

Introduction

- Cue-exposure therapy (CET) is a treatment for disorders where Pavlovian conditioning is central to the aetiology. CET weakens association between the unconditioned and conditioned stimulus (CS) by putting the CS through extinction. CET is very successful for treating anxiety disorders (Norton and Price, 2007), but thus far, has not been demonstrated to be as successful for treating AD (Conklin and Tiffany, 2002). We refer to this as the Alcohol Cue-Exposure Therapy Paradox (ACETP).
- One possible reason for the ACETP is Resistance-to-Extinction (REC). REC is when CSs extinguish at a slower rate when put through extinction. Rodents who were exposed to binge drinking in adolescence have been shown to have increased REC (Gass et al, 2014). The effects of alcohol and addiction on REC in humans has not been investigated.
- Therefore, the aim of the present studies is to investigate extinction rates of neutral cues in people with different alcohol drinking histories. Study 1 looked at differences between light and heavy social drinkers and study 2 looked at difference between alcohol-dependent and non-dependent. It was hypothesised that heavy drinkers/ dependent will have weaker extinction than light drinkers.

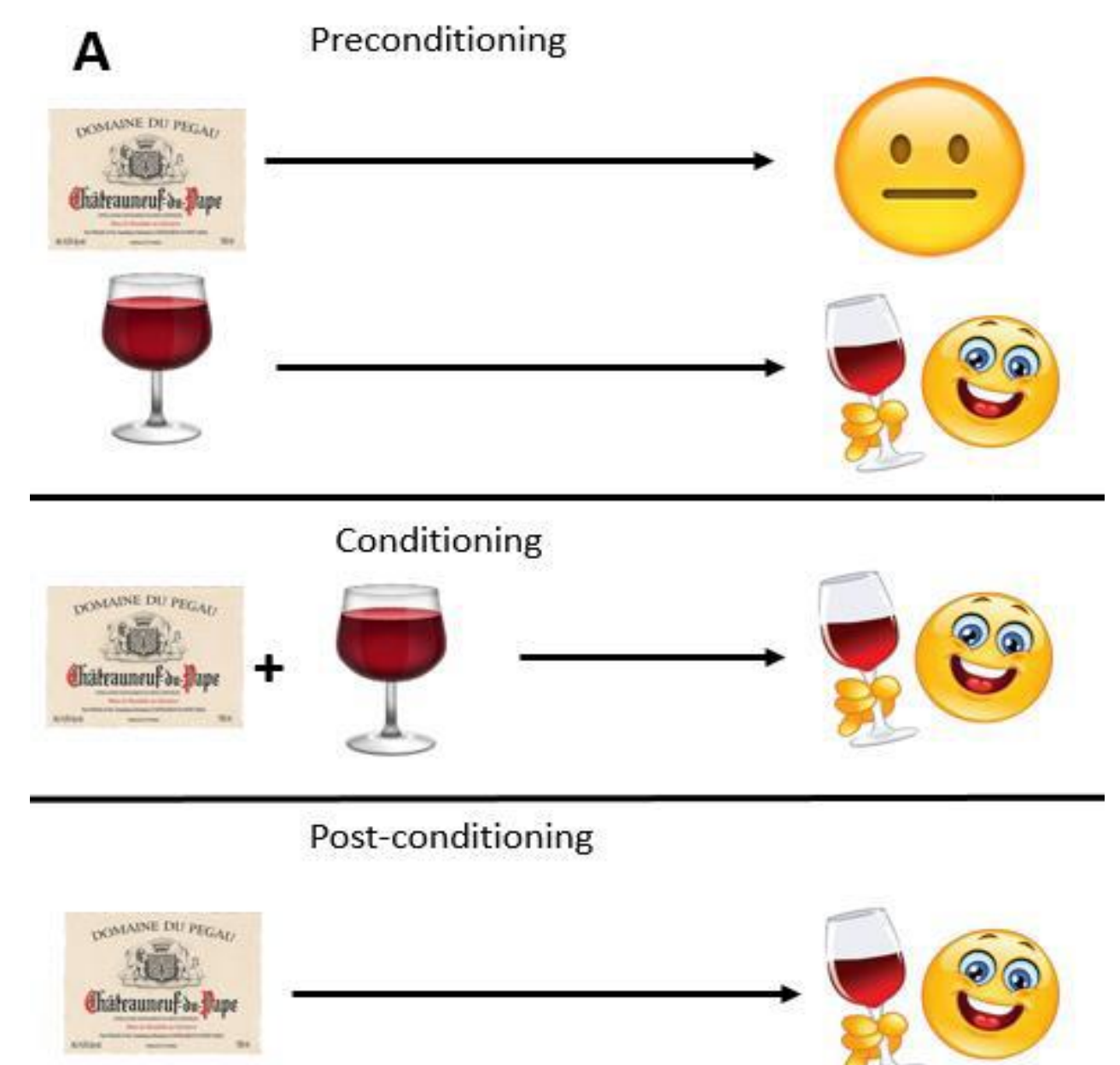


Figure 1: A schematic of Pavlovian conditioning for AD.

Study 1: Methods

Participants

74 Participants (54 female) with a mean age of 20.4 years were recruited from University of Southampton.

Computer Task

The computer task is designed to help study learning. 4 cues fall from the top of the screen to the bottom where they can trigger a sensor. Design is summarised in table 1.

Table 1: Summary of Experimental Design

Stage 1	Stage 2	Stage 2a	Stage 3
A: A → X (x10)	B: A → Z (x8)		C: A → Z (x2)
A: B → Y (x20)	B: B → Y (x8)		
A: C → Z (x20)	B: C → Z (x8)		
A: G → X (x10)		B: G → Z (x2)	

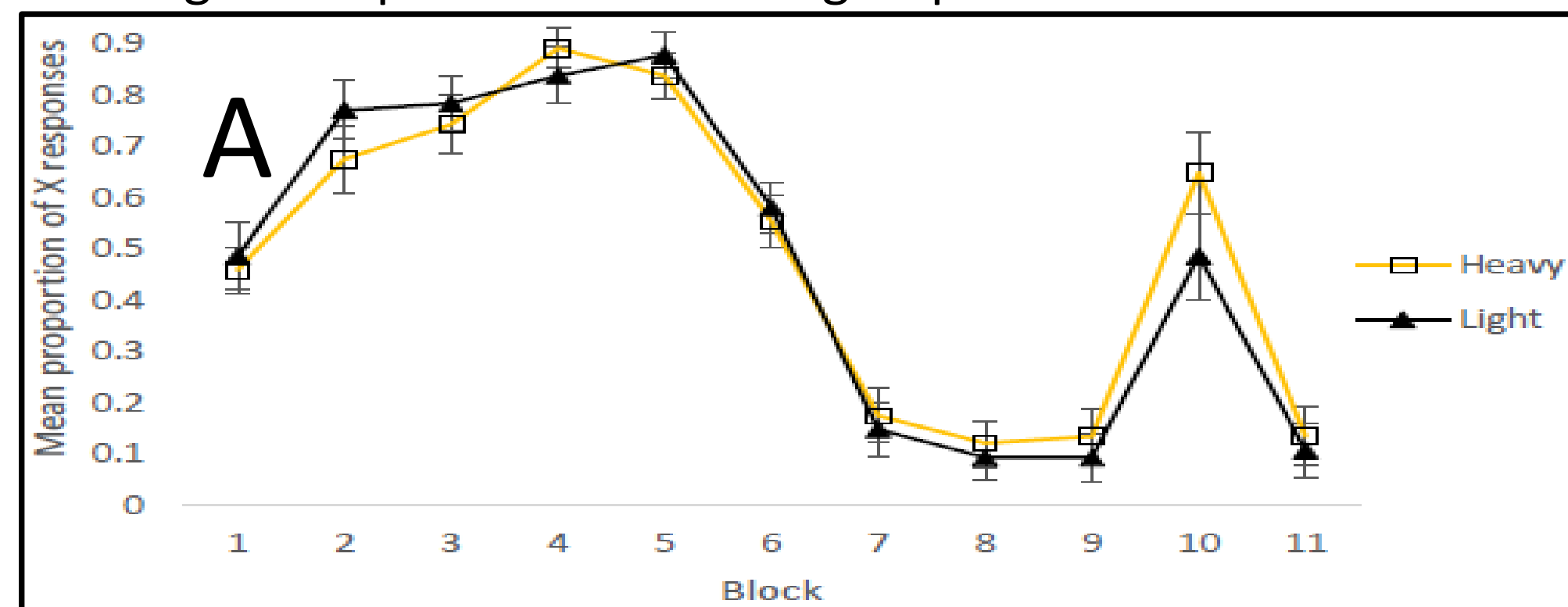
Note. A: A → X (x10) indicates context A, cue A, outcome X present on 10 trials. Outcomes X and Y randomised as X=red and Y=green or vice versa and outcome Z indicates no outcome. Trial orders are randomised within blocks.



Figure 2: Display of computer task

Study 1: Results and Conclusion

There was a significant main effect of block for acquisition ($F(3.13, 225.5) = 26.5, p < .001$), extinction ($F(2.65, 190.7) = 120.8, p < .001$) and the recovery test ($F(1, 72) = 51.2, p < .001$), but not a significant main effect of group or interaction of block and group indicating successful learning was equal between each group.



There was a significant main effect of block for acquisition ($F(3.26, 234.5) = 21.7, p < .001$) and the summation test ($F(1, 72) = 78.2, p < .001$), but not a significant main effect of group or interaction of block and group indicating successful learning was equal between each group and context B acquired inhibitory strength.

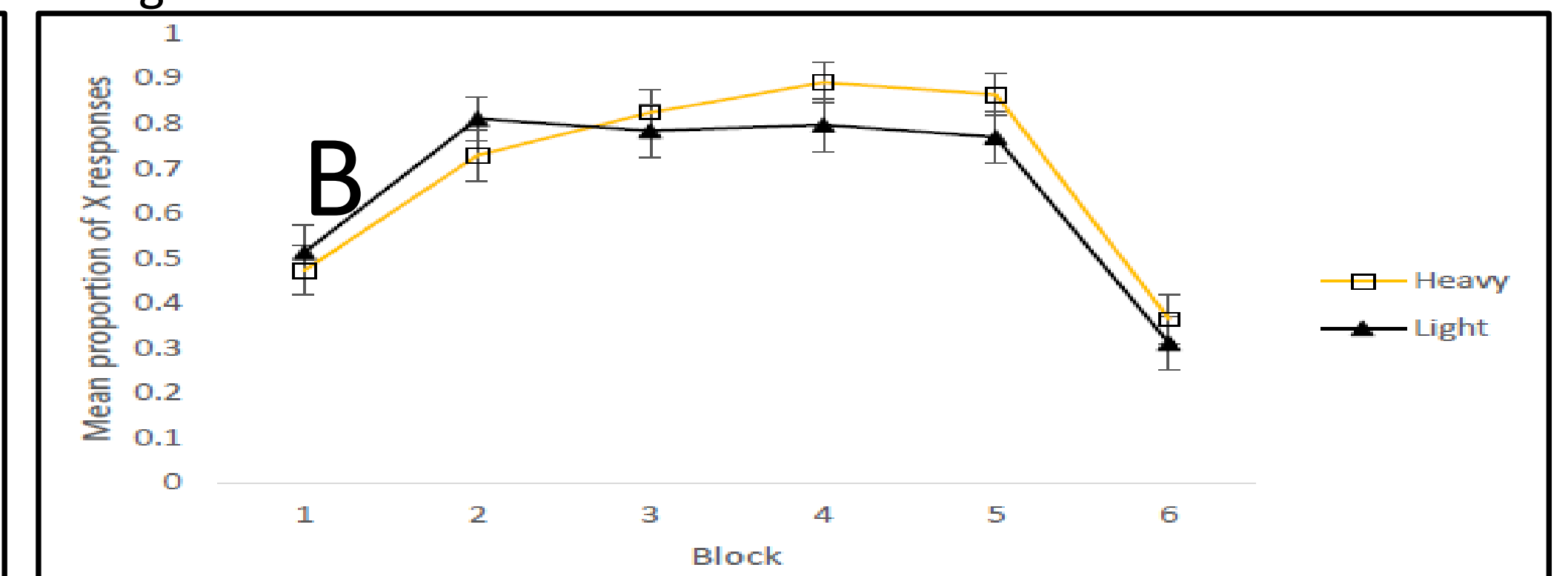


Figure 3: Line graphs displaying the mean appropriate X responses. Panel A is for cue A and panel B is for cue G. There were no statistically reliable differences between light and heavy drinkers in either extinction, response recovery, or inhibition. Further research will compare these learning processes in alcohol dependent patients with a non-clinical controls.

Study 2

Study 2 is a replication of study 1, with alcohol dependent participants and age and sex matches controls. The dependent group is selected if they are <1 year abstinent and the control group is selected if they score ≤ 5 on AUDIT-C. The data collection for Study 2 is currently still in progress. We aim to get 51 for the experimental group & 51 for the control group. To date 29 of the experimental group and 5 controls have been collected. Therefore, figure 4 displays a line graph of the means of cue A for the 29 from the experimental group compared to 29 randomly selected participants from study 1

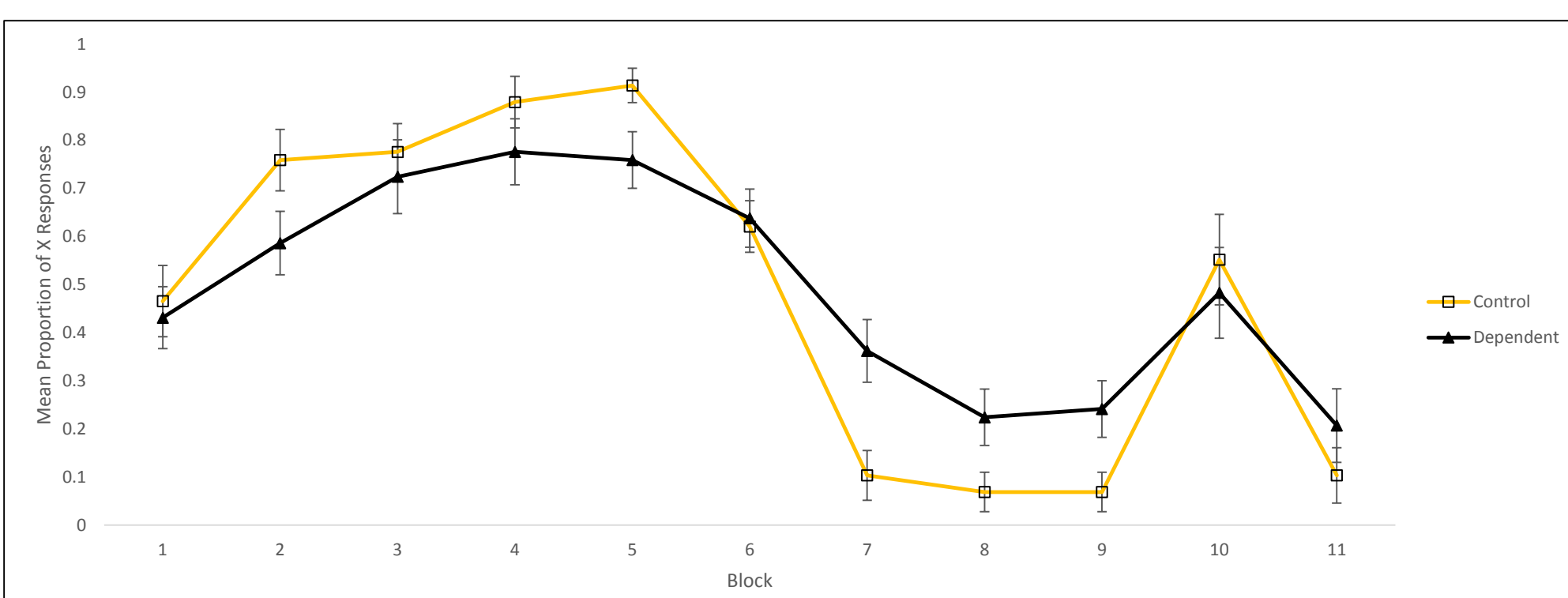


Figure 4: Line graphs displaying the mean appropriate X responses for cue A

These preliminary results indicate individuals with alcohol dependence who are recently abstinent could be worse at learning to extinguish conditioned responses of generic cues.

References:

- Conklin and Tiffany (2002). *Addiction*, 97 (2), 155-167.
- Gass et al. (2014). *Neuropsychopharmacology*, 39 (11), 2570-2583.
- Norton and Price (2007). *The Journal of Nervous and Mental Disease*, 95 (6), 521-531.

The authors have no conflict of interest to declare.