EMBO
Workshop

III LATIN AMERICAN WORM MEETING

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Abstracts Book

THIRD LATIN AMERICAN WORM MEETING

The field of biomedical research using *C. elegans* as a model organism has flourished in Latin America in the last decade. This is partly because of a sustained exchange of expertise and tools with the international community through regional meetings and courses. The global *C. elegans* community has made outstanding advances in the last decades, for example in understanding the neural dynamics of living and performing animals or the molecular nature of transgenerational memory of infection and abiotic stresses. Moreover, the nematode has been used as a remarkable biotechnological tool to help find new drugs for animal and plant parasites, and as a sensor for ecotoxicological studies. Our meeting will feature these and other topics that include neuronal regeneration and neuronal diseases, synaptic transmission, whole brain dynamics, epigenetic inheritance, interactions in natural environments among many others. Our previous two meetings, held in Uruguay and Argentina, were successful and instrumental in expanding the networking and training possibilities for students, postdoctoral researchers, and investigators alike. We believe this meeting is an ideal environment to present recent and exciting discoveries using this fantastic nematode.

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Neural modulation of systemic stress response requires the insulin like-peptide INS-3

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Throughout the animal kingdom, the perpetuation of the flight response leads to reduced ability to cope with environmental challenges, a drastic lifespan reduction, and an increase in disease susceptibility. We showed that, in *C. elegans*, the tyraminergic neuron RIM supplies a state-dependent neural switch between acute flight and long-term environmental stress responses. During the flight-stress response RIM neurons release TA, which stimulates the intestinal adrenergic-like receptor TYRA-3. This leads to DAF-2/Insulin/IGF-1 pathway activation and inhibition of cytoprotective mechanisms in the intestine and other tissues. We hypothesized that TYRA-3 stimulates the release of Insulin-Like Peptides (ILPs) from the intestine that can systemically activate the DAF-2 insulin/IGF1 receptors. We focused on strong agonist ILPs that are expressed in the intestine (INS-3, -4, -6, -32, and DAF-28). We found that *ins-3* mutants are resistant to both heat and oxidative stress, much like *tyra-3* mutants. Moreover, *ins-3* mutants are resistant to the impairment of stress resistance upon exposure to exogenous tyramine. In addition, *ins-3;tyra-3* double mutants are as resistant to environmental stress as single mutants, suggesting that both genes act in the same pathway. Since *ins-3* is expressed in neurons and the intestine, we performed tissue-specific rescue experiments. We found that expression of *ins-3* in the intestine restores stress resistance to wild-type levels. Taken together, our results suggest that intestinal activation of TYRA-3 by the escape neurohormone TA leads to INS-3 release which acts as an endocrine, autocrine, and/or paracrine signal to activate DAF-2 in different tissues.