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ABSTRACT BOOK

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teins are responsible for depositing H3K4me3, suggesting that the methylation status of histones may be involved in regulating freeze-thaw tolerance in *C. elegans*.

Collectively, our findings suggest that three factors, namely starvation, precise cuticular structure, and histone methylation status, contribute to the regulation of freeze-thaw tolerance in *C. elegans*.

1265V **1-Mesytil-3-(3-sulfonatopropyl)imidazolium protects against oxidative stress and delyas proteotoxicity in *C. elegans*** Natalia Andersen[1], Tania Veuthey[1], Gabriela Blanco[1], Gustavo Silbestri[2], Diego Rayes[1], Maria Jose De Rosa[11]INIBIBB. CCT-UNS, [2]INQUISUR. UNS-CONICET

Due to the increase in life expectancy, age-related neurodegenerative diseases (NDs) have become more prevalent. Conventional treatments fail to arrest or delay neuronal proteotoxicity that characterizes these diseases. Due to their diverse biological activities, imidazole rings are intensively explored as powerful scaffolds for the development of new bioactive molecules. By using *C. elegans*, our work aims to explore novel biological roles for these compounds. To this end, we have tested the in vivo anti-proteotoxic effects of imidazolium salts. Since NDs have been largely linked to impaired antioxidant defense mechanisms, we focused on 1-Mesityl-3-(3-sulfonatopropyl) imidazolium (MSI), one of the imidazolium salts that we identified as capable of improving iron-induced oxidative stress resistance in wild-type animals. By combining mutant and gene expression analysis we have determined that this protective effect depends on the activation of the Heat Shock Transcription Factor (HSF-1), whereas it is independent of other canonical cytoprotective molecules such as abnormal Dauer Formation-16 (DAF-16/FOXO) and Skinhead-1 (SKN-1/Nrf2). To delve deeper into the biological roles of MSI, we analyzed its impact on previously established *C. elegans* models of protein aggregation. We found that MSI ameliorates β-amyloid-induced paralysis in worms expressing the pathological protein involved in Alzheimer's Disease. Moreover, MSI also delays age-related locomotion decline in other proteotoxic *C. elegans* models, suggesting a broad protective effect. Taken together, our results point to MSI as a promising anti-proteotoxic compound and provide proof of concept of the potential of imidazole derivatives in the development of novel therapies to retard age-related proteotoxic diseases.

1266V **The role of eIF3d in stress response** Jiaqing Lang Molecular and Cellular Function, University of Manchester, FBMH, School of Biological Science

Stress granules (SGs) are cytoplasmic ribonucleoprotein condensates that help reprogramme cells to adapt to and survive stress. Their dysregulation has been implicated in neurodegenerative diseases, cancer and ageing. SGs are conserved amongst eukaryotes and are composed predominantly of untranslated mRNAs, translation initiation complexes and RNA-binding proteins. They also interact with cellular signalling pathways to regulate changes in mRNA translation underpinning altered cell fate. The translation initiation factor eIF3d participates in canonical mRNA translation as a component of the eIF3 complex but can also directly bind to the 5' cap and recruit the rest of the eIF3 complex to a cohort of stress-responsive transcripts that promote cell survival. eIF3d and other eIF3 factors are also components of SGs and are required for their formation in cultured human cells. Furthermore, a pool of eIF3d is found in the nucleus, the function of which is unknown. These results hint at a role for eIF3d in coordinating different aspects of the stress response. However, much of our knowledge of stress-induced translational reprogramming as well as SG regulation and function are derived from cell-based studies, so it is important to understand this in the context of an organism. to We show that eIF3d and other eIF3 factors are required for SG formation in response to heat shock in *C. elegans*. We are now investigating how particular domains and post-translational modifications of eIF3d may play regulatory roles in the stress response and aim to uncover the relationship between SG function, eIF3d regulation and organism adaption over the lifespan of *C. elegans*. This research will provide new insights into the role eIF3d and SGs play in the integrated organismal response to both acute environmental insult and longer-term stress.

1267V **Effects of downstream mediators of DBL-1/BMP immune signaling on gut microbiome composition** Kenneth Trang[1], Siavash Karimzadegan[2], Barbara Pees[3], Michael Shapira[11]Integrative Biology, University of California, Berkeley, [2]University of California, Berkeley, [3]Evolutionary Ecology and Genetics, University of Kiel

The composition of the gut microbiome is determined by a complex interplay of diet, host genetics, microbe-microbe competition, abiotic factors, and stochasticity. Previous studies have demonstrated the importance of host genetics in community assembly of the *Caenorhabditis elegans* gut microbiome. More specifically, DBL-1/BMP/TGFb immune signaling has been shown to modulate microbiome composition, particularly affecting abundance of gut *Enterobacteriaceae*. Although *dbl-1(nk3)* and *sma-3(e491)* mutants, lacking the BMP-1-like ligand DBL-1 and its R-SMAD downstream transcriptional regulator, respectively, both exhibit a bloom of *Enterobacteriaceae* abundance, wildtype level control over *Enterobacteriaceae* abundance was primarily dependent on expression of *sma-3* in the epidermis, not in the intestine. This suggested that effects of Sma signaling on the gut microbiome were mediated by yet unidentified intestinal factors.

In the present study, we used RNA-seq gene expression analysis of wildtype, *dbl-1* and *sma-3* mutants, and a *dbl-1* over-expressing