Title. Acute exposure of Nicotine during Drosophila puncture injury activates an epidermal wound response.

Authors. Michelle T. Juarez[1]*, Chrissy Cherenfant[1]

Affiliations. [1] City College of New York, CUNY School of Medicine, Sophie Davis Program of Biomedical Education, New York, NY

*Corresponding Author *Email Address:* mjuarez@med.cuny.edu (MT Juarez)

Abstract.

Our body has many cellular functions to maintain homeostasis. One of these cellular functions is the mechanism of cellular repair and wound response. When localization of a wound response occurs only in damaged tissue, the body is then able to recover from injury such as, surgery or disease. Wound response can be visualized in *Drosophila melanogaster* embryos by wound-dependent and epidermal-specific fluorescent wound reporters. These reporters each display their respective phenotypes when activated, being restricted to a local surrounding the wound site or extending to a global area in all epidermal cells. External chemicals allow the testing of a diverse array of components that contribute to the regulation of wound response localization. Our hypothesis is that nicotine exposure to *Drosophila* embryos will activate a global wound response after acute exposure through microinjection. We may gain improved understanding of how external factors and human actions, like smoking, may ultimately affect healing after injury. Future experiments will focus on genetic components of nicotine signaling (e.g. Nicotinic acetylcholine receptors) and the interplay between activation of the wound response. Determining the effect of nicotine exposure on wound response in *Drosophila* will provide insight into the impact of commonly used drug on tissue repair.

Keywords.

Wound Response, Nicotine, Epidermal, Dopa decarboxylase, Nicotinic acetylcholine receptor

Current Funding.

NIAID R03AI117671
NCI U54CA137788/U54CA132378
NIMHD G12MD007603.