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Abstract

Posttraumatic Stress Disorder (PTSD) is associated with elevated noradrenergic signaling, which has an impact on emotional learning and memory. Fear extinction is thought to underlie the processes of exposure therapy, however the relationship between noradrenaline and extinction in PTSD is unclear. Participants with PTSD (n = 21), trauma-exposure without PTSD (TC; n = 36), and non-trauma-exposed controls (NTC; n = 27) completed a fear conditioning and extinction paradigm, and conditioned fear was indexed by skin conductance response (SCR). Salivary α-amylase (sAA) collected at baseline and immediately post-fear acquisition was used as an index of noradrenaline, and we examined whether sAA in response to fear acquisition was a moderator between fear extinction and PTSD symptoms. While there was a significant increase in sAA from baseline to post-fear acquisition, this was not modulated by group. Compared to TC and NTC, the PTSD group displayed a slower decline in SCRs during early extinction, which generalized across stimulus type, and was not