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Alcohol intoxication reduces visual sustained attention

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Abstract. Effects of alcohol intoxication on visual sustained attention were studied using a vigilance task entailing detection of degraded target stimuli. Data were obtained in separate sessions under four ethanol doses, ranging from 0 (placebo) to 1.05 g/kg lean body weight, with periodic maintenance dosing of 0.12 g/kg. Intoxication lowered the overall level of detection performance, and in addition produced dose-related increases in the rate of performance decrement over time. Analysis of performance data using techniques derived from Signal Detection Theory indicated that the decrements were due specifically to alterations in perceptual sensitivity. Examination of eye movements and blinks indicated that the effects of ethanol were not mediated peripherally. Rather, alcohol appears to have deleterious effects on central processing capacity and the availability of capacity over time. The alcohol-related failure of sustained attention may contribute to increased accident risk in tasks requiring continuous performance.

Key words: Alcohol – Ethanol – Vigilance – Sustained attention

It is well established that alcohol intoxication yields profound impairment in tasks, such as driving or piloting, that require continuous performance. Analyses by safety experts (see Linnoila et al. 1986) and reports from intoxicated subjects themselves (e.g., Fabian et al. 1983) ascribe much of the impairment to reduced attention. Despite the prevalence of this assumption, however, the supporting laboratory evidence is not extensive. The strongest evidence that alcohol affects attention comes from tasks that require "divided" attention, i.e., the allocation of attention amongst two or more simultaneous sources of information (Moskowitz and Depry 1968; Hamilton and Copeman 1970). Although recent reports generally agree with these early studies, they also emphasize that the susceptibility of such tasks to alcohol is likely to depend on idiosyncratic aspects of the stimuli, cognitive strategy and performance requirements (Landauer and Howat 1983; Mills and Bisgrove 1983; Moskowitz et al. 1985; Wilson et al. 1985; Miles et al. 1986; Lamb and Robertson 1987; Ward and Lewis 1987).

Data bearing on alcohol's effects upon the ability to sustain attention in continuous tasks (i.e., "vigilance") are notably inconsistent. Two principal aspects of sustained attention need be distinguished (Parasuraman 1979, 1984). One aspect is the overall *level* of performance in continuous monitoring tasks. Several studies have found no effect of ethanol on performance level in such tasks (Colquhoun 1962a; Moskowitz and DePry 1968; Pearson and Neal 1970; Talland 1966; Vogel-Sprott 1976), leading to the frequently-encountered generalization that ethanol's effects on simple tasks are minimal (e.g., Ritchie 1980). Other studies, however, have found evidence for impairment at moderate and high doses (Erwin et al. 1978; Tong et al. 1980; Jansen et al. 1985; Sahgal et al. 1986) and one study (Sahgal et al. 1986) has yielded a suggestion that low doses may slightly improve rather than impair vigilance.

The second principal aspect of sustained attention is the *decrement* in performance over time, which is the hallmark of attention failure in such tasks. Data from studies of alcohol's effects on the rate of decrement are, again, inconsistent, and include findings of no effect (Tong et al. 1980) or an actual improvement in performance with alcohol (Docter et al. 1966). Colquhoun (1962a, 1976) has reviewed evidence suggesting that alcohol intoxication exacerbates the vigilance decrement, although the effects described are quite small. In a study reported by Erwin et al. (1978), alcohol was found to produce large and dose-related effects on the level of vigilance performance in a visual monitoring task, but no significant effect on rate of decrement. A re-examination of these data, however (Rohrbaugh et al. 1987a), discloses some evidence that alcohol acted to hasten the decrement early during the 30 min vigilance period. More recently, Gustafson (1986a, b) has reported that simple reaction time to repetitive visual and auditory stimuli is slowed over time by alcohol.

A variety of factors may underlie the inconsistencies among these results. The various studies differ widely with respect to such critical features as dose, timing and duration of vigilance period, and stimulus, task and response variables, and they are amenable to correspondingly diverse interpretation. It is also important to note that the separate entities of vigilance level and decrement are conceptually distinct, and that they may be differentially affected by various task and subject characteristics (Parasuraman 1979, 1984; Nuechterlein and Dawson 1984).

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Here we report findings which demonstrate that alcohol intoxication can have appreciable effects on both the level of performance and the rate of performance decrement. These findings were obtained in a vigilance task described by Nuechterlein et al. (1983), whose usefulness in the study of sustained attention is supported by a variety of theoretical and empirical considerations (see also Nuechterlein and Dawson 1984; Parasuraman 1984, 1985). The task has the additional advantage of yielding a performance decrement over comparatively brief periods (about 8 min in the present experiment), so that performance changes could be studied without the confounding effects of large simultaneous changes in blood alcohol levels, or the appearance of alcohol-induced sleep episodes (Rundell et al. 1972; see Erwin et al. 1978). Performance data were analyzed using measures derived from Signal Detection Theory (Green and Swets 1966), so as to allow aspects related separately to sensitivity and response biases to be distinguished. We also have attempted to distinguish the possible role of peripheral contributions (in the form of eye blinking and movements) to performance changes. Additionally, the effects of alcohol were studied after four separate doses (including placebo) so as to disclose any threshold or non-monotonicity of effect.

Methods

Subjects. Subjects were 12 male "social drinkers" (average age 23.2 years, range 21–32 years) who, on examination, were free of significant psychiatric or physical abnormality, and had no history of drug or ethanol abuse. Subjects denied drug use throughout the course of the experiment. This was confirmed by analysis of urine samples which yielded no detectable evidence of cocaine, marijuana, opiates, benzodiazepines or other common psychoactive drugs. All subjects gave informed consent after the nature of the procedures and attendant risks were described. The initial screening and examination sessions also included a test of ability to maintain ocular fixation and to withhold blinks during the vigilance runs, on the basis of which four subjects were excluded from participation in the study.

Vigilance task. The sustained attention task described by Nuechterlein et al. (1983) requires subjects to attend to visually-presented digits that are degraded by blurring the images. Performance is monitored by examining accuracy of responses to occasional target digits. Under such conditions, performance shows a rapid decrement over periods as short as 5–10 min. The brevity of the period over which performance declines, in addition to the finding that the pattern of decrement is reproducible over repeated administrations of the task (Nuechterlein et al. 1983), make the task well suited for the study of drug action.

The digit stimuli were white on black, projected at an intensity of 0.7 log units above threshold on a screen 190 cm in front of the seated subject. At this distance, the digits subtended 1.9° vertical by 1.4° horizontal visual angle. The images were degraded by interposing diffusing sheets of irregular plastic film in front of the projector lens. The amount of degradation was established empirically in pilot sessions so as to yield an initial performance level (in sober subjects) of about 85%. Digits were presented singly for a duration of 40 ms, at a rate of 1 digit per s. On a quasi-random 25% of the trials the target digit "0" was presented, to which the subject was instructed to respond with a button press. The non-target digits "1" through "9" were presented equally often in irregular order on remaining trials. A single run of trials comprised 486 digits (8.1 min), 120 of which were the target digit "0". The runs were not preceded within the session by any practice trials. Subjects were instructed to maintain their gaze throughout the runs on a centrally-located fixation cross, and to refrain from eye movements or blinks as much as possible. A sound attenuated chamber and low level masking noise were used to ensure that the stimuli were not accompanied by audible sounds.

Ethanol dosing and procedure. Following a practice session, four experimental sessions, each with a different dose of ethanol, were run at intervals of at least 3 days (usually weekly) under blind conditions and in counterbalanced order. The doses were 0 (placebo), 0.45 (low), 0.80 (medium) and 1.05 (high) g ethanol per kg lean body weight (LBW). Doses were administered as 95% ethanol in a constant volume (500 ml) with orange juice and consumed over a 30-min period. In addition to this initial loading dose, three maintenance doses of 0.12 g/kg LBW in 30–50 ml orange juice were consumed at 30-min intervals after the loading dose. In one subject whose BACs exceeded 100 mg% in the 0.80 g/kg LBW dosing condition, the respective doses were adjusted to 0, 0.35, 0.60 and 0.80 g/kg LBW. The resultant blood ethanol concentrations (BACs), were estimated from Intoximeter Alco-Sensor III breath alcohol analyzer readings taken at 30-min intervals. Subjective measures of intoxication were also obtained at 30-min intervals by asking subjects to mark an analog scale labeled from "not at all" to "very much." Performance data in the sustained attention task were obtained in two runs, beginning 90 and 180 min following the beginning of loading dose consumption. The time preceding the first vigilance run, and between the two runs, was occupied with judgment and movement tasks unrelated to the vigilance task.

Subjects were instructed to consume no ethanol for a period of 24 h preceding the testing sessions, and to eat a light, low-fat breakfast. Sessions were conducted in morning and early afternoon hours. No subject arrived at the laboratory with a detectable BAC.

Measures of eye movements and blinks. An electro-oculographic (EOG) record of eye movements and blinks was obtained from an electrode placed in the center of the forehead (Fpz in the 10–20 electrode placement system), with reference to linked earlobes. The EOG was amplified with a time constant of 10 s and an upper cutoff (−3dB) of 70 Hz, and digitized at a rate of 200 Hz for subsequent analysis.

Analysis of performance data. Rates for hits (i.e., proportion of target trials correctly responded to) and false alarms (i.e., proportion of non-target trials erroneously responded to), were computed on the basis of reaction time (RT) responses within an interval of 1000 ms following stimulus onset. Analyses of RT distributions in previous studies (Nuechterlein et al. 1983), and confirmed in the present study, have indicated that responses in this task are very rarely longer than the 1000 ms period. The hit and false alarm rates were computed separately for blocks of trials encompassing the initial (trials 1–162), middle (trials

163–324) and final thirds (trials 325–486) of each run. Data were analyzed using repeated measures ANOVAs (BMDP2V) applied to the factors of dose, trial block and run. The probability values reported here are adjusted using the Greenhouse-Geiser procedure.

Results

Blood alcohol content

The BACs associated with the four ethanol doses (as estimated by breath analysis) are depicted in Fig. 1. Also indicated are the two periods of vigilance performance measurement. The BACs associated with the four ethanol doses were significantly different [$F(3,33)=163.2$, $P<0.0001$], as were self-reports of subjective intoxication [$F(3,33)=50.47$, $P<0.0001$]. As shown in Fig. 1, the BACs associated with runs 1 and 2 showed several differences. Run 1 was given during the ascending limb of the BAC curve, whereas the BAC was presumably descending during run 2. Within dosage conditions the BAC differences between the beginnings of run 1 and 2 were small in comparison to between dosage differences but significant under the low and medium doses. Subjective intoxication did not differ between runs 1 and 2.

Performance measures

Figure 2 depicts the performance accuracy measures. There were no appreciable or statistically significant effects involving the main effect of run or its interaction with other variables for any measure based on performance accuracy, including hit rate, false alarm rate, or the derived measures A′, d′ or *beta*, as discussed below. In view of the overriding similarities in intoxication measures between runs 1 and 2, and the complete lack of any significant accuracy effect associated with the factor of run, the accuracy data are collapsed across runs in the ensuing presentation.

As shown in Fig. 2a, ethanol yielded dose-related overall decrements in hit rate [$F(3,33)=9.11$, $P<0.0001$], and performance declined across trial blocks under all doses ([$F(2,22)=30.32$, $P<0.0001$]. The deleterious effects of ethanol on sustained attention are apparent in the progressively sharp performance decline with increasing dose, as confirmed in a significant interaction between dose and trial block [$F(6,66)=3.68$, $P<0.01$].

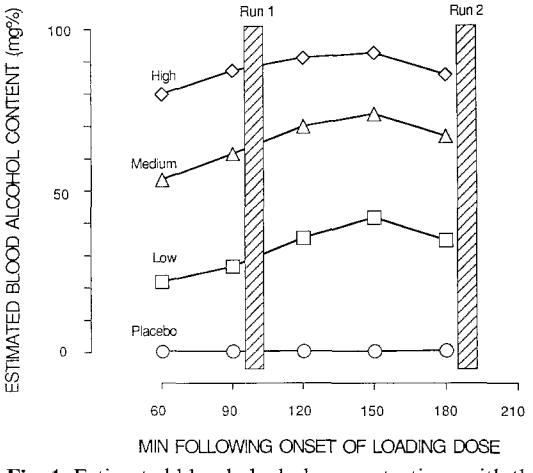

Fig. 1. Estimated blood alcohol concentrations with the four doses. The times of testing are indicated by the *vertical bars*

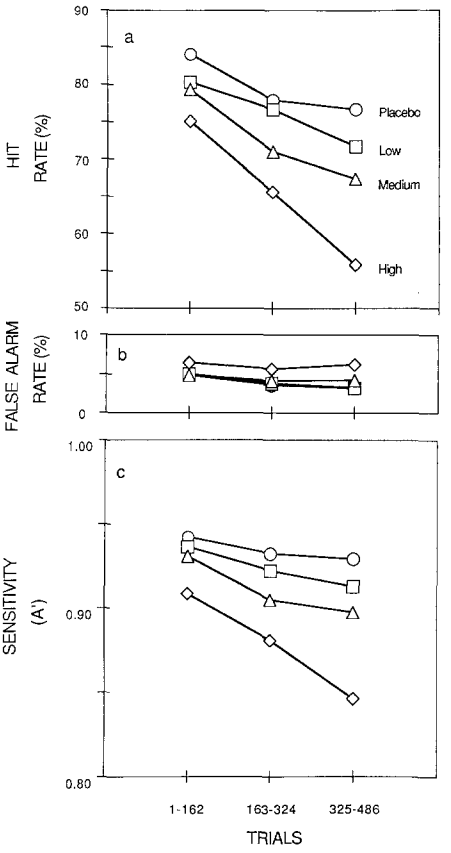

Fig. 2a–c. Performance measures as a function of dose and trial block: hit rate (**a**), false alarm rate (**b**), and the non-parametric index of sensitivity A′ (**c**). Each trial block corresponds to a period of 2.7 min

The accompanying false alarm rates were less affected by dose and trial block than were hit rates (Fig. 2b), although the modest growth in rate with increasing dose was significant [$F(3,33)=4.19$, $P<0.05$].

Criterion-free measures of sensitivity were computed from hit and false alarm rates using procedures from Signal Detection Theory (Green and Swets 1966). As is apparent in Fig. 2c, the critical interaction between trial block and dose is apparent in the A′ non-parametric index of sensitivity (Craig 1979) [$F(6,66)=3.62$, $P<0.05$], as are main effects of dose [$F(3,33)=8.86$, $P<0.001$] and trial block [$F(2,22)=32.51$, $P<0.0001$]. Analysis of the d′ measure yielded similar results, with significant effects for dose [$F(3,33)=7.14$, $P<0.005$], trial block [$F(2,22)=12.21$, $P<0.001$] and their interaction [$F(6,66)=2.62$, $P<0.05$]. The measure of criterion *beta* is presented in Table 1. This measure disclosed an increasing reluctance to respond across trial blocks [$F(2,22)=9.04$, $P<0.005$], with some suggestion of greater reluctance under the low dose [$F(3,33)=2.78$, $P<0.09$]. There was no suggestion of an interaction in *beta* between trial block and dose [$F<1.0$].

Mean reaction times (RTs) to hits and associated measures of variability are presented separately for runs 1 and 2 in Table 2. RTs showed a small monotonic increase from 504 ms with placebo to 543 ms under the high dose [$F(3,33)=5.21$, $P<0.03$]. There was no suggestion of an

Table 1. Mean values of criterion measure *beta* as a function of dose and trials (averaged over subjects), and associated standard errors

Dose	Trials		
	1–162	163–324	325–486
Placebo	4.65±1.30	6.38±1.83	5.92±1.30
Low	4.87±1.30	7.87±1.54	8.01±1.70
Medium	3.74±0.80	4.87±0.93	6.00±1.23
High	3.26±0.80	4.55±0.77	5.08±1.39

Table 2. Reaction time values (in ms) averaged over subjects as a function of dose, trials and run. The mean RT for each cell is given at the top with the associated standard error, and the attendant mean standard deviation of the contributory RT distributions is given below (in parentheses)

Dose	Trials		
	1–162	163–324	325–486
Run 1			
Placebo	498±12.6 (97± 8.8)	500±12.8 (102± 9.1)	524±13.9 (103±10.4)
Low	502±15.9 (83± 6.7)	517±17.0 (89± 7.7)	523±12.7 (90± 6.0)
Medium	526±17.9 (92± 8.0)	522±18.5 (90± 5.6)	546±21.4 (104±10.4)
High	542±17.3 (113± 9.1)	542±15.0 (95± 6.9)	565±16.7 (104± 6.5
Run 2			
Placebo	506±14.7 (90± 9.2)	496±11.4 (89± 6.9)	501±14.6 (81± 7.5)
Low	500±12.7 (102± 9.0)	506±13.4 (80± 5.4)	519±11.8 (86± 5.8)
Medium	517±18.1 (90± 6.4)	510±16.7 (91±10.2)	512±19.9 (93± 6.8)
High	534±19.0 (99± 6.9)	557±21.8 (115±10.7)	516±18.2 (118± 8.0)

RT effect associated with the factor of trial block [$F(2,22) = 1.71$] or the interaction between trial block and dose [$F(6,66) = 1.78$]. RTs were faster by 10 ms in run 2 than in run 1 [$(F(1,11) = 6.16,\ P < 0.03$] and they increased throughout the run by 22 ms in run 1 but remained stable in run 2 (as reflected in a significant interaction between run and trial block [$F(2,22) = 10.96,\ P < 0.0005$]). The associated standard deviations of the RT distributions were not significantly affected by any experimental variable. In conformance with previous evidence (Nuechterlein et al. 1983), examination of RT distributions disclosed that nearly all responses lay within a period spanning 150–1000 ms post stimulus. The RT distributions for false alarms on trials following missed targets were examined in detail for evidence of "late" hits, which would have been erroneously tabulated as false alarms. These distributions contained occasional responses with RTs less than 150 ms; the incidence of such trials, however, was negligible (less than 0.2%) and their proportion did not grow with increasing dose. A separate analysis of performance accuracy measures deleting trials with RTs <150 ms yielded the same pattern of results as reported here.

The possibility that the effects on performance accuracy were mediated peripherally in the form of increased blinking and/or fixation difficulty was assessed by visually scoring the EOG records for any evidence of eye blink or movement during a 250-ms period beginning with digit onset. Analyses of eye blink and movement rates yielded no evidence that they were responsible for the effects of dose or trial block on performance accuracy measures. The overall rate of blinks was low, particularly in comparison to the prevailing error rates. On average, blinks were in evidence on only 2.5% of target trials (never exceeding 4% in any condition), and the rate was not statistically affected by dose [$F < 1.0$], trial block [$F(2,22) = 1.47$] or their interaction [$F < 1.0$]. Even with the very conservative scoring epoch described above, blinks could account overall for only 5.5% of missed targets.

Discussion

In sum, these data show that ethanol intoxication produces dose-related impairment in the ability to sustain attention. A variety of mechanisms may contribute to this impairment. A portion of the impairment may be peripherally mediated, although our analysis of eye movements and blinks indicates that this portion was negligible. Under some conditions requiring saccadic following or rapid shifts in gaze, the well known effects of ethanol on eye tracking, convergence and accomodative functions (Stapleton et al. 1986) may exacerbate the performance decline by producing an equivalent to a degraded stimulus environment. The task used here, however, required predominantly static visual acuity functions, upon which ethanol's effects are minimal (Stapleton et al. 1986). Convergent information obtained from analysis of simultaneously-obtained event-related brain potentials (Rohrbaugh et al. 1987b) is consistent with the conclusion that the sensory input was not impaired at a peripheral level. In that analysis of brain potentials, which was based on a subset of trials selected to be free of electrical artifact, no effects of dose, trial block or their interaction were found for the N1 component, which is generally considered to reflect the quality of sensory input.

A more probable interpretation of the performance data is that the deleterious effects of ethanol reflect disruption of central processes. These effects are manifest both in the overall *level* of performance and in the rate of *decrement* over time. Psychophysiological and stress studies have shown that overall level of vigilance performance is controlled by basal arousal and related non-specific factors (Parasuraman 1984). The effect of ethanol on vigilance level thus may derive from the well-known sedative properties of ethanol (Erwin et al. 1978).

The sensitivity decrement over time reflects more specific aspects of the target discrimination task and the accompanying load on processing capacity (Parasuraman 1979, 1984, 1985). Our finding that ethanol potentiates this decrement has both theoretical and practical significance. On a theoretical level, our data suggest that ethanol diminishes central processing capacity and the availability of capacity over time. This interpretation of the decrement in terms of capacity loss is supported by data from a secondary task situation in which it was shown that the decrement is accompanied by an overall loss in responsivity to auditory probe stimuli as well as to the digit stimuli (Parasuraman

1985). It has been proposed that the principal capacity demands in this task are those that are needed to encode the degraded stimuli (Nuechterlein et al. 1983) and are called upon continuously by the rapid rate of stimulation (Parasuraman 1979, 1985). The present study differs thereby from most previous studies of the effects of ethanol intoxication on vigilance, which have used readily discriminable stimuli that are more amenable to automatic analysis and thus may be relatively impervious to the effects of ethanol (Fisk and Schneider 1982).

On a practical level, these data have relevance to the interpretation of accident statistics for driving and other tasks requiring continuous performance, to the extent to which such tasks place continuous demands on central processing capacity. Our finding that ethanol's effects are exacerbated with time on task has implications for the translation of discrete laboratory tasks to field studies in which performance demands are more likely to be continuous. Even though the skills involved might appear unaffected when probed for brief periods, the interactive effects of ethanol and time on task may well render significant impairment during typical field conditions. It is important to emphasize that evidence for such a decrement was obtained here over performance periods that were so short as to preclude marked changes in blood alcohol content or the appearance of drowsiness. The actual extent of impairment was probably underestimated by the present results, because of the likelihood that sleep episodes may intrude under conditions less rigorously controlled than those used here.

References

Colquhoun WP (1962a) Effets d'une faible dose d'alcohol et de certains autres facteurs sur la performance dans une tache de vigilance. Bull du C.E.R.P. 1:27–44

Colquhoun WP (1962b) Effects of hyoscine and meclozine on vigilance and short-term memory. Br J Ind Med 19:287–296

Colquhoun WP (1976) Estimation of critical blood-alcohol level in relation to tasks of sustained attention. In: Horvath IM (ed) Adverse effects of environmental chemicals and psychotropic drugs. Elsevier, Amsterdam, pp 69–83

Craig A (1979) Nonparametric measures of sensory efficiency for sustained monitoring tasks. Hum Factors 21:69–78

Docter RF, Naitoh P, Smith JC (1966) Electroencephalographic changes and vigilance behavior during experimentally induced intoxication in alcoholic subjects. Psychosom Med 28:605–615

Erwin CW, Wiener EW, Linnoila M, Truscott TR (1978) Alcohol-induced drowsiness and vigilance performance. J Stud Alcohol 39:505–516

Fabian WD, Fishkin SM, Williams HL (1983) Attentional absorbtion in marijuana and alcohol intoxication. J Abnorm Psychol 92:489–494

Fisk AD, Schneider W (1982) Type of task practice and time-sharing activities predict performance deficits due to alcohol ingestion. Proc Hum Factors Soc 21:926–930

Green DM, Swets JA (1966) Signal detection theory and psychophysics. Wiley, New York

Gustafson RD (1986a) Effect of moderate doses of alcohol on simple auditory reaction time in a vigilance setting. Percept Mot Skills 62:683–690

Gustafson RD (1986b) Alcohol and vigilance performance: effect of small doses of alcohol on simple reaction time. Percept Mot Skills 62:951–955

Hamilton P, Copeman A (1970) The effect of alcohol and noise on components of a tracking and monitoring task. Br J Psychol 61:149–156

Jansen AAI, deGier JJ, Slangen JL (1985) Alcohol effects on signal detection performance. Neuropsychobiology 14:83–87

Lamb MR, Robertson LC (1987) Effect of acute alcohol on attention and the processing of hierarchical patterns. Alcohol: Clin Exp Res 11:243–248

Landauer AA, Howat P (1983) Low and moderate alcohol doses, psychomotor performance and perceived drowsiness. Ergonomics 26:647–657

Linnoila M, Stapleton JM, Lister R, Guthrie S, Eckardt M (1986) Effects of alcohol on accident risk: epidemiology and laboratory studies. Pathologist 40:36–41

Miles C, Porter K, Jones DM (1986) The interactive effects of alcohol and mood on dual-task performance. Psychopharmacology 89:432–435

Mills KC, Bisgrove EZ (1983) Body sway and divided attention performance under the influence of alcohol: dose-response differences between males and females. Alcohol: Clin Exp Res 7:393–397

Moskowitz H, DePry D (1968) Differential effect of alcohol on auditory vigilance and divided-attention tasks. Q J Stud Alcohol 29:54–63

Moskowitz H, Burns MM, Williams AF (1985) Skills performance at low blood alcohol levels. J Stud Alcohol 46:482–485

Nuechterlein KH, Dawson ME (1984) Information processing and attentional functioning in the developmental course of schizophrenic disorders. Schizophren Bull 10:160–203

Nuechterlein KH, Parasuraman R, Jiang Q (1983) Visual sustained attention: image degradation produces rapid sensitivity decrement over time. Science 220:327–328

Parasuraman R (1979) Memory load and event rate control sensitivity decrements in sustained attention. Science 205:924–927

Parasuraman R (1984) Sustained attention in detection and discrimination. In: Parasuraman R, Davies R (eds) Varieties of attention. Academic Press, New York, pp 243–271

Parasuraman R (1985) Sustained attention: a multifactorial approach. In: Posner MI, Marin OSM (eds) Attention and Performance XI. Erlbaum, Hillsdale, New Jersey, pp 493–511

Pearson RG, Neal GL (1970) Operator performance as a function of drug, hypoxia, individual, and task factors. Aerospace Med 41:154–158

Ritchie JM (1980) The aliphatic alcohols. In: Gilman AG, Goodman LS, Gilman A (eds) The pharmacological basis of therapeutics, 6th edn. MacMillan, New York, pp 376–390

Rohrbaugh JW, Stapleton JM, Parasuraman R, Frowein HW, Eckardt MJ, Linnoila M (1987a) Alcohol intoxication in humans: effects on vigilance performance. Alcohol and Alcoholism [Suppl] 1:97–102

Rohrbaugh JW, Stapleton JM, Parasuraman R, Zubovic E, Frowein H, Varner JL, Adinoff B, Lane EA, Eckardt MJ, Linnoila M (1987b) Dose-related effects of ethanol on visual sustained attention and event-related potentials. Alcohol 4:293–300

Rundell OH, Lester BK, Griffiths WJ, Williams HL (1972) Alcohol and sleep in young adults. Psychopharmacology 26:201–218

Sahgal A, Wright C, Ferrier IN (1986) Desamino-D-arg^8 (DDAVP), unlike ethanol, has no effect on a boring visual vigilance task in humans. Psychopharmacology 90:58–63

Stapleton JM, Guthrie S, Linnoila M (1986) Effects of alcohol and other psychotropic drugs on eye movements: relevance to traffic safety. J Stud Alcohol 47:426–432

Talland GA (1966) Effects of alcohol on performance in continuous attention tasks. Psychosom Med 28:596–604

Tong JE, Henderson PR, Chipperfield GA (1980) Effects of ethanol and tobacco on auditory vigilance performance. Addict Behav 5:153–158

Vogel-Sprott M (1976) Coding and vigilance under alcohol. J Stud Alcohol 11:1581–1592

Ward TB, Lewis SN (1987) The influence of alcohol and loud music on analytic and holistic processing. Percept Psychophys 41:179–186

Wilson GT, Niaura RS, Adler JL (1985) Alcohol, selective attention and sexual arousal in men. J Stud Alcohol 46:107–115

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