Review of: "How low working memory demands and reduced anticipatory attentional gating contribute to impaired inhibition during acute alcohol intoxication"

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We are particularly interested in this paper because it explores a surprising finding in the alcohol-cognition literature we first stumbled on five years ago. The effect is an alcohol performance impairment that occurs only when task demands are relatively low. When the task is manipulated such that its cognitive load is increased, alcohol performance is equivalent to that of sober controls. We have since observed this exact form of interaction in two different contexts, during an attentional reaction time (RT) task (Bayless & Harvey, 2017) and in an inattentional blindness task (Harvey, Hyams & Bayless, 2018). We have also revealed a closely related phenomenon in a test of number judgment (see Harvey & Seedhouse, 2021). For alcohol to impair performance of an easy rather than more difficult version of a task is puzzling. We were therefore intrigued to discover Stock and colleagues had observed this effect in the context of response inhibition – the ability to withhold prepotent yet task inappropriate responses (Stock et al., 2014, 2016).

In their most recent study (the focus of this review) Stock et al. (2022) investigated the boundary conditions under which acute alcohol intoxication increases impulsive responses in a test of motor inhibition known as the Go/No Go task. Participants were presented with a series of target stimuli (the items 5G7R) in either their mirrored or (standard) non-mirrored form. For high-demand trials (Block 1) they had to make a (button press) response only on non-mirrored “Go” trials (which occurred 70% of the time), with a left-hand button for letter stimuli and a right-hand button for number stimuli. For remaining (mirrored) “No Go” trials this habituated motor response had to be withheld. For low-demand trials (Block 2), they had to respond to letters (Go trials) but not numbers (No-Go trials), using a left-hand button press for mirrored letters and a right-hand button press for standard (non-mirrored) letters. Participants were tested under sober then alcohol intoxicated conditions. Task difficulty was manipulated by rotating each stimulus set such that it imposed either a low (30^o), medium (90^o) or high (150^o) working memory load. A counterintuitive pattern of results emerged with alcohol reducing inhibition of “No Go” responses significantly more often for trials imposing the lowest (Block 2, 30^o) rather than highest (Block 1, 150^o) mental workload.

We interpreted our alcohol-task difficulty interactions within the framework of *alcohol myopia theory* – the idea that alcohol depletes cognitive resources such that the focus of attention is restricted to only the most important or immediate stimuli (Steele & Josephs, 1990). Our simple suggestion was that alcohol impairs performance under low but not high working memory demands because high load tasks are so challenging they leave little spare attentional capacity for the drug to

deplete (Harvey et al., 2018; Harvey & Seedhouse, 2021). Stock et al. (2022), however, offer a more nuanced possibility. They suggest that motor responses to their easier Go trials are largely automated, thus demanding stronger inhibitions for successful No-Go trials – a cognitive control process which alcohol presumably weakens. For the harder task, a degree of complex cognitive control is needed even for Go trial responses, which the authors suggest is sufficient to disrupt automated responding itself thus negating the effects of alcohol.

While developed to explain inhibitory task performance, this theory generalises to varying levels of cognitive control across other tasks (cf. Bayless & Harvey, 2017; Harvey et al., 2018). In testing it, Stock et al. (2022) use EEG data to examine the neurophysiology of response inhibition under alcohol for low and high working memory demand versions of the Go/No Go task. They focus on event-related alpha and theta band activity – theta oscillations due to their association with cognitive control, inhibitory processes and WM; and alpha oscillations due to their association with WM and attentional control mechanisms. The authors suggest alcohol related effects on inhibitory processes are mediated by prefrontal cortical networks associated with WM and inhibitory processes, comprising the superior and middle frontal gyrus, and the inferior frontal gyrus. With the timing of these effects being either during the actual process of inhibitory control (up to 500ms after No Go stimulus presentation), or during the interval before the subsequent stimulus, reflecting alcohol myopia driven impairments to the anticipatory filtering (gating) of attention. Under low load conditions participants showed less theta desynchronisation when intoxicated than when sober (localised to the temporal cortex), indicating poorer attentional control under alcohol. Under high load conditions, however, less theta synchronisation (localised to central sites) was recorded when participants were intoxicated than when sober. This indicates increased attentional control under alcohol, consistent with the behavioural data. As expected, the authors also found the strongest source-level theta synchronisation under alcohol during low load trials, reflecting poorer inhibitory processing under these conditions. Plus, more synchronised task-related alpha band activity under alcohol for low load trials, suggesting less attentional gating under these conditions (localised to temporal and inferior frontal cortices).

The problem with this account, however, is that it hinges on the assumption that low demand Go responses are predominantly automated, yet no behavioural or neural evidence is presented to support this claim. The analysis in the main paper focuses on false alarm rates and the underlying neural processing for inhibitory No-Go trials only. But Go trial measures are also needed to evaluate the impact alcohol had on these. It is possible, for example, that some participants sought to avoid errors of commission by adopting a more conservative and therefore effortful Go response strategy, particularly with under demanding task conditions. We note the authors' supplementary Go trial analysis, which indicates that alcohol had a significant effect on Go trials for both trial blocks. But data in this are collapsed across easy (30^{o}) and hard (150^{o}) task conditions. What is needed is a signal detection theory (SDT) analysis incorporating both Go and No-Go performance within the same inferential test. Specifically, comparing rates of Go trial hits, Go trial misses, No Go false alarms and correct No Go rejections (for details see, Young, Sutherland & McCoy, 2017). This approach, along with a complementary analysis of reaction time (RT) data would provide a more complete understanding of alcohol effects and potential compensation effects across variations in task complexity. Further limitations to the study are outlined below.

Two participants were excluded from the behavioural analysis for having Go trial scores substantially lower than the mean, though these participants were not excluded from the EEG analysis. This seems an odd decision so clarification on it would be helpful. A potentially more significant omission is the exclusion of the medium task difficulty data (trials at 90^{o} rotation) for which, in our view, no convincing reason is given. Responses to these trials should be incorporated into the main analysis, ideally using the SDT approach described above.

Localisation of EEG signatures indicated some unexpected sources. The neural substrates for response inhibition and anticipatory gating are thought to be mostly in the prefrontal cortices, however, key differences between the alcohol and sober conditions were localised to temporal sites. The authors suggest that, in sequential tasks, aspects of response inhibition processing occur after the inhibitory act, which may therefore relate to bottom-up attentional processing common to occipital sites. However, a more elaborate discussion of this unexpected localisation of activity is warranted.

EEG analyses are conducted on correct responses only, however, behavioural data are based on false-alarm rates, i.e., incorrect responses. In other words, the behavioural focus of alcohol's effect is on inhibition failures, while the neural focus is on inhibition success. It is not clear why this is the case. Were correct response trials emphasised to help identify the subsequent preparatory responses anticipated (e.g., attentional gating, etc.)?

Alpha and theta band differences were found within a 500-1500ms time window, a latency corresponding to aspects of cognitive rather than motor processing, the latter being captured earlier (within 600ms of stimulus onset). The authors suggest that effects in later time windows may represent preparatory processes (in advance of subsequent trials), such as proactive control. This is an interesting idea that might have been addressed more specifically by analysing the time window time-locked to before the stimulus onset. For comparison, it may also be more reliable to run such an analysis on incorrect vs. correct trials.

Finally, it would be of interest to mention whether there was an alcohol effect overall on the EEG signal, and if so what the characteristics of this were.

References

Bayless, S. J. & Harvey, A. J. (2017). Testing alcohol myopia theory: Examining the simultaneous effects of alcohol intoxication on central and peripheral attention. *Perception*, *46*, 90-99. doi:10.1177/0301006616672221

Harvey, A. J., Bayless, S. J. & Hyams, G. (2018). Alcohol increases inattentional blindness when cognitive resources are not consumed by ongoing task demands. *Psychopharmacology*, *253*, 309-315. https://doi.org/10.1007/s00213-017-4772-9

Harvey, A. J. & Seedhouse, M. (2021). The influence of alcohol and cognitive capacity on visual number judgements. *Perception*, 50, 39-51. doi: 10.1177/0301006620984105

Steele, C. M., & Josephs, R. A. (1990). Alcohol myopia: Its prized and dangerous effects.*American Psychologist*, *45*, 921–933. https://doi.org/10.1037/0003-066X.45.8.921

Stock, A. K., Schulz, T., Lenhardt, M., Blaszkewicz, M., & Beste, C. (2016). High-dose alcohol intoxication differentially modulates cognitive subprocesses involved in response inhibition. *Addiction Biology*, *21*, 136-145.

Stock, A. K., Riegler, L., Chmielewski, W. X., & Beste, C. (2016). Paradox effects of binge drinking on response inhibition processes depending on mental workload. *Archives of Toxicology*, *90*, 1429-1436.

Young, M. E., Sutherland, S. C., & McCoy, A. W. (2018). Optimal go/no-go ratios to maximize false alarms. Behavior Research Methods, *50*, 1020-1029. https://doi.org/10.3758/s13428-017-0923-5