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In Sleep Lost, Emotions Become Unrecognized: Commentary on Minkel et al.'s, "Emotional Expressiveness in Sleep-Deprived Healthy Adults"

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In Sleep Lost, Emotions Become Unrecognized:  
Commentary on Minkel et al.’s,  
“Emotional Expressiveness in Sleep-Deprived Healthy Adults”  

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Emotions are powerful things. Not only do they modulate our cognition, but, perhaps more critically, they motivate our actions; and not just any actions—the most optimal actions. In other words, emotions are designed to make us do things. Consider the last time you saw a child’s face with tears rolling down his or her cheeks as they sobbed. You wanted to do something; you wanted to help. Emotions, therefore, are of profound importance to the ubiquity of species imbued with them. Their physiological antecedents reach far back in evolution—a telling sign of their utility.

It is for these reasons that the pioneering and elegant work of Minkel, Htaik, Banks, and Dinges (this issue), examining the impact of sleep deprivation on the recognition and expression of human emotions, is so striking in result and concerning in ramification. To set the stage of appreciation, we can consider lessons from neurology. Patients who suffer an inability to recognize certain emotions due to lesions in key limbic and prefrontal brain regions display profound social dysfunction, being unable to detect and, subsequently, be guided by relevant affective cues (Damasio, 2000). Developmental disorders, such as autism, afford further evidence for impaired social interaction caused by abnormal affective processing, particularly facial expressions carrying emotional significance (Sasson et al., 2007).

The work of Minkel et al. (this issue) suggests we should now add the brain state of sleep deprivation to this list of circumstances where expressions and subjective perceptions of
emotion become adversely compromised. In the study, following a baseline assessment under sleep-rested conditions, participants were either sleep deprived for one night or again slept normally, all occurring in a lab under carefully controlled experimental conditions. To examine the impact on emotional processing, participants were shown evocative film clips that were either positive in affect (amusing) or negative in affect (sad). While they viewed these film clips, participants themselves were videotaped, and their face expressiveness was coded using a validated assessment procedure.

Relative to the sleep-rested condition, sleep deprivation imposed a significant blunting in the facial expressiveness of emotion displayed by participants to the amusing film clips and, to a lesser degree, the sad film clips also. This blunting could be due to the participants themselves not experiencing the emotions as significantly. Alternatively, participants may simply not be able to physically express emotions that they nonetheless may have been generating. This is where the researchers’ decision to collect additional data proved insightful. Not only were the facial expressions of emotions coded, participants also subsequently rated the degree of emotion they themselves perceived—a subjective complement to the objective facial-coding measure. Although not reaching significance, the concomitant decrease in subjective assessment supported the possibility that the impaired expression of emotion was related to a genuine decrease of subjective feelings.

This still does not necessarily mean that the emotions were not as—or even more—strongly registered in the brain and body of the sleep-deprived participants. It could be that those signals are present, but that key regions of the sleep-deprived brain, particularly areas of the prefrontal cortex where those emotional signals are integrated, are impaired. Indeed, the prefrontal cortex appears to be especially sensitive to the impact of sleep deprivation (Dahl, 1996; Horne, 1993; Thomas et al., 2000, 2003; Yoo, Gujar, Hu, Jolesz, & Walker, 2007). Future work attempting to simultaneously assess neural, peripheral, and subjective measures will prove valuable in this regard. Nevertheless, these findings complement and, more important, extend emerging experimental work demonstrating that sleep deprivation compromises the ability to detect selective face expressions of emotion in others, particularly in certain ranges of affect intensity (van der Helm, Gujar, & Walker, 2010). They add behavioral and subjective insights onto the emerging descriptions of neural (Yoo et al., 2007) and physiological (Franzen, Buysse, Dahl, Thompson, & Siegle, 2009) abnormalities of emotional reactivity associated with sleep loss.

Parenthetically, Hollywood knows all too well the power of facial emotions, both in their deliberate presence and conspicuous absence. It is no coincidence that the classical silver screen villain is so often masked. Consequently, not only is the visual identity of the rogue character withheld from us, but the salient signals of any emotional expressiveness are also obscured. We are, therefore, unable to emotionally “read” such individuals. It is unnerving, and puts us at a disadvantage for understanding the individual, predicting the individual, and orchestrating behavior toward that individual. In many ways, it is this same compromising shroud that the report of Minkel et al. (this issue) teaches us about sleep deprivation, with very real ecological relevance. Consider professional and societal circumstances where sleep deprivation is common. In many of these, be it doctors and medical staff, military personnel or new parents, the expression and recognition of emotional signals—as well as the need to be guided by them—is utterly critical. Worryingly, it is just these processes that appear compromized by lost sleep.
As with all good pioneering research, the study by Minkel et al. (this issue) raises more questions than it provides answers, offering fruitful targets for future studies in larger sample sizes. We currently know little about the underlying peripheral and central nervous system mechanisms leading to abnormalities of emotion processing caused by sleep deprivation. Luckily, there is rich neuroscientific literature describing the brain circuits and systems that support the panoply of emotions we experience, as well as their associated neurochemistry. Taking advantage of this knowledge and translating it to the domain of sleep research should offer fertile means for an improved neurobiological account of such impairments.

Similarly, although we continue to learn about the detrimental affective impact caused by the absence of sleep, we know far less about the proactive benefit of sleep on emotional brain function when it is obtained. What is it about sleep—its stages, its brain oscillations, its neurochemical cocktail, its cycling nature, and its timing—that seemingly resets the default balance of next-day emotional stability? Conversely, why do some emotional events we experience while awake impact our sleep at night, whereas others do not? Is it their intensity, novelty, salience, valence, time proximity to sleep, relationship with past experience, or degree of unresolved understanding? We need look no further than clinical circumstances, such as posttraumatic stress disorder, to appreciate this wake–sleep reciprocity of emotion interaction.

This rich litany of questions is testament to the thought-provoking work of Minkel et al. (this issue). Such findings emphasize that, beyond the role of sleep in cognitive processes such as learning and memory, an equally intimate but less defined functional relationship exists between sleep and affective brain function. If correct, and when considering the continued erosion of sleep time throughout industrialized nations, the clinical, societal, professional, and public health ramifications of this association should stir emotions in us all.

REFERENCES


