No Evidence That Sleep Deprivation Effects and the Vigilance Decrement Are Functionally Equivalent: Comment on Veksler and Gunzelmann (2017)

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Abstract

Veksler and Gunzelmann (2017) make an extraordinary claim, which is that sleep deprivation effects and the vigilance decrement are functionally equivalent. Extraordinary claims require extraordinary evidence, which is missing from Veksler and Gunzelmann’s study. Their behavioral data offer only weak theoretical constraint, and to the extent their modeling exercise supports any position, it is that these two performance impairments involve functionally distinct underlying mechanisms.

Keywords: Psychomotor vigilance; Sleep deprivation; Vigilance decrement; Cognitive modeling

Veksler and Gunzelmann (2017) claim that performance impairments due to sleep deprivation are functionally equivalent to what is often known as the vigilance decrement, the performance impairment associated with passage of time in a vigilance task (as performed by people who are not sleep deprived). This is an extraordinary claim. Sleep deprivation has profound effects on the brain (e.g., Thomas et al., 2000), yet the vigilance decrement is measured under rested conditions. By analogy, children and older adults both perform worse than young adults on many cognitive tests, but their brains are different and no one would claim that the two deficits are caused by the same mechanism. And yet, Veksler and Gunzelmann’s claim implies, for example, that we can learn how people perform when they are sleep deprived by studying how they perform when they are not.

Veksler and Gunzelmann (2017) rest their case on a modeling exercise. They adapt a model previously developed to account for sleep deprivation effects (Gunzelmann, Gross, Gluck, & Dinges, 2009) to account for the vigilance decrement. One problem is that the
adapted model has low identifiability, meaning that it is difficult to distinguish from alternative models based on its fit to data. In general, the more complex the data, the fewer models can fit the data, so the stronger the case for one that does. The most complex data presented by Veksler and Gunzelmann are the response time (RT) distributions in their fig. 4. At first blush, the changing shape of the distributions across task blocks (as the vigilance decrement sets in) is impressively complex. In fact, however, this pattern is generic. To illustrate, I developed a simple model of my own, a gamma distribution with shape parameter 3 and a scale parameter that increases with task block. Fig. 1 plots the output, which reproduces the changing shape of the RT distributions as well as Veksler and Gunzelmann’s model does, showing that these distributions offer only weak constraint on models of the underlying cognitive mechanisms. The gamma distribution is a cognitive model in the sense that it represents a succession of processing stages (enumerated by the shape parameter) each with variable duration (the variability being indexed by the scale parameter). Here, the model says that there are three processing stages, and that the vigilance decrement is caused by an increase in system noise across task blocks.

Another problem is that Veksler and Gunzelmann’s (2017) modeling exercise actually suggests that sleep deprivation effects and the vigilance decrement are functionally distinct, just as the behavioral effects simulated by the two models are completely dissimilar. The sleep deprivation model of Gunzelmann et al. (2009) simulates lapses, defined behaviorally as trials in a simple detection task (the Psychomotor Vigilance Task, or PVT) on which RT is greater than 500 ms. (These long RTs are taken to reflect a failure to timely detect the stimulus.) At least two components of the model are necessary to simulate lapses. One is the

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Fig. 1. Plots of a gamma distribution matching the data in fig. 4 of Veksler and Gunzelmann (2017). Each curve represents the response time distribution from a block of performance on the Psychomotor Vigilance Task. The value of the gamma shape parameter is 3 for all curves. The value of the gamma scale parameter is 2, 2.25, 2.5, 2.75, 3, and 3.25 for the curves with the highest to the lowest peaks, respectively. The function is evaluated at $x = 0, \ldots, 40$, with the rightmost value representing the sum of the function evaluated at $x = 21, \ldots, 40$. 
microslapse, a single 50-ms system cycle on which no processing occurs. The other component is a contingency mechanism that links a microslapse on one system cycle to an increase in the probability of a microslapse on the next system cycle (Gunzelmann et al., 2009, p. 892). This contingency causes the model to periodically descend into a sleep-like state in which nothing gets done for a series of consecutive cycles. Such a series represents a lapse. After a lapse, some other component mechanism reverts the probability of a microslapse to baseline (Gunzelmann et al., 2009, p. 894), representing a transition back to wakefulness before any next lapse.

In the vigilance model, the contingency between system cycles seems to be vestigial, perhaps reflecting the low rate of lapses in the authors’ vigilance data (also recorded using the PVT, though in longer sessions). At the peak of the vigilance decrement, lapses were only about half as frequent as after a single night of sleep deprivation (5.23% vs. 9.03% of trials; Veksler & Gunzelmann, 2017). The lapse rate under vigilance conditions may even be beside the point, as we have no evidence that behavioral lapses during rested performance reflect anything like the physiological brain states associated with lapses after sleep deprivation.

Instead of simulating lapses, the vigilance model simulates the vigilance decrement. To do this, it uses a new mechanism that is not present in the sleep deprivation model. This new mechanism increases the baseline probability of a microslapse (Veksler & Gunzelmann, 2017, Eq. 2) gradually across the full, 35-min duration of a session, with no periodic reversions to wakefulness. This gradual, monotonic state change looks nothing like a lapse.

Thus, there is a functional double dissociation between the two models. The sleep deprivation model has a mechanism (the contingency between system cycles) that simulates lapses but not the vigilance decrement. The vigilance model has a different mechanism (change in baseline probability across a session) that simulates the vigilance decrement but not lapses. This dissociation means that sleep deprivation effects and the vigilance decrement are functionally as well as behaviorally distinct.

Veksler and Gunzelman will say that their claim of functional equivalence rests on the microslapse mechanism, which does play a role in simulating both sleep deprivation effects and the vigilance decrement. However, the microslapse mechanism also plays a role in simulating other, very different effects. In a model of mine, microslapses play a role in simulating a perceptual encoding effect in which stimulus processing is more efficient the shorter the stimulus duration (Altmann & Gray, 2008). If one accepts Veksler and Gunzelmann’s claim, this perceptual encoding effect is functionally equivalent to sleep deprivation effects and the vigilance decrement, which makes no sense. Like Navon’s (1984) theoretical soup stone, microslapses have little explanatory value on their own, without an appropriate theoretical garnish. The garnishes here suggest that sleep deprivation effects and the vigilance decrement are functionally distinct.

Veksler and Gunzelmann (2017) propose that performing a vigilance task for 35 min has the same effect on the brain as sleep deprivation. This is an extraordinary claim that requires extraordinary evidence, yet what evidence there is contradicts the claim.
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References