MICROBIOLOGY SOCIETY

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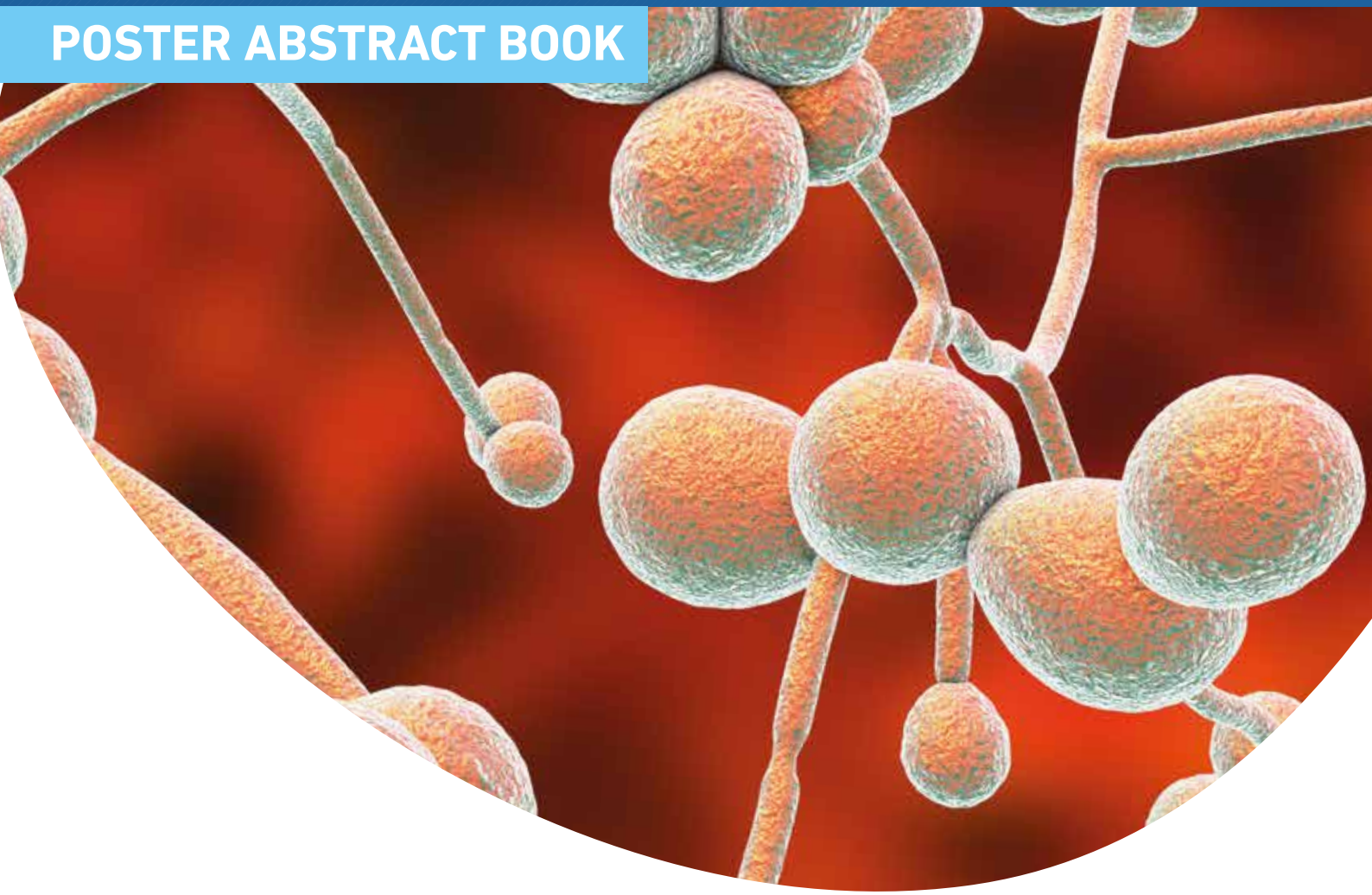
2019

British Yeast Group: Discovery to Impact

26–28 June 2019 County Hotel, Newcastle, UK

POSTER ABSTRACT BOOK



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Poster no: 1

Assessing the ecological role of yeasts in the human gut

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Abstract

Background:

The influence of gut microorganisms in health and disease is well documented, however, the role of yeasts has not been as widely studied as bacteria, despite associations with gastrointestinal disorders, such as Irritable Bowel Syndrome (IBS). For instance, *Candida albicans* is an opportunistic pathogen that has been linked to gastrointestinal symptoms.

Methods:

Urine, stool and blood samples will be collected from 40 healthy controls, 40 IBS and 40 inflammatory bowel disease (IBD) patients for microbial and metabolic analysis using primarily flow cytometry-fluorescence *in situ* hybridisation (FC-FISH) and proton nuclear magnetic resonance (^{1}H-NMR) spectroscopy, respectively. Calprotectin and anti-*Saccharomyces cerevisiae* antibodies (ASCA) levels will assess gastrointestinal inflammation. Statistical analysis will identify yeasts and their metabolites associated with the cohorts of interest.

Preliminary results:

Preliminary tests to assess the presence and functionality of yeasts in healthy and IBS faecal samples using *in vitro* batch culture fermentation highlighted significant metabolic differences between the phenotypes: Trimethylamine (TMA), short chain fatty acids (SCFA) and ethanol predominantly distinguished in the IBS metabolic profile, compared to gamma-amino-N-butyrate (GABA) in the healthy donor. The addition of *C. albicans* shifted the healthy phenotype to resemble the IBS donor, whereas nystatin shifted the IBS phenotype towards the healthy metabolic profile.

Conclusion:

This study will further define the preliminary results that indicate the implication of yeasts in IBS pathogenesis. Understanding the role of yeasts within the healthy and diseased human gut is necessary to develop targeted therapies to improve patient's quality of lives and relieve burdens on the economy and healthcare systems.

Poster no: 2

Investigating the role of RNA-protein interactions in the control of gene expression noise

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Abstract

Noise is an important factor in the molecular biology of many different systems. Here we consider the yeast *Saccharomyces cerevisiae*, and investigate control of gene expression noise as a result of post-transcriptional regulation. Although it has been previously observed that RNA-binding proteins are generally less “noisy” than other proteins, whether RNA-protein interactions could directly affect gene expression noise is not known. In this project we take up this issue and focus in particular on the 3’ untranslated region (UTR) of mRNA transcripts, which is a possible region for auto-regulation through interactions with the encoded RNA-binding protein. Therefore, we are currently generating mNeonGreen-tagged strains in budding yeast, preserving the endogenous 3’ UTRs while allowing sensitive quantification of GFP expression with high-throughput flow cytometry. We further aim to mutate binding sites for RBPs on target 3' UTR motifs using CRISPR-Cas9 mediated knock-out/knock-in experiments, and investigate its effect in gene expression noise. The data produced will clarify the contribution of post-transcriptional noise in gene expression.

Poster no: 3

The response regulator Ssk1 orchestrates stress specific changes in Candida albicans SAPK pathway architecture.

Alison Day[1], Deborah Smith[1], Carmen Herrero-de-Dios[2], Alistair Brown[2], Jan Quinn[1]

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Abstract

The *C. albicans* Hog1 stress activated protein kinase (SAPK) is essential for stress-adaptation and virulence in this human fungal pathogen, however, its structural and functional conservation with analogous human SAPKs (p38/JNK) makes the development of antifungal drugs that specifically target fungal SAPKs challenging. There is therefore much interest in identifying fungal-specific SAPK regulators as potential drug targets. Such targets include two-component related phosphorelay systems which are used by fungi, but not mammals, to sense and relay stress signals to SAPK modules. In *C. albicans,* the phosphorelay system is comprised of three histidine kinases, a phosphotransfer protein (Ypd1), and the response regulator Ssk1. *C. albicans* can tolerate loss of Ypd1 and unexpectedly inactivation of *YPD1* enhances *C. albicans* virulence. Thus in this study we have explored the role of Ssk1 in Hog1 regulation to assess its suitability as an antifungal target.

Excitingly, we find that Ssk1 is a master regulator of Hog1. Cells lacking Ssk1 display impaired Hog1-activation and overlapping stress-sensitive phenotypes with *hog1Δ* cells in response to diverse stress-inducing agents with the exception of osmotic stress. We present data illustrating that Ssk1 functions as a core regulator of Hog1 by acting as a scaffolding protein to promote interactions between the Ssk2 and Pbs2 kinases within the SAPK module. This interaction is maintained following exposure to multiple stresses, however, osmotic stress drives the phosphorylation of Pbs2 and triggers its release from the Ssk1 scaffold. These findings question the dogma that stress-induced activation of Hog1 depends on the activation of upstream kinases. d.

Poster no: 4

The HIRA histone chaperone is required for the reversibility and longevity of quiescence in *S. pombe*.

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Abstract

Quiescence (G0) is the reversible suspension of cell division. Many cells in the human body are quiescent and the ability to enter and exit this state appropriately is essential for tissue repair and regeneration. For eukaryotic microbes, quiescence represents a mechanism that facilitates survival in adverse nutritional conditions. In *Schizosaccharomyces pombe* nitrogen-source depletion causes entry into a long-lived quiescent G0 state in which cells remain viable for weeks. We find that the reversibility and longevity of this quiescent G0 state is dependent upon the conserved replication-independent histone chaperone HIRA. HIRA is not required for entry into G0 or the induction of autophagy. However, although G0 cells lacking HIRA function retain metabolic activity they rapidly lose the ability to resume proliferation. After a short G0 HIRA mutants are able to resume cell growth in response to the restoration of a nitrogen source, but do not efficiently re-enter the cell cycle. This correlates with a failure to induce the expression of MBF transcription factor-dependent genes such as *cdc18*, *cdc22* and *cdt1* that are critical for S phase. In addition, G0 cells lacking HIRA rapidly progress to a permanently arrested state in which they can no longer re-initiate growth following nitrogen source restoration. Analysis of a conditional *hip1* allele is consistent with these findings and indicates that HIRA is required for efficient exit from quiescence and prevents the premature onset of senescence.

Poster no: 5

Molecular Dissection of aneuploidy and spindle checkpoint in Cryptococcus neoformans.

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Abstract

Background: Cryptococcosis is a dynamic fungal infection caused by a pathogenic yeast *Cryptococcus neoformans.* This yeast demonstrates noticeable ploidy shifts during *in vivo* pulmonary infection providing a model yeast to study aneuploidy and cell cycle checkpoints.

Methods: Comparative growth rates in the presence of anti-microtubule drugs of H99S strains with several checkpoint protein mutant strains including mad2Δ, bub1Δ, bub1kd & mps1Δ have been assayed using micro colony assays and tecan plate reader. Relative aneuploidy rates will be quantitated by monitoring GFP marked chromosome loss rates of different mutant strains. Spindle checkpoint proteins, including the Bub and Mad proteins, and their interactions during spindle checkpoint activation will be studied using immuno-precipitations and mass spectrometry.

Results: Several checkpoint mutant strains specially *bub1* mutant are very sick in growth assays compared with wild type H99S strain. Both *bub1* and *mps1* mutants are very sensitive to anti-microtubule drugs.

Conclusion: Cryptococcus having noteworthy ability to change ploidy status is an excellent model yeast to study aneuploidy associated cellular fitness. Phenotypes of checkpoint mutant strains suggest critical role(s) in cellular function and checkpoint activation. Further study is needed to explore checkpoint protein interactions and their molecular mechanism in the activation of the spindle assembly checkpoint in *Cryptococcus neoformans.*

Poster no: 6

Genomic diversity and complexity within the Saccharomyces complex

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Abstract

The Saccharomyces complex is a well-studied group of yeasts of considerable academic and industrial importance. Many species within the complex have no genome sequence publicly available and have yet to be formally compared at the genomic level with related species. We have recently sequenced the genomes of 40 species from 11 clades within the Saccharomyces complex. Considerable genomic diversity was observed in this dataset, including varying predicted genome size (9-22Mbp), coding proportion (42-76%) and gene number (4,194-11,001).

We evaluated the completeness of our gene sets and draft genome assemblies using the BUSCO tool, with a few species found to have a large number of duplicated and missing genes. These same species also had larger than average genome sizes and numbers of genes, which could indicate a duplication or hybridisation event. The total GC content was also found to vary significantly across the dataset, from *Hanseniaspora uvarum* (31.3%) to *Lachancea thermotolerans* (46%). GC content in the coding regions versus the whole genome was also observed, which could indicate differences in evolutionary pressures between species according to their environmental niches.

Here, we highlight some of the key differences found within this fascinating dataset, investigating the origins of some of the more extreme results and touching upon the opportunities and challenges they present in investigating the evolution of these yeasts.

Poster no: 7

A CRISPR-Cas9 strategy to elucidate the effect of histone modification on meiotic recombination initiation in fission yeast

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Abstract

Meiotic recombination, the reshuffling of genetic material between homologous chromosomes during meiosis, is a key process to enable faithful segregation of chromosomes during gamete production. Recombination initiation is a tightly regulated process, and the frequency of recombination varies markedly throughout the genome, creating regions of hot- and cold-spot recombination activity. Although many factors have been implicated in this regulation, the process is not yet fully understood. This project proposes to elucidate how local chromatin structure and histone post-translational modifications regulate the initiation of recombination. Histone modifications (e.g. histone acetylation, histone methylation) can induce changes of local chromatin structures that allow for better or worse access of the recombination initiation machinery to the DNA. Here, we explore whether local enrichment of histone modifiers is sufficient to effect changes in recombination frequency. We will thus fuse particular modifiers to a catalytically inactive version of the Cas9 protein, target the fusion proteins to hot- and cold-spot loci in fission yeast (*Schizosaccharomyces pombe)* via the guide RNA component, and measure changes in recombination frequency using a recombination assay based on nutritional markers. This CRISPR-based method constitutes a new tool to improve our understanding of regulation of meiotic recombination initiation and hot-spot determination and to identify candidate factors involved in the process, and can have potential biotechnological applications by allowing us to increase or decrease recombination in a targeted and localised way.

Poster no: 8

Investigation into polyphosphate mobilization and inositol pyrophosphate synthesis in regulating phosphate homeostasis in the fungal pathogen Candida albicans.

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Abstract

The human fungal pathogen *Candida albicans* is a metabolically versatile organism that can survive various stress conditions imposed by its human host. It causes systemic candidiasis in immunocompromised patients and is accountable for over 400,000 life-threatening infections per annum. Recent evidence has shown that the acquisition of nutrients such as phosphate play an important role in stress resistance and virulence of *C. albicans*. Phosphate homeostasis is mediated by the PHO pathway and recent studies in the model yeast *Saccharomyces cerevisiae,* indicate that inositol pyrophosphate molecules, specifically IP_7 molecules, play a key role in phosphate sensing and signalling. Many regulatory proteins involved in phosphate homeostasis contain SPX domains that form a binding interface for IP_7 molecules, the levels of which decrease in phosphate limiting conditions. The aim of this study is to investigate the importance of inositol pyrophosphate molecules in phosphate signalling in *C. albicans* by disrupting the kinase enzymes involved the synthesis of in IP_7 molecules, and its precursor IP_6. The IP_7 inositol pyrophosphate is synthesized by the Kcs1 Inositol hexakisphosphate kinase which converts IP_6 to IP_7, whereas IP_6 is converted from IP_5 by the Ipk1 inositol pentakisphosphate kinase. Strains lacking *KCS1* have been constructed, however *IPK1* appears to be an essential gene. Thus a conditional mutant in which *IPK1* is under the *MET3* promoter is under construction. A phenotypic analysis of these mutant strains will be presented.

Poster no: 9

Stop and dismiss: INO80 chromatin remodeller promotes early termination of mRNA synthesis

Sara Luzzi[1], Camille Gautier[2], Sarah Greener[1], Kang Hoo Han[3], Jack Darke[1], Rossana Piccinno[4], B. Franklin Pugh[3], Antonin Morillon[2], Manolis Papamichos-Chronakis[1]

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Abstract

RNA quality control is essential for functional gene expression. In eukaryotes unproductive transcription by RNA polymerase II (RNAPII) is subjected to premature termination followed by RNA degradation. Nevertheless the molecular mechanism of RNAPII removal from chromatin during transcription termination are largely unknown. ATP-dependent chromatin remodellers have been widely implicated in transcriptional regulation although their role in co-transcriptional RNA quality control is unclear. Here I report that in *Saccharomyces cerevisiae cerevisiae* premature transcription termination of aberrant mRNAs is coupled to promoter-proximal pausing of RNA Polymerase II and regulated by the evolutionarily conserved ATP-dependent chromatin remodeling complex INO80. Disruption of INO80 leads to enhanced RNAPII pausing proximally to promoters and accumulation of unproductive nascent mRNA transcripts. Cells that are inactive for INO80 exhibit impaired transcription elongation, as well as defective transcription termination. Mechanistically INO80 associates with the RNA quality control machinery facilitating recruitment of the RNA surveillance factor Nab2 to chromatin and its interaction with the histone variant H2A.Z. An acetyl-mimic mutant of H2A.Z rescues the defects in transcription elongation and RNA degradation observed in the absence of INO80. Finally, histone hyperacetylation by deletion of the HDAC Rpd3S subunit *RCO1* reduces RNAPII pausing and rescues functional mRNA expression when INO80 is inactive. Overall our work reveals a chromatin-mediated mechanism for quality control of mRNA transcription that depends on evolutionary conserved factors and that hence has relevance in the study of gene expression in higher organisms and in disease.

Poster no: 10

Hog1 signalling in the emerging multidrug resistant fungal pathogen Candida auris

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Abstract

The emerging fungal pathogen *Candida auris* is responsible for recent global outbreaks caused by genetically divergent clades that, alarmingly, display multi-drug resistance to currently available antifungals. Although not much is known about the pathobiology of this emerging pathogen, we recently found that the Hog1 stress-activated protein kinase (SAPK) in *C. auris* promotes stress resistance and virulence. SAPKs are evolutionary conserved pathways employed by fungi to promote survival in hostile environments. The aim of this project is to characterise the upstream signalling proteins that relay stress signals to *C. auris* Hog1. In all eukaryotes, SAPK pathways are comprised of three tiers of kinases with upstream MAPKKK and MAPKKs relaying signals to the terminal SAPK. However, unique to fungi and plants, is an upstream two-component related signalling cascade that transmits signals to the SAPK module, comprising of a histidine kinase, a phosphorelay protein and a response regulator. In *C. auris*, the proteins that regulate Hog1 activation are unknown. However, bioinformatics analyses of the *C. auris* genome has allowed for the identification of putative homologues of key Hog1 regulatory proteins including the Pbs2 MAPKK, the Ssk2 MAPKKK, the Ssk1 response regulator, the Ypd1 phosphorelay protein and the Sln1 and Nik1 histidine kinases. Mutational analyses is currently underway to dissect the role of these proteins in Hog1 regulation. Understanding how the Hog1 SAPK is regulated in *C. auris* may facilitate the identification of fungal specific anti-fungal targets that can be exploited to tackle the threat of this emerging drug resistant pathogen.

Poster no: 11

Manipulating Meiotic Double-strand Break Formation using a Modified CRISPR/Cas9-system in Fission Yeast

Samantha Jacqueline Mpaulo, Alexander Lorenz

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Abstract

Meiosis is critical to the success of sexual reproduction. It not only ensures the faithful transmission of the hereditary material from one generation to the next, but also enables the generation of new gene combinations that have the potential to enhance the genetic diversity amongst members of a given species. An integral aspect of meiosis is the programmed formation of DNA double-strand breaks (DSBs), which occur non-randomly and with a higher frequency at specific sites across the genome, known as meiotic DSB hotspots. Following DSB induction, these breaks are repaired by meiotic recombination preferentially using the homologous chromosome as the template. The repair can result in either crossover or non-crossover products, both of which may be associated with a gene conversion event. Only crossover events establish connections between the parental chromosomes, known as chiasmata. These represent sites of reciprocal exchange between parental chromosomes, and are essential for correct meiotic nuclear divisions.

To better understand the regulatory mechanisms that underpin various aspects of meiotic recombination (DSB formation and repair type), we investigate how manipulating DSB delivery influences repair outcome. We achieve this by combining a meiotically expressed CRISPR/Cas9-system with a genetic recombination assay, in fission yeast. This approach allows for the induction of targeted DSBs independent of the meiotic DSB formation machinery, which will provide novel insight into the mechanisms driving certain types of repair outcome.

Poster no: 12

Evaluating the use of variation graphs for the characterisation of yeast rDNA arrays

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[1]Quadram Institute Bioscience, Norwich, United Kingdom. [2]Earlham Institute, Norwich, United Kingdom

Abstract

Over the past decade, we have gained considerable insight into the identification of sequence variation within the rDNA array of *Saccharomyces cerevisiae* and its closest wild relative, *Saccharomyces paradoxus*. Yet considerable challenges remain in the computational characterisation of this complex genomic region. This study aimed to evaluate the use of variation graphs for this purpose, formally comparing their effectiveness with traditional linear approaches.

Specifically, we aimed to identify both partial and fixed variants (i.e. pSNPs, SNPs, pINDELs and INDELs) in the rDNA arrays of 10 diverse, haploid *Saccharomyces cerevisiae* strains with high quality genomic datasets. We constructed two computational pipelines using two highly different approaches. The first pipeline used the BWA read mapper and the BCFtools variant caller to identify variants against the linear S288c reference, with the second pipeline using the vg tool to call variants against a graphical reference (either based on a graphical representation of the S288c genome or a *Saccharomyces cerevisiae* pan-genome).

The results showed that the graph-based pipeline was able to identify more variants than the linear pipeline, and in particular partial variants, while also missing some key variants identified by BWA/BCFtools. A major discrepancy between the two pipelines was found in the read coverage at loci where the vg pipeline identified variants. In the coming months, we aim to investigate the cause of these differences and to develop a new graph-based computational pipeline that can accurately identify the full range of sequence and copy number variation within this key genomic region.

Poster no: 13

Investigating the Apoptotic Function of Human ECE-1 isoforms by Using a Yeast-based System

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Abstract

The B cell lymphoma 2 (*Bcl-2*) family consists of a number of pro-apoptotic members (e.g. *Bax*) and anti-apoptotic members (e.g. *Bcl-xL*), which regulate apoptosis by activating mitochondrial events. The heterologous expression of human *Bax* and *Bcl-xL* in budding yeast (*Saccharomyces cerevisiae*) resulted in similar effects to those in mammalian cells. Human endothelin converting enzyme 1 (ECE-1) is involved in proteolytic processing of endothelin precursors, which are related to many diseases such as cardiac defects and autonomic dysfunction. However, it is unclear whether ECE-1 is associated with apoptosis. ECE-1 contains four isoforms, namely ECE-1a, b, c and d.

ECE-1a is expressed at the plasma membrane, while ECE-1b is only intracellularly expressed. The expression of ECE-1c is in another case that usually occur on cell surface. The latest discovered isoform, which is ECE-1d, it widely distributes both cell surface and intracellular area. For example, it was detected at Golgi and several endosomal structures. Therefore, we are interested in studying whether the four isoforms have different function.

In current study, the human ECE-1 isoforms were transformed individually into W303 strains containing an integration of the coding sequence of human *Bax*. With the expression of *Bax* and ECE-1 isoforms, which were both controlled by a Gal1 promoter, the growth defects were observed and detected by spot test assay, recording the growth curves and cell viability assay by measuring colony forming units (CFU). Furthermore, the gene expression level would be detected by RT-PCR and dPCR in the future.

Poster no: 14

DEVELOPMENT OF A NEW HIGH-THROUGHPUT METHOD FOR SCREENING LARGE YEAST LIBRARIES FOR USE IN THE BEVERAGE INDUSTRY

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Abstract

High-throughput techniques have become crucial for the discovery of new strains suitable for the beverage industry. These approaches allow assessment of critical traits for fermented beverages such as; stress tolerance, growth, viability, vitality and aromatic compound production in a fast and effective manner.

The growth rate of 24 *Saccharomyces* and non-*Saccharomyces* yeast strains were evaluated in industrially relevant media. High-throughput robotic platforms, the ROTOR HDA and PIXL, were used to pin colonies on solidified synthetic apple juice (SAJ), malt extract (ME), apple juice (AJ) and wort (WO) media. During incubation at 20 and 25 ºC for up to 240 hours, the plates were imaged, and analysed via PhenoBooth and the *ggplot2* R package. The results were corroborated via liquid growth.

Pichia scullata Y-7663 and *Hanseniaspora vineae* Y-17530 showed the highest growth values in ME, WO and AJ; with final colony diameters double that of the *Saccharomyces cerevisiae* ESM356-1 control. However, the former two strains displayed defective growth in SAJ. *Kluyveromyces lactis* Y-1140 exhibited high growth performance, especially in ME and WO. Interestingly, the ale strain *S. cerevisiae* Y-11875, showed significantly higher growth in SAJ and AJ medium than in ME and WO. Better adaptation to growth in wort conditions was expected.

This trial shows that the fitness of yeast strains can be assessed in solid media representing industrial conditions. The method can be up-scaled to a wider format, opening the possibility of screening a thousand-fold libraries of industrially relevant and/or novel yeast isolates in a fast, cost-effective manner.

Poster no: 15

Regulation of ubiquitin pathway enzymes determines cellular responses to reactive oxygen species

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Abstract

Cells are exposed to reactive oxygen species (ROS) from internal processes such as respiration or from environmental sources. High levels of ROS cause oxidative stress, damaging many cellular components such as DNA, proteins and lipids, and as a consequence oxidative stress has been linked to the pathology of many age-related diseases. In contrast, low levels of ROS have been found to perform vital roles in cellular signalling, regulating processes such as the cell cycle. Therefore, it is essential that cells are able to distinguish both the type and concentration of ROS in order to elicit an appropriate response. Ubiquitin and ubiquitin-like (Ubl) proteins are reversible protein modifications that regulate many essential cellular processes. Interestingly, many of the enzymes involved in the conjugation and deconjugation of these dynamic post-translational modifications utilise catalytic cysteine residues, raising the possibility that these pathways function in ROS sensing mechanisms. Indeed, work in our lab utilising *Saccharomyces cerevisiae* as a model organism supports this hypothesis. Here we present evidence that specific deubiquitinases (DUBs), the enzymes responsible for removing ubiquitin from modified substrates, are distinctly regulated by the type and concentration of ROS. Significantly, dysregulation of human homologues of ROS-regulated *S. cerevisiae* DUBs have been linked with several common diseases and thus our findings potentially have wider implications for human health.

Poster no: 16

Proteomic analysis of lifespan extension by dietary restriction

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Abstract

Ageing leads to the eventual deterioration of both biochemical and physiology function and is the main risk factor for many neurodegenerative and cardiovascular diseases. While the underlying aspects of ageing are not fully understood, current research has linked the insulin/IGF-1 signalling (IIS) and nutrient response pathways to be a key influencer in a diverse range of species including yeast, worms, flies and rodents, providing evidence for a universal mechanism of ageing. This is further supported through dietary restriction (DR) experiments that have been shown to increase lifespan by up to 30% and reduce age-related pathologies of model organisms. In this study, to shed light on the mechanisms behind DR, liquid chromatography-mass spectrometry (LC-MS) was performed on *Saccharomyces cerevisiae* that had been grown under standard 2% glucose or lifespan-extending DR 0.05% glucose conditions. Our results show large fold increases in a range of proteins closely linked to mitochondria function and biogenesis as well as in general metabolism. This is consistent with the shift from fermentation to respiration that accompanies glucose limitation. However, we also found a number of non-mitochondrial proteins to be significantly altered in expression level due to DR, notably small heat shock proteins. Here we show that this increase in small heat shock protein expression plays a functional role in the increased longevity caused by DR.

Poster no: 17

The INO80 ATP-dependent chromatin remodeller promotes selective autophagy under metabolic stress conditions.

David Shapira[1], Anne Lafon[2], Manolis Papamichos-Chronakis[1]

[1]Newcastle University, Newcastle Upon Tyne, United Kingdom. [2]Institut Curie, PSL Research University, Paris, France

Abstract

Autophagy is an evolutionarily conserved catabolic process essential for cellular metabolism, homeostasis and stress response. Autophagy mediates both non-selective degradation of cargoes, as well as selective, ubiquitin-dependent degradation of proteins and organelles, such as mitochondria. While the autophagy pathway itself is well studied, the contributions of epigenetic and chromatin-related mechanisms in regulation of selective autophagy remains poorly understood.

The evolutionarily conserved ATP-dependent chromatin remodelling INO80 complex has recently been implicated in metabolic gene regulation, however, its role remains unclear. Here we report that in budding yeast, INO80 regulates selective autophagy under metabolic stress conditions. Using a combination of ubiquitin proteomics and cellular biology assays we demonstrate that, while autophagy is properly induced in cells lacking INO80, loss of INO80 leads to defective nucleophagy, reticulophagy and mitophagy. Our transcriptomic analysis revealed that INO80 promotes the expression of a group of genes involved in the multivesicular body (MVB) sorting pathway for protein degradation and our functional analysis demonstrated that this is required for selective autophagy of mitochondria. Genome-wide association analysis by ChIP-seq under nutrient stress conditions revealed selective enrichment of INO80 at general amino acid control (GAAC) genes, providing evidence for a direct role for INO80 under starvation response. Finally, we show that loss of INO80 leads to a profound increase in lethality under nutrient starvation or upon chemical inhibition of the mTORC pathway by rapamycin. Our results suggest that INO80 coordinates amino acid biosynthesis with selective autophagy, revealing a novel epigenetic mechanism that controls cellular metabolism.

Poster no: 18

The activity of *S. pombe* LTR retrotransposons is activated in response to rapamycin.

Tsun Ho Chan, Simon Whitehall

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Abstract

Long terminal repeat (LTR) retrotransposons are mobile genetic elements that are present in the genomes of most eukaryotes. They are closely related to retroviruses and mobilize through an RNA intermediate. The uncontrolled mobilization of retrotransposons is potentially harmful to the integrity of the genome and so the activity of these elements is subjected to strict host cell controls. We are using the fission yeast, *Schizosaccharomyces pombe* to study the signalling pathways that regulate the activity of LTR retrotransposons. We have found that the expression and mobilization of the *Tf2* LTR retrotransposons is activated in response to exposure to the immunosuppressant drug rapamycin. Rapamycin binds to the conserved FKBP12 protein (called Fkh1 in *S. pombe*) and the resulting FKBP12-rapamycin complex inhibits the kinase activity of the conserved the TORC1 complex. This suggests that *Tf2* activity is under the control of the TORC1 signalling network which is a master regulator of cellular responses to nutrient and energy availability. However, the inhibition of TORC1 activity using a *tor2* temperature sensitive allele or a direct chemical inhibitor (Torin) did not activate either the expression or mobilization of *Tf2* elements. Therefore, rapamycin may be controlling *Tf2* activity via a TORC1-independent pathway. We are currently defining this pathway and find that it is dependent upon the FKBP12 protein, Fkh1 and the Forkhead transcription factor, Fhl1.

Poster no: 19

Dissecting the role of Peroxiredoxins in regulating conserved ROS-activated kinases

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Abstract

In order to protect against oxidative damage, cells have evolved a host of ROS-detoxifying enzymes. These include peroxiredoxins, a highly conserved family of thioredoxin peroxidases. Unexpectedly, given their role in lowering H_2O_2 levels, peroxiredoxins have been shown to be required for the activation of conserved stress-activated MAPKs in response to ROS in yeast[1] and human[2] cells. For example, we have previously shown that the single 2-Cys peroxiredoxin in *S. pombe*, Tpx1, but not its thioredoxin peroxidase activity, is required for the H_2O_2-induced activation of the p38/JNK-related MAPK, Sty1. Our findings revealed that Tpx1 forms H_2O_2-induced disulphide bonds with cysteines in Sty1[1], which suggested that Tpx1 may directly regulate Sty1 through these complexes. However, the mechanisms by which Tpx1-Sty1 disulphide complexes alter Sty1 function have remained unclear. Sty1, like its mammalian counterparts, has a number of important functions, including roles in coordinating cell growth, division, stress resistance and longevity in response to a variety of nutritional and stress stimuli. Our data suggests that disulphide complexes with Tpx1 are important for a subset of these roles. Intriguingly, our proteomic studies have identified multiple protein kinases that form disulphide complexes with Tpx1, these include kinases with established roles in regulating cell division and ageing. Here, we will present data suggesting that interactions with Tpx1 play important roles in regulating the activities of these kinases.

1. Veal et al. (2004) *Molecular Cell*, 15(1), pp. 129-139.

2. Jarvis et al. (2012) *Free Radical Biology and Medicine*, 53(7), pp. 1522-1530.

Poster no: 20

Inhibition of Drug-Resistant *Candida glabrata* by Killer Toxins Produced by *Saccharomyces cerevisiae*

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Abstract

Combatting the spread of drug-resistant microbes requires new antifungal compounds with novel mechanisms of inhibition. Our lab investigates natural, proteinaceous toxins that are coded by double stranded RNA satellites found within *Saccharomyces cerevisiae*. Commonly known as "killer yeasts", toxin-producing strains of *S. cerevisiae* have been found to inhibit the growth of many important fungal pathogens. To assess the value of killer yeasts for medical application we tested over 6,000 interactions between killer yeasts and various important fungal pathogens. To determine the susceptibility of fungi to killer toxins, we competed killer yeasts against a dilute lawn of a fungal pathogen; an active toxin produces a zone of growth inhibition in the lawn around the killer yeast. We determined that *Candida glabrata* is broadly susceptible to killer toxins, while other *Candida* species showed little or no susceptibility. Of the 90 strains of *S. cerevisiae* tested against *C. glabrata* seven were capable of inhibiting all clinical strains of drug-resistant *C. glabrata* available from the Center for Disease Control and the U.S. Department of Agriculture. In our evaluation of these toxins as potential antifungal therapeutics we confirmed their inhibitory capability against *C. glabrata* under physiological conditions. Based on the results of these tests and prior knowledge about killer yeasts, we believe that these toxins show a potential as antifungal therapeutic precursors to treat recalcitrant infections caused by *C. glabrata*.

Poster no: 21

Novel Double-Stranded RNA Viruses Discovered within *Saccharomyces cerevisiae*

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Abstract

Saccharomyces yeasts harbor many different viruses and parasitic genetic elements, including double-stranded RNA (dsRNA) viruses from the family *Totiviridae*. The identification of novel dsRNA viruses in yeasts has been constrained by the lack of effective protocols for the unbiased preparation and sequencing of dsRNAs. We have developed a next-generation sequencing method that enables the amplification and sequencing of yeast dsRNAs. Using this method, we have performed a metagenomic screen of more than 600 strains of *Saccharomyces cerevisiae* for the presence of novel dsRNA viruses. Surprisingly, we have identified several novel bipartite dsRNA viruses from the family *Partitiviridae* within different strains of *S. cerevisiae*. Partitiviruses have never been described within *Saccharomyces* yeasts or within the wider Saccharomycotina taxonomic subdivision of the Ascomycota phylum. We confirmed the presence of these partitivirus dsRNAs using reverse transcriptase-PCR and have been successful in purifying viral particles from infected yeasts. After deep sequencing of purified dsRNAs, we have identified three species of virus that have weak homology (20-35% amino acid identity) to viruses of the genus *Cryspovirus*, which replicate within the protozoan pathogen *Cryptosporidium parvum*. Partitiviruses can modulate the virulence and fecundity of plant and human pathogens, respectively. The discovery of partitiviruses in *S. cerevisiae* will enable the study of how partitiviruses interact with the host cell environment and alter cellular physiology.

Poster no: 22

Drug resistant *Candida* colonization among ICU patients in a Nigerian teaching hospital.

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Abstract

Introduction: Colonisation of body surfaces with resistant strains is an initial step in the pathogenesis of drug resistant invasive *Candida* infections. Nigeria has little data on the species distribution and resistance pattern of colonising *Candida* species in intensive care unit(ICU) patients. This study set out to investigate the burden of *Candida* colonisation in the ICU of a tertiary health institution in Ile-Ife, Nigeria.

Methodology: At admission, 72 hours and 7 days, swabs of skin, oropharynx, perianal region, surgical wounds, tracheal aspirates and urine samples taken from adult ICU patients were cultured on media selective for fungi. *Candida* species were identified by standard microbiological methods. Sensitivity to fluconazole, voriconazole and caspofungin was determined by disc diffusion according to CLSI guidelines. Strain interrelatedness was investigated by Randomly Amplified Polymorphic DNA (RAPD).

Results: Of 744 samples from 110 patients, 142 samples (18.3%) yielded *Candida*. The average colonisation index increased with duration of admission (p=0.001). Most frequently colonized were the oropharynx (39.5%) and the perianal region (22.4%). Species isolated were *C. albicans* 97 (68.3%), *C. tropicalis* 20 (14.1%), *C. glabrata* 12 (8.5%), *C.parapsilosis* 7 (4.9%) and *C. krusei* 6 (4.2%). About 1 in every 3 isolates was resistant to fluconazole (42/142; 29.6%). In addition, resistance to voriconazole was 12/142 (8.5%) while 10/142 (7%) were resistant to caspofungin. RAPD showed identical strains across several body sites of many patients.

Conclusion: *Candida* species with high rates of resistance to the first line antifungal agent colonise and are transmitted among ICU patients. These can potentially cause difficult to treat invasive infections, underscoring a need for better infection control practices.

Poster no: 23

DNA checkpoint kinases as peroxide sensors and regulators of virulence attributes in the human fungal pathogen *Candida albicans*.

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Abstract

The ability of the major human fungal pathogen, *Candida albicans,* to sense and respond to reactive oxygen species (ROS) is essential for virulence, as ROS are antimicrobial weapons employed by innate immune cells. We have previously shown that H_2O_2 triggers activation of the DNA damage checkpoint kinase Rad53 in *C. albicans* which leads to a morphological switch with daughter buds displaying continuous polarized growth. Furthermore, we found that the thioredoxin Trx1 is a negative regulator of H_2O_2-induced Rad53 activation, and that *trx1Δ* cells are filamentous due to the constitutive activation of Rad53 (*Mol Cell Biol* 30; 4550).

Here we have examined the mechanisms underlying H_2O_2-induced activation of Rad53, and its importance during pathogenesis. We find that Rad53 is robustly activated following phagocytosis by macrophages and, interestingly, that the DNA-damage sensing kinases $Mec1^{ATR}$ and $Tel1^{ATM}$ regulate different Rad53-mediated responses. For example, $Mec1^{ATR}$-induced activation of Rad53 is vital for survival following H_2O_2 exposure and within the hostile environment of the phagosome, whereas $Tel1^{ATM}$ regulates H_2O_2-induced polarized growth. Excitingly, we find that $Tel1^{ATM}$ is oxidised following H_2O_2 stress, and that Trx1 functions to reduce $Tel1^{ATM}$. Thus the filamentous growth phenotype of *trx1Δ* cells may reflect an accumulation of oxidised $Tel1^{ATM}$.

Collectively these results reveal the unprecedented finding that there is a bifurcation of Rad53 mediated responses depending on the upstream activating kinase, which together co-ordinate *C. albicans* responses to host-derived ROS.

Poster no: 24

Modification of *Candida albicans* cell wall by commensal gut bacteria.

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Abstract

The human gut is populated with a vast community of microbes, the microbiota. Fungi comprise 0.1% of the total gut microbiota. Some of these fungi exist as benign members, however others such *Candida albicans* can undergo a pathogenic switch causing disease. The fungal cell wall is the first target for immune system recognition. Recent studies have suggested that *Candida* is decorated with different cell wall epitopes within different physiological niches, due to the impact of carbon source and oxygen availability on cell wall remodelling. Here we hypothesize that resident gut bacteria also play a major role in fungal cell wall remodelling and immune recognition. Data from our lab has shown that a common bacterium from the gut, *Bacteroides thetaiotaomicron* (*Bt*), produces an extensive repertoire of degradative enzymes to breakdown the Candida cell wall. Recently, we have identified novel enzymes in *Bt* from the glycoside hydrolase family 130 (GH130), which specifically target β1,2-linked mannan, a unique feature of *Candida* mannan. We have deleted multiple fungal mannan specific loci in *Bt* and examined the ability of deletion strains to utilise *Candida* mannan as a carbon source. These data suggest that *Bt* contains multiple pathways to degrade the *Candida* cell wall. Now we are systematically dissecting the impact of mannan degradation on the physiology of the fungus. This will provide insights into how two prominent members of the gut microbiota interact with each other, how the *Candida* cell wall is modified in the anaerobic environment of the gut, and the importance of this in promoting immune homeostasis.

Poster no: 25

Investigating the role of yeasts as potential probiotics in controlling *E. coli* in chicken gut

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Abstract

Chicken is a major food source for humans and a valuable source of protein, therefore, the production industry is keen to constantly improve healthy growth of poultry. The use of antibiotics in animal feed as growth promotors allowed the development of antimicrobial resistance. This health risk problem led to the ban of antibiotic use in animal feed which has driven an increase in endemic production related infections such as Colibacillosis. As a result, a need for alternative therapies to control such infections is necessary (Castanon, 2007). Recent studies have focused on modulating the normal intestinal microflora in humans and animals using live microbial cultures known as probiotics, which are defined by the World Health Organization (WHO) as "live micro -organisms which, when administered in adequate amounts, confer a health benefit on the host" (FAO and WHO, 2001). *Saccharomyces boulardii* has been used as a probiotic with some success (Czerucka & Rampal, 2002) and this study is focusing on the investigation of some yeast-like strains that were isolated from chicken tissues or surroundings for their probiotic potential in controlling gut pathogens such as Avian Pathogenic *Escherichia coli* (APEC).

Aim: Isolation of yeast and yeast like microorganisms from chicken gut and surroundings to test isolates for potential probiotic activity against avian pathogenic *E. coli* (APEC) by performing agglutination and inhibition assays.

Poster no: 26

Response of *Paracoccidioides* spp. to oxidative stress: The role of superoxide dismutases (SODs)

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Abstract

Dimorphic human pathogenic fungi belonging to the *Paracoccidioides'* genus are the causative agents of paracoccidiodomycosis (PCM), an endemic disease widespread in Latin America, acquired by inhalation of conidia. Previous studies in *Paracoccidioides* have identified some genes involved during some events underlying the host-pathogen interactions; however, the molecular mechanisms that rule the response of the fungus to oxidative stress have been poorly understood. To counteract the Reactive Oxygen Species (ROS) set by the host and by its own metabolism, *Paracoccidioides* expresses several antioxidant enzymes, among them superoxide dismutases (SODs) are predominant. SODs neutralize $O_2^{\bullet -}$ and convert them into H_2O_2 and molecular oxygen (O_2). Through bioinformatic analyses, we identified six isoforms, afterwards, we determined gene expression levels of each gene through RT-qPCR. Further analyzes of SODs gene expression led us to suggest that Pb*SOD1* and Pb*SOD3* could assist in combating the superoxide radicals generated during the host-pathogen interactions. Subsequently, we developed *PbSOD1* and *PbSOD3* knockdown strains and evaluated them in conditions mimicking conditions at which the fungus would be exposed once inside the host. We showed that these genes were involved in the response of the fungus against host effector cells, particularly the oxidative stress response, and in a mouse model of infection. Protein sequence analysis together with functional analysis of knockdown strains led us to suggest that *PbSOD3* is linked with an extracellular activity while *PbSOD1* seems more related to intracellular requirements of the fungus.

Poster no: 27

The role of the outer mitochondrial membrane protein Porin in cell death and ageing

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Abstract

The Outer Mitochondrial Membrane (OMM) contains channels to regulate metabolic flux, such as the Voltage-Dependent Anion Channel (VDAC) also referred to as Porin in *Saccharomyces cerevisiae*. Porin has been reported to play a role in apoptosis and oxidative stress, but surprisingly little is known about the role of VDAC in Mitochondrial Morphology, Mitophagy, Autophagy or ER-Mitochondria connections (The ER-mitochondria encounter structure, ERMES). We present evidence that Porin is associated with the ERMES complex and that its deletion results in aggregation of mitochondria that mimics the loss of ERMES components. This aggregation is associated with a loss of respiration, reduced capacity for autophagy, an increase in peroxisome number and elevated necrosis. We have also found that overexpression of Porin leads to activation of the cell wall integrity pathway, flocculation and vacuole fragmentation. Our experiments suggest that Porin plays a critical role in the maintenance of mitochondrial function and the regulation of several important signalling mechanisms. These functions may be linked to as yet undefined roles for Porin in lipid metabolism and the assembly of stress regulatory modules on the mitochondrial surface.

Poster no: 28

Temperature adaptation of *Saccharomyces* yeasts: a molecular approach

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Abstract

In nature, the species of *Saccharomyces* sensu stricto complex have adapted to different ranges of optimal growth temperatures; and temperature variability has a key role on the maintenance of biodiversity. Some species live in sympatry because they respond differently to thermic conditions; on the other hand, some species have similar optimal growing temperatures but occur in different habitats. The process of yeasts adaptation to a wide range of different thermal niches have not been extensively studied and understood, especially from a molecular approach.

Of the eight species of the *Saccharomyces* sensu stricto group, we are comparing the expression of six genes previously identified as partially responsible for cryo-tolerance (*ADH3*, *ADH5*, *GUT2*, *NMA1*, *YND1* and *FAA1*), in warm and cold conditions in rich media. We observe that the cold-tolerant species showed higher expression of the candidate genes compared with thermo-tolerant species, except for *S. paradoxus*, a thermo-tolerant species, whose expression profile is similar to a cold-tolerant. Then, we measured the gene expression of the candidate genes between populations of *S. paradoxus* from different locations, and we compared with the gene expression profile of *S. kudriavzevii* populations, a strict cold-tolerant species. Thus, we observed that the level of expression of the candidate genes varies between populations of *S. paradoxus*, and not between *S. kudriavzevii* populations.

In addition, we knocked-out the candidate genes, creating heterozygous and homozygous deletion mutants of each species and then, we compared the fitness with the natural isolated wild-type yeast strains at cold and warm temperatures. The absence of the candidate genes affects the fitness at cold, but also, increase the fitness at warm temperatures in cold-tolerant species compared to the natural isolated strain. The increasing on the fitness of the deletion mutants occurred on different temperatures above 30°C, directly related to the optimal growth temperature of the cryo-tolerant species.

This study will allow us to understand more precisely the impact of temperature fluctuation on species of the *Saccharomyces* sensu stricto group, and observe the role of genes that increase the cold tolerance under a gradient of temperatures. Also, under climate change conditions, we will have a better understanding of how species of the *Saccharomyces* sensu stricto group coexist and how the biodiversity is maintained, using a molecular approach.

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