# The effects of a bacterial endotoxin LPS: neuromuscular junction and cardiac function in Drosophila melanogaster and Phaenicia sericata larvae.

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***Abstracts will be considered for both poster and platform presentations***

***Neurophysiology***

Gram negative bacterial septicemia inflicts humans as well as other animals. The immunological response to bacterial infection activates cascades of defense cytokines and antibody formation (1). Two common culprits in mammals are Pseudomonas aeruginosa (P.a.) and Serratia marcescens (S.m.)(2-5). The induced cytokines and defense response to the surface antigens on bacteria accounts for some of the immune response but also the secretion of lipopolysaccharides (LPS) is responsible for a large degree of the immune response. The direct action of bacterial LPS endotoxin was shown to enhance synaptic transmission and hyperpolarize the membrane potential at low dosage but block glutamatergic receptors and decrease observable spontaneous events at a high dosage. The dosage effects are LPS type specific. The hyperpolarization is not due to a voltage gated potassium channels or due to activation of nitric oxide synthase (NOS). Comparative effects of LPS on heart rate (HR) were

examined in larvae. Acute direct exposure of in situ heart tubes with saline containing at 500 µg/ml LPS from two common bacterial stains (P.a. and S.m.) showed a dose-dependent effect on HR but different responses for the two fly models. LPS is likely altering ionic balance of the pacemaker potential by inducing a hyperpolarization of the cardiac muscle. Currently, we are investigating research with lower doses of LPS. The significance of these findings is a better understanding the direct mechanism of action of LPS on synaptic function and effects on HR induced without the effect of an immune response as occurs in intact animals. Knowing the acute and direct actions of LPS exposure on larvae in these species may aid in understanding the underlying mechanisms in other animals during septicemia. We thank Ms. Kameron Roach, Ms. Amanda Paschal, Ms. Alexandra Stanback, Mr. Jaylen Scott and Ms. Nicole Audia for helping in conducting these experiments. Funding was provided by personal funds (R.L.C.), student tuition, and a “Sustaining Excellence-2014” competition grant from the Howard Hughes Medical Institute (Grant #52008116) awarded to the Univ. KY (VM Cassone, PI). The authors confirm that the HHMI funder had no influence over the study design and content.