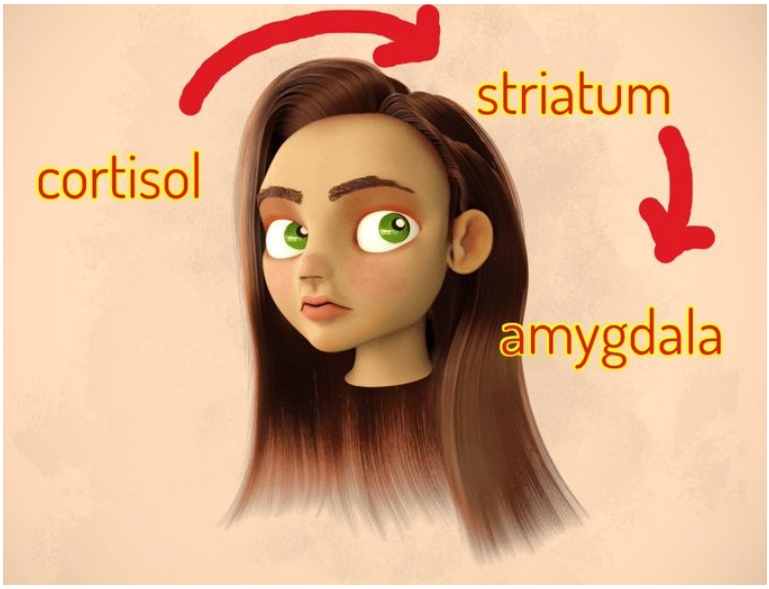

Stress-dependent cortisol modulation promote instrumental conditioning through inhibitory dopamine activity in the paraventricular nucleus of the thalamus.

Jack Bortone
jack@open-neurosecurity.org
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Abstract

Chronic intermittent hypoxia (CIH)-induced noradrenergic dysregulation is associated to increased disinhibitory dopamine activity in the paraventricular nucleus of the midline thalamus (PVT). It appears likely that chronic hypoxia increases cortisol production through a disinhibitory reuptake mechanism associated with a locus coeruleus (LC-NE) mediated rise in extracellular dopamine levels. Consequently, chronic intermittent hypoxia (CIH) with a surgical mask may enhances stress-dependent cortisol responses (c-Fos expression) in the paraventricular nucleus of the thalamus (PVT) through dopaminergic projections from the locus coeruleus. [reference]

Hypoxia-mediated cerebral hypometabolism

Hypoxia-induced changes to noradrenergic signaling may modulate the sympathetic nervous system thereby altering cerebral blood glucose levels in

the midbrain region (striatum and hippocampus). Secondly, the upregulation of cerebral blood flow (CBF) in the striatum caused by hypoxia-induced sympathetic activity is influenced by nitric oxide (NO)-mediated metabolic changes (ie: SpO2/FiO2) in tissues.

Hypoxia-induced instrumental conditioning

The persistent stress-induced dopamine (D2) modulation in the PVT may creates a motivational conflict associated to the neurocircuitry of fear extinction learning and may promote passive avoidance mecanism.

Effects of chronic social isolation in womens experiencing intimate partner violence (IPV)

Chronic social isolation (SI) may promote stress-dependent inhibitory control of fear extinction memory and reinforce passive coping mechanisms in womens experiencing intimate partner violence (IPV). Likewise, chronic social isolation may enhance COVID-19 associated instrumental conditioning through the persistent modulation of locus coeruleus (LC) activity thereby increasing cortisol levels.

Conclusion

Hypoxia-mediated noradrenergic dysregulation is driven by increased basolateral amygdala-striatum reactivity altering the dopamine-noradrenaline response (LC-NE) following chronic episodes of mild and intermittent hypoxia (IH), independently of pulse oxymetry status. Stress-dependent cortisol modulation of the striatum may promotes aversive and instrumental coping mechanisms through inhibitory connectivity from the locus coeruleus.

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