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Meeting Abstract

P3.49 Friday, Jan. 6 **Sirtuin-induced protein deacetylation affects the heat shock response in blue mussel congeners (*Mytilus*)** *BEAM, M*; ZUZOW, M; TOMANEK, L; California Polytechnic State Uni., San Luis Obispo; California Polytechnic State Uni., San Luis Obispo; California Polytechnic State Uni., San Luis Obispo* mbeam@berkeley.edu

The warm-adapted Mediterranean blue mussel species *Mytilus galloprovincialis* invaded southern California during the last century and has since replaced the cold-adapted native *M. trossulus* from its southern range, possibly due to climate change. Based on previous proteomic analyses, we hypothesized that the more heat-sensitive *M. trossulus* switches from NADH-producing metabolic pathways that may generate reactive oxygen species (ROS) to NADPH-producing pathways that are able to scavenge ROS during severe heat stress (32°C). We further linked these changes to the activity of the mitochondrial NAD-dependent deacetylase, sirtuin-5, which has been shown to regulate many metabolic pathways. To test the latter hypothesis, we repeated the experiment for both species by exposing gill tissues to 37°C, 28°C, 32°C and 35°C (heating rate of 6°C/h) seawater for 1 h with a subsequent 24 h recovery at 13°C under constant aeration. In a parallel set of incubations we added suramin, a potent sirtuin inhibitor, to characterize the effect of sirtuins on the stress response. Applying a gel-based proteomic analysis and mass spectrometry, we found that sirtuin inhibition affected 19% and 25% of all protein changes during heat stress in the warm-adapted *M. galloprovincialis* and the cold-adapted *M. trossulus* (excluding 35°C), respectively. Identified proteins function as molecular chaperones, in proteolysis, signaling, ROS scavenging, energy metabolism, and cytoskeletal dynamics. The number of proteins that were affected by sirtuins doubled in *M. trossulus* at 35°C, suggesting possible thermal damage of proteins or a role of internal lysine-acetylation in protein degradation as has been shown for N-end lysine-acetylation.