May 20th, 12:00 PM - 3:00 PM

Roles of ubiquitin and stress in diacetyl chemosensation of C. elegans

Ellen Zocher  
*Western Washington University*

Nelson Ruth  
*Western Washington University*

Marissa Hogg  
*Western Washington University*

Follow this and additional works at: [https://cedar.wwu.edu/scholwk](https://cedar.wwu.edu/scholwk)

Part of the [Biology Commons](https://cedar.wwu.edu/scholwk/2016/Day_two/13), and the [Higher Education Commons](https://cedar.wwu.edu/scholwk/2016/Day_two/13)

Zocher, Ellen; Ruth, Nelson; and Hogg, Marissa, "Roles of ubiquitin and stress in diacetyl chemosensation of C. elegans" (2016). *Scholars Week*. 13.  

This Event is brought to you for free and open access by the Conferences and Events at Western CEDAR. It has been accepted for inclusion in Scholars Week by an authorized administrator of Western CEDAR. For more information, please contact [westerncedar@wwu.edu](mailto:westerncedar@wwu.edu).
**BACKGROUND**

Ubiquitin is a small protein that can be attached to other proteins in a cell, tagging them for destruction. The process of adding ubiquitin to a protein substrate (ubiquitination), and the subsequent trafficking and degradation of this substrate, is a principle regulator of the abundance and activity of many proteins across all forms of life. We are examining the role and dynamics of this regulatory system in the olfactory neurons of the model organism Caenorhabditis elegans, specifically the olfactory receptor protein ODR-10, which allows the worm to detect diacetyl, a volatile compound that is produced by the bacteria the worm eats. Without ODR-10, worms cannot properly perform "chemotaxis"—detection and movement toward food (Bargmann et al., 1996).

The ubiquitin-mediated degradation pathway is known to regulate other cell-surface receptors in neurons (Kowalski et al., 2011), and by testing the diacetyl-sensing ability of worms with mutations in this pathway, we found that abnormal ubiquitination in the ODR-10 expressing neuron ("AWA") leads to reduced diacetyl detection in the worm (Fig. 1), suggesting that the ubiquitin pathway is indeed involved in regulating ODR-10 (Fig. 2).

To further characterize how the ubiquitin degradation pathway functions to regulate the worm’s sense of smell and the trafficking of ODR-10, we put worms with ubiquitin pathway mutations under conditions of food stress and compared both their food-seeking behavior and ODR-10 abundance/localization within the AWA neuron to the wild-type strain.

**RESULTS**

**Figure 1.** Chemotaxis is impaired in worms with overexpression of ubiquitin.

**Figure 2.** Representation of the regulatory control of ODR-10 by ubiquitin. VPS4 is a protein that allows ubiquitinated proteins to enter the lysosome for degradation. Such systems are constantly acting to regulate protein amounts.

**Figure 3.** Chemotaxis indexes of Wild-type and DN-VPS4-4 worms under fed/starved conditions. (B) ODR-10::GFP +/+ fluorescence assay results. See figure 5 for key.

**Future Directions**

Based on these data, there are many ways we can continue to explore this pathway. We can continue to study chemotaxis ability and imaging simultaneously on strains that have other mutations along the ubiquitin-mediated degradation pathway, such other enzymes that regulate ubiquitin or the ESCRT proteins, (E2/E3, pre-β-arrestin kinases) or we could stress the worms in different ways (heat, axonic, osmolality) and observe their ODR-10 abundance/localization and chemotaxis abilities.

We would like to thank Lina Dahlberg for her patience and guidance and the Office of Research and Sponsored Programs for providing necessary funding to carry out these tests.