Further Evidence That Sleep Deprivation Effects and the Vigilance Decrement Are Functionally Equivalent: Comment on Altmann (2018)

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Abstract

Veksler and Gunzelmann (2018) argue that the vigilance decrement and the deleterious effects of sleep loss reflect functionally equivalent degradations in cognitive processing and performance. Our account is implemented in a cognitive architecture, where these factors produce breakdowns in goal-directed cognitive processing that we refer to as microlapses. Altmann (2018) raises a number of challenges to microlapses as a unified account of these deficits. Under scrutiny, however, the challenges do little to discredit the theory or conclusions in the original paper. In our response, we address the most serious challenges. In so doing, we provide additional support for the theory and mechanisms, and we highlight opportunities for extending their explanatory breadth.

Keywords: Fatigue; Vigilance; Sleep deprivation; Computational model

1. Introduction

Veksler and Gunzelmann (2018) present a computational model providing evidence that the vigilance decrement manifests as disruptions to goal-directed cognitive processing and performance that are functionally equivalent to those brought on by the deleterious effects of sleep loss. Altmann (2018) challenges the conclusions of Veksler and Gunzelmann (2018) based on a number of specific critiques. Some of these represent reactions to choices that were made in the writing of the original paper. For instance, we opted to include relatively little discussion of the limitations of a number of alternative
mechanisms to address identifiability (but see Gunzelmann, Gross, Gluck, & Dinges, 2009). We also included limited discussion of recent neuroscience results that provide converging evidence for our account (e.g., Krueger et al., 2008; Van Dongen, Belenky, & Krueger, 2011). On the other hand, several of the critiques require more careful consideration. We take up these in turn in the sections that follow.

2. Gamma distributions provide an alternative account

Altmann (2018) argues that the response time distributions for the Psychomotor Vigilance Test (PVT), reported in Veksler and Gunzelmann (2018) and modeled by us with microlapses, can be modeled more simply with a Gamma distribution. It is true that the Gamma distribution has a shape that is qualitatively similar to the response time distributions presented in Veksler and Gunzelmann (2018). However, it differs in certain respects that make it a poor model of response times in the PVT specifically, and of fatigue effects more generally. Most important, it produces no false starts, which are a key indicator of degraded performance in the task. In addition, it fails to fit the PVT response time distributions without additional parameters. Moreover, the parameters say nothing about the underlying processes, which reveals a fundamental issue with Altmann’s argument. Specifically, mathematical functions used to characterize response time distributions have no capacity at all to generalize to other tasks such as the Mackworth Clock Task (Mackworth, 1948). Generalizability to tasks that explicitly investigate the vigilance decrement is a crucial test of the model and theory, and it is a major focus of Veksler and Gunzelmann (2018). The fundamental limitation of Gamma distributions in this regard only reinforces the theoretical contribution of our research in helping to unify these two research domains.

3. Different theories account for the impact of sleep loss versus the vigilance decrement

Altmann provides several specific arguments suggesting that the account presented in Veksler and Gunzelmann (2018) is theoretically distinct from the theory of how sleep loss impacts cognitive performance. To paraphrase, Altmann (2018) suggests that lapses in the “sleep” model are a function of the capacity for microlapses coupled with a mechanism that increases the likelihood of a microlapse when one occurs within a trial (i.e., the “contingency” mechanism). He contrasts this with the “vigilance” model, which he suggests accounts for deficits through manipulation of the baseline probability of microlapsing.

Altmann is mistaken in his understanding of the mechanisms and their roles in the performance of the model. In fact, both the sleep and vigilance models manipulate the baseline probability of microlapses and both models incorporate the contingency mechanism. The baseline probability of a microlapse is influenced in the vigilance model by an
exponential function that controls changes to the utility value and the utility threshold as time on task accumulates. This is based on evidence that exponential functions capture the general shape of the decline observed in vigilance tasks (Giambra & Quilter, 1987). Analogously, we use biomathematical models of alertness (e.g., McCauley et al., 2013) to set utility and threshold values for each session in the sleep model (Walsh, Gunzelmann, & Van Dongen, 2017). They capture the shape of the decrements observed in tasks like the PVT resulting from the interacting effects of sleep loss and circadian rhythms across sessions (e.g., Van Dongen, 2004). Walsh et al. (2017) did not model intrasession changes in PVT performance, in part because PVT sessions tend to be shorter in sleep research (10 min) than what we used (35 min; Veksler & Gunzelmann, 2018). However, we have explored the conjunction of sleep loss and time on task in the PVT in the past (Gunzelmann, Moore, Gluck, Van Dongen, & Dinges, 2011).

At the same time, the contingency mechanism plays a crucial role in both models. When a microlapse occurs, the probability of a subsequent microlapse increases at a rate governed by a parameter we refer to as $FP_{dec}$. Critically, the value for $FP_{dec}$ is identical in both the sleep and vigilance models (0.98; Walsh et al., 2017; Veksler & Gunzelmann, 2018), which is held constant across levels of sleep loss in Walsh et al. (2017) and across time on task in Veksler and Gunzelmann (2018). In addition, in both models the probability of a microlapse reverts to the baseline value at the start of each trial. Without the contingency mechanism, the model does not produce the pronounced increase in lapses observed with time on task (Fig. 1). Far from being vestigial, this component of the model is critical in accounting for the time on task effect in the PVT (Veksler & Gunzelmann, 2018), just as it is necessary to capture the deleterious effects of sleep loss (Walsh et al., 2017).

4. Microlapses are generic enough to account for almost anything

Finally, Altmann (2018) argues that microlapses have little explanatory power, citing some of his earlier work as an illustration of how flexible they are. To consider this argument, it is necessary to understand the model in Altmann and Gray (2008) in more detail. Importantly, despite Altmann’s claim, they did not implement microlapses. They implemented a production, which they refer to as “unrelated-process.” Also, Altmann (2018) indicates that the mechanism helped to “account for a perceptual encoding effect…” (p. 3), but this is a little ingenuine. The real contribution of the unrelated-process production was to account for degradations in attentional focus in trials with longer intervals between the cue and stimulus presentations. Specifically, they associate production utilities related to cue encoding with “preparedness, which is aversive to maintain over time” (Altmann & Gray, 2008), p. 610). Later, they describe the function of the unrelated-process as accounting for situations where “attention lapses” (p. 612).

There are some similarities in the consequences for model performance between what Altmann and Gray (2008) implemented and microlapses, but their mechanism does not capture the “aversiveness” of sustaining attention over time. That is, the utility values in
their model fluctuate due to noise, but they do not change systematically over time. Veksler and Gunzelmann (2018) propose mechanisms that do capture such dynamics. Indeed, rather than stripping our theory of its explanatory power, Altmann (2018) provides an argument for the further generalizability of our mechanisms to other phenomena associated with “attention lapses.”

One may conclude from this that Altmann’s claim that microlapses are an overly general mechanism is surely correct. However, we disagree. We argue that degradations in performance brought on by the demands of maintaining attentional focus can operate at multiple timescales depending on the demands of the task and the limitations of the human cognitive system. Our theory is that the degradations manifest as breakdowns in the capacity for focused, goal-directed processing, which we implement as microlapses. Veksler and Gunzelmann (2018) demonstrate the explanatory power of this perspective in the context of time on task in the PVT and the vigilance decrement in the Mackworth

Fig. 1. Proportion of responses identified as lapses (response times greater than 150 ms) across blocks as a function of whether the “contingency” mechanism is enabled.
Clock Task, whereas other research has provided evidence for the theory in sleep loss and circadian rhythms (Walsh et al., 2017). We would find it unsurprising if similar dynamics were at play in human performance in Altmann and Gray (2008), where participants were asked to complete nearly 3,000 trials of an attention-demanding laboratory task in an experiment lasting 45 minutes. However, that is an issue that is outside the scope of our current research effort.

5. Conclusion

Altmann (2018) presents a number of challenges to the research and conclusions described in Veksler and Gunzelmann (2018). The most critical of these relate to the nature of the mechanisms, the degree to which they are consistent across the two domains, and whether they are constrained enough to inform our understanding of phenomena in the sleep loss and vigilance literatures. In this reply, we have reemphasized the consistency of the model and mechanisms across contexts, and we have discussed the central theoretical claims that may generalize further. We do not claim to have the final word in understanding the nature of fatigue effects from sleep loss and time on task. However, our model does illustrate that a single set of mechanisms can account for phenomena in both domains. We are humbled by Altmann (2017) characterization of this claim as “extraordinary” (p. 3), but we believe that the theory and the mechanisms are consistent with a growing body of empirical results from both domains.

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Note

1. Although the Gamma distribution has a similar shape, a scaling constant is needed to align the Gamma distribution with human response times. This improves the fit overall, but it further precludes the model from capturing the fastest response times of human participants.

References


