Traumatic stress history interacts with sex and chronic peripheral inflammation to alter mitochondri

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Exposure to life-threatening events can result in mental disorders such as Posttraumatic Stress Disorder (PTSD). In addition, inflammatory disorders have been associated with PTSD. Given this, along with the notion that mitochondrial function and synaptic connectivity may be impacted by PTSD and inflammation, Shaw and colleagues investigated how traumatic stress modulates mitochondrial bioenergetics at the synapse in response to sex and inflammation. Males exposed to chronic (predatory) stress and chronic lipopolysaccharide injections demonstrate a deficit in synaptosomal respiration while in females, chronic stress or chronic LPS was sufficient. Moreover, males demonstrate an increase in central and peripheral inflammation and reactive oxygen in response to stress and chronic LPS, while females do not. In response to acute LPS injections, females demonstrate an increase in peripheral inflammatory markers, however, chronic LPS failed to stimulate an increase in peripheral and central inflammatory factors or reactive oxygen. These findings provide further insight into how sex, inflammation, and stress mediate changes in mitochondrial bioenergetics and how chronic stress and inflammation may contribute to altered synaptic function.

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