Alcohol misuse appears to be rising to the extent that the UK Government considers tackling problem drinking as a social and health priority (Prime Minister’s Strategy Unit, 2004). The alcohol-related death rate in the UK has increased from 6.9 per 100,000 population in 1991 to 12.9 in 2005 (Office for National Statistics, 2006). While the dangers of binge drinking have been highlighted (e.g. Pincock, 2003), 35% of all men (42% of those aged 16–24 years) and 20% of all women (36% of those aged 16–24 years) still reported exceeding daily benchmarks on at least 1 day in the previous week to the 2005 Great Britain General Household Survey (Goddard, 2006). A recent government report estimated the societal costs of heavy drinking, including costs to the NHS in treating alcohol-related injuries and illnesses (estimated at £1.7 billion per year), costs associated with alcohol-related crime and disorder (estimated at £7.3 billion per year), and costs due to lost productivity through illness and absence from work (estimated at up to £6.4 billion per year; Prime Minister’s Strategy Unit, 2004). The latter estimate includes the costs to the economy of alcohol-related deaths. A further, less visible societal cost arises because of alcohol-related impairment in the morning after an evening’s binge drinking—due to the so-called alcohol hangover.

Estimating the societal costs of hangover is prone to inaccuracies when one considers that hangover effects may include lateness, accident risk, poorly performed work and disputes (Crofton, 1987) in addition to absenteeism. Even the recent report by Pittler et al. (2005) cites hangover cost estimates (£2 billion) that were made 20 years ago and without reference to the original author’s caveat concerning the crude nature of the costings (Crofton, 1987). A recent report by the Prime Minister’s Strategy Unit (2004) estimates that the cost to the UK economy of alcohol-related absenteeism from work (due in part to hangover) is between £1.2 and £1.8 billion per year. However, this estimate does not take into account effects of depleted worker performance and includes lost days due to long-term health problems associated with alcohol dependency. In the grey literature, the BBC (BBC, 2004) cites research carried out by an employment agency estimating that hangovers cost the UK economy £2.8 billion a year due to the average of 2.3 sick days per person per year, augmented by a further 2.5 days per year that workers spend, on average, hungover on the job. Wiese et al. (2000) cited an estimate of hangover costs to the US economy of $148 billion a year, but this estimate was criticized by Becker (2001). A fair conclusion would be that hangover costs are indeterminate but significant.

Several reviews of the hangover state have been undertaken. Finnigan and Hammersley (1992) report findings from a small number of hangover and performance studies, although this review has now become dated. In a brief review of hangover appearing as an editorial piece, Calder (1997) recounted the findings of several performance studies but did not conduct any critical evaluation. Swift and Davidson (1998) gave a detailed account of the mechanisms and mediators of hangover but did not discuss performance effects. Wiese et al. (2000) conducted a detailed systematic review of the causes, pathophysiological characteristics and treatment of alcohol-induced hangover. They defined hangover as ‘the presence of at least two symptoms (out of: headache, poor sense of overall well-being, diarrhea, anorexia, tremulousness, fatigue, and nausea) occurring after the consumption and full metabolism of alcohol with sufficient severity to disrupt the performance of daily tasks and responsibilities’ (p. 898). However, the notion that...
hangover disrupts performance of certain tasks is not as safe an assumption to make as it might at first appear. Certainly, Wiese et al. (2000) do not make a convincing case. They reviewed several studies claiming to show performance effects of hangover, but they recounted findings without any critical evaluation, and they omitted a large number of published peer-reviewed hangover performance studies.

The present review offers a more critical analysis of all published peer-reviewed papers on hangover performance that we could find. For the purposes of this review, performance effects are defined primarily as changes in cognitive functioning assessed using cognitive tests. However, more everyday aspects of performance, such as driving or performance in a management-style decision-making game are also considered. We began by searching the relevant studies and produced many false positives (e.g. non-performance based studies). Therefore, while some papers were identified from database searches, the majority were identified from references cited in papers already obtained. Some further papers were identified using citation searching of obtained papers (i.e. searching forwards for papers that cite the papers we had obtained). This was done using the MIMAS Web of Knowledge database.

We briefly outline the biological mechanisms that may underlie hangover effects before embarking on a detailed review of the literature on the performance effects of alcohol hangover. We set out to include all the peer-reviewed studies published to date but it is possible that one or two of the less well-cited studies will have been omitted.

MECHANISMS

Several pathophysiological changes that both follow and outlast acute alcohol intoxication (i.e. are present after all the acute alcohol has been metabolized) may give rise to the alcohol hangover. Wiese et al. (2000) note increased levels of acetaldehyde, hormonal alterations due to deregulated cytokine pathways and the inhibition of the availability of glucose via a process mediated by insulin. Calder (1997) lists additional phenomena associated with dehydration, metabolic acidosis, disturbed prostaglandin synthesis, increased cardiac output and vasodilation. Other potential mechanisms include sleep deprivation and insufficient eating (Verster et al., 2003). Calder (1997) suggests that the complex organic molecules found in alcoholic beverages known as congeners may have an important role in producing hangover effects because some, such as methanol, are metabolized to the notably toxic substances formaldehyde and formic acid. Congeners tend to be present in greater concentrations in darker drinks (e.g. whisky) compared with clear drinks (e.g. vodka).

These mechanisms have in common the prediction of the presence of hangover effects when the blood alcohol level (BAL) has returned to zero, after having become elevated during and following the drinking episode. Indeed, a defining characteristic of alcohol hangover effects is their presence at zero BAL. This is necessary for research purposes in order to distinguish between hangover and acute alcohol intoxication. However, there appears to be inconsistency in the way that some of these physiological changes would be predicted to affect cognitive functioning. For example, sleep deprivation is known to impair executive functioning (e.g. Jones and Harrison, 2001). On the other hand, increased cardiac output is associated with improved cognitive performance (e.g. Tomporowski and Ellis, 1986). The wide range of mechanisms and the lack of a unitary direction of effect make predicting the effects of hangover complex. Fisk and Scerbo (1987) found that, during acute alcohol intoxication, controlled processes were affected more than automatic processes, implicating an effect on executive function. Such an effect may persist into the hangover phase. Likewise, Jones and Harrison (2001) found executive function decrements following modest amounts of experimentally induced sleep loss, one of the mechanisms thought to underlie hangover. Therefore, executive function, or certainly higher cognitive functions, should be considered as candidate functions to be affected by hangover.

Humans have been drinking alcohol, and presumably experiencing hangovers, since the first mead was brewed from fermented honey around 8000 BC (Meyer and Quenzer, 2005). Nevertheless, although a number of studies have been carried out addressing hangover effects on cognition and performance, we argue in the following sections that their interpretation is severely limited due to a variety of methodological considerations.

LABORATORY STUDIES

We identified 27 English language peer-review studies that have investigated some aspect of psychological performance during alcohol hangover following controlled alcohol ingestion. Typically, in a between-subjects design, alcohol is given to one group and placebo is given to another group. After the passage of sufficient time for the acute intoxication effects to wear off (typically 11 hours), one or more cognitive/performance tests are applied. Decrements in the alcohol group relative to the placebo group are interpreted as hangover effects. A similar procedure may be applied in a related design where an analogous comparison is made but the same participants are tested twice, once following alcohol and once following placebo. However, close reading reveals that these relatively straightforward designs have not been well implemented in practice in this literature, with the majority of studies having basic methodological shortcomings.

Several early studies either present no data at all or do not present inferential statistical analyses of the data, and it is not possible to estimate statistics, such as effect sizes, based on the information presented (Carroll et al., 1964; Ekman et al., 1964; Ideström and Cadenius, 1968; Dowd et al., 1973). Future studies clearly were able to learn from these pioneering efforts. Nevertheless, without data or a rigorous data analysis it is not possible to draw meaningful conclusions from these studies. In an even earlier ground-breaking study Takala et al. (1958) compared hangover performance in medical students with non-hangover performance in a group of psychology and technical students. Unfortunately, in comparing intact groups of participants it is not possible to distinguish hangover effects from pre-existing participant differences.

Some other studies used a no-alcohol control condition rather than a placebo (Yesavage and Leirer, 1986; Taylor et al., 1996; Kruijsselbrink et al., 2006). In the laboratory, placebo controlled trials have become the gold standard for research into effects of imbibed substances such as alcohol. Results from laboratory
studies that do not implement a placebo control have little credibility. Without a placebo, participants would have known when they were consuming alcohol and so any effects shown could be expectancy effects, rather than genuine effects arising from the experimental treatment. In the case of alcohol, which is renowned amongst the general public for producing performance decrements, participants may unwittingly have put in less effort when completing the performance tests following alcohol ingestion. Morrow et al. (1990) did employ a placebo but they used a repeated measures design without properly controlling for condition order effects. Participants becoming fatigued during the experiment could explain their findings.

Another common problem is not verifying that BALs have returned to zero at the time the performance testing is carried out. As already mentioned, a defining characteristic of alcohol hangover effects is their presence at zero BAL. Ensuring BAL genuinely is zero (or so low as to be at the limits of detection) is necessary for research purposes in order to distinguish between hangover and acute alcohol intoxication. Lemon et al. (1993) purport to assess hangover effects without verifying that BAL is zero. In addition several supposed hangover studies report carrying out performance testing at elevated BALs. Such studies are likely to be picking up acute alcohol intoxication effects and cannot be interpreted as showing genuine hangover effects (Kelly et al., 1970; Seppela et al., 1976; Collins, 1980; Myrsten et al., 1980; Kim et al., 2003). Several other studies that are sometimes cited as assessing hangover effects were actually concerned with acute alcohol effects on the descending limb of the blood-alcohol curve. As these studies conducted testing at raised BALs, they do not elucidate alcohol hangover effects (Ekman et al., 1963; Jones and Vega, 1972; Peeke et al., 1980; McCaul et al., 1991; Millar et al., 1999).

Seven laboratory hangover studies are sufficiently rigorous to warrant serious attention (Collins et al., 1971; Collins and Chiles, 1980; Roehrs et al., 1991; Chait and Perry, 1994; Streufert et al., 1995; Finnigan et al., 1998; Verster et al., 2003). One further laboratory study that we have criticized for not verifying zero BAL at testing will also be reviewed further. Lemon et al. (1993) found no effects and so this study is immune from the criticism that, in not verifying zero BAL, hangover results are contaminated by acute alcohol intoxication effects. Key aspects of the design, procedure and results of these eight studies are summarized in Tables 1 and 2.

Each study involved the administration of between 0.7 and 1.6 g/kg of alcohol (median 1 g/kg, roughly equivalent to 9 units, that is 3 pints of typical 5% ABV lager, or one bottle of 13% ABV wine), as pure ethanol or vodka mixed with orange juice, tonic, lime or lemonade. The placebo was usually orange juice or another mixer with a small quantity of alcohol to provide an appropriate olfactory sensation. The majority of studies employed all-male samples, the consumption-to-test interval ranged from 7.5–12 hours (median 10 hours), and appropriate steps were taken to control condition order effects. Each study verified that BAL was zero at testing (except Lemon et al., 1993, but see above). A range of cognitive and other performance tests were applied across the memory, attention, processing speed, executive function and psychomotor domains, although attention was tested in more studies than any other function.

Only two of the studies showed hangover effects. In a between-subjects design, Verster et al. (2003) showed poorer delayed recall of items from a 15-word list the morning after 1.4 g/kg of alcohol consumption (mean recall 9.4 items, SD 3.4) compared with placebo (mean recall 11.5 items, SD 3.5). Four of the eight rigorous laboratory studies assessed divided attention (or dual task performance which amounts to the same thing) but only Roehrs et al. (1991) showed a significant hangover decrement, with null effects in the three other studies (Lemon et al., 1993; Chait and Perry, 1994; Finnigan et al., 1998). This low ‘hit rate’ of effects across studies questions the reliability of the finding by Roehrs et al. and this reliability is further questioned by the small sample size those investigators employed. Therefore, there are only limited data to support the hypothesis that hangover produces decrements in divided attention. Only one of the eight rigorous laboratory studies showed a significant hangover decrement in delayed recall, although this was the sole study to have assessed long-term memory (Verster et al., 2003). At this juncture it is worth considering the extent to which long-term memory decrements have been shown in the less rigorous studies. Takala et al. (1958) noted a poorer rate of improvement due to practice (later trial performance was compared with earlier trial performance) on a visual search task after 1.3 g/kg beer, although, as already stated, this result was based on an intact groups comparison and it is possible that the technical students in the control group had generally superior visual search ability relative to the medical students in the hangover group. Ekman et al. (1964) assessed immediate and 7-minute delayed recognition of letter pairs but neither presented nor analysed the test scores. A small effect is claimed but it is not clear whether this is an acute intoxication effect. In a related design, Kim et al. (2003) showed long-term memory ‘decrements’ following 1.5 mg/kg alcohol consumption. However, again it is not clear whether this is an acute intoxication effect.

We find these mainly null findings surprising and strongly suspect their explanation lies with study insensitivity rather than a genuine absence of hangover effects. There are three reasons for this. First, five of the above studies employed crossover placebo designs, yet such designs have been argued to be unsuitable for ingestion studies. A combination of proprioceptive changes due to alcohol ingestion and inferential reasoning on the part of participants allows some participants to determine which is the placebo condition and diminishes study sensitivity (Finnigan and Hammersley, 1992). Second, all the above studies employ the pharmacological model of drug action, i.e. the alcohol is administered as a single large dose under as close to possible double-blinded conditions. However, this model may not be applicable to study the social phenomenon of drinking alcohol (Finnigan and Hammersley, 1992). For example, in real life people may control their drinking rate so that they can continue to function socially and will often take food with the alcohol. Third, it is possible that the alcohol doses employed in laboratory studies are insufficient for a hangover to occur; insufficient; due to research-ethics-imposed consumption limits. Nevertheless, five of the identified eight rigorous laboratory studies assessed subjective hangover symptoms and in all cases hangover symptoms were detected (Collins and Chiles, 1980; Roehrs et al., 1991; Streufert et al., 1995; Finnigan et al., 1998; Verster et al., 2003). It is possible that the alcohol dose required to produce cognitive hangover effects is larger than that for somatic hangover symptoms.
Table 1. Summary of design and procedural aspects of the eight rigorous laboratory-based hangover studies

<table>
<thead>
<tr>
<th>Country</th>
<th>Funder</th>
<th>Design</th>
<th>Participants</th>
<th>Male:female ratio</th>
<th>Age Range</th>
<th>Alcohol consumed (g/kg)</th>
<th>Alcoholic drink</th>
<th>Placebo drink</th>
<th>Restricted before morning test session</th>
<th>Consumption to test interval (h)</th>
<th>BAL zero at test</th>
<th>OJ = orange juice</th>
</tr>
</thead>
<tbody>
<tr>
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<td></td>
<td></td>
</tr>
<tr>
<td>Collins et al. (1971)</td>
<td>USA FAA Army and Navy</td>
<td>Independent</td>
<td>20 college students</td>
<td>20/0</td>
<td>Range 21–30</td>
<td>1.6 (2 ml/kg)</td>
<td>Vodka and OJ</td>
<td>OJ with few drops rum extract and colouring</td>
<td>–</td>
<td>10 (max)</td>
<td>Yes (at 8 and 10 h post-drink)</td>
<td>0.25 %</td>
</tr>
<tr>
<td>Collins and Chiles (1980)</td>
<td>USA NIH, NIAAA</td>
<td>Related</td>
<td>11 healthy pilots</td>
<td>7/4</td>
<td>Mean 39.6 (SD not stated)</td>
<td>0.8</td>
<td>Vodka and tonic</td>
<td>Rum extract and colouring</td>
<td>Coffee/caffeine</td>
<td>7.5</td>
<td>Very low (under 0.007%)</td>
<td></td>
</tr>
<tr>
<td>Roehrs et al. (1991)</td>
<td>Australia Federal Office of Road Safety, Canberra</td>
<td>Related</td>
<td>5 healthy males</td>
<td>5/0</td>
<td>Range 21–34</td>
<td>0.5–1.0 (max 7–8 units)</td>
<td>Ethanol and OJ</td>
<td>Tonic with three drops of ethanol floated on OJ</td>
<td>Caffeine</td>
<td>9</td>
<td>11</td>
<td></td>
</tr>
<tr>
<td>Lemon et al. (1993)</td>
<td>USA NIDA, USPHS</td>
<td>Independent</td>
<td>64 healthy males</td>
<td>64/0</td>
<td>Mean 24.6 (SD 6.6)</td>
<td>1.0</td>
<td>Ethanol, tonic and lime</td>
<td>Ethanol and OJ</td>
<td>–</td>
<td>10</td>
<td>Not stated</td>
<td></td>
</tr>
<tr>
<td>Chait and Perry (1994)</td>
<td>USA NIDA</td>
<td>Related</td>
<td>14 healthy volunteers</td>
<td>10/4</td>
<td>Mean 24.5 (SD 21–34)</td>
<td>1.0</td>
<td>Ethanol, tonic and peppermint extract</td>
<td>Tonic and peppermint extract sprayed with atomized 10% ethanol on OJ</td>
<td>Eating, smoking, coffee</td>
<td>10</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td>Streufert et al. (1995)</td>
<td>Scotland AERC</td>
<td>Related</td>
<td>21 healthy professionals</td>
<td>21/0</td>
<td>Under 46 (Mean not stated)</td>
<td>1.0</td>
<td>Ethanol, tonic and peppermint extract</td>
<td>Tonic and peppermint extract sprayed with atomized 10% ethanol on OJ</td>
<td>Eating and drinking anything but water</td>
<td>10</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td>Finnigan et al. (1998)</td>
<td>USA</td>
<td>Related</td>
<td>40 male healthy heavy social drinkers, non-smokers</td>
<td>40/0</td>
<td>Mean 25.6 (SD 18–41)</td>
<td>0.7 (100 mg/100 ml blood)</td>
<td>Vodka, water and diabetic OJ</td>
<td>Diabetic OJ, rim swabbed with vodka and lozenges</td>
<td>–</td>
<td>10</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td>Verster et al. (2003)</td>
<td>Netherlands</td>
<td>Independent</td>
<td>48 healthy moderate drinkers</td>
<td>24/24</td>
<td>Mean 21.9 (SD 17.1)</td>
<td>1.4</td>
<td>Ethanol and OJ</td>
<td>OJ and Grand Marnier essence consumed with nose clip</td>
<td>Eat, smoke, coffee, excessive water</td>
<td>10</td>
<td>Yes</td>
<td></td>
</tr>
</tbody>
</table>
While some aspects of the design of laboratory studies can easily be addressed to improve sensitivity, research ethics limitations on alcohol dose and the limitations of the pharmacological model of drug action applied to alcohol ingestion studies have led some investigators to look beyond the laboratory.

**NATURALISTIC ALCOHOL CONSUMPTION**

Using an alternative approach, three studies have investigated the cognitive consequences of hangover subsequent to more naturalistic consumption, i.e. after participants have been allowed to drink what and when they choose. One of these showed decrements on several tests of attention—but, as zero BAL at testing was not confirmed, these could be due to acute alcohol intoxication rather than hangover effects (Anderson and Dawson, 1999). McKinney and Coyle (2004) used a related design with n = 48 and found impairments for free recall of a word list, delayed recognition of words in the list, and both simple and complex reaction times. Testing was carried out at least 7 hours after reported consumption of an average of 1.6 g/kg of alcohol and BAL was zero at testing for all except two participants whose readings were very low. Finnigan et al. (2005) used a between-subjects design with n = 25 in the hangover group but showed no cognitive effects at zero BAL the morning after reported consumption of an average of 1.7 g/kg of alcohol. We estimate the power of these studies to be 0.94 and 0.47, respectively, for the detection of medium-sized effects using two-tailed hypotheses (Cohen, 1988). Therefore the absence of effects in the latter study, in common with the results. In these studies participants were always informed at the outset that hangover effects were to be assessed and they knew which was the hangover condition. Under these circumstances expectancy effects are likely to have significantly contaminated the results. Therefore, while naturalistic alcohol consumption studies can be considered as being suggestive of hangover effects they should not, on their own, be interpreted as providing definitive evidence of hangover effects.

**REVISITING LABORATORY STUDIES LACKING A PLACEBO CONTROL**

Earlier we argued that findings from laboratory studies that did not employ a placebo control were likely to be biased as any effects found were likely to be contaminated by expectancy effects, rather than genuine effects arising from the experimental treatment. However, we went on to review naturalistic alcohol consumption studies on the grounds that these studies do not employ the pharmacological model of drug action and so may have greater sensitivity relative to laboratory studies employing
Table 3. Summary of design and procedural aspects of the five naturalistic alcohol consumption hangover studies

<table>
<thead>
<tr>
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<th></th>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>Country</td>
<td>Sweden</td>
<td>Sweden</td>
<td>South Africa</td>
<td>Northern Ireland</td>
<td>Scotland</td>
</tr>
<tr>
<td>Funder</td>
<td>None stated</td>
<td>None stated</td>
<td>None stated</td>
<td>Related</td>
<td>AERC</td>
</tr>
<tr>
<td>Design</td>
<td>Related</td>
<td>Related</td>
<td>Mixed</td>
<td>Related</td>
<td>Independent</td>
</tr>
<tr>
<td>Participants</td>
<td>22 healthy volunteers</td>
<td>24 healthy volunteers</td>
<td>16 hangover versus 10 controls</td>
<td>48 students</td>
<td>25 hangover versus 33 control versus 13 acute + hangover (volunteers)</td>
</tr>
<tr>
<td>Male:female ratio</td>
<td>16/6</td>
<td>23/1</td>
<td>8/8 vs. 5/5</td>
<td>15/33</td>
<td>36/35</td>
</tr>
<tr>
<td>Age</td>
<td>Range 19–38</td>
<td>Range 22–46</td>
<td>Mean 21.7 (SD 1.1)</td>
<td>Mean 23.4 (SD 5.3)</td>
<td>Mean 24.3 (SD ns)</td>
</tr>
<tr>
<td>Alcohol consumed</td>
<td>0.6 approx. (based on mean peak BAL 147 mg%)</td>
<td>0.7 approx. (based on man peak BAL 176 mg%)</td>
<td>1.0 (minimum)</td>
<td>1.6 (mean)</td>
<td>1.75 (mean)</td>
</tr>
<tr>
<td>Alcoholic drink</td>
<td>Beer, wine, spirits</td>
<td>Beer, wine, spirits</td>
<td>Not reported</td>
<td>Not reported</td>
<td>Not reported</td>
</tr>
<tr>
<td>Control condition</td>
<td>Abstention</td>
<td>‘Non-alcoholic drinks’</td>
<td>Abstention</td>
<td>Abstention</td>
<td>Abstention</td>
</tr>
<tr>
<td>Restricted before morning test session</td>
<td>None stated</td>
<td>None stated</td>
<td>Caffeinated drinks</td>
<td>None stated</td>
<td>None stated</td>
</tr>
<tr>
<td>Consumption to test interval (h)</td>
<td>8+</td>
<td>14 (at 2 pm)</td>
<td>12–16</td>
<td>7+</td>
<td>Not stated</td>
</tr>
<tr>
<td>BAL zero at test</td>
<td>Yes</td>
<td>Yes (at 2 pm)</td>
<td>Not stated</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>

Table 4. Summary of cognitive domains and tests used in the five naturalistic alcohol consumption hangover studies (underlined tests showed hangover decrements)

<table>
<thead>
<tr>
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<tbody>
<tr>
<td>Memory</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>Word recall, delayed word recognition</td>
<td>Probe (short-term memory recall)</td>
</tr>
<tr>
<td>Attention</td>
<td>–</td>
<td>–</td>
<td>Digit symbol modalities test, PASAT, Letter cancellation, star cancellation, symbol cancellation</td>
<td>Reaction time (simple and 5-choice), selective attention, divided attention, spatial attention, sustained attention, 5-Choice reaction time</td>
<td>Dual task tracking/ RT, Vigilance (repeating three-digit numbers)</td>
</tr>
<tr>
<td>Processing speed</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Executive function</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>5-Choice reaction time</td>
<td>–</td>
</tr>
<tr>
<td>Psychomotor</td>
<td>Car driving (cone avoidance)</td>
<td>Driving simulator – cover 20 km as fast as possible</td>
<td>–</td>
<td>Stroop</td>
<td>–</td>
</tr>
</tbody>
</table>

such models. Nonetheless, these naturalistic consumption studies also did not control expectancy effects. In the interests of balance the findings of the non-placebo controlled laboratory studies are considered in this section as further examples of suggestive hangover effects, albeit effects which may be primarily due to expectancy processes.

Yesavage and Leirer (1986) found evidence of poorer performance piloting a flight simulator in 10 male navy pilots 14 hours following administration of approximately 1 g/kg of alcohol served as ethanol added to a soft drink. Blood alcohol was zero. Taylor et al. (1996) did not show any decrements on a flight simulator in 23 male and female pilots 8 hours following ingestion of 0.6 g/kg of alcohol served as ethanol and diet soda. Individual blood alcohol levels were not reported, but the mean BAL had dropped to zero 1 hour prior to testing. Kruijsselbrink et al. (2006) showed an increase in choice reaction time errors in 12 female students 8.5 hours following consumption of approximately 1.2 g/kg of alcohol served as beer, compared with after abstaining. Blood alcohol was zero.

In summary, these studies have shown decrements in ability to pilot a simulated aircraft and in attentional processing (choice reaction time) at alcohol doses ranging from 1 to 1.2 g/kg. The study employing a much smaller quantity of alcohol (0.6 g/kg) did not show any effect – probably due to the low alcohol dose. However, it must be remembered that these studies did not control expectancy effects pertinent to alcohol consumption, and this is likely to have biased these participants’ performance to a sub-optimal level.

DISCUSSION

In the Introduction we highlighted executive function as likely to be susceptible to hangover effects, based on analogy with research into acute alcohol and sleep deprivation. However, only one study has assessed executive functioning during hangover and no effect was observed. This may be due to insensitivity associated with the manner of assessment – using a management simulation game rather than using well-validated neuropsychological testing. Future research should employ well-validated executive function tests.

Encouragingly, there is some consistency in the kinds of cognitive effect shown across the laboratory and naturalistic alcohol consumption studies. Verster et al. (2003) and McKinney and Coyle (2004) showed hangover-related memory
decrement. The latter study also shares the finding of attention decrements with the laboratory-based studies of Roehrs et al. (1991) and Kruisselbrink et al. (2006). A convergent finding from differing methodologies is known as triangulation and is considered to be a sign of validity in psychological research (e.g. Howitt and Cramer, 2005). As more studies are carried out, there may be a continued trend of convergent findings, which could lead future reviewers to be able to reach more definitive conclusions. Nevertheless, we have identified some serious problems with the two methodological approaches used to study hangover effects.

Rigorous laboratory-based studies, where participants are blinded to alcohol consumption, have tended not to show effects of hangover on performance. This insensitivity may arise partly because the pharmacological model of drug action, where a certain drug dose is predicted to affect aspects of behaviour, may be of little relevance to normal drinking (Finnigan and Hammersley, 1992). This is because the typical laboratory-based controlled intake study ignores potentially important everyday aspects of drinking that are usually set by the drinker – for example, the number and types of drink consumed, the pace of consumption, whether food is also consumed, and the social setting. On the other hand, naturalistic alcohol consumption studies, which allow for all these factors, have tended to show effects of hangover on performance. However, as participants were unblinded in these studies, the significant results are likely to be contaminated by expectancy effects. Finnigan and Hammersley (1992) reviewed all the acute alcohol studies published from 1980 to 1991 and this remains the most-up-to-date, comprehensive review published. They concluded that the research is ‘ambitious rather than rigorous’ (p. 74) and ‘while ample evidence exists that alcohol is capable of impairing performance… it is premature to conclude that it invariably does so’ (p. 74). The criticisms they raise with respect to acute alcohol studies may also be applied to the hangover studies reviewed here. In both literatures there are studies with flaws in basic experimental design, such as very small sample size and ineffective control of expectancy, for example by not including a placebo. Additionally, in both literatures the majority of publications describe laboratory-controlled alcohol consumption studies employing the pharmacological model of drug action. As outlined in the previous paragraph, this model may be of little relevance to normal drinking. Finnigan and Hammersley (1992) argued that: ‘Natural intoxication may lead to more impairment, less impairment or different impairments’ (p. 78). This is likely to be true for hangover effects as well as for acute intoxication.

CONCLUSION

One could say that the literature on performance effects of the alcohol hangover resembles a Catch 22. Each of the two methodological approaches employed in this literature has its own interpretative problems. Controlled-intake laboratory-based studies appear to lose a significant quantity of variability attributable to user-controlled aspects of social drinking. On the other hand, data from naturalistic alcohol consumption studies are likely to be contaminated by expectancy effects. Currently there is little definitive empirical evidence determining what, if any, effects on performance arise as a result of the alcohol hangover. In this respect, the hangover and performance literature resembles the acute alcohol intoxication and performance literature, which has also yielded largely inconclusive data. Future research must overcome the shortcomings of previous research identified in this review if a full understanding of the performance effects of the alcohol hangover is to be gained. For naturalistic drinking studies, the main issue is controlling expectancy effects. A novel approach would be for future studies to exploit the predictability with which social drinking occurs, e.g. on Friday evenings. Such regularity could be used to assess hangover effects in individuals believing that they are taking part in research with other aims, by simply inviting them to attend for testing on mornings likely to follow an evening spent drinking, and on mornings likely to follow an evening of abstinence. Not all participants would have hangovers but those that do would arrive in the laboratory in a frame of mind similar to any hangover person arriving at work or college – aiming to have a reasonable go under the circumstances. This scenario is potentially very useful for understanding hangover effects and could contribute a further strand to a convergent alcohol hangover and performance literature.

REFERENCES


