Title:	Physiological ROS controls Upd3-dependent modeling of ECM to support cardiac function in Drosophila
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Abstract:	Despite their highly reactive nature, reactive oxygen species (ROS) at the physiological level serve as signaling molecules regulating diverse biological processes. While ROS usually act autonomously, they also function as local paracrine signals by diffusing out of the cells producing them. Using in vivo molecular genetic analyses in Drosophila, we provide evidence for ROS-dependent paracrine signaling that does not entail ROS release. We show that elevated levels of physiological ROS within the pericardial cells activate a signaling cascade transduced by Ask1, c-Jun N-terminal kinase, and p38 to regulate the expression of the cytokine Unpaired 3 (Upd3). Upd3 released by the pericardial cells controls fat body–specific expression of the extracellular matrix (ECM) protein Pericardin, essential for cardiac function and healthy life span. Therefore, our work reveals an unexpected inter-organ communication circuitry wherein high physiological levels of ROS regulate cytokine-dependent modulation of cardiac ECM with implications in normal and pathophysiological conditions.
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