

## Conclusion

School age children (4-14 years old) restrained with a seat belt were 2-10 times as safe as unbelted children in car crashes and were at least as well protected as adults wearing seat belts. Despite these benefits, 40% of children in our study were unbelted. Urgent efforts should therefore be made to increase the use of seat belts by school age children. However, it is not clear if the degree of protection afforded by such belts could be improved. Given the impact of childhood injury on potential life lost, further research and development of highly effective restraints designed for school age children is warranted.

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Contributors: see bmj.com.

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# Driver sleepiness and risk of serious injury to car occupants: population based case control study

Jennie Connor, Robyn Norton, Shanthi Ameratunga, Elizabeth Robinson, Ian Civil, Roger Dunn, John Bailey, Rod Jackson



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## Abstract

**Objectives** To estimate the contribution of driver sleepiness to the causes of car crash injuries.

**Design** Population based case control study.

**Setting** Auckland region of New Zealand, April 1998 to July 1999.

**Participants** 571 car drivers involved in crashes where at least one occupant was admitted to hospital or killed ("injury crash"); 588 car drivers recruited while driving on public roads (controls), representative of all time spent driving in the study region during the study period.

**Main outcome measures** Relative risk for injury crash associated with driver characteristics related to sleep, and the population attributable risk for driver sleepiness.

**Results** There was a strong association between measures of acute sleepiness and the risk of an injury crash. After adjustment for major confounders significantly increased risk was associated with drivers who identified themselves as sleepy (Stanford sleepiness score 4-7 v 1-3; odds ratio 8.2, 95% confidence interval 3.4 to 19.7); with drivers who reported five hours or less of sleep in the previous 24 hours compared with more than five hours (2.7, 1.4 to 5.4); and with driving between 2 am and 5 am

compared with other times of day (5.6, 1.4 to 22.7). No increase in risk was associated with measures of chronic sleepiness. The population attributable risk for driving with one or more of the acute sleepiness risk factors was 19% (15% to 25%).

**Conclusions** Acute sleepiness in car drivers significantly increases the risk of a crash in which a car occupant is injured or killed. Reductions in road traffic injuries may be achieved if fewer people drive when they are sleepy or have been deprived of sleep or drive between 2 am and 5 am.

## Introduction

Published estimates of the proportion of crashes attributable to sleepiness vary more than tenfold, from 1-3% for the United States<sup>1</sup> to 10% in France<sup>2</sup> and 33% in Australia.<sup>3</sup> This variation reflects the quality of the data available as these figures are derived from descriptive information about crashes.

Measures of acute and chronic sleepiness, sleep restriction, sleep disorders, and work patterns that interfere with normal sleep have been associated with decreased performance in psychomotor tests and driving simulators<sup>4-8</sup> and with increased rates of crashes in selected populations.<sup>9</sup> We examined the association of these sleep related characteristics with the risk of

Division of Community Health, University of Auckland, Private Bag 92019, Auckland, New Zealand

Jennie Connor  
senior lecturer in epidemiology

Shanthi Ameratunga  
senior lecturer in epidemiology

Rod Jackson  
professor of epidemiology

Elizabeth Robinson  
biostatistician

Institute for International Health, University of Sydney, Newtown, New South Wales 2042, Australia

Robyn Norton  
professor of public health

continued over

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Department of  
Surgery, Auckland  
Hospital, Private  
Bag 92024,  
Auckland, New  
Zealand

Ian Civil  
*director of trauma  
services*

Department of Civil  
and Environmental  
Engineering,  
University of  
Auckland

Roger Dunn  
*associate professor*

Bailey Partnership,  
Porirua 6006, New  
Zealand

John Bailey  
*scientist*

Correspondence to:  
J Connor  
j.connor@  
auckland.ac.nz

crashes in which a car occupant is injured or killed in a regional population.

## Methods

We carried out a population based case control study in the Auckland region of New Zealand between April 1998 and July 1999. The region includes urban, suburban, and rural areas and has a population of about one million. The source population for study participants comprised drivers of light vehicles on public roads. We excluded all vehicles licensed as heavy vehicles, taxis, and emergency vehicles. We applied the definitions of geographical boundaries, time period, eligible vehicles, and eligible roads in an identical manner to cases and controls.

**Selection of cases**—We prospectively identified all drivers or passengers in eligible vehicles who were admitted to hospital or died as the result of a car crash in the study region through daily surveillance and case finding in the region's four trauma hospitals and single coroner's office. In each case the driver of the vehicle was the key informant, whether or not the driver was injured, unless the driver was killed (see below).

**Selection of controls**—The control group comprised a sample of car drivers representative of all time spent by people driving on the region's roads during the study period. They were identified by cluster sampling of drivers at 69 randomly selected sites on the road network (for details see the long version of this paper on [bmj.com](http://bmj.com)). Surveys were carried out at an average of one a week and recruitment approximately matched accrual of cases.

**Data collection**—Interviews with case drivers were conducted face to face in hospital or by telephone at home. Proxy interviews were sought for drivers who sustained fatal injuries or were too ill to participate. For control drivers we obtained contact details, suitable interview times, and results of a breath test for alcohol at the roadside recruitment sites. Interviews were conducted by telephone. Many (65%) of the interviews were carried out in the 48 hours after the crash or survey. The highly structured interview was based on a questionnaire covering the circumstances of the current trip and many usual behaviours and background characteristics of the drivers. Questions related to the time of the crash in cases were indexed to the time of the roadside survey for control drivers, and the questions related to sleep comprised only a small part of the interview.

**Measures of driver sleepiness**—We used the Stanford sleepiness scale, a self rating scale, to quantify progressive steps in acute sleepiness. Respondents chose one of seven hierarchical statements that most closely described their level of alertness immediately before the crash or survey.<sup>10</sup> We used the Epworth sleepiness scale to measure chronic or usual daytime sleepiness.<sup>11</sup> We obtained the start and finish times of all sleep periods in the 24 hours before the crash or survey and the number of full nights of sleep (at least seven hours, mostly between 11 pm and 7 am) in the previous week. Participants were asked about symptoms of obstructive sleep apnoea and work patterns including types of shift.

**Confounding variables**—Potential confounders that we considered in the analysis were age, sex, socioeconomic status, ethnicity, alcohol consumption, use of recreational drugs, time spent driving per week, vehicle speed, average traffic speed, type of road, and how long the person had been driving that day. We collected self reported data for all confounders except traffic speed and road type, which were ascertained from environmental surveys. We also obtained objective alcohol measurements with breathalyser tests for control drivers, and results of hospital blood tests, police blood tests, or police breathalyser results for drivers involved in crashes.

**Analysis**—To account for the sampling design we weighted control data. We calculated odds ratios using unconditional logistic regression. The confounders we included in the analyses are shown in table 1. We used self reported data on alcohol consumption for the statistical modelling. We calculated population attributable risk estimates and their 95% confidence intervals.<sup>12 13</sup>

## Results

We identified 615 eligible cases during the study period. These crashes resulted in 683 admissions to hospital with non-fatal injuries and 63 deaths. Two thirds of the deaths (43) and 60% of admissions to hospital (405) involved drivers. Of the 615 case drivers or proxies eligible for the study, 30 (5%) declined to participate, and 14 (2%) could not be contacted. Fifty seven of the case interviews (10%) were completed by a proxy respondent. Of the 746 control vehicles selected, 94 declined (12%), 60 were untraceable (8%), and four could not give informed consent because of language difficulties (<1%).

Table 1 shows the distributions of measures of sleepiness and potential confounders.

There was a strong association between the level of acute driver sleepiness, as measured by the Stanford score, and the risk of injury crash (table 2). The comparison of drivers who identified any degree of sleepiness (score  $\geq 4$ ) with drivers who identified themselves as alert or relaxed (score < 4), which is most relevant to usual driving, resulted in an eightfold increase in risk.

The two direct determinants of acute sleepiness that we measured, sleep deprivation and time of day, were also strongly associated with the risk of an injury crash. The most sleep deprived drivers (three hours or less) had a high risk of injury crash, but there were only two control drivers in this group (adjusted odds ratio 47, 95% confidence interval 11 to 195). Drivers with five to seven hours of sleep were not at any greater risk than those with more than seven hours. The risk associated with driving between 2 am and 5 am was more than five times that of other times of day, but we found no increase in risk associated with the secondary circadian dip in mid-afternoon (data not shown). We observed no increase in risk with measures of chronic sleepiness. We found no major alteration in the effect estimates when we excluded proxy respondents or restricted analyses to drivers who had not been drinking.

The population attributable risk is the proportion by which the incidence of injury crashes would be

reduced if a specific risk factor was eliminated from the population. If we assume that the associations are causal and unconfounded, the population attributable risks were 11% (8% to 15%) for feeling sleepy (Stanford score 4-7 *v* 1-3), 8% (5% to 13%) for sleeping less than five hours in the previous 24 hours, and 7% (4% to 11%) for driving between 2 am and 5 am. The population attributable risk for having at least one of these three risk factors was 19% (15% to 25%).

## Discussion

We found a strong association between acute sleepiness in car drivers and the risk of a crash in which a car occupant was injured or killed that was independent of the effects of acute alcohol consumption and other major confounding factors. Decreased levels of self reported alertness were associated with increased risk. There was an eightfold increased risk if drivers reported sleepiness, an almost threefold risk for drivers who were driving after five hours or less of sleep, and a five fold risk for driving between 2 am and 5 am. In contrast, we found no significant increase in risk with measures of chronic sleepiness.

### Possible bias

It is unlikely that our results can be explained by selection bias as we identified all cases and a representative sample of controls from the study region over the study period and obtained high response rates. We also minimised information bias by using standard interviews and a reference point for acute exposures (crash or survey). Biases may remain however, particularly recall bias, even though sleepiness was not an identified focus of the study. The risk associated with a Stanford score of  $\geq 4$  is the measure most likely to be affected by recall bias and could, therefore, be somewhat inflated. The measurement of acute sleep deprivation, based on start and finish times of sleep periods, is less likely to be biased. We verified time of day, which has been used as a proxy measure of crashes related to sleepiness in previous research,<sup>14</sup> though the precision of estimates involving time of day was reduced by the clustering in time of control recruitment. Chronic sleepiness is difficult to measure by self report,<sup>7</sup> and the lack of effect associated with measures of chronic sleepiness in this study may be due in part to the methods used. Some studies have found the Epworth sleepiness scale to be an insensitive measure,<sup>15</sup> whereas others have found it to be associated with risk of crash (although not injury).<sup>16</sup> The use of symptoms alone as an indicator of obstructive sleep apnoea may have resulted in misclassification<sup>17</sup> that could have affected the validity and precision of the associated risk estimate.

Only two previous case control studies of car crashes have examined factors related to sleep<sup>15 18</sup> and only one of them measured acute sleepiness and sleep deprivation as exposures.<sup>18</sup> Although the outcome measure was crash rather than injury and the setting rural rather than predominantly urban, this recent study from Washington state found a significant increase in risk associated with nine or less hours sleep in the previous 48 hours and with self reported sleepiness, broadly consistent with our results.

**Table 1** Distribution of measures of sleepiness and confounding variables for drivers in crashes that resulted in injury to car occupant (cases) and representative sample of drivers not involved in crashes (controls)\*

	Cases (n=571)	Controls (n=588)
Stanford sleepiness scale:		
1 (most alert)	175 (34.3)	322 (54.4)
2-3	272 (53.3)	256 (44.6)
4-7	63 (12.4)	8 (1.0)
Sleep in previous 24 hours:		
>5 hours	464 (87.7)	554 (96.9)
≤5 hours	65 (12.3)	30 (3.1)
Time of day:		
2 am-5 am	46 (8.1)	17 (0.4)
2 pm-5 pm	107 (18.7)	136 (27.1)
Other	418 (73.2)	435 (72.5)
Epworth sleepiness scale:		
<10 (normal)	516 (96.1)	531 (90.8)
10-15	17 (3.2)	48 (7.9)
16-24	4 (0.7)	6 (1.3)
Sleep in previous week:		
At least 1 full night†	474 (88.6)	544 (92.3)
No full night's sleep	61 (11.4)	44 (7.7)
Regular loud snoring:		
No	375 (70.2)	379 (62.8)
Yes	159 (29.8)	204 (37.2)
Triad of sleep apnoea symptoms‡:		
No	528 (98.1)	581 (98.4)
Yes	10 (1.9)	6 (1.6)
Night work§:		
No	511 (90.6)	520 (91.9)
Yes	53 (9.4)	61 (8.1)
Sex:		
Women	198 (34.7)	226 (41.3)
Men	373 (65.3)	362 (58.7)
Age group (years):		
15-24	195 (34.2)	91 (13.7)
25-34	133 (23.3)	125 (22.3)
35-44	85 (14.9)	154 (24.5)
45-54	61 (10.7)	107 (19.6)
55-64	39 (6.8)	80 (14.2)
65-80	58 (10.2)	31 (5.6)
Self reported alcohol consumption in previous 6 hours (standard drinks):		
0	418 (76.4)	537 (94.7)
1	15 (2.7)	15 (1.8)
2	20 (3.7)	21 (2.7)
≥3	94 (17.18)	5 (0.8)
Measured alcohol level (mg/100 ml):		
0	263 (63.7)	474 (98.0)
1-50	32 (7.8)	9 (1.5)
>50	118 (28.6)	3 (0.5)
Educational level:		
≤3 year high school (left at age ≤15 years)	251 (44.4)	157 (25.6)
>3 years high school	137 (24.2)	154 (25.1)
Further education	178 (31.5)	276 (49.3)
Ethnicity:		
White/European	313 (54.8)	444 (74.7)
Maori	117 (20.5)	61 (9.2)
Pacific Islands	86 (15.1)	36 (6.1)
Other	55 (9.6)	47 (10.0)

\*Proportions of controls are adjusted for sampling design. Column totals may differ due to missing data.

†At least seven hours, mostly between 11 pm and 7 am.

‡Witnessed episodes of choking and breathing pauses during sleep and regular loud snoring.

§Included rotating shifts with nights, permanent night shift, and other work patterns that required regularly starting before 6 am or finishing after midnight.

### Implementation of findings

Our study shows that acute sleepiness makes a considerable contribution to the burden of car crash injuries in this population. Moreover, reductions in the prevalence of specific behaviours may result in

**Table 2** Association of variables related to sleep with risk of crash in which car occupant was injured. Figures are adjusted odds ratios and 95% confidence intervals for multivariable models\*

	Whole study population
<b>Acute sleepiness</b>	
Stanford sleepiness scale:	
1 (most alert)	1
2-3	1.7 (1.1 to 2.5)
4-7	11.0 (4.5 to 27.2)
Stanford sleepiness scale:	
1-3	1
4-7	8.2 (3.4 to 19.7)
Sleep in previous 24 hours:	
>5 hours	1
≤5 hours	2.7 (1.4 to 5.4)
Time of day:	
2-5 am	5.6 (1.4 to 22.7)
Other	1

\*Logistic regression analysis included age group, sex, educational level, ethnicity, and self reported alcohol consumption in all models. Time of day included in all models except Stanford score, for which it is a determinant.

reduction in injuries or death of up to 19%. It provides some simple evidence based messages to disseminate with regard to specific driver behaviours in place of general advice against driving while sleepy. The priority given to developing and implementing interventions to prevent crashes related to sleepiness needs to reflect the contribution of driver sleepiness to the overall burden of injury from car crashes, and any such interventions should target the specific behaviours where there is evidence of potential benefit.

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## What is already known on this topic

Driver sleepiness is considered a potentially important risk factor for car crashes and related injuries but the association has not been reliably quantified

Published estimates of the proportion of car crashes attributable to driver sleepiness vary from about 3% to 30%

## What this study adds

Driving while feeling sleepy, driving after five hours or less of sleep, and driving between 2 am and 5 am were associated with a substantial increase in the risk of a car crash resulting in serious injury or death

Reduction in the prevalence of these three behaviours may reduce the incidence of injury crashes by up to 19%

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## One hundred years ago

### The problem of the premature infant

The premature infant is born with the skin and the skeleton, and the organs of a seven-months fetus. He is called upon to play the part of a newborn infant with the *personalia* of a fetus. He is admirably fitted to continue living in the uterus, but is ill provided to meet the exigencies of an extra-uterine existence. He is suddenly forced into surroundings of a kind which impose upon him urgent calls to which he is little able to respond. His tissues have not had time to mature, and he is not ready for so complete a change in environment. He is like some dweller in the hot

plains of India who has been transported in a moment of time on some "magic carpet of Tangu" to the chill summits of the "frosty Caucasus;" with no opportunity for acclimatization such as a gradual transit affords; he is suddenly submitted to the severe strain which so marked a change in surroundings entails; it is possible that the marvellous adaptive mechanisms of the human body will overcome the difficulties of adjustment of capabilities to requirements, but there will be danger till this condition of physiological equilibrium is reached. (BMJ 1902;:1196)