Sleep rebound induced by immobilization stress: Neuronal consequences

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Stress has been shown to affect the normal functioning of many systems of the body, including the nervous system, the endocrine system, and the immune system. It is now clearly established that an acute stress activates the hypothalamus-pituitary-adrenal (HPA) axis to increase circulating glucocorticoids. On the other hand, the brain targets for glucocorticoids normally participate in negative feedback regulation of the HPA axis. Moreover, an increased need for sleep during the recovery phase occurs after stressful experience. The sleep rebound occurring after stress being part of the restorative process necessary to compensate for stress overshoot. Following one-two hours immobilization stress, slow wave sleep (SWS) and rapid eye movement (REM) durations are augmented. A beneficial action of the stress-induced sleep rebound (during waking period) on brain homeostasis and behavioral adaptation is therefore commonly accepted. Our investigations indicate that this effect is mediated by neuronal activation observed in the hypothalamic paraventricular and noradrenergic locus coeruleus (LC) after immobilization stress and in the preoptic area (POA) and arcuate nucleus (ARN) during stress-related sleep rebound. An unbalanced in the above processes may contribute to pathological outcomes, such as depression and anxiety.

Keywords: Stress; Sleep; PVN; ARN; LC