

P042 An integrin-containing muscle adhesion complex opposes muscle protein degradation in *C. elegans*

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Across phyla, spaceflight induces changes in muscle, including decreased mass and strength. Returned *C. elegans* display uncoordinated movement and decreased expression of ~100 'muscle' genes, many are likewise decreased in returned mammals. We find that acute treatment of adult *C. elegans* with RNAi against one of these genes, *unc-97*(PINCH/LIM-domain), causes muscle protein degradation. Acute RNAi treatment against any of another eight genes, whose products are likewise members of an integrin-containing muscle adhesion complex, also causes degradation. Experiments using a temperature-sensitive mutation in one of these genes, *unc-112*(MIG-2), confirm that members of the complex normally act to oppose this degradation. Suggesting specificity in the regulation of degradation, we find that RNAi against the gene for another complex member, *unc-95*(LIM-domain), fails to cause muscle protein degradation. In *C. elegans*, AChR-Ca⁺⁺ and IGFR-FGFR signalling networks control muscle protein degradation via proteasome and non-proteasome dependent mechanisms, respectively. Drugs, mutations, and RNAi treatment known to block degradation in *C. elegans* muscle fail to block degradation triggered by acute loss of members of the attachment complex. Current goals include determining the identity and regulation of the relevant protease(s).