Title:	Cell Adhesion-Mediated Actomyosin Assembly Regulates the Activity of Cubitus Interruptus for Hematopoietic Progenitor Maintenance in Drosophila
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Abstract:	The actomyosin network is involved in crucial cellular processes including morphogenesis, cell adhesion, apoptosis, proliferation, differentiation, and collective cell migration in Drosophila, Caenorhabditis elegans, and mammals. Here, we demonstrate that Drosophila larval blood stem-like progenitors require actomyosin activity for their maintenance. Genetic loss of the actomyosin network from progenitors caused a decline in their number. Likewise, the progenitor population increased upon sustained actomyosin activation via phosphorylation by Rho-associated kinase. We show that actomyosin positively regulates larval blood progenitors by controlling the maintenance factor Cubitus interruptus (Ci). Overexpression of the maintenance signal via a constitutively activated construct (ci.HA) failed to sustain Ci-155 in the absence of actomyosin components like Zipper (zip) and Squash (sqh), thus favoring protein kinase A (PKA)-independent regulation of Ci activity. Furthermore, we demonstrate that a change in cortical actomyosin assembly mediated by DE-cadherin modulates Ci activity, thereby determining progenitor status. Thus, loss of cell adhesion and downstream actomyosin activity results in desensitization of the progenitors to Hh signaling, leading to their differentiation. Our data reveal how cell adhesion and the actomyosin network cooperate to influence patterning, morphogenesis, and maintenance of the hematopoietic stem-like progenitor pool in the developing Drosophila hematopoietic organ.
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