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Proteomic analysis of the decapod molting gland (Y-organ) in response to PKG inhibition

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Physiological regulation of molting in decapods is predominantly coordinated by two hormones, the peptide molt-inhibiting hormone (MIH), and steroid molting hormones termed ecdysteroids. MIH is produced and secreted by the X-organ/sinus gland complex located in the eyestalk ganglia, and negatively regulates the production of ecdysteroids in the molting gland (Y-organ, YO). MIH signaling begins with a cAMP-dependent triggering phase followed by a cGMP-dependent summation phase which ultimately leads to inhibition of mTORC1. Previous work revealed that two cGMP-dependent protein kinases (PKG1 and PKG2) have opposing roles in modulating ecdysteroidogenesis via MIH signaling in YOs. Specifically, PKG1 plays a dominant role in MIH signaling by inhibiting ecdysteroid synthesis, while PKG2 counters that inhibition and maintains basal ecdysteroidogenesis in the intermolt YO. This study aims to use sample multiplexing alongside phosphopeptide enrichment in LC-MS/MS to identify potential substrates of PKG in the YO. Transcript expression of PKG1 is two orders of magnitude greater than that of PKG2 in the intermolt YO, and preliminary proteomic data identified peptides from PKG1 but not PKG2. This data indicates that protein expression may roughly follow transcript expression for the two PKG isozymes, providing a basis for the differential effects of their opposing roles. Supported by NSF grants to DM (IOS-1922701) and LT (IOS-1922718 & IOS-23221487).