulation of virulence. We showed that CgYps1-11 yapsins are required for suppression of the spleen tyrosine kinase (Syk)-dependent pro-inflammatory response (IL- 1β production) in human THP-1 macrophages. Additionally, we showed that CgYapsins are required for biofilm formation and colonization of murine organs in a disseminated candidiasis

model. Further, characterization of the secretome of *C. glabrata wild-type* and *Cgyps1-11Δ* mutant revealed that the wild-type secretome consisted of 119 proteins, which were primarily involved in cell wall organization, carbohydrate metabolism, proteolysis and translation processes. Strikingly, the *Cgyps1-11Δ* secretome was found to be 4.6-fold larger, and contained 65 differentially abundant proteins, as revealed by label-free quantitative profiling, with 49 and 16 being high- and low-abundant proteins, respectively, compared to the *wild-type* secretome. We will discuss how CgYapsins, being both bona-fide constituents and key modulators of the *C. glabrata* secretome, aid in establishment of successful infections.

Putting bits and pieces together: The saga of Arrestin Related Trafficking protein in *Saccharomyces cerevisiae*

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Nutrient uptake is one of the rate limiting steps in the growth and differentiation of organisms. In yeast, complex inter-connected signalling network brings about the preferential utilisation of nutrients. This network continuously monitors not only the quantity, but also the quality of the nutrients. For example, with respect to carbon, glucose is preferred whereas with respect to nitrogen, glutamine/ammonia are the most preferred nitrogen sources. In contrast, preferential utilisation of amino acids would be detrimental, as all of them are required for the normal growth. Our interest in nutrient induced regulation of growth and differentiation in *Saccharomyces cerevisiae* led to the isolation of MRG19, whose expression was tightly regulated by both carbon and nitrogen. We now know that MRG19 is a member of Arrestin Related Trafficking family of proteins (ARTs), which is conserved from yeast to humans. While searching a role for ECM21 (ART8), a paralogue of MRG19 (ART2) in a nutrient dependent growth, we stumbled upon the observation that ecm21Δ cells are defective in leucine utilisation, most likely because of the non-availability of BAP2, a high affinity leucine transporter. We found out that this defect is not linked to the ECM21 locus, but to an unknown locus piggybacking on ecm21Δ background. A second site suppressor of leucine growth defect in the above strain

was identified to be in BUL1, another member of ART family. Further analysis, indicated that BUL1 is the cognate arrestin for the down regulation of not only the high affinity leucine transporter BAP2 but also the low affinity leucine transporter BAP3. Ironically, BUL1 also down regulates GAP1 (general amino acid permease) and JEN1 (lactate permease). Recently, BUL1 has also been implicated in the rescue of protein trafficking and life span defect in a yeast strain disomic only for chromosome 5. Based on the above and the results obtained during this investigation, we propose that BUL1 might be a central component of amino acid signalling pathway, which in turn co-ordinates carbon and nitrogen induced cellular processes.

Repeat to Reap it: Systems-level understanding of the role of amino acid repeats

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Abnormal expansion of amino acid homorepeats such as polyglutamine have been linked to detrimental effects such as neurodegenerative diseases in humans. Such identical runs of amino acids are low-entropy sequences with low information content. Why, then, are homorepeats prevalent in eukaryotic proteomes? In this talk, I will discuss how integrating diverse large-scale datasets spanning sequences, structures, phenotypes, molecular networks and evolution in budding yeast, has informed us about the consequences of homorepeat emergence. Importantly, the presence of homorepeats increases the functional versatility of proteins by mediating protein interactions and facilitating spatial organization in a repeat-dependent manner. During evolution, homorepeats are preferentially retained in proteins with stringent proteostasis, which might minimize repeat-associated detrimental effects such as unregulated phase separation and protein aggregation. Thus, homorepeats are distinct modules that are often retained in stringently regulated proteins. Their presence facilitates rapid exploration of the genotype–phenotype landscape of a

population, thereby contributing to adaptation and fitness. I will also discuss about our ongoing research on the functional implications of co-occurrences of different amino acid homorepeats in the same protein.

Loss of F-box Motif encoding gene SAF1 and RRM3 together leads to synthetic growth defect and sensitivity to hydroxyurea and methyl methane sulfonate in *S. cerevisiae*

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SAF1 gene of *S. cerevisiae* encodes for F-box motif containing protein. The N- terminus F-box motif of Saf1 interacts with Skp1 subunit of the SCF-E3 ligase and C-terminus with Aah1 (adenine deaminase) for ubiquitination and degradation by 26S proteasome during phase transition from proliferation to quiescent phase due to nutrient stress. The replication fork associated Pif1 family helicase Rrm3 of *S. cerevisiae* function in removal of the non-histone proteins during replication fork movement. Here we have investigated genetic interaction between both the genes SAF1 and RRM3 for their role in growth fitness and genome stability. The *saf1Δrrm3Δ* showed the extremely slow growth phenotype in rich medium and sensitivity to genotoxic stress agents such as HU and MMS when compared with *saf1Δ*, *rrm3Δ*, and WT cells. The *saf1Δrrm3Δ* also showed the defects in nuclear migration as

evident by multi-nuclei phenotype and elevated frequency of Ty1 retrotransposition. Based on observations, we report that SAF1 and RRM3 function in parallel pathways for growth fitness and stability of the genome. (Acknowledgement: The authors acknowledge the support of Department of Biotechnology and SERB, DST, GOI and SMVDU, Katra, Jammu)

ST11/P72

Investigating the mechanism of regulation of cellular repertoire of *SKS1* mRNA in *Saccharomyces cerevisiae*

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Opportunistic human fungal pathogen *C. albicans* displays a morphological transition from yeast to hyphal state, which has been postulated to mimic its transition from avirulent to pathogenic form upon infection to the host tissue. Strikingly, the budding yeast *Saccharomyces cerevisiae* also exhibits similar transition from yeast to filamentous hyphal state upon growth under nutrient limiting conditions, thus serving as an excellent model system to study the genetics and molecular biology of pathogenic transition.

However, the mechanism of formation of these pseudohyphal filaments and the regulatory signal transduction pathways by which these signaling systems are integrated is poorly understood. The protein kinase Sks1p was implicated in the integration of signals for nitrogen and/or glucose limitation, resulting in pseudohyphal growth in both *S. cerevisiae* and *Candida albicans*. Thus, Sks1p constitutes a mechanism that integrates glucose-responsive cell signaling and pseudohyphal

growth, whose function is required for the virulence in *C. albicans*. However, the regulation of the expression of the *SKS1* gene is completely unknown. Recent findings in our laboratory indicated that *SKS1* is regulated at the post-transcriptional level via a combination of mRNA export and degradation. Data of the experiments substantiating our hypothesis will be presented.

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ST12/P44

Laboratory evolution of allopatric speciation in *Saccharomyces cerevisiae*

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In nature, organisms evolve to best fit their ecological niches, and while doing so, arrange themselves into discrete, reproductively isolated groups called species. While we know that an evolutionary response of an organism is shaped by adaptive mutations, random genetic drift, and bottlenecks, the fundamental forces driving speciation events are largely unknown. However, why an adaptive process defined by interaction between a genome and an environment, should dictate a process which is defined by interaction of two distinct genomes is not clear. While a number of theoretical models have been proposed to explain speciation, no systematic experimental characterization exists. This lack of understanding of speciation events forms the basis of our work.

In this context, we use *Saccharomyces cerevisiae* as the model system, and characterize contributions of adaptation towards

distinct environments and mutation-order towards dictating allopatric speciation. We evolve three parallel lines of haploid yeasts in glucose, galactose, glucose + galactose or melibiose as the carbon source for a few hundred generations. Degree of speciation was quantified by using decrease in mating efficiency. With this study we aim to systematically study speciation and decouple the relative contributions of factors contributing to speciation dynamics.

ST13/P43

Understanding the pathobiology of mixed-species candidiasis

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Candidiasis of tissues ranges from superficial mucocutaneous infection to invasive disease involving multiple organs. Access to the bloodstream requires penetration of mucosal barriers, and infection of internal organs requires invasion of endothelia, both of which depend on the yeast to hypha (Y-to-H) transition – a morphological switchover in *Candida.* The most prevalent species - *C. albicans* - undergoes morphological transition in response to various factors including growth condition and molecules of different origins. However, the second most prevalent species *C. glabrata* does not exhibit morphological switchover. *C. glabrata* is usually co-isolated with *C. albicans* from clinical samples. The frequency and potency of *Candida* co-infection make the communication between the two species intriguing. We hypothesized that inter-species interactions might be mutually beneficial for them to strike a harmony and enhance virulence. To better understand the pathobiology, various approaches such as mutant library screening,

biochemical and analytical chemistry studies and various co-culture assays are being utilized. Our preliminary findings show that *C. glabrata*, which does not form hyphae, rather has the ability to induce Y-to-H transition in *C. albicans*, with the help of a novel secreted molecule. Overall, findings from this study would help decipher the intricate molecular communication between different *Candida* species.

Regulation of mitotic timing: communication between CDK and MAPK

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Mitogen Activated Protein Kinases (MAPKs) play vital roles in multiple physiological processes. The MAPK Spc1 (human p38 homolog) in *Schizosaccharomyces pombe* regulates both cell division and stress response. Here, we present interesting findings about role of the MAPK Spc1 in regulating mitotic timing. Spc1 can sense aberrant activity of classical regulators of mitotic timing and can initiate mechanisms to rescue the cells from deleterious effects of aberrant mitotic timing. A key effector of this rescue mechanism is the 14-3-3 homolog Rad24 which is an indirect negative regulator of the Cyclin dependent Kinase Cdc2. We found Cdc2 hyperactivation to be associated with a Spc1 dependent increase in *rad24*$^{+}$ expression. This mechanism was active exclusively in cells where both Cdc2 and Spc1 activities were high. Activation of Spc1 in cells with basal Cdc2 activity failed to activate this mechanism. Perturbing the balance of positive and negative regulators of Cdc2 to make Cdc2 hyperactive also led to increased activation of Spc1, even if the known environmental triggers for Spc1 are absent. Our observations indicate that Cdc2 hyperactivation

represents a new trigger for Spc1 activation and that the former can activate a new Spc1 dependent feedback loop for restoring its optimal activity.

Alteration in the fidelity of *HIS4* start codon selection influences 3-amino-1,2,4-triazole sensitivity in hyper-GTPase defective eIF5 protein

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The factor eIF5 plays critical role in maintaining the fidelity of AUG start codon selection by providing GTPase activating protein (GAP) function to hydrolyse the GTP into GDP and P_i by the eIF2 ternary complex. The hyperGTPase $eIF5^{G31R}$ mutant was originally isolated as a dominant Sui^{-} (Suppressor of initiation codon). The $eIF5^{G31R}$ mutation also showed sensitivity to 3-Amino-1,2,4-Trizole (3AT) drug and consistently showed Gcn^{-} phenotype by repressing the *GCN4* expression. Our data suggests $eIF5^{G31R}$ mutation utilizes the upUUG1-10 ORF from the 5′ regulatory region of the *GCN4* transcript and dissociates before reaching the main *GCN4* ORF. Interestingly, the 3AT sensitivity of $eIF5^{G31R}$ mutation was suppressed by $HIS4^{UUG}$allele which is ostensibly not a direct target of 3AT inhibition. A detailed molecular analysis suggested that the below critical threshold expression of $HIS4^{UUG}$ allele by $eIF5^{G31R}$ mutation caused additional de-repression of *GCN4* expression which in turn up-regulated *HIS3* expression to

overcome 3AT sensitivity. Consistently, the overexpression of eIF1 increases the stringency of AUG codon selection by shifting the equilibrium of 48S complex to Open/P_{OUT} conformation thereby suppressing the 3AT sensitivity of eIF5^{G31R} mutation.

Cohesin proteins control protein quality through misfolding, aggregation and accumulation

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Cohesin is a multisubunit protein which holds sister chromatids together. Mutations in cohesin network of proteins give rise to precocious separation of sister chromatids. These are characterized by developmental disorders that give rise to diseases referred to as Cohesinopathy. The diseased allele mutation of *eco1-W216G*, known as Roberts syndrome, is made in yeasts, analogous to humans. Gene expression analysis shows that genes involved in ER stress are elevated in these mutants. Proteostatic stress is increased in the Roberts allele. Overexpression of the *eco1-W216G* protein in the Roberts mutant, results in formation of aggregates. Activation of the UPR pathway lead to stabilization of misfolded proteins. In the Roberts mutant, there occurs a constant elevation of the UPR pathway. Screening against an overexpression library of Hsp40 chaperones, rescues the growth defect in these cells. The DnaJ domain chaperones that emerge as suppressors of the Roberts allele are Zuo1, Apj1, Caj1 and Jjj1. The Roberts mutant exhibits a translational

defect. Roberts mutants, where there occurs an overexpression Zuo1, the translational defects are rescued which could possibly explain the growth rescue. This could be one of the possible ways to explain the pathophysiology associated with the Roberts syndrome.

Poster Abstracts
ICBYFF-2019

P1

Deciphering the role of multicopy suppressor IZH2 in oxidative stress

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Towards identifying gene or genes that are involved in cigarette smoke-mediated cellular toxicity, we performed a multicopy gene suppression of cigarette smoke sensitivity in *Saccharomyces cerevisiae*. In this assay, an array of yeast transformant harboring an extra copy of a gene from yeast library was screened for resistance to cigarette smoke-induced cellular toxicity. At the end, we have come up with several candidates; one of them is *IZH2*. To further confirm, wild type yeast cells with or without multicopy of *IZH2* were treated with cigarette smoke and we observed that cells with IZH2 exhibited better survival. Since cigarette smoke is known to increase the amount of ROS and misfolded protein inside the cell, we measured both the amount of ROS and misfolded protein in CS-treated and -untreated cells. The cells with IZH2 exhibited lesser ROS and misfolded proteins and this reduction resulted in less CS toxicity in yeast cells. Towards gaining insight into

IZH2-mediated less ROS production, results showed that the presence of extra copies of IZH2 caused translocation of Fet3p, an iron transporter, from plasma membrane to vacuole in CS-treated IZH2 overexpressing cells and thus resulted in reduced Fenton reaction, reduced ROS production and less sensitivity to cigarette smoke.

P2

Role for the aspartyl protease, CgYps1, in regulation of the oxido-reductive status in *Candida glabrata*

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Candida glabrata, a unicellular budding yeast, is a constituent of the normal human gut microflora. However, it is capable of causing life-threatening bloodstream infections in immunocompromised individuals. Known virulence factors of *C. glabrata* include biofilm formation, intracellular replication and expression of adhesins. Additionally, a family of eleven GPI (glycosylphosphatidylinositol)-linked cell surface-associated aspartyl proteases (yapsins), that are encoded by *CgYPS1-11* genes, has been shown to be essential for virulence of *C. glabrata*. One of the aspartyl protease CgYps1 was found to be pivotal to maintenance of vacuolar homeostasis, survival in macrophages and murine model of systemic infection. To delineate the mechanism of CgYps1-mediated regulation of these cellular processes, we set out to identify substrates and interactors of CgYps1. For this, we

performed immunoprecipitation assay followed by mass spectrometry analysis, and identified a protein, belonging to the family of Flavodoxin-Like Proteins (FLP), as one of the interactor. Through in vitro and in vivo studies, we have shown CgYps1 physically interacts with FLP and cleaves at the C-terminus of FLP. Additionally, we have performed the structural and biochemical characterization of FLP protein. These data along with the importance of FLP-CgYps1 interaction in the physiology and virulence of *C. glabrata* will be presented.

FT1/P3

Vigilin protein Vgl1 is required for heterochromatin mediated gene silencing in *Schizosaccharomyces pombe*

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Heterochromatin is a conserved feature of eukaryotic genomes and regulates various cellular processes, including gene silencing, chromosome segregation and maintenance of genome stability. In the fission yeast *Schizosaccharomyces pom*be heterochromatin formation involves methylation of lysine 9 in histone H3 (H3K9) which recruits Swi6/HP1 proteins to heterochromatic loci. The Swi6/HP1-H3K9me3 chromatin complex lies at the center of heterochromatic macromolecular assemblies and mediates many functions of heterochromatin by recruiting diverse set of regulators. However, additional factors may be required for proper heterochromatin organization, but are not fully known. Here, using several molecular and biochemical approaches, we report that Vgl1, a member of a large family of multiple KH-domain proteins, collectively known as vigilins, is indispensable for heterochromatin mediated gene silencing in *S pombe*. ChIP

analysis revealed that Vgl1 binds to pericentromeric heterochromatin in an RNA dependent manner that Vgl1 deletion leads to loss of H3K9 methylation and Swi6 recruitment to centromeric and telomeric heterochromatic loci. Furthermore, we show that Vgl1 interacts with the H3K9 methyltransferase, Clr4 and that loss of Vgl1 impairs Clr4 recruitment to heterochromatic regions of the genome. These findings uncover a novel role for Vgl1 as a key regulator in heterochromatin-mediated gene silencing in *S pombe*.

P4

Ustilago maydis small heat shock protein UmHSP12 is a stress responsive protein

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Ustilago maydis is a basidiomycetes smut fungi that develops a biotrophic interaction with its host plant *Zea mays*. During initial colonization the fungus utilizes varied molecular strategies to counteract the host defence responses and establish biotrophy. In this study we report the functioning of UmHSP12 under such conditions thereby establishing it as a stress responsive protein in *Ustilago maydis*. The expression of UmHsp12 is differentially regulated during infection and its absence causes reduction in the virulence of the fungus. A detailed bioinformatic analysis of the primary and secondary structure of UmHSP12 protein revealed its intrinsically disordered nature. Nevertheless, Circular Dichroism analyses of recombinant UmHSP12 in the presence and absence of SDS showed that the protein gets more structured in a hydrophobic environment. Thus the possibility of UmHSP12 in stabilizing different membrane structures of the fungus under

conditions of stress is discussed. Accordingly, confocal imaging of UmHSP12_iLOV fusion protein showed the localization of the protein in the cellular membranes in addition to the cytosol. Besides membrane stabilization, a role of the protein in maintaining the intracellular protein homeostasis is also demonstrated in this study.

P5

The phosphatidylinositol 3-phosphate 5-kinase, CgFab1, is essential for antifungal tolerance in the pathogenic yeast *Candida glabrata*

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Increasing resistance of the human opportunistic fungal pathogen *Candida glabrata* towards the azole and echinocandin antifungals, which target ergosterol biosynthesis and the cell wall, respectively, poses a serious clinical challenge. In the current study, we have deciphered the role of phosphoinositide signalling in antifungal resistance. We show that CgFab1, a putative phosphatidylinositol 3-phosphate 5-kinase, is essential for azole and echinocandin tolerance in *C. glabrata*. Further, we demonstrate that the widely-used azole antifungal, fluconazole, promotes actin cytoskeleton reorganization, and genetic (*CgFAB1* disruption) or chemical (latrunculin B) perturbation of actin structures results in azole susceptibility. Consistently, distribution of the actin depolymerization factor CgCof1 [PI(3,5)P2-binding protein]

and the actin filament-capping protein CgCap2 was altered upon both *CgFAB1* disruption and fluconazole exposure. These data implicate CgFab1 in azole tolerance through actin network remodeling. Lastly, through genetic and biochemical analysis, we showed an essential requirement for CgFab1 and its activity regulators CgVac7 and the scaffolding protein CgVac14, for maintenance of the cell wall chitin content, and survival of the cell wall, fluconazole and caspofungin stress. Altogether, our data underscore that CgVac7, whose homologs are absent in higher eukaryotes, may represent a promising target for antifungal therapy.

P6

Insights on the mechanism of calmodulin and calcium/calmodulin-dependent kinases in regulating stress tolerance and sexual development in *Neurospora crassa*

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Transient increase in calcium concentration is detected by multiple Ca^{2+} sensing proteins triggering versatile Ca^{2+} signaling events. Calmodulin (CaM), the main receptors for Ca^{2+}, binds to calcium with four EF hand domain and activates various downstream signalling molecules involved in regulation of different cellular pathways. Calcium/calmodulin dependent kinases (Ca^{2+}/CaMKs) are serine /threonine kinases that have a CaM binding domain, they are capable of phosphorylating broad range of substrates. In this study, we used a repeat-induced point (RIP) mutated strain of CaM and knockout mutant of four C*a*$^{2+}$/CaMKs to study the role in stress tolerance and sexual development. These mutants showed reduced survival rate under induced thermotolerance, hydrogen peroxide induced oxidative stress and reduced growth rate under DTT induced ER stress. Female and male fertility assay

showed that *cmd*^{RIP} mutant was female sterile and the vegetative hyphae of the mutant could not support the development of the newly fertilized wild type perithecia. The expression profiles of *cmd* and *ca*$^{2+}$*/camks* genes studied under heat stress and in sexual development condition showed increased expression. The expression analysis of *hsp80*, a heat shock protein that regulates the heat shock response pathway showed decreased expression in the mutants suggesting that CaM and C*a*$^{2+}$/CaMKs are important for stress tolerance and sexual development in *Neurospora crassa.*

FT2/P7

Role of kinetochore in maintaining chromosome condensation in meiosis

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Faithful chromosome segregation during mitotic and meiotic cell divisions is the key to resist aneuploidy that can lead to myriad disease states including cancer and infertility. Since condensation of the chromosomes plays pivotal role during segregation, revealing determinants of condensation is crucial to understand mechanism of chromosome segregation. The attachment of the chromosomes to the microtubules during segregation is mediated by a supra-molecular multi-protein complex called kinetochore formed at the centromere of each chromosome. While essential kinetochore proteins have been shown to influence condensation in mitosis, we earlier observed that the mutation in non-essential kinetochore proteins affects rDNA condensation only in meiosis. From this we hypothesized that although complete disruption of the kinetochore hinders chromosome condensation, kinetochore has additional meiosis specific role in condensation as we argue that the level of condensation might be more in meiosis

than mitosis. To examine our hypothesis we directly measured in vivo chromosome condensation using homoFRET based assay in *S. cerevisiae* in the wild type and in the kinetochore mutant of a key non-essential protein, Ctf19. Interesting, as hypothesized, we observed a significant increase in chromosome condensation in meiotic metaphase I compared to mitotic metaphase in the wild type. We also observed that *ctf19* mutant was compromised in condensation in meiosis but not in mitosis. These observations were further corroborated with reduced association of condensin (Brn1) with the chromatin in the mutant compared to the wild type, as measured by chromosome spread and chromatin immunoprecipitation assays. How mechanistically kinetochore promotes condensin targeting to the chromatin is under investigation. While we and others have earlier demonstrated more significant meiotic functions of the mitotically non-essential kinetochore proteins in budding yeast, in this work we further extend that knowledge to reveal a meiotic specific chromosome condensation function of kinetochore.

P8

Screening *Trichoderma* strains for rhizosphere competence and induction of biotic stress tolerance in castor (*Ricinus communis L.*)

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Trichoderma spp. are known to be promising biological control agents, mycoparasitizing plant pathogenic fungi and minimizing plant diseases. Identifying effective *Trichoderma* strain(s) could serve as potential non-hazardous alternative to the usage of chemical pesticides. In our study, six *Trichoderma* strains were characterized by analyzing morphological and molecular variations. Screening studies were carried to test root colonizing ability in a castor genotype, DC 107. Of the six strains, four strains which showed significant colonization of castor roots [*Trichoderma harzianum* N13 (MH356723.1; MK411202.1), *Trichoderma asperallum* TV5 (MH393299.1; MK411216.1) *Trichoderma asperallum* TV2B16 (MH393229.1; MK411214.1) and *Trichoderma asperallum* 7316

(MH236427.1; MK411201.1)] were screened for their root and soil colonizing ability along with growth promoting properties and biotic stress tolerance against two important fungal pathogens, *Fusarium oxysporum* fsp. *ricini* and *Alternaria ricini*. Compared to controls, significant decline in colony forming unit (CFU) count in *Trichoderma*-treated bulk and rhizosphere soil, besides mycoparasitism against *F. oxysporum and A. ricini* was observed. Further, castor seed bio-priming with *Trichoderma* strains showed an increase in shoot length by 46.6, 28.9 and 21% in 7316, N13 and TV5 treated DCS-107 respectively compared to controls suggesting these strains either individually or in combination, could confer resistance to pathogens besides enhancing crop growth.

P9

Role for the lipid kinase, CgVps34, in trafficking of the membrane transporters in *Candida glabrata*

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Candida glabrata is an opportunistic human fungal pathogen that causes mucosal and disseminated infections. The ability of *C. glabrata* to survive and replicate in host macrophages requires both effective antifungal response-counteracting and micronutrient acquisition mechanisms. CgVps34 is the sole class III phosphatidylinositol 3-kinase, which phosphorylates phosphatidylinositol (PI) to phosphatidylinositol-3-phosphate (PI3P), and essential for intracellular survival. In this study, we have examined the role of CgVps34 in micronutrient transport and homeostasis mechanisms in *C. glabrata*. The *Cgvps34*Δ mutant showed growth defects in medium containing surplus iron, copper and zinc metal ions. We showed that this growth attenuation was due to defective retrograde trafficking of high-affinity respective ion transporters from the plasma membrane to the vacuole, in response to excess metal ions. We took a

proteomic approach to delineate the molecular basis underlying defective endocytic trafficking of high-affinity ion transporters and identified protein interactors of CgVps34 kinase and PI3P lipid. We identified a total of 125 CgVps34 interactors, of which 5 are involved in intracellular protein transport. The role of these proteins, along with CgVps34, in endocytic recycling/degradation of ion transporters, as an adaptive response of *C. glabrata* cells to changes in environmental ion conditions, will be presented.

P10

Low hydrostatic pressure inhibit morphogenesis in *Candida albicans*

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We have evaluated impact of low hydrostatic pressure (LHP) on *Candida albicans* morphogenesis in response to L-proline at 37ºC using an *in vitro* study. *C. albicans* showed LHP dependent modulation in morphogenesis. Most dramatic effect was observed at 1800 Pa i.e. 13.5 mmHg (less than the lowest capillary hydrostatic pressure); wherein hyphae induction was inhibited by 99%, pseudohyphe increased to 91% without affecting percentage of budded and un-budded cells. However complete inhibition of hyphae and pseudohyphe formation was observed at 3000 Pa. LC-MS/MS analysis revealed significant modulation in level of 30 proteins (53% up regulated and 47% down regulated) involved in different biological processes, in response to LHP (1800 Pa). Dog1 & LELG_01648, reported to be associated with morphogenesis were down regulated by 8.62 and 4.55 fold respectively. It suggests that LHP inhibit hyphae induction by activating antagonistic pathway (as Dog1 is regulated by Nrg1 and Tup1) while induce pseudohyphe by down regulating Kel1 (12 fold), maintaining cell shape & Cht3 (4 fold), separating mother and daughter cells. Up regulation of Mic19, an important component

of MICOS complex maintaining crista junctions, inner membrane architecture and proper mitochondrial functions could be a compensatory response to LHP induced strain on mitochondrial membrane structure and function. It was supported by enhanced biosynthesis of phosphatidyl choline required for strengthening membranes through Sam2 up regulation. Similarly, LHP affect vacuolar trafficking and proper folding of proteins, as level of components involved in transport and proteolysis of aberrant and unfolded proteins has gone up. In addition to metabolic processes (carbohydrate, amino acid, nucleotide), gene expression, energy generation in *C. albicans*. This is the first report on modulation of *C. albicans* morphogenesis by LHP along with an insight into the mechanism of action. Our results cites the significance of capillary hydrostatic pressure in maintaining *C. albicans* cells in yeast form during blood stream infections.

P11

Deletion of F-box motif encoding *YDR131C* and retrograde signaling related *RTG1* gene together leads to cell size enlargement and stress tolerance phenotype in *Saccharomyces cerevisiae*

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The YDR131C gene encodes for a protein product which contain N-terminal F-box motif which interacts with Skp1 subunit of E3 ligase for ubiquitination and subsequent degradation of target proteins by 26S proteasome complexes. The retrograde signalling pathway is well conserved from yeast to humans who controls the gene expression leading to cell adaptation during stress conditions and prevents the cell death. The retrograde signelling related trasncritpin factor Rtg1 is encoded by *RTG1* gene in *S.cerevisiae*. In this study, we investigated the binary genetic interaction between both the (*YDR131C* and *RTG1*) genes by knocking out from BY4741 genetic background, followed by spot assay for cellular growth response to stress agents i.e. genotoxic and alternate carbon source utilization. We observed that the *ydr131cΔrtg1Δ* cells showed enlarged cell size with increased growth phenotype on

YPD medium when compared with *ydr131cΔ, rtg1Δ,* and WT cells. The *ydr131cΔrtg1Δ* cells also exhibited the tolerance phenotype to MMS, glycerol, galactose, and acetic acid when compared with *ydr131cΔ, rtg1Δ,* and WT cells. However, *ydr131cΔrtg1Δ* cells showed sensitivity to hydroxyurea. Based on observations we suggest that *YDR131C* and *RTG1* interact genetically to regulate the cell size and stress response. The underlying mechanisms of the observed phenotypes need further investigation in future.

(**Acknowledgement:** The authors acknowledge the support of Department of Biotechnology, SERB, DST, GOI and SMVDU, Katra, Jammu & Kashmir)

ST6/P12

Nucleolar size regulates nuclear envelope shape in *Saccahromyces cerevisiae*

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Nuclear shape and size is cell type-specific. Change in nuclear shape is seen during cell division, development, and pathology. The nucleus of *S. cerevisiae* is spherical in interphase and becomes dumb-bell shaped during mitotic division to facilitate the transfer of one nucleus to the daughter cell. As yeast cells undergo closed mitosis, the nuclear envelope remains intact throughout the cell cycle. The pathways that regulate nuclear shape are not well characterized. The nucleus is organized into various sub-compartments, with the nucleolus being the most prominent. We have initiated a focused genetic screen for nuclear shape defects in *S. cerevisiae* to ask if the nucleolus influences the nuclear shape. We find that increasing nucleolar volume triggers non-isometric nuclear envelope expansion resulting in an abnormal nuclear envelope shape. We further show that the tethering of rDNA to the nuclear envelope is required for the appearance of these extensions.

P13

Functional specificity and evolution of Cwc23: A ubiquitous and non-canonical J-domain protein essential for pre-mRNA splicing

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Hsp40s (also called J-domain proteins because of the presence of a characteristic J domain) act as obligate co-chaperones of Hsp70s. The N-terminal J domain of Hsp40 interacts with ATPase domain of Hsp70, while the C-terminal is involved in recognition of client proteins. Cwc23 is one of these J domain proteins, and is essential for viability in *S. cerevisiae*. It is a known splicing factor and via its C-terminus interacts with Ntr1 (NTC complex protein), a spliceosome disassembly factor. This interaction is very critical for the functionality of Cwc23 and thus the C-terminal truncation mutant of Cwc23, unable to interact with Ntr1, shows gross pre-mRNA splicing defects as well as accumulation of the intron lariat. Cwc23 orthologs identified in higher eukaryotes have also been shown to be physically associated with spliceosome. This suggests towards their role in the splicing

process, however there is no information on the involvement of any Hsp70: J domain protein system in spliceosomal remodeling in higher eukaryotes. In the present study, we are trying to understand how the structural differences in the Cwc23 are modifying its function in more complex eukaryotes and whether the increasing complexity has any effect on the J domain requirement in these organisms.

P14

Systems biology of pathogenesis mechanisms of fungal pathogen *Candida albicans*

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Predicting pathogenicity of a microbe is a difficult but an essential biomedical challenge. I aim to address this Grand Challenge in the context of human fungal pathogen *Candida albicans*. Like any pathogen, *C. albicans* have evolved ingenious ways to invade, survive and replicate inside hosts to successfully establish/disseminate infection. I will illustrate the design principles of two central virulence factors of *C. albicans*, namely, their adaptive ability to phagosomic stresses and morphogenetic plasticity.

Host immune cells attack and kill invading micro-organisms by imposing a combination of different stresses upon them. When sequestered inside a phagosome, *C. albicans* activate an array of different stress response pathways (SRPs), including, oxidative, nitrosative and osmotic SRPs in order to evade host immune attack. I will explore the synergistic/antagonistic,

time- and dose-dependent interactions of these SRPs to combinatorial perturbations, by means of systems theoretic approach.

Morphogenetic plasticity is an essential virulence factor of *C. albicans*. Morphogenetic transition in *C. albicans* is majorly governed by cAMP-Protein Kinase A, MAPK, pH, embedded, cell cycle arrest and negative regulation pathways. A grand mathematical model of Candida's morphogenesis network is constructed and analysed to achieve a holistic understanding of cellular-processes governing morphogenesis.

FT3/P15

D-amino acids poisoning in an opportunistic fungal pathogen *Candida albicans*.

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Proteins are made of 20 genetically encoded amino acids. Except for glycine, each amino acid is chiral, existing in two stereoisomeric forms- dextrorotatory (D-) and laevorotatory (L-) forms. During protein biosynthesis in a cell, several checkpoints ensure to prevent D-amino acids from getting incorporated into a nascent polypeptide. One such checkpoint is D-aminoacyl-tRNA deacylase (*DTD*), an enzyme evolutionarily conserved across all domains of life. DTDs specifically hydrolyze the D-aminoacyl-tRNA ester bond but do not act on L-aminoacyl-tRNA molecules. While molecular mechanisms are relatively well-established, *in-vivo* effects of the loss of *DTD* function is unexplored in most organisms. Using a genetic approach, we deleted the functional *DTD* gene from an opportunistic fungal pathogen *Candida albicans* that can undergo various phenotypic transitions depending on environmental cues. Upon contact with a biotic or abiotic

surface, *C. albicans* can form a multilayered biofilm structure. Our results suggest that *C. albicans dtd* null strains fail to form intact biofilm in the presence of D-leucine. However, planktonically growing *C. albicans cells* utilize D-leucine even when L-leucine biosynthetic pathway is blocked. This indicates that additional checkpoints are active during the planktonic phase to prevent D-leucine misincorporation and utilize D-leucine as a source for L-leucine synthesis.

ST8/P16

Scd6 and Psp2 are multicopy suppressors of clathrin deficiency

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Clathrin, one of the major vesicular coat proteins, plays a major role in endocytosis, sorting, and retention of proteins at the trans-Golgi network. Upon deletion of Chc1 (Clathrin Heavy Chain), *Saccharomyces cerevisiae* can either be viable or inviable, depending upon the genetic background.

Scd's (suppressors of clathrin deficiency) are the plasmids that were identified to rescue the growth defect associated with Chc1 depletion in *scd*1-i state. Scd6 is one of the multi-gene suppressors identified, having a total of 8 genes. We report here that YPR129W, one of the genes on that construct is necessary and sufficient to suppress the growth defect of Chc1 depleted cells. We have also identified another novel suppressor, Psp2 (Polymerase Suppressor), which upon overexpression can also rescue the growth defect of Chc1 depleted cells.

Scd6 and Psp2 both are known to localize to RNA granules, and both have C-terminal RGG motif. Interestingly, for both Scd6 and Psp2, RGG motif was found to be the most important domain for the suppression activity. Here, we hypothesize that these proteins might be working by reducing the protein load upon vesicular trafficking pathway somehow to balance the effect of CHC1 depletion leading to growth rescue.

ST3/P17

Atg11, an autophagy regulator, is required for high fidelity chromosome segregation

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Autophagy is an evolutionarily conserved process that maintains the turnover of cytosolic components. Recently autophagy related (Atg) proteins are shown to play unconventional roles including maintenance of genomic stability. Using data mining, we find several Atg proteins physically or genetically interact with proteins of chromosome segregation machinery in *Saccharomyces cerevisiae*. In this work, we sought to determine the role of Atg proteins in chromosome segregation in *S. cerevisiae*. By screening a large collection of *atg* mutants, we identified *atg11* to be the most sensitive to thiabendazole. *atg11* displayed increased

minichromosome loss, and a delay at G2/M stage in the presence of a microtubule poison thiabendazole. Importantly, loss of Atg11 protein function resulted in shorter spindles in large-budded cells suggesting its possible role in regulating spindle dynamics. By analyzing a series of double deletion mutants of *atg11* and motor proteins, we report previously unknown genetic interactions between Atg11 and Kar3. We show that *atg11 kar3* displays slow growth and increased percentage of mitotically arrested large-budded cells having shorter spindle (1-4 μm) at 37°C. Thus, we report a previously unknown moonlighting function of Atg11 together with Kar3 in regulating the spindle length dynamics in *S. cerevisiae* during vegetative growth under nutrient-rich conditions.

P18

Loss of Glyoxylate metabolic switch regulator *UCC1* and La-motif encoding *SRO9* genes together leads to MMS and H_2O_2 stress tolerance in *Saccharomyces cerevisiae*

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Glyoxylate pathway is activated in response to mitochondrial dysfunction or in case of non-availability of glucose to metabolize non-fermentable carbon sources. In *Saccharomyces cerevisiae,* Ucc1*,* an F-box motif protein, encoded by *UCC1*, functions as a metabolic switch regulator by targeting the key glyoxylate enzyme, Cit2 for proteasomal degradation when glucose is present in the cell environment, resulting in inhibition of glyoxylate pathway activity. The La-motif protein Sro9, encoded by SRO9 is involved in bud formation; actin filament assembly and RNA biogenesis have been reported to physically interact with Ucc1. However functional implication of their interactions is not known. Here we have investigated the genetic interaction between *UCC1* and *SRO9* genes. The single and double gene deletions of *UCC1* and *SRO9* were constructed in BY4741

genetic background and evaluated for growth fitness in rich and on non-fermentable carbon sources solid media. The cellular growth response to genotoxic stress agents (Hydroxyurea, Methyl methanesulfonate) was also studied using spot assay. We observed that *ucc1Δsro9Δ* cells showed robust growth in the presence of 20mM acetic acid, 4% galactose, and 4% glycerol without dextrose when compared with WT, *ucc1Δ* and *sro9Δ* cells. The double mutant *ucc1Δsro9Δ* showed tolerance to MMS andH_2O_2 but sensitivity to hydroxyurea. The mechanism of stress tolerance conferred by the loss of both the genes needs to be investigated in the future.

(**Acknowledgement:** The authors acknowledge the support of Department of Biotechnology and SERB, DST, GOI and SMVDU, Katra, Jammu)

ST2/P19

Mechanism of DNA-Protein crosslink repair in pathogenic yeast *Candida albicans*

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Candida albicans is an opportunistic fungal pathogen exists as an integral part of the massive human microbial flora. It remains under the constant threat of DNA damages due to oxidative burst generated by innate immune cells and therapeutics such as radiation, cisplatin, AZT, etc. administered to immune-suppressed individuals. DNA-Protein Crosslinks (DPC) are one such damages where specific DNA binding proteins are covalently linked to DNA and form irreversible bulky adducts to induce genome instability and cell death. Underlying repair mechanisms of DPCs are elusive in *Candida sp*.. Currently, we are investigating involvement of three pathways in *C. albicans* namely NER, HR and DPCR. Unlike NER and PRR that target damaged portion of DNA segment, DPCR targets the protein part of the DNA-protein complex. In budding yeast and mammals, metalloproteases

like Wss1 *(alias* Sprtn*)* and Tdp*1* play essential role in DPCR. The gene encoding *TDP1*, is absent in *C. albicans*. Thus, the involvement of Wss1 dependent DPCR would become essential for *C. albicans* survival, colonization, and in the development of systemic candidiasis. Contrary to *S. cerevisiae*, homozygous deletion of *WSS1* strain alone is hypersensitive to both DNA replication inhibitor and DPC inducing agents but not to MMS, H_2O_2 etc. Detail characterisation of CaWss1 and its role in genome stability, filamentation, drug resistance and pathogenesis will be discussed.

FT19/P20

Characterizing the dual targeting and function of the peroxisomal protein Pex30

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Peroxisomes are single membrane-bound dynamic organelles whose number and function may vary according to the need of the cell. Peroxisomes interact with other surrounding organelles like mitochondria, lipid droplet, endoplasmic reticulum (ER) *etc* in order to optimize their multiple cellular functions. Pex30 is a peroxisomal protein that resides in the ER and associates with peroxisomes to regulate peroxisome biogenesis. Our study aims to understand the importance of this dual localization of Pex30 and for this we have constructed *Saccharomyces cerevisiae* strains expressing Pex30-GFP and GFP-Pex30. Fluorescence microscopy revealed an interesting difference in phenotype and localization pattern for both the fusion proteins. To understand the observed differences in phenotype and localization, we constructed systematic truncations of the protein. Each of these truncations showed a distinct phenotype and localization to peroxisome and ER.

Further to identify novel interacting partners of Pex30 and their potential role in the observed differential localization of Pex30, expression and purification of the full length protein and dysferlin domain alone is performed.

P21

Structural and functional analysis of seven transmembrane receptor protein *RTA3* in *Candida albicans*

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Rta1(**R**esistance **T**o **A**minocholestrol) family of proteins are unique to fungal kingdom and can be considered as potential therapeutic targets. In *S. cerevisiae* these super family of proteins are involved in lipid translocation. *C. albicans* has four genes of this family namely orf19.6224, *RTA2*, *RTA3* and *RTA4*. It is noteworthy that these proteins lack an overall sequence conservation with the classical GPCRs. Sequence homology of these Rta proteins revealed the presence of signature sequence. Published data from our laboratory has implicated Rta3, a 7-transmembrane receptor protein (7- TM), as the determinant of biofilm development in *C. albicans*. In addition, this protein also plays a role in maintaining the asymmetric distribution of phosphatidylcholine across the plasma membrane, highlighting the role of this protein in multiple regulatory pathways. Considering the relevance of Rta3 in *C. albicans*, we aimed to dissect the importance of the

conserved signature sequence of Rta3. Furthermore, the 7- TM receptor proteins are known to mediate their functions through the C-terminal domain. So in order to analyze the role of C-terminal in Rta3 function, we constructed C terminal truncation mutant (Δ C 414-464) to explore the relationship between its structure and function. Herein, we show that the strain lacking the signature motif of Rta 3 displayed increased susceptibility to miltefosine. Consistent with *rta3*Δ/Δ cells, H379R/E380D mutant also displayed increased internalization of NBD-labelled phosphatidylcholine compared to its parent strain, while the cells expressing Δ C 414-464 mutant behaves similar to the wild-type cells. Our data demonstrate the relevance of signature sequence of Rta3 family which will pave a way in developing a new drug target for future antifungal therapy.

P22

Cyclin synthesis and degradation in mitosis: Understanding new regulators

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Schizosaccharomyces pombe is an excellent model organism to study cell cycle progression. Earlier work from our lab identified transcription factor Atf1 to be one of the key regulators facilitating mitotic entry by increasing mitotic cyclin (Cdc13) expression in *S pombe*. Interestingly, Atf1 can also take part in cyclin degradation by interacting with Anaphase promoting complex, thereby facilitation mitotic exit. Completion of a cell cycle is coordinated by the effective regulation of the phase transitions. Atf1 being one of the key regulators controlling the mitotic timing. Stress dependent activation of Atf1 is mediated by phosphorylation events, but the exact role of phosphorylation status of Atf1 in regulating mitotic timing is poorly understood. Our study was undertaken to unravel these unknown mechanistic connections. We intended to study the role played by the phosphorylation mediated responses of Atf1 in regulating mitotic exit. A phosphorylation defective Atf1

mutant (with all the 11 sites mutated), namely Atf1-11M was used to study changes in metaphase to Anaphase transition, mitotic cyclin Cdc13 synthesis and degradation. Our results indicate that MAPK dependent phosphorylation of Atf1 may positively regulate its interaction with APC/C complex and promote mitotic exit in *S. pombe* cell cycle.

P23

Prediction and analysis of the secretome of an opportunistic fungal pathogen

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Aspergillus fumigatus and multiple other *Aspergillus* species cause a wide range of lung infections, collectively termed aspergillosis. Aspergilli are ubiquitous in environment with healthy immune systems routinely eliminating inhaled conidia, however, Aspergilli can become an opportunistic pathogen in immune-compromised patients. The aspergillosis mortality rate and emergence of drug-resistance reveals an urgent need to identify novel targets. Secreted and cell membrane proteins play a critical role in fungal-host interactions and pathogenesis. Using computational pipeline integrating data from high-throughput experiments and bioinformatic predictions, we have identified secreted and cell membrane proteins in ten *Aspergillus* species known to cause aspergillosis [1]. Small secreted and effector-like proteins similar to agents of fungal-

plant pathogenesis were also identified within each secretome. A comparison with humans revealed that at least 70% of *Aspergillus* secretomes have no sequence similarity with the human proteome. An analysis of antigenic qualities of *Aspergillus* proteins revealed that the secretome is significantly more antigenic than cell membrane proteins or the complete proteome. Finally, overlaying an expression dataset, four *A. fumigatus* proteins upregulated during infection and with available structures, were found to be structurally similar to known drug target proteins in other organisms, and were able to dock *in silico* with the respective drug.

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FT14/P24

Gcn4 mediates a methionine-dependent anabolic program by controlling amino acid supply

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Methionine – an important sulfur amino acid acts as a strong growth signal for cells. Here, anabolic processes like nucleotide biosynthesis, amino acid biosynthesis, along with ribosomal biosynthesis are all upregulated transcriptionally when methionine is abundant, even in otherwise aminoacid limited conditions. In this study, we discover the role of the conserved Gcn4/Atf4 transcriptional regulator, typically studied during starvation responses, in mediating the methionine induced anabolic response. Gcn4 protein is strongly induced by methionine, independent of carbon sources. Further, we find that Gcn4 primarily controls nitrogen-flow in the methionine dependent anabolic program. Using ChIP-seq and RNA-seq analysis we identify direct targets of Gcn4 during methionine-abundance. Gcn4 directly increases the expression of most of the amino acid biosynthetic genes, and indirectly controls

nucleotide biosynthesis, suggesting that Gcn4 controls nitrogen flow required for anabolism. Importantly, all genes involved in arginine, lysine and histidine biosynthesis are direct Gcn4 targets. Thereby, the loss of Gcn4 strikingly reduces nitrogen flux towards arginine, lysine and histidine biosynthesis, suggesting that Gcn4 is critical for the supply of these amino acids in the methionine-induced anabolic program. Interestingly, ribosomal genes remain induced by methionine in Gcn4-deficient cells, decoupling the anabolic transcriptional program from ribosomal gene transcription. However, despite higher transcript amounts in the presence of methionine, Gcn4-deficient cells show reduced translation of ribosomal genes, due to a supply deficiency of arginine and lysine. Collectively, Gcn4 controls the supply of nitrogen-precursors, thereby fueling the methionine-induced anabolic program in cells.

FT5/P25

eIF4G(Tif4631p): A major player in nucleating the CTEXT proteome

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In *Saccharomyces cerevisiae*, the Nuclear Exosome, TRAMP and CTEXT (**C**bc1-**T**if4631-dependent **Ex**osome **T**argeting) selectively degrades diverse classes of faulty messages generated due to inaccuracy associated with nuclear mRNP biogenesis. Recent studies established that beside TRAMP, CTEXT acts as an alternative cofactor, which assists the exosome to target and degrade specific classes of aberrant messages. Importantly, CTEXT consists of nuclear cap-binding complex Cbc1p, two shuttling proteins Tif4631p and Upf3p and recently identified Dbp2, an ATP-dependent RNA helicase.

However, whether these proteins exists as a single multiprotein complex is not known. Here, using a combination of biochemical and genetic approach we (i) define the composition of this protein-complex and (ii) investigate the role of Tif4631p/eIF4G and Cbc1 and its various active functional

domains in the DRN function as well as in the complex formation. Interestingly, Co-immunoprecipitation studies employing a C-terminally TAP-tagged Tif4631 protein indicated that three major proteins Cbc1p, Rrp6p and Nrd1pare associated with Tif4631p and Tif4631p, Cbc1p, Upf3p, Dbp2p and Red1p perhaps exists as one protein complex. Similar Co-immunoprecipitation studies using Cbc1-TAP protein as bait indicates that CTEXT exists in a novel multiprotein complex, which is nucleated by Tif4631p.The identification of the other interacting candidates is currently in progress.

FT11/P26

Identification of critical amino acid residues of calcineurin regulatory subunit (*cnb-1*) involved in stress response and cross talk with heat shock stress pathway via its target calcineurin responsive zinc finger protein (CRZ-1) in *Neurospora crassa*

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We have generated mutations in different EF hand domains in calcineurin B (*cnb-1*) gene via Repeat Induced point mutation (RIP), in the fungi *Neurospora crassa.* We have demonstrated role of these mutations in different stress responses – heat shock, calcium (Ca^{2+}) homeostasis, osmotic stress and regulation of circadian clock in *N. crassa*. The calcineurin stress response pathway is mediated via one of its target calcineurin responsive zinc finger protein (CRZ-1). The cross talk between the heat shock response pathway and calcineurin-CRZ-1 signaling pathway was established from the increased expression of *hsp80* in wild type and *CRZ-1* homokaryotic strain whereas expression decreased by 2 fold

in the *cnb-1*^{RIP} mutants and using potent calcineurin inhibitor FK506 results in 3-fold decrease in the expression profile both in wild type and *crz-1* homokaryotic strain under heat shock stress condition. Further binding of CRZ-1 in the promoter of *hsp80* has been proved by chromatin immunoprecipitation assay (ChIP) and the exact binding sequence will be identified by electrophoretic mobility shift assay (EMSA) in heat shock condition. The above observations altogether with the expression, interaction studies suggest the role of the mutated amino acid residues of *cnb-1* in different stress response and gives a better molecular insight into the heat shock response and calcineurin-CRZ-1 pathway.

FT7/P27

The bakers's yeast Msh4-Msh5 associates with double strand break hotspots and chromosome axis during meiosis to promote crossovers

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Segregation of homologous chromosomes during the first meiotic division requires at least one obligate crossover/exchange event between the homolog pairs. In the baker's yeast Saccharomyces cerevisiae and mammals, the mismatch repair related factors, Msh4-Msh5 and Mlh1-Mlh3 generate majority of the meiotic crossovers from programmed Double Strand Breaks (DSBs).

To understand the mechanistic role of Msh4-Msh5 in meiotic crossing over, we performed genome wide ChIP-sequencing and cytological analysis of the Msh5 protein in cells synchronized for meiosis. We found that the initial recruitment of Msh4-Msh5 occurs following DSB resection. A two-step Msh5 binding pattern was observed: an early weak binding at DSB hotspots followed by enhanced late binding upon formation of double Holliday junction structures. Msh5 also showed weak binding along the axis and along the centromeres. Msh5 binding was enhanced at strong DSB hotspots away from the chromosome axis consistent with a role for DSB frequency in promoting Msh5 binding. These data on the in vivo localization of Msh5 during meiosis have implications for how Msh4-Msh5 may work with Mlh1-Mlh3 to ensure Holliday junction resolution at the chromosome axis.

P28

Understanding the role of novel actors of drug resistance in clinical isolates of *Candida* species

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Frequency of opportunistic fungal infections has increased significantly in past few decades. Further fungi being eukaryotic in nature, the emergence of drug resistance in them poses an additional challenge for drug target discovery and to prevent host cytotoxicity, therefore necessitating the need for the development of drugs with enhanced efficacy and minimized cytotoxicity. To this end, present study was carried out, wherein we tried to evaluate drug resistance, the molecular mechanism/s behind it and screening of few novel chemical formulations that can assuage this drug resistance phenomenon in the clinical isolates of pathogenic *Candida* emerging from Kashmir division of Jammu & Kashmir. We procured clinical isolates from a tertiary care hospital, which

were identified and subjected to extensive drug susceptibility analysis, which revealed that many isolates belonging to different species of *Candida* like *C. albicans*, *C. glabrata*, *C. auris* and few others were resistant to antifungal drugs. Gene expression analysis of selected genes also revealed the upregulation of many drug resistance genes in few drug resistant clinical isolates. Furthermore, we screened some novel chemical formulations and found few to be very effective in alleviating the drug resistance in these isolates, suggesting their potential role in abrogation of fungal drug resistance.

Key words: Opportunistic fungi, drug resistance, drug target, *Candida*.

ST1/P29

DDK Hsk1 phosphorylates Sirtuin Hst4 and target it for degradation on replication stress to stabilize stalled DNA replication forks

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Sirtuins are highly conserved NAD+ dependent class III histone deacetylases which function in cellular processes including cell survival, DNA damage and repair. Our previous reports show that fission yeast *hst4*Δ deficient cells are sensitive to replication stress and Hst4p is downregulated on methyl methanesulfonate (MMS) treatment. The aim of the current study is to decipher the molecular mechanism of regulation of Hst4. DDK is a highly conserved kinase involved in the regulation of DNA replication. The replication dependent roles of DDK are very well studied across all model systems. However, whether DDK has any positive role in the replication stress response is a matter of debate. We found that Hst4 is targeted for degradation in a replication dependent manner upon HU and MMS stress. Next, we found out for the first time that DDK phosphorylates Hst4 at serine residues to target it for proteolysis via SCF ubiquitin ligase. This process is

independent of intra-S phase checkpoint activation. The non-degradable mutant of Hst4 (4SA-Hst4) have persisting DNA damage foci and show defects in stalled fork recovery and proper DNA damage bypass.

We also show that fork protection complex stability at the chromatin is affected in 4SA-Hst4 mutant leading to these defects. Overall, this study identifies a novel role of DDK in replication fork stabilisation independent of its role in intra-S phase checkpoint pathway. Since, sirtuins are deregulated in cancer, this study helps in identifying similar mechanistic roles in higher eukaryotes.

P30

Flocculation of *Saccharomyces cerevisiae* is dependent on activation of Slt2 and Rlm1 regulated by the Cell Wall Integrity pathway

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Flocculation is an essential characteristic of yeast cells required for survival under adverse conditions. The multicellular structure (flocs) of yeast provides a suitable microenvironment to enhance the chances of survival during stress conditions. Although the signaling events triggering flocculation have been studied earlier, molecular mechanism remained elusive. In present study, we used flocculating sen1 mutants to identify the mechanism of flocculation. Based on the abnormal cell surface morphology and constitutive phosphorylation of Slt2 in flocculating Sen1 mutant cells, we assumed that flocculation is regulated by the Cell Wall Integrity (CWI) pathway. Up-regulation of *FLO* genes in wild type cells was observed upon activation of the CWI pathway either by chemical treatment or by deleting Slt2 phosphatase (Msg5). By using Slt2 mutants our study reveals that active state of Slt2 is indispensable for flocculation. The flocculation was reduced after deletion of *SLT2* or *RLM1*. Further we revealed

overlapping binding sites for Rlm1 and Tup1 at the promoters of almost all the *FLO* genes. Finally, we show higher Rlm1 and lower Tup1 occupancy at the promoters of *FLO1* and *FLO5* in flocculating cells. Altogether we demonstrate that CWI MAPK (Slt2) pathway use a non-catalytic mechanism to activate the transcription of *FLO* genes.

P31

Deciphering the role of Pol32, the non-essential subunit of DNA polymerase delta in pathogenesis of *C. albicans*

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Accurate and processive DNA synthesis by DNA polymerases (Pol) during DNA replication, repair and recombination is essential for lowering the rate of spontaneous (/damaged) induced mutations, suppressing mutagenesis in yeast and carcinogenesis in vertebrates. Polδ is a high fidelity processive enzyme, equivocally accepted to play a key role in all these DNA transaction processes. Not surprisingly, several mutations in Polδ from mouse and human have been mapped to cause various cancers. Yeast Polδ is a trimeric complex consisting of catalytic Pol3 and accessory Pol31 and Pol32 subunits. Pol3 is the largest subunit possesses both polymerizing and exonuclease activities. While Pol3 and Pol31 are essential subunits, Pol32 is dispensable for cell survival. Loss of proofreading activity in Pol3 increases the mutation rate to 100 folds. Similarly, any compromise in subunits

interaction in Polδ reduces processive DNA synthesis, resulting in frequent stalling of replication fork causing strands break. Chromosomal plasticity and genomic instability are frequently found in clinical isolates of pathogenic yeast *C. albicans*, which is responsible of 40-50 % mortality rate due to systemic candidiasis. Since, Polδ has not been characterized in this fungus so far, in this study; role of Pol32 in genome stability, morphogenesis, drug resistance and pathogenicity of *C. albicans* will be discussed.

P32

Diversity and ecology of manglicolous filamentous fungi isolated from Sundarban mangrove ecosystem, India

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Mangrove ecosystem of Sundarban is the world's largest mangrove biomes located both in India and Bangladesh. Since mangrove is detritus based ecosystem, variety of both marine and terrestrial fungi commonly referred as the "manglicolous fungi" are thriving in this ecosystem as the major decomposer. These fungi are adapted to adverse environmental conditions of high salinity, frequently inundated soft-bottomed anaerobic mud and contribute in global nutrient cycle by producing a plethora of extracellular enzymes such as xylnase, pectinase, cellulose, laccase etc which have great ecological and economical importance. Although several studies have been reported to decipher the overall biodiversity richness of this World Heritage Site, the diversity and ecology of microfungi in the Indian Sundarban region has not been much explored. Therefore, the present study aims to study the ecology and diversity of microfungi associated with Sundarbans mangrove forest of India. In this study, sediment and water samples were collected from three sites of Sundarban i.e. Jharkhali, Bali and Canning. Totally 63 fungal isolates were obtained from both sediment (50 isolates) and water samples (13 isolates) of these three sites out of which Bali island possessed highest fungal population i.e. 1.5×10^5 CFU/g in sediment and 3.2×10^4 CFU/ml in water sample. Colony morphology and microscopic analysis of fungal isolates revealed that majority of fungal isolates belonged to *Aspergillus*, *Penicillium*, *Fusarium* and *Trichoderma.* Among 63 isolates, 44 isolates were fast growing producing fruiting bodies with 48 hours. Thus, 44 fungal isolates were screened for

biofilm forming ability and iron oxide nanoparticle synthesis ability. It was found that only 12 fungal isolates were strong biofilm former as well as able to synthesize iron oxide nanoparticles rapidly which belonged to *Aspergillus niger* BSC-1, *Fusarium* sp. BSP-5, *Aspergillus niger* BSP-6, *Penicillium* sp. BSP-8, *Aspergillus niger* BWC-1, *Trichoderma* sp. CNSC-2, *Aspergillus flavus* CNSP-1, *Fusarium* sp. CNSP-5, *Fusarium proliferatum* JKSC-1, *Aspergillus flavus* JKSC-7, *Aspergillus niger* JKSP-5, *Aspergillus niger* JKWP-1. Further, these fungi exhibited excellent heavy metal resistance capacity i.e. 500-1000 ppm of four heavy metal salts such as potassium dichromate ($K_2Cr_2O_7$), lead nitrate ($PbNO_3$), copper sulphate ($CuSO_4$) and nickel chloride ($NiCl_2$). Out of these 12 isolates, *Trichoderma* sp. CNSC-2 and *Aspergillus flavus* JKSC-7 showed laccase activity suggesting potential role in degradation of lignin rich organic compounds. Therefore, the present study depicted possible role of manglicolous fungi in ecosystem sustenance and besides having role in biotechnological applications.

Key words: manglicolous fungi, microfungi, biodiversity, biofilm, Sundarban, mangrove.

FT4/P33

Stress: MAPK Spc1: Mitotic entry decisions

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Mitogen-activated protein kinases (MAPKs) play important roles in mitotic entry decisions and during physiological stress conditions. In *Schizosaccharomyces pombe* the MAPK Spc1 (p38 homologue) is known to both promote and delay mitotic entry. It was reported that a moderate increase in Spc1 activity promotes mitotic entry (nutrient stress) whereas a higher increase leads to activation of the G2/M checkpoint (oxidative, genotoxic or heat stress). Recently our laboratory showed, that in response to oxidative stress Spc1 targets the 14-3-3 protein, Rad24, independently of Srk1, leading to relocalization of Cdc25 and thus leading to mitotic inhibition. However, it is interesting to understand how the same protein which once serves to block mitotic entry in a particular kind of stress can also promote mitotic entry in another type of stress (nutrient stress).It's important to understand the intricacies, and how the same set of proteins behave in this alternate form of stress. Also, it was known that

absence of Wee1 is an important manifestation of many forms of cancer, and due to Warburg effect, lactate is an important byproduct of metabolism and so it's important to understand how Spc1, or it's downstream proteins behave in such conditions so that, further alterations can be designed to diagnose certain cancers.

P34

A role for the yeast PCNA unloader Elg1 in eliciting the DNA damage checkpoint

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The DNA polymerase clamp PCNA ring plays central roles during DNA replication and repair. The yeast Elg1 RFC-like complex (RLC) is the principal unloader of chromatin-bound PCNA, and thus plays a central role in maintaining genome stability. Here we identify a role for Elg1 in the unloading of PCNA during DNA damage. Using DNA damage checkpoint (DC)- and replication checkpoint (RC)-inducible strains, we show that Elg1 is essential for eliciting the signal in the DC branch. In the absence of Elg1 activity the Rad9 (53BP1) and Dpb11(TopBP1) adaptor proteins are recruited, but fail to be phosphorylated by Mec1(ATR), resulting in lack of checkpoint activation. The chromatin immunoprecipitation of PCNA at the Lac-operator sites reveals that accumulated local PCNA influences the checkpoint activation process in *elg1* mutants. Our data suggest that Elg1 participates in a mechanism that

may coordinate PCNA unloading during DNA repair with DNA damage checkpoint induction.

FT8/P35

Virulence function for ExAsp1, a secreted aspartyl protease from *Ustilago maydis*

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Ustilago maydis is a biotrophic plant pathogen that causes smut disease in maize. During colonization of the host and subsequent establishment of the disease *U. maydis* very efficiently utilizes its repertoire of secreted effector proteins. In this study the effector function of one of the secreted proteins from *U. maydis* is reported. ExAsp1 is an aspartyl protease that expresses only during in planta growth of the pathogen. The protein was found to be secreted very efficiently in *in vitro* secretion assays. ExAsp1 also contributes to the pathogenicity of the fungus. *U. maydis* deletion mutants lacking *ExAsp1* gene showed much reduced virulence compared to the wild type *U. maydis* strains. This study also explored any functional redundancy between ExAsp1 and other secreted aspartyl proteases in terms of their overall effect on the virulence of the fungus. Finally, the possible roles of ExAsp1 in the

pathogenicity of *U. maydis* are discussed based on the identification of candidate substrates of the protein.

P36

Intra-specific variation in LOH and mutation rates among *S. cerevisiae* strains

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A growing body of evidence suggests that mutation rates can show intra-specific variation within a species due to differences in genetic background. We estimated genome wide variation in mutation rates and spectrum within isogenic and hybrid strains of *Saccharomyces cerevisiae*. Mutation accumulation lines of the hybrid *S. cerevisiae* strains - RM11/S288c (S/R) and S288c/YJM789 (S/Y) were analyzed along with their isogenic diploid parents RM11, S288c and YJM145. The hybrid backgrounds further allowed us to look at patterns of loss of heterozygosity (LOH). The S/R hybrid showed fewer loss of heterozygosity (LOH) events compared to the S/Y hybrid. These results suggest that LOH rates vary between the two *S. cerevisiae* genetic backgrounds. Further, we observe up to 8-fold difference in single nucleotide mutation (SNM) rates among the different *S. cerevisiae* genetic backgrounds. Our

results demonstrate genome wide differences in both the rates and spectrum of gross chromosomal stability changes and SNMs between commonly used *S. cerevisiae* strains.

FT9/P37

Identifying the mRNA targets of eIF4G-binding translation repressor protein, Scd6

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Translation control plays a key role in various cellular processes. Upon stress, the translation can be repressed leading to the formation of repression ribonucleoprotein complexes (mRNPs). The RGG-motif-containing yeast Scd6 protein acts as a translational repressor. Scd6 represses translation by binding to the eIF4G subunit of the eIF4F cap-binding complex via its RGG motif, thereby forming a translation repression complex. Post-translational modifications of RGG-motif proteins are recently reported to play an important role in translational control e.g. arginine methylation of Scd6 has been studied and found to augment its repression activity by enhancing its interaction with eIF4G. Scd6 undergoes self-association via RGG-motif and competes

with eIF4G binding. The mechanism of translation repression by Scd6 is known but the mRNA targets of Scd6 are not yet studied. It is important to study the mRNAs repressed by Scd6 as it would identify the cellular processes targeted by Scd6. It could also establish the conditions under which Scd6 could function as a repressor. In the light of this objective, we are currently testing certain mRNA candidates which could be potential targets of Scd6 under oxidative stress.

Keywords: MRNPS, SCD6, Translational Repression etc.

FT13/P38

Screening and identification of multicopy suppressors of the genotoxic sensitivity associated with absence of ELL in *Schizosaccharomyces pombe*

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Transcription is one of the early steps of gene expression in all cells. Research has shown that regulation of gene expression occurs at both initiation and elongation steps of the transcription process. ELL (Eleven-nineteen Lysine-rich Leukaemia) family of proteins function as transcription elongation factors increasing the rate of transcription elongation by RNA polymerase II. They are also constituents of different elongation complexes. Our work has demonstrated that *S. pombe* cells lacking *ell*$^{+}$ display sensitivity towards genotoxic agents. To analyze the molecular mechanism(s) underlying this function of ELL, we performed a multicopy suppressor screen to identify genes whose overexpression could restore normal growth of the *ell* deletion mutant in the presence of genotoxic agents. Our screen has identified *epe1*$^{+}$, *SPAC922.04*$^{+}$, *hus5*$^{+}$, *toa2*$^{+}$ and *mug164*$^{+}$candidate genes, and the extent of rescue by few of them has been studied. We

have further carried out a more detailed analysis of two of these suppressors, and checked their effect on rescuing other phenotypes associated with lack of *ell*$^{+}$ in *S. pombe.* Taken together, our results show that increasing transcription may be one mechanism associated with suppression of various *ell* deletion phenotypes.

FT10/P39

Novel function of fission yeast sirtuin Hst4 in regulation of DNA replication.

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Chromatin organization in eukaryotes impact DNA based processes such as replication, transcription, and damage repair. Sirtuins are a highly conserved group of histone deacetylases (HDACs) that plays crucial role in important cellular processes including cell survival, DNA Replication and repair and maintain genome stability by deacetylating histones and non-histone proteins. In our previous study, we have reported that deletion mutant of fission yeast sirtuin hast4 show S phase delay and reduced DNA synthesis. This study investigates the molecular function of Hst4 in DNA replication by assessing BrdU incorporation as G2 synchronized WT and hst4D mutant cells progress through the S phase to measure global replication rate using flow cytometry. A delay in S- phase entry and defect in the rate of replication was observed on hst4 deletion. on the reduction in DNA synthesis observed in the hst4 mutants could be due to defective DNA replication origin firing and/or fork progression. Hence, we checked the role of

hst4 on origin firing using BrdU-IP followed by semi-quantitative PCR at few early and late origins and observed early origin to be less active at the mid-S phase in hst4 deficient strain, indicating the role of hst4 in defining the replication origin firing pattern in S. pombe. BrdU-IP-Seq to map the origin firing pattern at the whole genome is currently under process. Knowledge from this study is expected to be conserved in human system and could be used in designing therapeutics against pathophysiology where this pathway is deregulated.

ST9/P40

Metabolic constraints determine the self-organization of specialized, heterogeneous cell groups

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How phenotypically distinct states in clonal cell populations emerge and stably co-exist is an open question. We find that within a mature, clonal yeast colony growing in glucose limited conditions, cells organize into phenotypically distinct groups exhibiting opposite metabolic states. Beginning in a uniformly gluconeogenic state, cells exhibiting a contrary, high pentose phosphate pathway (PPP) activity state, spontaneously appear and proliferate, in a spatially constrained manner. Gluconeogenic cells in the colony produce and provide a resource, which we identify as trehalose. Above threshold concentrations of trehalose, cells switch to the new metabolic state and proliferate. This creates a self-organized system, where cells in this new state are sustained by trehalose consumption. In ongoing studies, we investigate how such a

state-controlling resource is itself modulated. We find that metabolite plasticity of an amino acid, aspartate, is a key determinant. Aspartate acts as a carbon substrate for trehalose production in gluconeogenic cells. Contrastingly, aspartate is differentially utilized by cells that show high PPP activity, as nitrogen donor for nucleotide synthesis. This metabolite plasticity enables the cell community as a whole to bet-hedge, with distinct advantages of each phenotypic state, allowing overall survival and growth of the colony

P41

Understanding the role of CDK hyperactivation as a novel trigger for MAPK activity in *Schizosaccharomyces pombe*

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Fission yeast, *Schizosaccharomyces pombe* has been extensively used to elucidate regulatory mechanisms associated with fundamental cellular processes in eukaryotes. The Mitogen activated protein kinase (MAPK) Spc1 (human p38 homolog, a well established target for cancer therapy) has multi-facet role in regulating cell division in *S. pombe* as well as in controlling the Core Environmental Stress Response(CESR).For any eukaryotic cell, checkpoint activation and gene expression modulation represent key determinants of cellular survival in adverse conditions. The former is mainly regulated by Cyclin dependent kinases (CDKs) while the latter is largely controlled by MAPKs. Cell cycle progression and MAPK dependent gene expression however are linked in during normal conditions or an unperturbed cell cycle. While MAPK mediated regulation of cell cycle is well known, the influence of mitotic CDKs (Cdc2 in *S.*

pombe) in stress responsive gene expression regulation is not known. We have strong experimental evidence that such a mechanism operates in *Schizosaccharomyces pombe*. Our observations pointed to the possibility of stress responsive transcription being controlled by the functional interaction between Cdc2 and Spc1 and also that Cdc2 activity might play a role in changing the way Spc1 engaged with its target promoters. Our work presents evidences that show how changes in Cdc2 activity can alter the Spc1 dependent gene expression programs in *S. pombe.* Our results unravel the complete picture of the functional cross talks between Cdc2 and Spc1 regulated physiological events that sustain stress response and ensure cell survival in stress conditions.

ST7/P42

Role of eisosome proteins, Pil1 and Lsp1, mitophagy and cell death in *Saccharomyces cerevisiae*

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Plasma membrane of yeast requires high degree of organization as it carries out the diverse array of functions apart from forming the protective barrier around the cell. Eisosomes are a complex of cytoplasmic proteins which mark the sites of endocytosis at the plasma membrane. In addition, they are also known to regulate the cellular levels of phosphatidylinositol(4,5)bisphosphate [PI(4,5)P2], sphingolipid homeostasis, and maintain membrane reservoirs for plasma membrane expansion. Pil1 and Lsp1 are the major components of eisosomes. Though eisosomes are static structures, recent study has shown that Pil1 appears to come on and off these structures. Our experiments show that Pil1 and Lsp1 localize to mitochondria. Also, we demonstrate that Pil1 overexpression induces cell death which can be rescued by human anti-apoptotic protein bcl-xl. Interestingly, we find that Pil1 is also required for autophagy as well as mitophagy. Hence, overall study shows that Pil1 plays various non-

canonical functions in cell apart from its function as eisosome component.

ST13/P43

Understanding the pathobiology of mixed-species candidiasis

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Candidiasis of tissues ranges from superficial mucocutaneous infection to invasive disease involving multiple organs. Access to the bloodstream requires penetration of mucosal barriers, and infection of internal organs requires invasion of endothelia, both of which depend on the yeast to hypha (Y-to-H) transition – a morphological switchover in *Candida.* The most prevalent species - *C. albicans* - undergoes morphological transition in response to various factors including growth condition and molecules of different origins. However, the second most prevalent species *C. glabrata* does not exhibit morphological switchover. *C. glabrata* is usually co-isolated with *C. albicans* from clinical samples. The frequency and potency of *Candida* co-infection make the communication between the two species

intriguing. We hypothesized that inter-species interactions might be mutually beneficial for them to strike a harmony and enhance virulence. To better understand the pathobiology, various approaches such as mutant library screening, biochemical and analytical chemistry studies and various co-culture assays are being utilized. Our preliminary findings show that *C. glabrata*, which does not form hyphae, rather has the ability to induce Y-to-H transition in *C. albicans*, with the help of a novel secreted molecule. Overall, findings from this study would help decipher the intricate molecular communication between different *Candida* species.

ST12/P44

Laboratory evolution of allopatric speciation in *Saccharomyces cerevisiae*

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In nature, organisms evolve to best fit their ecological niches, and while doing so, arrange themselves into discrete, reproductively isolated groups called species. While we know that an evolutionary response of an organism is shaped by adaptive mutations, random genetic drift, and bottlenecks, the fundamental forces driving speciation events are largely unknown. However, why an adaptive process defined by interaction between a genome and an environment, should dictate a process which is defined by interaction of two distinct genomes is not clear. While a number of theoretical models have been proposed to explain speciation, no systematic experimental characterization exists. This lack of understanding of speciation events forms the basis of our work.

In this context, we use *Saccharomyces cerevisiae* as the model system, and characterize contributions of adaptation towards distinct environments and mutation-order towards dictating allopatric speciation. We evolve three parallel lines of haploid yeasts in glucose, galactose, glucose + galactose or melibiose as the carbon source for a few hundred generations. Degree of speciation was quantified by using decrease in mating efficiency. With this study we aim to systematically study speciation and decouple the relative contributions of factors contributing to speciation dynamics.

P45

Investigating the role of Nrd1p-Nab3p-Sen1p (NNS) complex in the function of the nuclear exosome in *Saccharomyces cerevisiae*

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In the nucleus of *Saccharomyces cerevisiae*, aberrant/defective messages generated due to error-prone nuclear mRNP biogenesis events are selectively degraded by the nuclear exosome and its co-factors, TRAMP (**TR**f4p/5p-**A**ir1/2p-**M**tr4p-**P**olyadenylation), CTEXT (**C**bc1p **T**if4631p dependent **EX**osome **T**argeting) and NNS (Nrd1p-Nab3p-Sen1p) complex. Choice of the faulty mRNA target is a key event in the specificity of the mRNA surveillance and in the fidelity of gene expression in eukaryotes, which currently remains an enigma. TRAMP assists exosome to degrade faulty messages generated at the early phase of mRNP biogenesis, whereas CTEXT helps exosome to degrade defective messages generated in the later phase of mRNP biogenesis. Unlike TRAMP and CTEXT, NNS complex is required for the degradation of all kinds of aberrant messages. However, the

functional relationship between these co-factors is largely unknown.

Here, we were trying to investigate the functional relationship between NNS complex with the nuclear exosome/TRAMP/CTEXT. Previous data indicated that perhaps NNS complex plays a crucial role in the co-transcriptional recruitment of TRAMP and CTEXT complexes onto various kinds of aberrant messages. Note that NNS itself is recruited onto various kinds of aberrant messages in either RNA polymerase II dependent or independent manner. However, the exact order of co-transcriptional recruitment of NNS, TRAMP, CTEXT and Exosome onto diverse kinds of aberrant messages is currently unknown. So, we would like to study the order of their co-transcriptional recruitment onto various aberrant messages in a systematic manner.

P46

Identification of centromeres in the rapidly emerging multidrug resistant pathogen *Candida auris* reveals centromere inactivation during speciation

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The haploid ascomycete *Candida auris* has recently emerged as a nosocomial pathogen in four different geographical zones simultaneously, raising global concern. Biochemical similarities to the members of closely related *Candida haemulonii* complex, multidrug resistance and resistance to common sterilization agents render it a difficult organism to detect, treat and eradicate. As a step towards understanding its karyotype plasticity, we identified seven centromeres corresponding to seven chromosomes in *C. auris.* Centromeres in the East Asian clade (the first isolation was in Japan in 2009) were found to be involved in rearrangements, thus setting this clade apart from rest of the clades. This cements the current notion of the Japanese clade being closest to the ancestral *C. auris*

genome. Centromeres in three related species- *C. haemulonii*, *C. duobushaemulonii* and *C. pseudohaemulonii*- were computationally identified. The centromeres in all these species are similar in features. They are small regional, ORF-free, GC-poor regions which are devoid of associated repeats. We compared these centromere neighbourhoods with those in Clavispora, a sister clade. We report an instance of centromere inactivation in *C. auris* and the related species with respect to Clavispora, leading to specific chromosome number reduction. We also map synteny breakpoints to centromere vicinity suggesting centromere fragility.

FT6/P47

To gain insights into the Drp1 related disorders through analyses of equivalent orthologous mutations in budding yeast Dnm1

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Evolutionarily conserved GTPase family member, dynamin related protein 1 (Drp1) together with its accessory proteins forms a complex regulatory network that is essential to maintain the dynamic behaviour of organelles like mitochondria and peroxisomes. Random mutations in Drp1 contribute to the pathophysiology of several diseases that also includes neurodegenerative disorders. The yeast homologue dynamin like protein 1 (Dnm1) enables us to perform mutations similar to the disease causing mutations in Drp1 to understand the underlying mechanisms that regulate the function of the protein and organelle dynamics controlled by this protein.

In this study we used budding yeast Dnm1 as a model to gain insights into the mechanistic details caused by specific Drp1 related mutations associated with diseases like infantile Parkinson's, childhood epileptic encephalopathy, refractory

epilepsy and horizontal nystagmus. Orthologous disease mutations Dnm1-C481F, Dnm1-R438C, Dnm1-G397D and Dnm1-A430D in a plasmid expressing full length Dnm1 fused to GFP were obtained by site directed mutagenesis. The obtained plasmids were transformed into *Saccharomyces cerevisiae* and analysed for mitochondrial and peroxisomal morphology and intracellular localization and dynamics of Dnm1. Furthermore, effect of the mutations on the structure and/or function of the protein are currently being studied by in silico analyses.

ST5/P48

Relevance of 7-transmembrane receptor protein Rta2 in coordinating endoplasmic reticulum stress responses in *Candida albicans*

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C. albicans genome has three Rta1-like genes– *RTA2*, *RTA3* and *RTA4*. This fungal Rta1-like family of proteins may function as novel potential antifungal targets as they are unique to the fungal kingdom and do not have a murine or human homolog. Both Rta2 and Rta4, the downstream effector molecules of calcineurin pathway, are upregulated upon tunicamycin (TM) exposure. Rta2 is requisite to cope with tunicamycin induced ER stress in a Hac1-independent manner. Additionally, it also helps the cells to regain ER homeostasis after ER stress by attenuating the unfolded protein response. Transcriptional profiling of *rta2*Δ/Δ cells revealed genes enriched for the gene ontology (GO) processes related to biofilm formation, ribosomal biogenesis, cell wall and mitochondrial function. Consistent with the differential regulation of biofilm associated

genes in the transcriptional profiling data, *rta2*Δ/Δ showed in vivo biofilm defect. Sequence homology of these Rta proteins revealed the presence of signature sequence, evolutionarily conserved in the extracellular loop of these 7-transmembrane receptor proteins and the least conserved cytosolic C-terminal region. We show that the signature sequence of Rta2 do have a key role in providing tolerance to tunicamycin and promotes the cells to regain ER homeostasis, thus serving as a potential target for the antifungal drug development. This study for the first time shows the relevance of a 7-transmembrane receptor protein Rta2 during ER stress in *C.* albicans, with implications in future antifungal therapy.

P49

A blueprint of the protein secretion machinery in *Neurospora crassa*

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Protein secretion is a fundamental process for host pathogenesis, cellular communication and maintaining cellular homeostasis. In eukaryotes, protein secretion involves a network of organelles from cytoplasm to cell membrane. Elucidating the protein secretion machinery in filamentous fungi has critical applications for the development of hypersecretion strains for novel enzymes and/or understanding the host-pathogen interactions in fungal diseases. In this direction, we have built the first genome-scale model of the protein secretion system in *Neurospora crassa*. Our computational pipeline involves a combination of genomics tools and literature mining. Firstly, we have manually compiled and curated from literature the different components

of classical secretion pathway in *N. crassa,* with experimental and/or computational evidence supporting the annotation. This led to predict functions for several proteins with unknown function in *N. crassa* genome. Secondly, we have captured the protein sorting process by organizing the components into reactions or mechanisms. Post reconstruction, downstream analysis of RNA-seq and ChIP-seq data within the network context has shed new insights on the regulation of the protein secretion system in *N. crassa*. Overall, this integrative analysis provides a systems perspective on protein secretion machinery in *N. crassa* and will have future impact on functional genomics of filamentous fungi.

ST4/P50

Hsp110 mediated proteasomal degradation of Hsp70 chaperone associated substrates in yeast

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Proteins are essential bio-molecules that exist in all living cells and are critical to all physiological processes. However, many cellular events such as molecular crowding, translational errors, thermal stress, and oxidative damage subject the newly synthesized and the natively folded proteins to off-pathway reactions, which produce misfolded proteins and create proteotoxicity. Cells are equipped with several classes of regulatory components to buffer proteotoxic stress produced by misfolded protein species. The major regulatory components include, molecular chaperones, the ubiquitin proteasome system (UPS), and the autophagy system. Molecular chaperones and UPS plays essential role in the selective degradation pathways including both native and misfolded cellular proteins. Proteins belonging to Hsp70 chaperone and Hsp110 families (Sse1 and Sse2 proteins in yeast Saccharomyces cerevisiae) functions in the UPS mediated protein quality control pathway. However, the

molecular link between Hsp70-Hsp110 chaperone complex and UPS is a poorly understood mechanism. In the current study we report that Hsp110 protein function in the turnover of aggregation prone proteins involving both ubiquitin dependent and –independent proteasome substrates. Hsp110 is essential to keep Hsp70 associated substrates soluble and interacts with 19S regulatory particle of the proteasome, suggesting coordinated recruitment of Hsp70-substrate complexes to 26S proteasome for proteolysis. By using a highly defined ubiquitin independent proteasome substrate, we found that introduction of a single Hsp70 binding site render its degradation dependent on Hsp110. The findings define how cellular proteins use Hsp110 to get to the proteasome and to get rid of Hsp70 chaperones that sterically block degradation with profound implication for understanding cellular protein quality control and stress management.

P51

Transcriptome-wide alternative splicing and isoform usage during the biofilm growth phase in *Candida glabrata*

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Biofilm formation is considered as a potential virulence factor in the pathogenesis of *Candida glabrata*. Nevertheless, our understanding of how different genes coordinately regulate this complex process is rudimentary. To delineate this, transcriptomics based approach such as high-throughput RNA Seq by Next Generation Sequencing methods is the best option.

In the present study, we have not only investigated the transcriptomics profiles by quantification of gene expression and the analysis of differential gene expression (DGE) between experimental conditions (planktonic vs. biofilm growth phase) of *Candida glabrata*. In addition to this, we have also

identified, annotated, and visualized the alternative splicing and isoform switches with functional consequences (loss or gain of a protein domain, etc.). Isoform Switch AnalyzeR, which is the latest R based open-source tool of Bioconductor, was used to analyze the RNA-Seq data generated from the mature biofilm growth phase of *Candida glabrata*.

Alternative splicing is the process responsible for determining the diversity in the eukaryotic proteome and differential usage of isoform from the same gene plays a significant role in the stress and disease condition of a cell. So in our study, we attempted to decipher the transcriptome-wide alternative splicing and isoform usage in the biofilm growth phase of a clinical isolate of *Candida glabrata* isolated from Candidemia patient.

FT12/P52

A genome-wide screen identifies several genes essential for maintaining nuclear architecture and uncovers novel pathways in *S. cerevisiae*

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Nucleus, the largest organelle of the cell, dictates a plethora of cellular processes. It houses the chromatin, bordered by a double membrane bilayer. The outer and the inner nuclear membranes are continuous except at the sites where nuclear pore complexes are embedded. Overall nuclear organization is dependent on proper metabolite exchange, signal transduction, membrane re-modelling events and autophagy related processes among others. Efficient regulation of these activities is, to a large extent, dependent on the architecture of the nucleus.

An evaluation of the genome-wide screen undertaken in lab to look for genes involved in maintenance of nuclear architecture revealed that gene products that participate in diverse processes and localize to various non-nuclear sites affect the

localization of inner nuclear membrane protein- Esc1 and distribution of nuclear pore complexes. This includes genes associated with chromatin remodelling, ribosome biogenesis, kinases and their regulators, membrane and protein homeostasis, and Ulp1- a SUMO protease. The role of non-essential genes in maintaining nuclear shape and function in yeast will be presented.

P53

Proximity and homology guided complex-translocations among the centromeres drive karyotype evolution and centromere type transition in *Candida* species complex

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Despite their functional conservation, the centromeres remain one of the fastest-evolving loci among the closely related species. One prominent case of centromere type transition is found within closely related *Candida* species complex: *Candida albicans* carries epigenetically regulated unique centromeres but *Candida tropicalis* carries homogenized inverted repeat (HIR)-associated centromeres capable of *de novo* Cse4 recruitment on the CEN-ARS plasmid *in vivo*. To study this rapid transition in centromere structures, we assembled the *C. tropicalis* genome in seven gapless chromosomes by combining short-read and long-read sequencing technologies together with chromosome

conformation capture sequencing (3C-seq). Using this assembly, we demonstrate spatial clustering of centromeres as well as telomeres in *C. tropicalis*. Mapping of the inter-chromosomal synteny breakpoints (ICSBs), signature of ancient translocations, in the genome of *C. tropicalis* with reference to *C. albicans* shows an enrichment specifically near the centromeres and telomeres over the chromosomal arms. Similarly, we found ICSBs at the HIR-associated loci on seven out of eight chromosomes in *Candida parapsilosis* genome, with reference to *Candida orthopsilosis.* Finally, identification of cross-species conserved HIR-associated centromeres in *Candida sojae* and *Candida viswanathii,* suggests complex-translocations among the ancestral HIR-associated centromeres initiated a transition from HIR-associated ancestral type to unique centromeres in *Candida* species complex.

P54

Histone H4 dosage modulates DNA damage response in the pathogenic yeast *Candida glabrata* via homologous recombination pathway

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Candida glabrata, a nosocomial fungal bloodstream pathogen, causes significant morbidity and mortality in hospitals worldwide. The ability to replicate in macrophages and survive a high level of oxidative stress contributes to its virulence in the mammalian host. However, the role of DNA repair and recombination mechanisms in its pathobiology is still being discovered. Here, we have characterized the response of *C. glabrata* to the methyl methanesulfonate (MMS)-induced DNA damage. We found that the MMS exposure triggered a significant downregulation of histone H4 transcript and protein levels, and that, the damaged DNA was repaired by the homologous recombination (HR) pathway. Consistently, the reduced H4 gene dosage was associated with increased HR frequency and elevated resistance to MMS. The genetic analysis found CgRad52, a DNA strand exchange-promoter

protein of the HR system, to be essential for this MMS resistance. Further, the tandem-affinity purification and mass spectrometry analysis revealed a substantially smaller interactome of H4 in MMS-treated cells. Among 23 identified proteins, we found the WD40-repeat protein CgCmr1 to interact genetically and physically with H4, and regulate H4 levels, HR pathway and MMS stress survival. Controlling H4 levels tightly is therefore a regulatory mechanism to survive MMS stress in *C. glabrata*.

P55

Deletion of the non-essential Rpb9 subunit of RNA polymerase II results in pleiotropic phenotypes in *Schizosaccharomyces pombe*

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The Rpb9 subunit is the only non-essential subunit of *Schizosaccharomyces pombe* RNA polymerase II. However, its functions are poorly characterized in *S. pombe*. The results presented in this study demonstrate that Rpb9 is involved in regulating growth under optimum and certain stress conditions, as well as in regulating transcriptional elongation in *S. pombe*. Deletion mutant analysis show that the region of the Rpb9 subunit spanning the amino-terminal zinc finger domain and the linker region of Rpb9 is able to rescue the phenotypes associated with *rpb9*$^{+}$deletion. Our computational analysis reveals that the two largest subunits of RNA polymerase II, Rpb1 and Rpb2, seemed to lie in close proximity to the Rpb9 subunit. Moreover, the interaction of Rpb9 with the Rpb1 and Rpb2 subunits is not equally distributed over the three different

domains of Rpb9. The N-terminal zinc finger domain protrudes from the structure and is perhaps necessary for primary recognition of DNA, while the C-terminal is a stabilising secondary DNA recognition motif, and the linker region may be required more to anchor the N-terminal than the C-terminal domain to the rest of the polymerase enzyme. Furthermore, the functions of this subunit are partially conserved in budding yeast and humans.

P56

Understanding the role of Wat1 in TOR pathway and in cellular homeostasis

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The coordination between cell growth and nutrient availability is very essential for cell survival. TOR pathway (nutrient sensing) is highly conserved from yeast to mammals and form two distinct complexes TORC1 and TORC2. Both complexes contain four evolutionary conserved regulatory subunits. TORC1 consists of Tor2, Raptor homolog Mip1, Wat1/pop3, Toc1, and Tco89 subunit while TORC2 contain Tor1, Rictor homolog Ste20, sin1, Wat1 and Bit6. Wat1 is a significant component of TOR pathway and has pleiotropic roles. Wat1, a component of both TOR complex exhibit synthetic lethality with checkpoint kinase Chk1 and present in a complex containing phosphoinositol kinase family proteins Tor1 and Tor2. We have shown that in response to stress the Wat1 protein is phosphorylated at S116 residue. Further, the phosphodeficient mutant protein exhibit reduced expression as compared to wild type which was restored to normal level in a

phophomimetic*wat1S116D* mutant. Genetic interaction analysis suggest that wat1 works upstream to the other TOR complex proteins tor1, ste20 and sin1. Co-immunoprecipitation data revealed the physical interaction of Wat1 with Sin1 and wat1 -17 mutant was unable to interact with Sin1. We have also observed the interaction of Wat1 with Chk1 protein. Previously we have shown that wat1 is required for oxidative stress response and maintain cellular homeostasis. Disruption of wat1 leads to the formation of ROS and cell death with fragmented nuclei at non permissive temperature. Using mitochondrial membrane potential sensitive dyes, we have shown that in the absence of Wat1 the mitochondrial membrane potential is reduced suggesting that it might be involved in maintaining the mitochondrial integrity.

P57

Siz2 promotes rDNA stability by modulating levels of Tof2 in *Saccharomyces cerevisiae*

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rDNA stability in budding yeast is regulated by multiple converging processes including post-translational modifications such as SUMOylation. We find that the absence of a SUMO E3 ligase, Siz2, results in increased unequal rDNA exchange. We show that Siz2 binds to the RFB at the rDNA and also controls the homeostasis of Tof2 protein. siz2Δ results in increased accumulation of total Tof2 in the cell and a consequent increase in the binding of Tof2 at the rDNA. Overproducing Tof2 ectopically or conditional over-expression of Tof2 also resulted in higher levels of rDNA recombination suggesting a direct role for Tof2. Additionally, our ChIP data indicates that the accumulation of Tof2 in a siz2Δ resulted in an enhanced association of Fob1, an RFB binding protein at the rDNA at the RFB. This increased Fob1 binding may have

resulted in the observed rDNA recombination. Our work demonstrating that the Tof2 levels modulate recombination at the rDNA will be presented.

Key words: rDNA recombination, Siz2, Ulp2, Fob1, Tof2, SUMOylation

P58

Protein profiling of *Aspergillus parasiticus* var. *globusus* on exposure to Amphotericin B

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Aspergillus parasiticus var. globusus (ATCC151517) is highly toxigenic strain which produces four types of aflatoxins (AFB1, AFB2, AFG1 and AFG2) pose a potential risk to human health. However, the aflatoxin production is initiated with sporulation. Amphotericin B is most preferred drug of choice against *Aspergillus* infections. Thus, present study we have planned studies on proteomic changes associated with modulation, morphological and physiological features of *Aspergillus parasiticus* in response to Amphotericin B. *Aspergillus parasiticus var. globusus* grown on PDA containing amphotericin B (7.5/ml) yielded 1.6gm of mycelia after five days of incubation at 28 $\pm$ 2 while that of control yielded 2.5gm of mycelial mat. Although biomass was more in case of control, protein yield was more in test sample. SDS page analysis

localize nine bands in test and eight bands in control. It means eight common bands were observed in presence and absence of amphotericin B. Among the common band No. 1,4,6,9 are overexpressed in control while remaining four 2, 3,7,8 are overexpressed in presence of amphotericin B indicating effect of amphotericin B on protein expression. In addition to these bands no. 5(rf .392) is missing from control. In present study Amphotericin B inhibited sporulation and aflatoxin biosynthesis while aerial hyphae were induced.

Key words: - Aspergillus, Aflatoxin, Amphotericin B, Conidia, protein profiling.

ST10/P59

Glycosylphosphatidylinositol-linked aspartyl proteases regulate the secretome of the pathogenic yeast *Candida glabrata*

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Candida glabrata is an opportunistic human fungal pathogen, which accounts for upto 30% of total *Candida* bloodstream infections. A family of eleven putative GPI (glycosylphosphatidylinositol)-anchored aspartyl proteases, also referred to as CgYapsins, is essential for intracellular survival and pathogenesis of *C. glabrata*. Using an approach of molecular, transcriptomic and proteomic analysis, we elucidated the mechanism underlying CgYapsin-mediated regulation of virulence. We showed that CgYps1-11 yapsins are required for suppression of the spleen tyrosine kinase (Syk)-dependent pro-inflammatory response (IL- 1β production) in human THP-1 macrophages. Additionally, we showed that CgYapsins are required for biofilm formation and colonization of murine organs in a disseminated candidiasis

model. Further, characterization of the secretome of *C. glabrata wild-type* and *Cgyps1-11Δ* mutant revealed that the wild-type secretome consisted of 119 proteins, which were primarily involved in cell wall organization, carbohydrate metabolism, proteolysis and translation processes. Strikingly, the *Cgyps1-11Δ* secretome was found to be 4.6-fold larger, and contained 65 differentially abundant proteins, as revealed by label-free quantitative profiling, with 49 and 16 being high- and low-abundant proteins, respectively, compared to the *wild-type* secretome. We will discuss how CgYapsins, being both bona-fide constituents and key modulators of the *C. glabrata* secretome, aid in establishment of successful infections.

FT15/P60

Aspergillus terreus strain improvement for biodiesel production using Agro-waste substrate

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Biodiesel (fatty acid methyl- or ethyl-esters or FAME) has been proved to be a promising alternative fuel derived from the cellular lipid of various yeast and filamentous fungi. For the efficient application of these oleaginous fungi, we carried out mutagenesis of *Aspergillus terreus* by random chemical mutagenesis approach using Methy - 2-Nitro-1-Nitrosoguanidine (MNNG). Screening for lipid content of the mutants was done by Nile red flourimetery followed by confirmation experiments on sugarcane bagasse substrate.

Sugarcane bagasse detoxified liquid hydrolysate as the complex carbon sorce along with a lipid accumulation media showed FAME yields of 17.93 and 36.5mg/g in the wild type and TB21 mutant of *A. terreus* respectively. FAME yield of TB22 mutant was almost two times (40.2mg/g) as compared to the wild type (19.9 mg/g), when grown on acid hydrolysed solid bagasse residue.

Fungal biorefinery's time and cost efficiency was boosted by generating FAME by direct in-situ transesterification method from fermented fungal biomass. Time course experimentation of the wild type gave an insight into the sugar utilization pattern and simultaneous biomass and FAME accumulation in the fungi. This preliminary data would aid in fermentation media optimization by PBD and fuel properties analysis experiments which are in the pipeline.

Keywords: *Aspergillus terreus*. Chemical Mutagenesis. Agro-residues. In situ acid transesterification. FAME. Biodiesel.

P61

Saccharomyces boulardii: A systematic review on current scenario on metabolic engineering of probiotic yeast strain

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Saccharomyces boulardii (S. boulardii) is a probiotic yeast strain and has nearly cent percent homology to *Saccharomyces cerevisiae (S. cerevisiae)* but the small differences in genome architecture somehow leads to their probiotic characters such as resistance to high acidic environment, survival in high temperature and inability to form ascospores. *S. boulardii* is generally used to cure different GIT-related disorders and pathogenic infections. However, recent studies also showed that *S. boulardii* is well suited as a vector for therapeutic purposes. Hence, the metabolic engineering of *S. boulardii* with the help of different genome editing techniques such as UV mutagenesis, transformation, CRISPR-Cas9 system are used to develop auxotroph, recombinant therapeutic molecules and vectors that produces molecules of interest. Furthermore, there is more need to focus on diverse aspect of *S. boulardii* and enhance their usability as a probiotic

as well as therapeutic molecules, and also to make auxotroph which is not relay on antibiotic markers. To study a lot about molecular mechanisms and its machineries lead to morphological and physiological changes under different stress condition may allow us to know the behaviour of cell survival of this probiotic strain.

P62

Investigating the role of PEX25 in peroxisome biogenesis in yeast

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Peroxisomes are single membrane bound dynamic organelles. The ability of yeast peroxisomes to proliferate depending on the growth media has made them attractive system for studying molecular details of peroxisome biogenesis. Peroxisomes multiply by growth and division *i.e.* by the fission of pre-existing peroxisomes. In yeast, the peroxins which play a vital role in peroxisome proliferation belong to the Pex11 family (Pex11, Pex25 and Pex27). The role of Pex11 in peroxisome biogenesis is studied extensively, while the role of Pex25 in this process is not fully understood. Another interesting finding is the role of peroxisome proliferation in ageing of yeast cells.

In this study we aim to characterize the role of Pex25 in peroxisome biogenesis and ageing in *Saccharomyces cerevisiae*. For this, expression and localization of C and N-terminal GFP fusion plasmids of Pex25 were studied in wild

type cells under peroxisome inducing and non-inducing conditions. In addition, the expression of both the constructs was also analyzed in strains deleted for the interacting partners of Pex25. The chronological and replicative lifespan of *pex11*, *pex25* and *pex27* cells was analyzed to understand the role of peroxisomes in ageing.

P63

Investigating the role of peroxisomes in Parkinson's disease

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Parkinson's disease (PD) is a neurodegenerative disorder of the central nervous system which is characterized by the presence of α-synuclein protein inclusion (lewy bodies) in the brain and loss of dopaminergic neurons in substantia nigra. The etiology and pathogenesis of PD is still unclear. However, large number of evidences suggest imbalance between the production and elimination of ROS as one of the major contributors to the pathogenesis of PD. ROS is produced by two cellular organelles, mitochondria and peroxisomes and dysfunction of any of these leads to accumulation of ROS. Most PD related studies are limited to mitochondria and not much is known about the role of peroxisomes. ROS production and scavenging is one of the most important functions of peroxisomes.

Hence, in this study we investigated the effect of peroxisome proliferation and function on α-synuclein aggregation and toxicity in yeast models of PD. For this, a construct with GFP tagged α-synuclein under control of GAL promoter was made and transformed into mutants defective in peroxisome biogenesis and protein import. Cell growth, cell viability, expression and aggregation of α-synuclein were analysed in the mutant cells to understand the role of peroxisomes.

FT16/P64

Understanding the role of Exocyst: a Tethering Complex in Autophagy

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Macroautophagy or autophagy is an evolutionarily conserved intracellular degradation process in eukaryotes. Autophagy involves the formation of double membrane vesicles called autophagosomes that capture cytosolic cargoes and deliver it to vacuoles or lysosomes for degradation and the broken down byproducts are eventually recycled. Yeast as a model system has played a critical role in identifying the genes involved in autophagy. Despite being one of the most extensively studied fields the exact mechanism of autophagosome biogenesis is not well understood. To find new players in autophagy, a genetic screen carried out in our lab identified several essential and non-essential genes. Among these, a subset of genes that are part of the tethering complex exocyst were further characterized. Exocyst is known to tether vesicles to plasma membrane and is critical for the secretory pathway. The temperature sensitive mutants of exocyst subunits showed

blocked autophagy at non-permissive temperature. However, the exact mechanism by which exocyst is involved in autophagy is still a mystery. I will highlight some of the biochemical and fluorescence microscopy data that has led to our current working model. This model proposes the existence of an autophagy specific exocyst sub-complex which is involved in early steps of autophagosome biogenesis.

P65

Identification of novel inhibitors targeting 14-alpha demethylase (Erg11p) of *Candida albicans* using high throughput virtual screening combined molecular dynamics simulations (HTVS-MDS) approach

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14-alpha demethylase (Erg11p) is the specific drug target for antifungal drug azoles; which is the primary drug of choice in the treatment of Candida infections. Resistance to these antifungal drugs are increasing enormously. Therefore, there is an urgent need in finding alternative antifungal drugs to combat against this resistant pathogen. In this study, we used a high throughput virtual screening combined molecular dynamics simulation (HTVS-MDS) approach to find the novel inhibitors for 14-alpha demethylase of *Candida albicans*. Crystal structures of Erg11p (5FSA, 5TZ1, and 5V5Z) were used in this study. Ligand molecules were obtained from the enamine advanced collection database. Molecule screening was done using a three step (HTVS, SP and XP) docking method present in the Glide, Schrödinger suite 2017-1.

Docking results identified three least binding energy molecules Z14003605, Z54358605, and Z1444489827, which is common in all the three structures. Molecular dynamics simulations for 25 nanoseconds revealed that all the three molecules showed a stable root mean square values. Molecules that showed greater binding interaction with Erg11p from the highest to lowest are Z54358605, Z1444489827, and Z14003605 respectively. In our conclusion, these molecules may be potent inhibitors of 14-alpha demethylase and further experimental verification will be done in future.

P66

The high-osmolarity glycerol (HOG) MAPK pathway regulates iron homeostasis and virulence in *Candida glabrata*

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Mitogen-activated protein kinase (MAPK)-mediated signalling is essential for cell adaptation to various extracellular stresses. In this study, we have investigated the functions of the high-osmolarity glycerol MAPK, CgHog1, in the adaptation and survival of *Candida glabrata* to limited and surplus iron environment. It was shown earlier that the *Cghog1Δ* mutant contained high amounts of intracellular iron and expressed higher levels of cell surface adhesins. Consistently, the *Cghog1Δ* mutant was hyper-adherent to epithelial cells. In the current study, we identified the interactome of CgHog1, via immunoprecipitation-mass spectrometry approach, under low- and high-iron conditions. We found a set of 33 proteins to interact with CgHog1. One of the identified interactor was CgSub2, a putative DEAD box-RNA helicase. After verifying CgHog1-CgSub2 interaction, we created and characterized a conditional mutant of *CgSUB2*. We showed that *CgSUB2*

depletion render *C. glabrata* cells hypo-adherent, owing to diminished expression of cell surface adhesins. Additionally, we showed that CgSub2 interacts genetically with CgHog1. We also identified 55 proteins that interact uniquely with CgSub2 in the *Cghog1Δ* mutant. These findings along with phosphosite-mapping of CgSub2 will be presented.

P67

Evaluation of pathogenic potential of various DNA polymerase knock-out strains of *Candida albicans* and its implication in the development of live attenuated antifungal vaccine

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Candidemia is a bloodstream fungal infection caused by Candida species. Even with proper antifungal drug treatment, mortality rates remain high at 40–50%. Adverse side effects, toxicity, and emergence of drug resistance limit the use of currently available drugs. Therefore, to reduce the incidence and mortality, active research is needed to improve diagnostics to allow more rapid implementation of appropriate therapies, discover effective antifungal agents with less severe side effects, and to develop immunotherapies. A suitable anti-Candida vaccine could help in reducing global burden of both systemic and vaginal candidiasis bringing about substantial socioeconomic benefits. Since, we could generate an array of DNA polymerase null *C. albicans* strains, and some of these

strains exhibit morphological defects, the long term goal of our laboratory is to identify and characterize pathogenic potential of genetically modified *C. albicans* strains as a resource for the development of live attenuated vaccine in order to achieve anti-fungal immunity not only in immunocompetent hosts but also for immune-compromised individuals. A preliminary study of these knock outs will be discussed.

P68

Understanding the molecular mechanism of zinc transporter genes in *Neurospora crassa*

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Zinc is an essential nutrient for all cells, and various zinc transporters function to transport zinc to different cellular compartment. The zinc regulated gene 17 (ZRG17) belonging to cation diffusion facilitator (CDF) family of proteins localized in the endoplasmic reticulum helps in maintaining the zinc ion homeostasis and expression in zap1 dependent manner. Using in silico analysis we have identified a probable zinc finger transcription factor *znf14* to be the *zap1* homolog in *Neurospora crassa* (*N. crassa*) having 46% sequence similarities with *zap1* of *Saccharomyces cerevisiae*. Mutant of *znf14* have shown slow growth, stunted aerial hyphae under zinc deficient condition, and expression of *znf14* was increased in response to low zinc in wild type. Also, the double mutant of *zrg17* with *msc-2* did not show any additives phenotypes suggesting that these two might be functioning in the same pathway rather than parallel pathway. Therefore, we are

investigating probable interaction of ZRG17 with MSC-2.This study will provide us with a better understanding to the molecular function of ZRG17 in maintaining zinc ion homeostasis.

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Direct transesterification for biodiesel production using wet biomass of *Yarrowia lipolytica* NCIM 3589 and its mutant grown on waste cooking oil

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Direct transesterification step for biodiesel production using wet biomass of wild type and C7 mutant of *Yarrowia lipolytica* grown on widely available waste cooking oil (WCO) is studied.

Physiochemical characterization of WCO substrate showed batch to batch variation in WCO properties but that doesn't affect FAME yield and its quality in both of wild type and mutants.

Different methods for biodiesel production carried out and the biomass grown on lipid accumulation medium with WCO gave a high FAME yield of 0.42 g L^{-1} in 6 h at 60^0C with 30 ml methanol and acid catalyst (Conc. H_2SO_4, 3 ml g^{-1}) by direct transesterification of wet biomass method. This FAME yield was comparatively higher than FAME obtained from two step

transesterification reaction and direct transesterification of dry biomass which is 0.15 g L^{-1} and 0.22 g L^{-1} respectively.

The FAME profile and its physico-chemical properties were found to be suitable for biodiesel production. Thus, the direct one-pot in situ transesterification reaction using wet biomass of *Y. lipolytica* grown on WCO provides a high yield of biodiesel with potential applicability at industrial level while simultaneously addressing the management of this pollutant.

FT17/P70

Ubiquitin mediated clearance of prion protein in vivo

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Protein aggregation disorders such as Alzheimer's, Parkinsons and Prion diseases results in progressive loss of neuronal structure, inducing early ageing, loss of body control and cognitive failure. Prion diseases are misfolding disorders caused by accumulation of infectious form of the normal prion protein. These diseases pose a great threat to humans resulting in severe epidemics such as kuru and can be transferred horizontally amongst animal species. Despite being discovered years ago, mechanism of prion propagation and transmission is not well understood and has limited novel advances in targeted drug development. Strategies involving chemical compounds like anthracyclines or porphyrins and gene knockout using peptide aptamers have been formulated but none have shown remarkable success. Investigations involving ubiquitin are known to clear the protein aggregates associated with neurodegenerative disease. Infact, Ubiquitin proteasome system is shown to be the major degradation pathway for removal of endogenous and overexpressed levels of alpha- synuclein in living mouse brain.

The present study represents a significant step towards understanding the clearance mechanism of aggregating protein, Sup35 in Saccharomyces cerevisiae using ubiquitin mediated approach. Sup35/[PSI+] is perhaps the best-characterized yeast prion and notably, drugs against Sup35/[PSI+] are also actively effective in mammalian prions in cell-based assays.

Keywords: Protein aggregation, clearance, Prion disease, *Saccharomyces cerevisiae*, Ubiquitin

P71

CgYOR1 mediated azole resistance in *Candida glabrata* via TOR and calcineurin cascade.

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The mortality rate due to fungal infections is consistently increasing and posing a serious threat to human health which is further aggravated by the continuously evolving multi-drug resistance. Among fungal infections different *Candida* species are leading cause of invasive candidiasis, wherein *C. glabrata* ranks 2nd to 3rd. *C. glabrata* has intrinsic azole resistance. GOF mutations in transcription factor Pdr1, which upregulates ABC transporters is one of the frequent mechanism of azole resistance in clinical isolates of *C. glabrata*. Therefore, regulatory and functional aspects of ABC transporters need to be addressed in greater detail.

In this study, we screened disruptome library of all the ABC transporters of *C. glabrata* for sensitivity on antifungals, and

only some of them displayed susceptible phenotypes. *CgCDR1* is the major transporter involved in MDR and can mask the contribution of other transporters. Therefore, we analyzed the transporter deletions in *Cgcdr1Δ* background, which indicated a transporter *CgYOR1* could contribute in azole resistance. Reduced efflux activity of *Cgcdr1Δ* /*Cgyor1Δ* was also in agreement with its involvement in azole resistance. Additionally, *CgYOR1Δ* displayed sensitivity to myriocin, rapamycin and FK520, suggesting that TOR signaling, calcineurin pathway and *CgYOR1* mediated azole resistance could be interlinked in *C. glabrata.*

ST11/P72

Investigating the mechanism of regulation of cellular repertoire of *SKS1* mRNA in *Saccharomyces cerevisiae*

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Opportunistic human fungal pathogen *C. albicans* displays a morphological transition from yeast to hyphal state, which has been postulated to mimic its transition from avirulent to pathogenic form upon infection to the host tissue. Strikingly, the budding yeast *Saccharomyces cerevisiae* also exhibits similar transition from yeast to filamentous hyphal state upon growth under nutrient limiting conditions, thus serving as an excellent model system to study the genetics and molecular biology of pathogenic transition.

However, the mechanism of formation of these pseudohyphal filaments and the regulatory signal transduction pathways by which these signaling systems are integrated is poorly understood. The protein kinase Sks1p was implicated in the integration of signals for nitrogen and/or glucose limitation, resulting in pseudohyphal growth in both *S. cerevisiae* and *Candida albicans*. Thus, Sks1p constitutes a mechanism that

integrates glucose-responsive cell signaling and pseudohyphal growth, whose function is required for the virulence in *C. albicans*. However, the regulation of the expression of the *SKS1* gene is completely unknown. Recent findings in our laboratory indicated that *SKS1* is regulated at the post-transcriptional level via a combination of mRNA export and degradation. Data of the experiments substantiating our hypothesis will be presented.

P73

The E3 ubiquitin ligase Pib1 regulates effective gluconeogenic shutdown upon glucose availability

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Cells use multiple mechanisms to regulate their metabolic states in response to changes in their nutrient environment. However, our knowledge of the mechanisms mediating the glucose dependent downregulation of the gluconeogenic transcription factors during 'glucose repression' is limited. Using a major gluconeogenic transcription factor Rds2 as a candidate, here we identify a novel role for the E3 ubiquitin ligase Pib1 in regulating the stability and degradation of Rds2. Glucose addition to cells growing in glucose limitation results in rapid ubiquitination of Rds2, followed by its proteasomal degradation. Through in vivo and in vitro experiments, we establish Pib1 as the ubiquitin E3 ligase that regulates Rds2 ubiquitination and stability. Notably, this Pib1 mediated Rds2 ubiquitination depends on the phosphorylation state of Rds2, suggesting a cross-talk between ubiquitination and phosphorylation to achieve a metabolic state change. Using stable-isotope based metabolic flux experiments we find that

the loss of Pib1 results in an imbalanced gluconeogenic state, regardless of glucose availability. Our results reveal the existence of a Pib1 mediated regulatory program that mediates glucose-repression when glucose availability is restored.

FT18/P74

Understanding the functional relationship between the bZIP transcription factors Atf1 and Pcr1 in regulation of cell division in *Schizosaccharomyces pombe*

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Regulation of cell cycle phase transition is crucial for growth and survival of all living organisms and involves periodic transcription of a number of regulators of cell cycle and division, proliferation and apoptosis, where bZIP domain containing transcription factors play an important role. These transcription factors are found in all organisms and are known to regulate expression of many important cell cycle related genes. Also any aberrations in their function may lead to cancer and various other diseases. One such bZIP transcription factor found in *Schizosaccharomyces pombe* is Atf1 (homologous to ATF2 in mammals) which is known to regulate cell cycle phase transitions and upon overexpression can override G2/M and G1/S checkpoints. In stress conditions, Atf1 activity is indispensable for cell survival. So a mechanism is required to selectively inhibit the cell cycle related activity of

Atf1 during stress. There comes into the scenario, another bZIP transcription factor Pcr1, a binding partner of Atf1, whose expression also increases during stress and inhibits recruitment of Atf1 on promoters of cell cycle related genes to block the phase transitions. In an unperturbed cell cycle, the expressions of Atf1 andPcr1 are periodic in nature and reciprocal to each other. We are trying to investigate how the two transcription factors are able to function without being under the regulation of the other and what impact does the balance in their periodic expression on cell cycle progression in stress conditions.

P75

Studies on the regulation of UPR signaling by nuclear exosome/DRN in *Saccharomyces cerevisiae*

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In *Saccharomyces cerevisiae*, nuclear mRNA degradation controls the regulation of Unfolded Protein Response (UPR) by regulating the degradation of the precursor-*HAC1* mRNA, encoding its key transcription activator Hac1p. UPR is an intracellular signaling pathway that responds to stress causing a burden of unfolded proteins (ER stress) in the ER lumen. In the absence of the stress, the *HAC1* pre-mRNA undergoes a rapid decay by the nuclear exosome/DRN thus producing little or no Hac1p protein output. Under the stress, the decay diminishes, thus producing more Hac1p protein, which transcriptionally activates UPR. In this studies we further investigate how the regulation of differential decay of pre-*HAC1* RNA is accomplished by studying the (i) cross-talk between the Hac1p and different mRNA decay factors by employing co-IP, and (ii) differential recruitment of a variety of nuclear exosome and other decay factors onto this precursor RNA under both stress and unstressed conditions using RNA

immunoprecipitation. In addition, the immunoprecipitated RNA will also be analyzed by qRT-PCR to evaluate the specificity of the interaction. Collective findings should advance our understanding of the molecular mechanism of regulation of activation and attenuation of UPR. Notably, both mRNA degradation and UPR potentially impact the normal cellular hygiene and, consequently, tissue homeostasis. Malfunctions in the exosome and UPR functions have both been linked to cancer, diabetes and neurodegenerative disorders. Therefore, understanding how the balance of UPR tips towards causing a pathophysiological UPR may help develop a potential therapeutic strategy for the treatment of a diverse array of these diseases.

P76

An analysis of *Candida glabrata*-epithelial cell interaction

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Candida glabrata is a haploid budding yeast which resides as a commensal in the oral cavity, and gastrointestinal and genitourinary tracts in healthy humans. It also causes opportunistic mucosal and systemic fungal infections in immunocompromised patients. Phylogenetically, *C. glabrata* is closely related to the non-pathogenic yeast *Saccharomyces cerevisiae*, and these two yeasts share a common ancestor. Although antifungal resistance and intracellular survival mechanisms of *C. glabrata* are currently being studied extensively, the interaction of *C. glabrata* with host epithelial and endothelial cells remains largely unexplored. An interaction with tissue-resident immune cells as well as epithelial and endothelial cells is pivotal to the colonization and invasion of host niches. In the current study, I have established two in vitro model systems, using kidney epithelial cell line A498 and stomach epithelial cell line AGS, and showed that *C. glabrata* displays robust adherence to both cell lines, and a

family of eleven cell surface-associated aspartyl proteases modulates this process of epithelial cell adhesion. Additionally, I have investigated the role of CgYps7 aspartyl protease in tissue adherence, as this protease is uniquely required to survive cell wall stress in vitro. These results along with cytokine secretion upon *C. glabrata*-epithelial cell co-incubation will be presented.

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Chromosome end-adjacent regions (EARs) promote high density of DNA breaks on short chromosomes during sexual reproduction

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Infertility, spontaneous fetal loss and birth defects in humans result mainly from mistakes in chromosome inheritance during meiosis, the cell division that forms eggs/sperm. Faithful inheritance in meiosis requires crossover links between homologue pairs that are created by induction of numerous potentially genotoxic programmed DNA double-strand breaks (DSBs). Consequently, distribution of DSBs to all chromosomes irrespective of their length is critical for fidelity of their inheritance. Additionally, to avoid excessive damage, feedback mechanisms down-regulate DSBs likely in response to initiation of crossover repair. In *Saccharomyces cerevisiae*, this regulation requires the removal of the conserved DSB-promoting protein Hop1/HORMAD during chromosome synapsis. We have identified privileged end-adjacent regions (EARs) spanning roughly 100 kb near all telomeres that escape

DSB downregulation. These regions retain Hop1 and continue to break in pachynema despite normal synaptonemal complex deposition. Differential retention of Hop1 requires the disassemblase Pch2/TRIP13, which preferentially removes Hop1 from telomere-distant sequences, and is modulated by the histone deacetylase Sir2 and the nucleoporin Nup2. Importantly, the uniform size of EARs among chromosomes contributes to disproportionately high DSB and repair signals on short chromosomes in pachynema, suggesting that EARs partially underlie the curiously high recombination rate of short chromosomes.

FT20/P78

MRX8 is essential for Cox1p translation in *Saccharomyces cerevisiae*

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Mitochondrion harbors the OXPHOS machinery that couples majority of ATP production when cells feed on non-fermentable carbon source. *Saccharomyces cerevisiae* utilizes this machinery when glucose is exhausted at the point of diauxic shift. This metabolic switching from fermentable to non fermentable carbon source requires huge cellular reprogramming where a specific gene expression pattern is upregulated in order to support increased biosynthesis and assembly of OXPHOS subunits. GTPases are a major class of proteins required for mitochondrial translation regulation in response to nutrient signaling. *MRX8*, a YihA class of GTPase, has orthologue in bacteria, yeast and vertebrates including humans but none in invertebrates. We have shown that this novel protein Mrx8p helps in cellular adaptation to utilize non-fermentable carbon source. It does not utilize energy from GTP

hydrolysis to carry out a mechanical activity when associated with the ribosomes but rather utilizes nucleotide binding to regulate optimal maintenance of Cox1p translation. Interestingly its association with the mitoribosomes is independent of the guanine nucleotide bound. Finally, we have shown that *MRX8* function is conserved between yeast and humans as human form of Mrx8p partially complements loss of growth on glycerol in *Δmrx8* cells and hMrx8p is localized to mitochondria in mammalian cells.

P79

Raptor/Kog1 regulates AMPK/Snf1 activation for metabolic rewiring under nutrient limitation

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For efficient cell growth, integration of external nutrient and metabolic inputs by various signalling pathways into metabolic outputs is crucial. Two signalling pathways that have essential roles in maintaining metabolic homeostasis are TORC1 and AMPK/ Snf1 pathway. TORC1 is typically active under nutrient rich conditions, whereas the yeast AMPK (Snf1) activity is required under nutrient (ATP) limitation. This suggests opposite regulation of downstream pathways by TORC1 and Snf1, leading to the idea of opposing roles of these pathways. Here, we have identified a mutant of Kog1 (Raptor)-an essential component of TORC1, which revealed a specific growth defect exclusively under glucose and amino acid limitation, where TORC1 kinase activity is low. Combining metabolic flux and comparative transcriptome analysis, we find this growth defect to be a result of delayed metabolic rewiring

under nutrient limitation. This results in an imbalance in the routing of carbon flux towards amino acid biosynthesis and gluconeogenesis. The delayed transcriptional response in Kog1 strikingly correlates with Snf1 dependent targets for the gluconeogenic genes. Further, the Kog1 mutant revealed altered post-translational modifications on Snf1, and subsequent altered phosphorylation status of the primary Snf1 targets – Mig1 and Cat8. Collectively, we uncover a novel mechanism of Kog1 dependent activation of Snf1, which is required for maintaining appropriate carbon flux towards amino acid biosynthesis and gluconeogenesis. Kog1 thereby regulates growth under glucose and amino acid limitation via Snf1.

P80

Understanding the role of phospholipase C-1 and secretory phospholipase A2 in circadian clock and biomass degradation in *Neurospora crassa*

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Neurospora crassa presents a unique model organism to understand the Calcium (Ca^{2+}) signaling machinery with 48 Ca^{2+} signaling proteins involving in the pathway, which is more in number than those identified in rice blast fungus *M. grisea* and the most extensively studied model organism *S. cerevisiae*. However, a little is known about the physiology of Ca^{2+} signaling machinery in *N. crassa* or any other related fungi compared to plants and animals. Previous studies from our laboratory showed *N. crassa* Ca^{2+} signaling proteins phospholipase C-1 (PLC-1) and secretory phospholipase A2 (sPLA2) are important for survival under multiple stress conditions. Here, we show that a strain lacking *plc-1* exhibited defects in circadian clock and *plc-1* regulates the clock function by maintaining a proper level of *frq* and *wc-1*. Recently, *N. crassa* is gaining importance as a model organism to obtain a

more detailed understanding of the physiology of lignocellulose degradation across fungi as near full genome deletion set is available. Surprisingly, when grown on 2 % cellulose as the sole carbon source, the Ca^{2+} signaling mutant Δ*splA2* consumed cellulose at a faster rate compared to the wild type, exhibited significantly increased protein secretion and displayed ER response.

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School of Life Sciences, University of Hyderabad (UoH)

School of Life Sciences was founded in 1977 with the vision of promoting inter-disciplinary teaching and research. The School offers both MSc and PhD programs through the five departments: Department of Biochemistry, Department of Plant Sciences, Department of Animal Biology, Department of Biotechnology and Bioinformatics and Department of Systems and Computational Biology. The School has been funded by DST under the FIST programs, by the UGC under the SAP and by the BUILDER program of the DBT. Support from these external sources along with the support for infrastructure from the University has enabled the setting up of state-of-art facilities to promote cutting edge multidisciplinary research and teaching.

Centre for DNA Fingerprinting and Diagnostics (CDFD)

The Centre for DNA Fingerprinting and Diagnostics (CDFD) was established as an autonomous institute of the Department of Biotechnology (DBT), Ministry of Science & Technology, Govt. of India, in the year 1996. CDFD adopts a hybrid model amalgamating services and research, with both components complementing and enriching each other. The main objectives of CDFD are to provide DNA fingerprinting services to law enforcing agencies, to establish DNA diagnostic methods for detecting genetic disorders, to use DNA fingerprinting techniques for authentication of plant species (e.g. basmati rice), and to undertake fundamental and applied research in frontier areas of modern biology.

Centre for Cellular and Molecular Biology (CCMB)

The Centre for Cellular & Molecular Biology (CCMB) is a premier research organization and a constituent laboratory of the Council of Scientific and Industrial Research (CSIR), India. CCMB was set up initially as a semi-autonomous Centre on April 1, 1977 with the Biochemistry Division of the then Regional Research Laboratory (presently, Indian Institute of Chemical Technology, IICT) Hyderabad forming its nucleus and Dr P M Bhargava heading the new Centre. 1981-82, the CCMB was accorded the status of a full-fledged national laboratory with its own Executive Committee and Scientific Advisory Council. The ongoing research programmes at the CCMB are in three major categories – high quality basic research in the frontier areas of modern biology, research relevant to societal needs, and application-oriented research towards commercialization. These include the areas of biomedicine & diagnostics, evolution & development, gene regulation in prokaryotes and eukaryotes, host-parasite interactions, membrane biology, protein structure, bio informatics, functional genomics, theoretical biology, etc. In recognition for its outstanding contribution to modern biology, CCMB has received many prestigious international and national awards, and has been chosen as a Centre of Excellence by UNESCO Global Network for Molecular and Cell Biology MCBN.