Caenorhabditis elegans is a model organism for studying genetics and neuroscience.

C. elegans is frequently studied to understand how genes and the environment interact to produce new phenotypes. We take advantage of an organism-wide stress response and genetic tools that provide an excellent model for studying how phenotypes are impacted by stress.

Stress-resistant dauer stage

Crowded conditions and lack of food cause C. elegans to enter an alternative stress-resistant stage called dauer.

Dauers undergo multiple morphological changes including, thickening of the cuticle, radial shrinkage, and neuroplasticity. Dauer morphology provides resistance to 1% sodium dodecyl sulfate (SDS). [1]

There is extensive remodeling of the IL2 neurons during dauer

Outside of dauer the six IL2 dendrites are unbranched. During dauer they form elaborate branches that cover the head of the animal. Once the animal recovers from dauer, the branches resorb and return to their unbranched morphology.

Are ire-1 mutants true dauers?

We did a 1% SDS test to isolate dauers of each allele. Exposure to 1% SDS is a common way to isolate true dauers. None of the (v33) animals survived and only 3 of the (ok799) animals survived. Interestingly, these results contradicted literature that stated (v33) mutants form 100% dauers. This led us to question whether these animals were true dauers. We then performed SDS dose response assay to assess susceptibility. We used the following SDS concentrations: 1%, 0.5%, 0.2%, 0.1%.

cauliflower

PC: 002666

Pablo Garcia

Stress Induced Remodeling in the Nematode C. elegans

Becky Rose, Rebecca Androwski, and Nathan E. Schroeder

Lincoln Land Community College, Springfield, Illinois

Neuroscience Program, University of Illinois at Urbana-Champaign

Department of Crop Sciences, University of Illinois at Urbana-Champaign

Stress Induced Remodeling in the Nematode C. elegans by Rebecca Androwski, and resumes normal development. During the six IL2 dendrites are unbranched. During the larva, a post mutation is required for IL2 branching. We found allelic differences between ire-1(ok799) and ire-1(v33). We did a 1% SDS test to isolate dauers of each allele. Exposure to 1% SDS is a common way to isolate true dauers. None of the (v33) animals survived and only 3 of the (ok799) animals survived. Interestingly, these results contradicted literature that stated (v33) mutants form 100% dauers. This led us to question whether these animals were true dauers. We then performed SDS dose response assay to assess susceptibility. We used the following SDS concentrations: 1%, 0.5%, 0.2%, 0.1%.

ire-1 is required for IL2 branching

The candidate gene, ire-1, encodes a PTEN homolog in C. elegans. PTEN is a critical component of human neurological health. Mutations in PTEN lead to abnormal neuronal growth and neurological dysfunction and are hypothesized to be a cause of autism spectrum disorder. When C. elegans IRE-1/PTEN is disrupted, we found IL2 dendrite branching defects.

IRE-1 activates a stress response that plays a critical role in regulating dendrite growth

We looked at two variations of ire-1, (v33) and (ok799). We found branching defects in both alleles. However, we found these defects at low penetration where only 20% of the animals had severe defects.

Figure 1: Environmental stress causes an alternative larval stage called dauer. Upon return to favorable environmental conditions the animal recovers from dauer and resumes normal development.

Figure 2: Neuroplasticity of IL2 neurons during dauer. The IL2 densities (A) are unbranched in non-dauer animals. During dauer, the densities (B) branch extensively. White arrow indicates primary dendrite. Red arrow indicates body wall branch during dauer. The IL2s are labeled using klp-6::gfp. Scale bar is 10 µm.

Figure 3: The dauer mutant showed a lack of IL2 branching. The IL2s are labeled using klp-6::gfp. Scale bar is 10 µm.

Figure 4: ire-1(ok799) and ire-1(v33) are both large deletions. We found that IRE-1 (v33) and ire-1(ok799) have been linked separately to dauer formation and to neuronal defects, respectively. We found branching defects in both alleles. However, we found these defects at low penetration where only 20% of the animals had severe defects.

IRE-1 is a critical component of the IL2 branching pathway. We found allelic differences between ire-1(ok799) and ire-1(v33). ire-1(ok799) appears to have an intermediate resistance to SDS as compared to non-dauer animals. ire-1(v33) appears to lack resistance to SDS as compared to non-dauer animals.

Figure 5: (v33) and (ok799) are representative of severe branching defects (A,B). The IL2s are labeled using klp-6::gfp. Scale bar is 10 µm.

Figure 6: Analysis and Tukey’s Multiple Comparison Test were performed. In (ok799), n=20 for 0.2% and n=60 for 0.5%, 1x/20: n=60 for all treatments. Dauer is negative control; non-dauer is positive control. (ok799), there was a significance between the non-dauer and 0.5% treatment but no significance between non-dauer and 0.2%. For (v33), there was no significance between non-dauer and any treatment. Different letters indicate statistical significance.

Figure 7: We did a 1% SDS test to isolate dauers of each allele. Exposure to 1% SDS is a common way to isolate true dauers. None of the (v33) animals survived and only 3 of the (ok799) animals survived. Interestingly, these results contradicted literature that stated (v33) mutants form 100% dauers. This led us to question whether these animals were true dauers. We then performed SDS dose response assay to assess susceptibility. We used the following SDS concentrations: 1%, 0.5%, 0.2%, 0.1%.

Figure 8: SDS Results

Acknowledgments

Financial support was provided by the National Science Foundation under grant #NSF REU 1559906/1559929, as part of the Phenotypic Plasticity Research Experience for Community College Students, through the University of Illinois at Urbana-Champaign Institute for Genomic Biology and Parkland College. http://precs.illinois.edu/