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BRIEF REPORT

Awareness of sleepiness when driving

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Abstract

The extent to which sleepy drivers are aware of sleepiness has implications for the prevention of sleep-related crashes, especially for drivers younger than 30 years old who are most at risk. Using a real car interactive simulator, we report on EEG, subjective sleepiness, and lane drifting (sleepiness-related driving impairment) from 38 sleep-restricted, healthy young adults undergoing nontreatment control conditions from three (unpublished) investigations using the same experimental protocols for assessing various drinks intended to alleviate sleepiness. Participants drove 2 h during midafternoon under monotonous conditions. For all studies, subjective sleepiness and EEG activity indicative of sleepiness were highly correlated, with both changing concomitantly, along with lane drifting. Drivers had knowledge of their physiological sleepiness. There were indications that sugar content of these drinks may additionally affect sleepiness.

Descriptors: Subjective sleepiness, Electroencephalogram, Driving impairment, Sugar intake

After sleep-related road crashes, drivers usually deny knowledge of falling asleep at the wheel or feelings of sleepiness beforehand. One reason for this comes from laboratory studies showing that healthy people who fall asleep unexpectedly typically deny having been asleep if awoken within 2 min (e.g., Bonnet & Moore, 1982). As drivers cannot remain asleep for more than a few seconds without running off the road or having a crash, this may account for why their recollection of having slept is poor. Nevertheless, there is still the issue of their awareness of sleepiness beforehand.

With the possible exception of rare clinical conditions (e.g., narcolepsy), sleep is not known to occur spontaneously from an alert state, that is, healthy individuals do not experience unforewarned "sleep attacks." We (Reyner & Horne, 1998a) have shown in a non-EEG study that drivers who fall asleep at the wheel and drift out of their driving lane do have good knowledge of their level of sleepiness at the time, although they may underestimate the likelihood of actually falling asleep. Subsequently, however, they have a poor memory for the feeling of sleepiness itself. This is a common experience outside driving, as many of us cannot accurately remember how sleepy we were last night or when this became noticeable. Such recollection becomes more hazy as one thinks back further.

Greater clarification regarding the extent that drivers are aware of their sleepiness can be achieved by monitoring temporal trends in subjective sleepiness concomitant with EEG changes and sleepiness-related driving impairment. A study of long-distance truck drivers by Kecklund and Åkerstedt (1993) found that hourly subjective sleepiness ratings positively correlated with EEG alpha+theta power. Clearly, there is a need for more frequent measurements of both indices together so that changes with time and the interrelationship between the two can be ascertained. Here, we utilize recent, unpublished data that enable these comparisons to be made with young adults driving a full-size, interactive car simulator. All underwent the same experimental protocol in three comparable studies involving the consumption of noncaffeinated drinks that served as controls for other drinks containing caffeine.

Method

Participants

Sleep-related vehicle crashes typically occur with young adults (Horne & Reyner, 1995) and so we targeted this group. Participants were 21 men and 17 women, all aged between 20 and 28 years with a mean body mass index of 22.7 (*SEM* 0.6). They were healthy, medication-free, experienced drivers (driving >2 years, averaging >3 h/week), good sleepers, sleeping regular hours, and infrequent daytime nappers (less than once a month). They had Epworth Sleepiness Scale (Johns, 1991) scores within the normal range (mean 5.6, *SEM* 0.3). They were recruited by advertisement, screened by interview, had the procedures explained, signed consent forms, and were paid for their participation. These investigations had received approval from our University's ethics committee.

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Design and Procedure

On a preparatory day, drivers underwent a 2-h practice drive on the car simulator, and later underwent balanced treatment conditions (1 week apart), each of which included a control condition (see below) on which this report is based. Afternoon sleepiness was enhanced by sleep restriction to 5 h (delayed bedtime) the night before. Participants slept at home and wore wrist actimeters to ensure compliance with this sleep regimen. Actimeters were checked prior to the driving session. There was no alcohol intake for 36 h before each study and no caffeinated drinks after 18:00 h the evening before. Participants arrived at the laboratory at 13:00 h and had a standard light lunch consisting of one bread roll spread with butter, containing a slice of cheese, some lettuce, and tomato. Driving sessions began at 14:00 h, following a bathroom break. Driving began with a 30-min "warm-up" drive, followed by a 30 min rest when the drink was given while the driver remained in the driving seat listening to music and talking to the experimenter. The drink was consumed within 10 min of commencing this break. The 2-h continuous drive began at 15:00 h.

The Simulator

The simulator consisted of an immobile car with an interactive full-size computer-generated monotonous roadway having some gradual bends, projected on to a 2.0-m × 1.5-m screen located 2.3 m from the windscreen. There were two "up" and two "down" lanes with hard shoulders and simulated auditory "rumble strips." Participants sat in the driving seat and drove at their normal cruising speed. The legal speed limit in the United Kingdom is 70 mph, and our simulator drivers had to observe this limit. Although they almost invariably drove at this speed, there was some speed fluctuation, usually between 67 and 73 mph. Drivers had to stay within white lane markings. Lane drifting is the usual manifestation of sleepy driving. A car wheel crossing a lateral lane marking was identified as an "incident." On three occasions during the 2-h drive, a slower vehicle was encountered and had to be overtaken. An unobtrusive infrared camera filmed the driver's face, which was recorded with the roadway using a split-screen video display. Video data were analyzed by a skilled assistant blind to the experimental conditions to see whether incidents were due to: (a) poor steering (e.g., driver taking hand off steering wheel) or driver distraction (looking elsewhere), both of which were discounted; or (b) sleepiness (i.e., eye closure or vacant staring ahead). As a further guide, the EEG and EOG (see below) were checked, because typically during condition a there are saccadic eye movements and little alpha and theta EEG activity, whereas in condition b these EEG activities are present, often accompanied by slow, rolling eye movements typical of sleep onset. Quality checks on all these measurements were undertaken blind by a second investigator. Although driver distraction, as in condition a, might be associated with sleepiness, we only logged data that clearly indicated sleepiness, as in condition b. This simulator has a realistic driving setting and our findings are comparable with those from driving on a real road track (Baulk et al., 1998).

Subjective Sleepiness

Every 200 s drivers heard the monotonic word "sleep-check" spoken by the experimenter, to which the driver responded with a number from a modified version of the 9-point Karolinska Sleepiness Scale (Åkerstedt & Gillberg, 1990). This scale consisted of the following alert-sleepiness gradations: 1 = *extremely alert*, 2 = *very alert*, 3 = *alert*, 4 = *rather alert*, 5 = *neither alert nor sleepy*, 6 = *some signs of sleepiness*, 7 = *sleepy, no effort to stay awake*, 8 = *sleepy, some effort to stay awake*, and 9 = *very sleepy, great effort to keep awake, fighting sleep*. The scale and descriptors were located near the speedometer, within easy view.

EEG and EOG

Electrodes were attached for one channel of EEG (C_3-A_1). To identify slow eye movements, two channels of EOG were recorded (electrodes vertically offset from the two outer canthi and referred to the center of the forehead). The digitized EEG, sampled at 128 Hz, was recorded using "Labview" (National Instruments Inc., New Jersey) and spectrally analyzed using "Rhythm" (Stellate Systems, Quebec) in 4-s periods. Low and high bandpass filtering of the EEG at >30 Hz and <0.3 Hz removed slow eye movements and muscle artefact. However, there was little muscle artefact and it should be noted that drivers were seated comfortably in an ergonomically designed driver's seat (Ford Motor Co., UK). There was some unavoidable eyeblink contamination of the EEG that was mostly filtered out and does not bias the EEG outcomes (Horne & Reyner, 1996). We measured EEG alpha (8–11 Hz) and theta (4–7 Hz) power combined, which is positively associated with increasing sleepiness (Åkerstedt & Gillberg, 1990; Rechtschaffen & Kales, 1968). To remove individual differences in this EEG power and to facilitate comparison between conditions, these data were standardized within participants by expressing each epoch as a difference from the individual's mean EEG power value obtained for 30 min (eyes open, relaxed) prior to the drive divided by the standard deviation around that mean (Horne & Reyner, 1996). These standardized EEG power data were then averaged in 200-s epochs for comparison with the Sleepiness Scale values.

The Studies

The three control drinks and their particular study designs were as follows:

1. Decaffeinated coffee (250 ml) containing between 0 and 10 g sugar (to taste); Design: within subjects, double blind, with two other balanced conditions, $n = 14$ (7 men, 7 women).
2. Tea-flavored drink (250 ml) containing 25 g sugar (no other active ingredients); Design: within subjects, double blind, with one other balanced condition, $n = 12$ (6 men, 6 women).
3. Citrus-flavored drink (500 ml) containing 60 g sugar (no other active ingredients); Design: within subjects, double blind, with three other balanced conditions, $n = 12$ (8 men, 4 women).

The constituents of these drinks could not be varied, as these were control conditions for specific commercial drinks. The most obvious difference between them was in sugar content, and for this reason, drinks 1–3 will be identified respectively as low (sugar content), medium, and high. For this reason, we did not combine these data, especially as the changes with both the EEG and the Sleepiness Scale over the drive differed somewhat between the studies. These different patterns of change with independent groups further assist with evaluating the associations between the indices.

Results

The results are graphically summarized in Figure 1. Scales on the *y*-axes of the three graphs differ slightly to allow superimposition of the respective variables. This does not differentially affect the

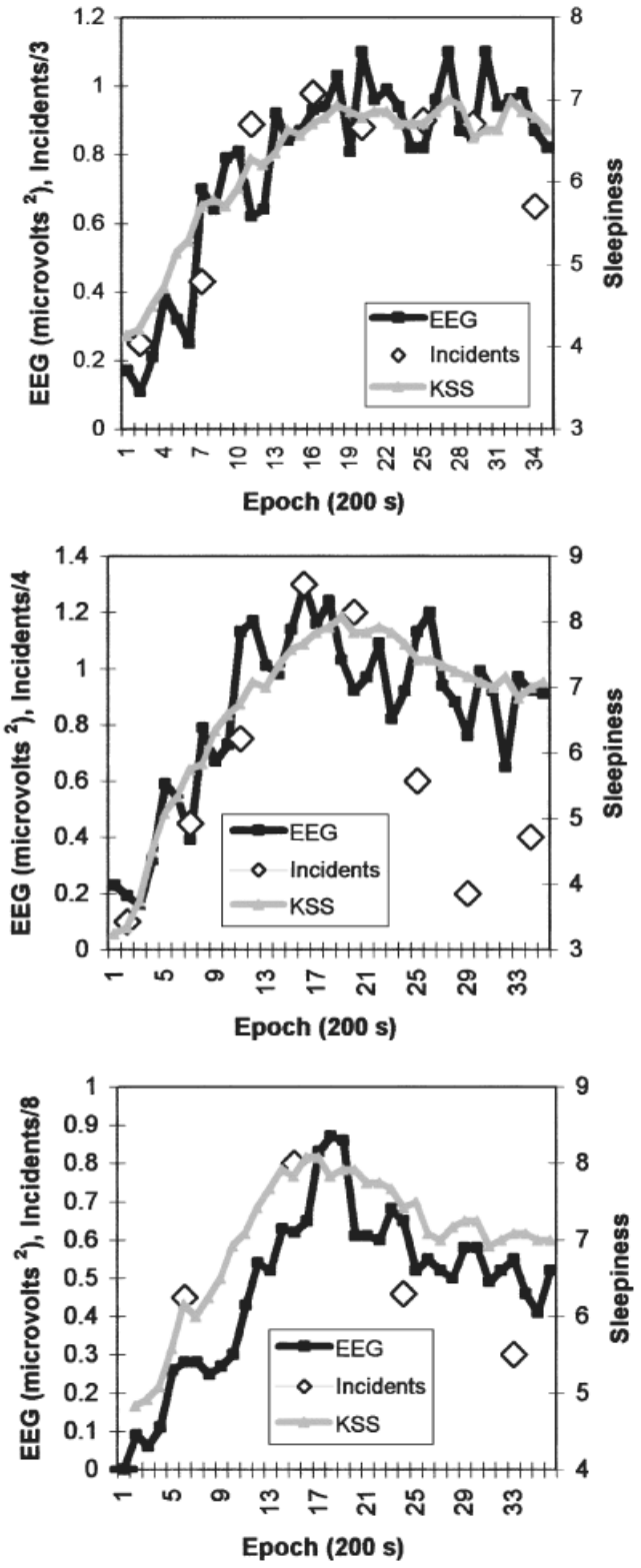

Figure 1. Mean changes in EEG power (4–11 Hz; increasing power = increasing sleepiness) and Karolinska Sleepiness Scale (KSS) ratings in 200-ms intervals during a 2-h drive following consumption of three different drinks identified as low, medium, and high (sugar content). Mean incidents (scaled down on the EEG axis) per 15- or 30-min period are also illustrated. Top panel: low drink; middle panel: medium drink; bottom panel: high drink. For each drink, the three indices are closely associated (see Results).

outcome for the correlations given below. In Figure 1 (top), showing EEG and Sleepiness Scale scores per 200 s for the low drink, it can be seen that the two trends are closely associated and highly correlated, $r = .93$, $df = 34$, $p < .001$. Variations in incident data, shown every 15 min, occur concomitantly with those of the other two indices. Collapsing the Sleepiness Scale and EEG data across 15-min epochs and correlating these values with incident data also gives significant outcomes (EEG vs. incidents: $r = .90$, $df = 6$, $p < .003$; Sleepiness Scale vs. incidents: $r = .88$, $df = 6$, $p < .004$). All three indices show initial increases in sleepiness associated with the "midafternoon circadian dip" in alertness.

The medium drink (Figure 1, middle) also shows a close temporal association between the Sleepiness Scale and EEG, $r = .88$, $df = 34$, $p < .001$, which is reflected by significant correlations with incidents when EEG and Sleepiness Scale data are collapsed into 15-min epochs (EEG vs. incidents: $r = .72$, $df = 6$, $p < .04$; Sleepiness Scale vs. incidents: $r = .71$, $df = 6$, $p < .05$). However, compared with the low drink, it is evident that incidents with the medium drink eventually decrease at a greater rate than do the trends with the Sleepiness Scale and EEG. For the high drink (Figure 1, bottom), the afternoon dip is more pronounced, with sleepiness decreasing in the latter part of the drive. Figure 1 (bottom) again shows similar and significant, $r = .92$, $df = 34$, $p < .001$, associations between the Sleepiness Scale and EEG. For technical reasons, incidents here were only logged in 30-min intervals, and no correlations with EEG and Sleepiness Scale were feasible. Nevertheless, there is a clear association between incidents and the two other variables.

When the Sleepiness Scale data for all three drinks were compared, as depicted in Figure 2, there was an unexpected but interesting difference between them: the greater the sugar content the higher were the Sleepiness Scale ratings. This association with sugar content may just be a coincidence (or otherwise—see Discussion). A repeated-measures ANOVA comparing the 36 mean Sleepiness Scale data points between the three drinks is significant, $F = 39.6$ (corrected df: 1.89, 64) $p < .001$, Huynh-Feldt $\varepsilon = 0.94$, and post hoc Tukey tests were significant, $p < .05$, between all three pairs. For comparative purposes, we have added a fourth set of Sleepiness Scale data to Figure 2, from the

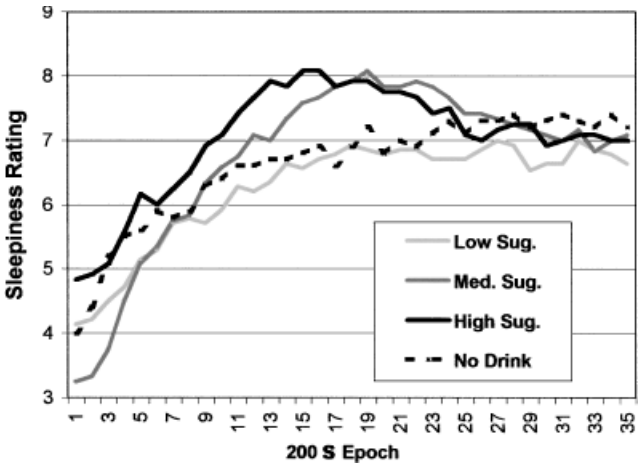

Figure 2. Comparison of Sleepiness Scale ratings for the three (low, medium, and high) drinks together with findings from another, no-drink study. There are significant differences between the low, medium, and high drink conditions (see Results). The no-drink and low drink conditions show similar, smaller rises in sleepiness.

control condition of a published study (Reyner & Horne, 1998b) where no drink or other treatment was given (there were two other within-subject, active-treatment, no-drink conditions), and using the same protocol as well as participants from the same subject pool. It can be seen that the Sleepiness Scale trends in these additional data are almost identical to those of the low drink.

We are unable to provide comparisons across these studies with incidents and EEG data because the assessments of both did change slightly from study to study (but remained constant within studies). However, the application of the Sleepiness Scale was constant throughout all studies.

Discussion

The close association between subjective sleepiness and the EEG for all three studies, even with the varying interstudy changes over time, indicates that our young adult drivers had knowledge of their neurophysiological level of sleepiness. Moreover, this knowledge was concurrent in that these indices changed together. For example, EEG sleepiness did not rise appreciably before the subjective measure. Driving incidents also closely followed both these indices, although for the medium drink the latter association was not as close for the second hour of the drive.

Within any one subject there can be quite large fluctuations in EEG power associated with the driver's admission of being very sleepy. EEG data in Figure 1 reflect these fluctuations superimposed on the general trend, which contrast with the more stable Sleepiness Scale data. It should also be remembered that each 200-s point for the EEG is derived from 50×4 s analysis periods, whereas for the Sleepiness Scale there is only one sample per 200 s. When our drivers began to drift out of lane while becoming sleepy, the EEG usually indicated a high level of alpha/theta. On making a correcting steering movement there was a transient arousal. If they happened to traverse a lateral rumble strip, then drivers heard the typical rumbling sound that also aroused them momentarily. On three occasions (around 200-s epochs 7, 13, and 23, depending on the driving speed) drivers also encountered a slow moving vehicle that they had to overtake. This also led to a transient arousal. It was rare (5% of occasions) for drivers actually to run into the back of these vehicles. Such EEG fluctuations therefore increased with rising incidents. Group averages of EEG data across an epoch reflect the influence of these fluctuations in arousal.

The difference in subjective sleepiness between the three drink conditions was an unexpected finding. It might be explained by their different sugar contents, as this is the most obvious difference between them. The addition of more data from a no-drink study (Figure 2) seems to endorse this interpretation. The medium and high drinks contained bolus amounts of sucrose in solution, consumed on a fairly empty stomach (the lunch consisting of only a small, single cheese roll consumed about 90 min earlier). Conversion of the sucrose to glucose and absorption in the stomach would have been rapid and well under way by the time the drive commenced (about 30 min later). A literature search on the effects of sugar/glucose loading on alertness/sleepiness produced equivocal findings for those studies using participants who were already alert and not previously sleep deprived. On the other hand, there is greater consistency among the few studies of people already sleepy before sugar/glucose intake (cf. Landstrom et al., 2000). Here, there is an initial arousing effect lasting 20–30 min followed by marked drowsiness, with the latter perhaps related to the insulin surge and other sequelae. As our drivers did not commence driving until at least this point in time, the initial effect would not be so evident. Bruck, Armstrong, and Coleman (1994), in a double-blind study involving EEG monitoring and administration of an auditory vigilance test to sleepy patients given 50 g glucose in solution versus an artificially sweetened drink (following a light lunch), reported a significant glucose-related enhancement of sleepiness. We are presently investigating more systematically this putative sleepiness-enhancing effect of bolus ingestion of sugars. It should be noted that our drinks were decaffeinated controls, and the addition of caffeine can effectively counteract this apparent action of sugar.

To return to our more important finding relating to the awareness of sleepiness, would our drivers have known that they were sleepy had we not asked them? If not, then one would have to accept that it is common for healthy drivers to have unforewarned and spontaneous "sleep attacks" eventuating in accidents. However, this seems unlikely, because, as we have previously shown (Reyner & Horne, 1998a), drivers who run off the road (rather than have a more minor lane "drift") in our simulator and actually "crash" have already reached Sleepiness Scale scores of 8 or 9, which embody "fighting" sleep. Under real driving conditions "fighting sleep" implies that the driver would be performing acts such as opening the window and so forth, in attempts to overcome sleepiness and, in our opinion, these acts are self-evident of sleepiness. Of course, it could be that by asking our drivers periodically to report their sleepiness we might have heightened their perception of sleepiness. This may be so, but it implies that when drivers reflect on whether they are sleepy, they may become more aware of any sleepiness. Given that sleepiness is a major cause of serious road accidents (Connor et al., 2002; Horne & Reyner, 1995; Maycock, 1996), educating drivers about the dangers of driving while sleepy may prompt them to ask this question of themselves more often, and in doing so, help prevent such accidents.

Finally, the level of sleepiness sustained by our drivers followed a night when sleep was restricted to 5 h. A further sleep limitation would have resulted in a greater level of afternoon sleepiness, and the greater likelihood of a more rapid onset of sleepiness. It could therefore be argued that our findings are only relevant to the level of sleepiness we induced, and that perhaps a sleepier driver has less forewarning and might even be "less responsible" for any accident. However, in that such drivers must knowingly have driven with inadequate sleep, they are already forewarned in this respect.

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