

Work. Stress. Sleep. Repeat. Breaking the cycle of mutually reinforcing work stress and sleep deprivation

*Work stress and sleep deprivation have accumulated effects on biological processes that maintain health. **Odessa Hamilton** summarises the evidence and explains why sleep should be a targeted behavioural treatment.*

The intersected costs of **stress at work** and **dysfunctional sleep** are substantial from a population perspective. Both are prevalent in societies with major global economies that drive decisions to maximise productivity to the detriment of sleep. The COVID-19 pandemic exposed variations in the magnitude of distress ([Hamilton et al., 2021](#)), with noteworthy disparities seen in occupational experiences between groups ([Baker, 2020](#)). Meanwhile, an estimated 25-30% of adults worldwide report persistent sleep issues, with 260 million people projected to be inflicted with sleep dysfunction by 2030 ([Stranges et al., 2012](#)).

Prolonged exposure to occupational stress is a recognised influencer of disease onset, progression, and severity, while expediting mortality ([Armon, 2009](#)). Equally, sleep is a key modulator of health, but its dysfunction is long known as an antecedent to disease across a spectrum of severity, with involvement also in all-cause mortality ([Brauchi & West, 1959](#)). These aggregate outcomes impose a great burden on society ([Jansson & Linton, 2006](#)). Still, despite their ubiquitous nature, both are widely neglected influencers of disease across the lifespan ([Prather, 2019](#)). But what starts the downward trajectory of effects remains unknown. Prognostic sequencing in either direction is theoretically plausible, which develops into a clinical uncertainty.

The chicken or the egg

Stress is as antithetical to sleep as sleep is to stress ([Jones & Gatchel, 2018](#)). There is robust evidence that stress and sleep deprivation frequently co-occur and are reciprocally reinforcing ([Heffner et al., 2012](#); [Stipp, 2019](#)), and yet we instinctively know this to be true through common experience. Feelings of stress can impede sleep onset and cause multiple wake periods that have us up at night – tossing and turning, fraught with a thousand thoughts that overwhelm our natural ability to sleep. Equally, experiences of occupational stress, arising from high demands or low decision latitude, is a salient trigger and maintainer of sleep deprivation ([Jansson & Linton, 2006](#)).

Conceivably all of us have felt how even one night of poor sleep interfered with how well (or not) we functioned at work the following day. Even the mere anticipation of excessive work demands has been described as a “mechanism of sleep interference” ([Stipp, 2019](#), pg. 104). At the same time, sleep plays a central role in affective processes that promote resilience and recovery from stressful experiences ([Germain, 2013](#)). Originating in the prefrontal cortex with connections to the amygdala, deep sleep (~20% of total sleep) and rapid eye movement-sleep (REM-sleep; 20-25% of total sleep) have been found to consolidate emotions following stress exposure to assist in the recovery of daytime stressors ([Stipp, 2019](#)). A lack of sleep, additionally, attenuates the psychological threshold for stress perception ([Minkel et al., 2012](#)); heightening our experience of stress, and rendering us less able to cope with everyday burdens, which ultimately leads us to poor occupational outcomes ([Godet-Cayré et al., 2006](#); [Uehli et al., 2014](#)).

Mutual physiological derangements

Associations between stress and sleep can be understood by the physiological derangements seen in both phenomena. One proposed pathway through which they are connected is in the mechanistic action of sleep-supporting neurally integrated immunity that anticipates threat ([Irwin, 2019](#)). Disruption to this process would lead to over-sensitised stress experiences. Experimental studies have also offered evidence that sleep deprivation alters baseline activity of the stress system, along with physiological responses to stress ([Meerlo et al., 2008](#)). Another popular proposition is the persistent overproduction of circulating pro-inflammatory factors (i.e., cytokines) and increased immune cell infiltration, that arise in response to the parallel action of stress and dysfunctional sleep. They together give reason for dysregulated inflammatory responses and prolonged states of systemic low-grade inflammation ([Irwin, 2008, 2019](#)). Yet, remarkably, there is indication of multi-directionality between stress, sleep and neuroimmunological processes, suggesting natural confounding that informs a positive feedback-loop perpetuating a downregulation of processes ([Dolsen et al., 2019](#)).

Psycho-neuro-immunology

As mutual cytokine inducers, stress and sleep have common deleterious health outcomes ([Irwin, 2008, 2019](#)), which comes as no surprise. As shared in an earlier [article](#), psychoneuroimmunology (PNI) is the process through which stress disrupts the functionality of various aspects of the integrative network between immunity, endocrinology, and the central nervous system (CNS), in maintaining health. PNI is an equally useful framework to understand how poor sleep activates inflammatory signalling pathways that elevate proinflammatory cytokines ([Heffner et al., 2012](#)). PNI research on the reciprocal connections between sleep, immunity and the CNS have demonstrated that sleep enhances immune defences and afferent signals from immune cells that subsequently promote sleep ([Besedovsky et al., 2019](#)). Similarly, poor sleep potentiates affective and biochemical stress responses ([Germain, 2013; Minkel et al., 2012](#)) that, in turn, contribute to poor sleep ([Jones & Gatchel, 2018](#)), while each upregulate inflammatory processes ([Irwin, 2008, 2019](#)).

Sleep soundly, live lively, work gainfully

Against this interdisciplinary backdrop, sleep is one component with compelling prescriptive properties; said to have robust efficacy as a targeted behavioural treatment ([Jackowska et al., 2013](#)). It has a protective effect and typically increases naturally to promote recovery ([Stipp, 2019](#)), and as a prototypical recovery activity, sleep confers salubrious effects on immunity ([Prather, 2019](#)). While the prescription of sleep is an unlikely stress panacea, it has the potential to alleviate many of its symptoms ([Germain, 2013; Jackowska et al., 2013](#)). Thus, the ultimate message is to prioritise sleep as an activity worthy of your attention. Sleep soundly to live lively and work gainfully!



Notes:

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