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## Alcohol retards visual recovery from glare by hampering target acquisition

ADAMS and Brown<sup>1</sup> showed that relatively small doses of alcohol produced "large, significant, dose-related increases in the time required to recover foveal contrast sensitivity following bright light exposure". As this effect of alcohol may be directly related to industrial and to car driving safety<sup>2</sup>, we set out to ascertain its precise origin. Adams and Brown concluded tentatively that the alcohol exerted its effect at the retinal level. The experiments presented here, however, implicate non-retinal mechanisms, as alcohol delays recovery from glare only when observers have difficulty localising or fixating the test stimulus.

The first part of our study was a replication of the Adams and Brown experiment. Our subjects were four male college students with normal (6/6) or corrected-to-normal visual acuity. On separate days subjects ingested either alcohol (1.0 ml per kg body weight) or placebo. The two treatments were given in random order. The alcohol dose was 95% ethanol diluted 2:1 with fruit drink so that the total volume (ml) was three times the subject's weight (kg). The placebo was an equivalent volume of fruit drink alone. The drink was consumed in 20 min from a paper cup containing two ice cubes. In addition, 2 drops each of ethanol and eucalyptus oil were placed on the lid of the cup to minimise olfactory cues to the presence of alcohol<sup>1</sup>.

Details of stimulus presentation are given in Fig. 1. Subjects practiced the detection task to achieve stable performance at the beginning of each experimental session; they then were tested before drinking, and 30, 75, 150, 240, and 330 min after drinking. At each of these times, three sets of measurements were taken with 5 min allowed between sets to allow complete recovery from the adapting luminance level. The session always started in the early afternoon, and subjects were instructed to eat lunch beforehand.

As expected, alcohol produced large, significant increases in the time required to recover foveal contrast sensitivity. In particular, as Fig. 1 shows, the difference between recovery times with and without alcohol is maximum at the sampling time 75 min after ingestion and declines thereafter, and is most pronounced at low target contrasts. Analysis of variance (ANOVA) indicates that the depicted interaction between dose, time, and contrast level is statistically significant ( $P < 0.001$ , d.f. = 20, 60).

A simple but trivial explanation of the results of Adams and Brown and of our replication would be that following alcohol ingestion observers become more cautious about reporting the visibility of the test target, thereby spuriously increasing recovery times<sup>3,4</sup>. We decided therefore to use a methodology<sup>5</sup> which could discriminate alcohol-induced changes in sensitivity from changes in criterion (that is, willingness to report).

Using the same apparatus as before but without a glare source, we tested seven male college students. On half of the 2-s trials the small flickering test spot actually was presented; on the remaining trials no spot was presented. The subject's task was to respond either 'yes' (a test spot had been presented) or 'no'. With each subject, 500 trials were run at various spot

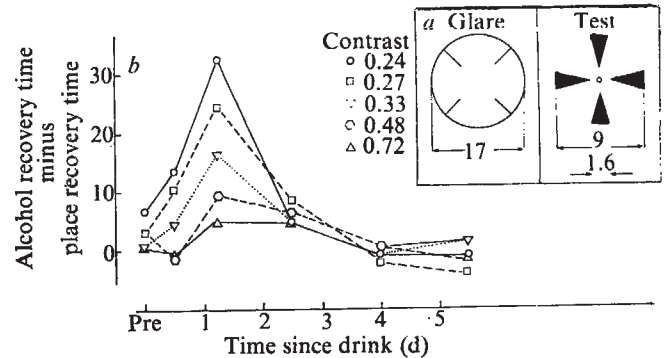


Fig. 1 a, The stimulus configuration of the  $8.6 \times 10^4$  cd m<sup>-2</sup> glare field on the left and the test field on the right. The 10 min arc test spot was located at the centre of the test field reference markers on a background of luminance 24.8 cd m<sup>-2</sup>. In the actual experiment, the glare field was located directly above the test field, which was at eye level. At the beginning of each measurement session, the subject fixated with his left eye the centre of the circular adapting (glare) field. The right eye was covered with a patch, and fixation was aided by four thin diagonally-orientated reference lines. Immediately after a 60-s exposure to the high intensity glare source, and with the eye-patch still in place, the subject looked directly below (straight ahead) and fixated the centre of the test field on which a test spot was presented intermittently (125-ms flashes at 4 Hz). The contrast of the test spot was under the experimenter's control. When the subject indicated orally that he saw the spot at the highest contrast level, the experimenter rotated a filter wheel one notch, reducing the target contrast a further step below the subject's threshold. When the subject's contrast sensitivity had recovered enough to restore the test spot to detectability, the spot contrast was reduced again. The time taken to recover to each of five predetermined contrast levels (0.72, 0.48, 0.33, 0.27, and 0.24) was obtained. These levels were chosen to give approximately equal intervals between each recovery point. b, Interaction of alcohol, contrast, and time for the first experiment. Each point is the mean difference (s) between recovery time with alcohol and recovery time with the placebo. Data from four subjects.

contrasts. Subjects were tested either following ingestion of alcohol (dose identical to that used before) or following ingestion of the placebo. Intermittent tests were carried out 30-140 min after ingestion, when glare recovery was most seriously retarded in our original experiment. The conditional probabilities of 'yes' responses in the presence of a test spot and 'yes' responses in the absence of a test spot were used to estimate<sup>6</sup> the observer's response criterion ( $\beta$ ). Analysis of variance indicated no significant alcohol-induced changes in  $\beta$  ( $P > 0.50$ ).

With response bias eliminated as an explanation, we sought to identify the source of the alcohol-induced delay in glare recovery times more precisely. Four new subjects participated in an experiment identical in every respect to our first, except that three sets of eye movement measurements (before, during, and after exposure to the glare) were taken every 15 min for 135 min following the 20-min drinking period. Standard electrooculographic<sup>6</sup> (EOG) methods produced records of eye position as a function of time. The resolution of our recordings was approximately one degree of visual angle.

With or without alcohol, very little eye movement occurred either before or during exposure to the glare source. But immediately after the 60 s of high intensity glare, there were many eye movements as if subjects 'hunted' for the flashing spot within the test field; after alcohol ingestion the eye movements were larger and more frequent. These eye movement patterns suggested that alcohol might combine with glare to prolong recovery time by impeding the subject's attempts to localise and fixate the target. Localisation would be hampered if alcohol ingestion were to disturb the subject's memory of precisely where the target would appear. Steady fixation would be hampered if alcohol were to impair control of the extraocular muscles<sup>7</sup>. Irregular visual fixation could produce enough

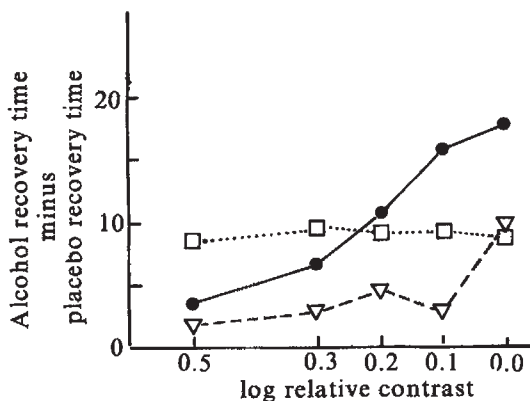
uncertainty about the target's spatial location to reduce its detectability<sup>8</sup>.

Three of the four subjects who participated in our original study were available for two additional experiments. In the first, the test spot locus was circumscribed by a small, dark circle, 12.5-arc min diameter. The circle was of high contrast, and clearly visible immediately after glare exposure even though the flashing test spot within was not. As a result, the subject would not have to remember where the target might appear thereby aiding target localisation. Details of the experimental design and procedure were as in the original experiment, except that the test spot luminance was three times that of the spot in the original experiment. In a second experiment, the test spot was enlarged (100-arc min diameter) to fill the entire area between the arrow reference marks (Fig. 1). Consequently, no matter where between the arrows the subject looked, his fixation would fall on some part of the flashing test spot. Enlarging the area of the test spot reduced its maximum luminance; to compensate, we reduced the background luminance to 0.89 cd m<sup>-2</sup>. In both these experiments, as before, glare recovery was measured with spots whose contrasts covered a range of 0.5 log units; a single set of five measurements was made before, and 30, 90, 150 and 210 min after drink ingestion.

As shown by Fig. 2, the effect of alcohol on recovery from glare was greatly reduced when either the small test spot was circumscribed by a clearly defined circle or the test spot was enlarged. With the enlarged test spot, no effects involving alcohol were statistically significant (ANOVA, all  $P > 0.5$ ). This is what would be expected if the alcohol-induced retardation of glare recovery in our original experiment had been partly caused by an unsteadiness of fixation which the enlarged test spot rendered unimportant. The contrast independent elevation shown in Fig. 2 is not statistically significant ( $P > 0.80$ ) and is due to greatly increased recovery times for just one subject. With the small, circumscribed test spot, one effect involving alcohol (the interaction of alcohol and contrast) was significant ( $P < 0.03$ , d.f. = 4, 8), due mainly to the increased recovery time at the lowest contrast level. This reduced effect of alcohol would be expected if in our original experiment alcohol had made subjects unsure about the location of the target and if the easily seen, high-contrast circle in the present experiment had made them less unsure. The small residual effect of alcohol may reflect difficulty in maintaining precise fixation following alcohol consumption, a problem that would be less critical with the enlarged spot.

Our experiments confirm that ingestion of alcohol causes a

**Fig. 2** Mean difference (s) between recovery time with alcohol and recovery time with the placebo. Data are shown for all five contrast levels and for all three glare experiments. The data have been normalised in both this figure and in all analyses of variance to produce 47.52-s average recovery time for each subject (the average for the original experiment). Only the data from those subjects who participated in all three experiments are shown. Data shown were collected at those times following alcohol ingestion which should have produced the largest effect of alcohol: 75 min for the original experiment (●), 90 min for the large spot (□), and 90 min for the circumscribed spot (△).



loss of visual sensitivity following glare. But non-retinal processes are primarily responsible for the effect of alcohol on glare recovery. We emphasise that the non-retinal original of this alcohol-induced visual disability does not lessen its potential for adversely affecting driving and related visual tasks. On the contrary, uncertainty about the location<sup>8</sup> of possible targets imposes an inescapable reduction of visual sensitivity of any driver; if alcohol either retards a driver's target acquisition<sup>9</sup> or causes him to mislocalise the target<sup>10</sup>, visibility necessarily will be reduced and his ability to respond to the target impaired. Moreover, we cannot rule out the possibility that in driving, the effect of alcohol that we studied can sum with other, previously identified perceptual consequences of alcohol ingestion<sup>11</sup>.

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## Visual discrimination between small objects and large textured backgrounds

THE remarkable ability of certain higher order insect visual neurones to discriminate between movement of a small object or target and a large textured background has been clearly demonstrated by Palka<sup>1,2</sup> and O'Shea and Fraser-Rowell<sup>3</sup>. In addition, movement of a large textured background such as a windmill pattern is inhibitory to detection of motion of a small target by this neurone, known as the descending contralateral movement detector (DCMD). The response of this identifiable neurone to motion of small objects is inhibited by rotation of the windmill pattern over a specific range of high spatial frequencies of the pattern, but is augmented by low spatial frequencies in the pattern. For this neurone we have quantitatively determined the spatial frequency at which the effect of the windmill pattern becomes inhibitory.

The DCMD is a visual interneurone in the ventral nerve cord which is excitatory to motor neurones involved in jumping. It originates in the protocerebrum where it receives excitatory input through an electrical synapse from the LGMD (lobular giant movement detector) which has a dendritic field in the shape of a fan across the entire lobula. The LGMD receives at least two kinds of inhibitory inputs: lateral inhibition on neurones peripheral to the fan of dendrites, and inhibitory input proximal to the convergence of the fan of dendrites (post-convergence inhibition)<sup>3,4</sup>. The post-convergence inhibition samples activity across the entire visual field but is effective only for higher velocities<sup>3,4</sup> of patterns than used in these experiments.

Responses of the DCMD in *Schistocerca gregaria* were obtained by conventional methods<sup>3</sup>. The receptive field of the DCMD is approximately a hemisphere, and motion detection