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Neuro-protective Effect of Alcohol in Dealings With Idiocy

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In social outings involving idiots, alcohol was found to protect neurons against apoptosis in smarter individuals. The delay in sensory relay caused by alcohol consumption is the likely mechanism for this phenomenon. Whilst neurons were still lost in the experimental group, the rate of loss was significantly lower than in controls or placebo alcohol free beer intervention groups. Exposed control groups lost 4 arbitrary neuronal density more than their respective alcohol test cohort. Results were significant to >0.0001. The authors stipulate that beer may save braincells under severe intellectual-vacuum stress.

Introduction

It is well recognised that in social dealings inane conversation is a fact of life. Whilst individuals often indulge in a variety of conversations during the course of socialising, entirely vacuous conversations will arise. Topics including what the latest non-celebrity has scandalously done and other trivialities are the most frequently occurring examples.

As the conversation enters the realm of irrelevance, a feeling of brain-death is often reported amongst some participants. Those with higher intellectual requirements are more likely to report the sensation of cerebral liquefaction. Studies have not determined the cause of this phenomenon, nor strategies to overcome this social disease.

Therapeutic coma in the treatment of rabies is a controversial field. Though no trials have been conducted, the methods of inducing the coma have not been universal. Clinical anecdotes of untreated patient survival in two such cases points to a potential CNS protection via shutting down periphery nerve impulse relay. No neural transmission may act to cease the movement of the virus to the cerebellum.

Similarly to rabies, chronic stupidity exposure causes neuronal plaques. Relayed into the brain via the sensory cranial nerves (namely number VIII the vestibulocochlear nerve), blocking nerves transmission is the key to preventing stupidity induced brain damage. Advances in the therapeutic coma offer a prototype prophylactic method to save brains.

Methods

Four-hundred and fifty people were selected for the study. The participants were stratified by a modified intelligence test. Three intelligence categories (High, Intermediate and Low) were created. Cutoffs were 100+ for High, 80-100 medium, <80 for low. One third of participants belonged to each category.

Participants in each category were further divided into three intervention groups, alcohol, alcohol free and control. Intervention was provided for one month after which time neuronal density was measured. An arbitrary

neuronal density measure was used for enumeration purposes.

Upon completing measurement of baseline neuronal density, participants were exposed to three hours of conversation topics associated with neuronal death. These conversations covered the Kardashians, Jersey Shore, Lady Gaga's newest costume monstrosity and popular culture.

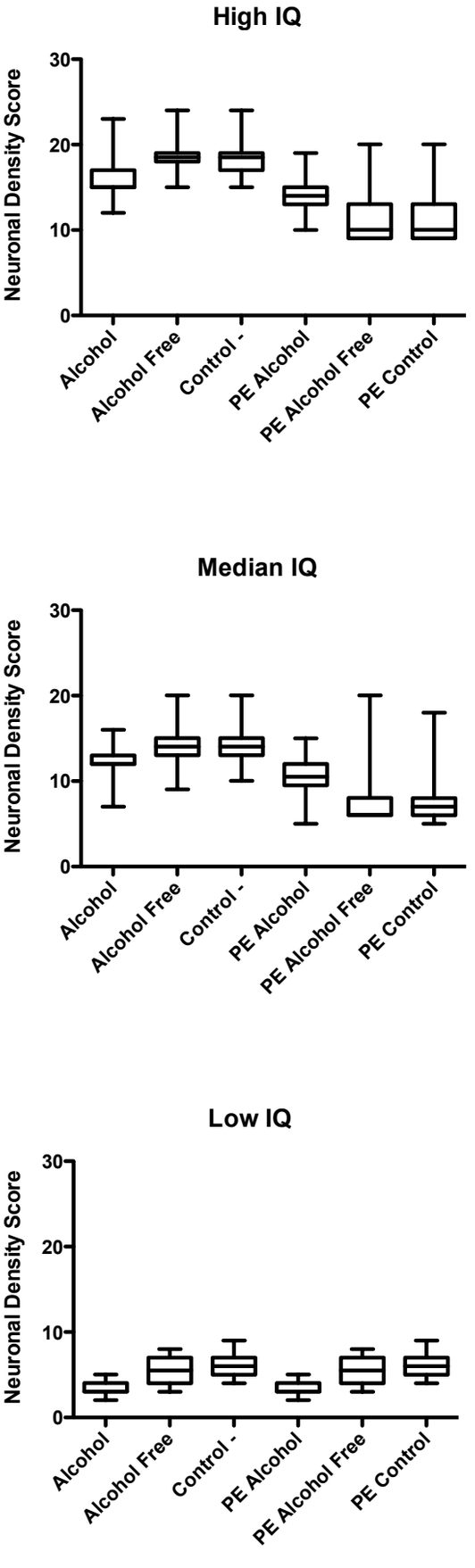
During the exposure, social interaction between participants was monitored and analysed.

Population statistics for each sub-groups' intervention cohorts were calculated and Mann-Whitney tests conducted. Results were deemed significant if $p \leq 0.05$.

Results

High IQ Population

Population statistics for the pre-exposure high IQ group showed a significant difference in neuronal density existed between the alcohol treated and control cohorts (Alcohol: 16.10 ± 2.06 Control: 18.54 ± 1.88, $p < 0.001$). There was no significant difference in neuronal density between alcohol free cohort (18.62 ± 1.69 $p = 0.84$) and the control cohort.



Right: Box plot of neuronal density scores of population treatment cohorts before and after exposure. Pre-exposure groups receiving alcohol (Alcohol), alcohol free beer placebo (Alcohol Free) and untreated control (Control) are denoted by their respective group descriptor. Post exposure groups receiving alcohol (PE Alcohol), alcohol free placebo (PE Alcohol Free) and control (PE Control) are denoted by their respective group descriptor. Top: High IQ cohort. Middle: Median IQ cohort. Bottom: Low IQ cohort.

Post-exposure, the alcohol exposed cohort lost fewer neurons than the control and alcohol free cohorts. Final density scores for Alcohol, alcohol free and control were 14.14 ± 1.84, 11.40 ± 2.63 and 11.36 ± 2.694 respectively. No significant difference was observed between the alcohol free and control post-exposure cohorts, however, alcohol was significantly neuroprotective $p < 0.0001$ in the high IQ population.

Median IQ Population

Similarly to the high-IQ population, alcohol initially reduced neuronal density compared to the negative control (11.88 ± 1.88, 14.20 ± 2.02 $p < 0.0001$). No such difference was observed in the alcohol free cohort (14.08 ± 2.27, $p = 0.97$).

Post exposure, alcohol was observed to display statistically significant neuro-protective effects when compared to the negative control (10.28 ± 2.18, 7.18 ± 2.26, $p < 0.0001$). Again, there was no significance between the negative control and the alcohol free intervention (7.06 ± 2.12 $p = 0.79$).

Low IQ

Alcohol significantly reduced neuronal density compared to the negative control (3.32 ± 0.65, 6.10 ± 1.13 $p < 0.0001$) and alcohol free intervention (5.42 ± 1.50 $p < 0.0001$). The alcohol free intervention had a mildly statistically significantly lower neuronal density than the control cohort ($p = 0.042$).

Post-exposure the same results were found as in the pre-exposure group, almost like the author sloppily copied and pasted the data.

Discussion

Alcohol was observed to act as a neuro-protective agent when high and median IQ populations were exposed to stupid conversation. It appears the standard reaction of reaching for a drink in these tedious and mind-numbing conversations is indeed a protective act, saving the brain from damage.

Stupidity whilst arbitrary, can usually be defined as talking about something of no consequence in conversation. These superficial topics especially those the groups were exposed to damaged the brains of those who were not chronically exposed. Chronic exposure granted a strong degree of resistance to the brain damaging effects, but it did come with high neurological penalties.

The mechanism behind alcohol's neuro-protective function is believed to be similar to the therapeutic coma in rabies. This shutting down of the vestibularcochlear nerve may result in a male selective-hearing like state. Male selective-hearing is a known way to maintain sanity and plausible deniability when attempting to shirk tasks, and is therefore a suitable model for further investigation.

Of note, one subject in the median IQ alcohol free post-exposure cohort was able to increase neuronal density. Observation of the individual's social interactions during the exposure point to trolling as a possible explanation. Attempting to intellectually confuse the exposure participant potentially allowed for stimulation and neuronal growth.

The lower IQ group with the exception of the control cohort, were accidentally unblinded to the intervention they received. It is believed that this explains the significantly lower IQ observed in the alcohol free cohort, as only an idiot would choose such a beverage. Ironically, this mistake payed dividends for those who committed it.