Effects of Acute Alcohol Consumption on Measures of Simulated Driving: A Systematic Review and Meta-Analysis

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ABSTRACT

Driving simulators are used in a wide range of research settings to help develop an understanding of driver behavior in complex environments. Acute alcohol impairment is an important research topic for traffic safety and a large number of studies have indicated levels of simulated driving impairment imposed by alcohol across a range of performance outcome variables. The aim of the present study was to examine the impact of acute alcohol consumption on simulated driving performance by conducting a systematic review and meta-analysis of the available evidence. The online databases PubMed (MEDLINE), Web of Science (via Thomas Reuters) and Scopus were searched to identify studies that measured simulated car driving performance under control ('no alcohol' or 'placebo alcohol' ingestion) and intervention (acute alcohol ingestion) conditions, using repeated-measures experimental designs. Primary research outcomes were standard deviation of lane position (SDLP) and standard deviation of speed (SDSP); (total number of lane crossings (LC) and average speed (Speed) were secondary research outcomes). Meta-analytic procedures were used to quantify the effect of acute alcohol consumption on vehicle control, and to determine the influence of methodological variables (i.e. the duration of the simulated driving task, the limb of the BAC curve (ascending vs. descending) and the type of driving simulator employed (i.e. car vs. PC-based)) on the magnitude of the performance change due to alcohol consumption. 423 records were screened, and 50 repeated-measures trials ($n = 962$ participants, 62% male) derived from 17 original publications were reviewed. 37 trials ($n = 721$ participants) used a 'placebo alcohol' comparator to determine the effect of alcohol consumption on SDLP (32/37) and SDSP (22/37). Alcohol consumption significantly increased SDLP 4.0 ± 0.5 cm (95% CI: 3.0, 5.1) and SDSP 0.38 ± 0.10 km·h⁻¹ (95% CI: 0.19, 0.57). Regression analyses indicate BAC ($p = 0.004$) and driving simulator platform ($p < 0.001$) influence the magnitude of the SDLP change, such that higher BAC levels and the use of PC-based driving simulators were associated with a larger performance decrements ($R^2 = 0.80$). The limb of the BAC curve and the duration of the driving task did not significantly alter the magnitude of the performance change. Eleven trials ($n = 205$ participants) used a 'no alcohol' comparator to measure the effect of alcohol consumption on SDLP (10/11); few trials assessed SDSP (3/11). Alcohol consumption resulted in a small significant increase in SDLP under these conditions (standardized difference in means = 0.23, 95% CI: 0.06, 0.39). These results demonstrate that lateral (SDLP and LC) and longitudinal (SDSP) vehicle control measures in a driving simulator are impaired with acute alcohol consumption. However, SDLP is a more sensitive indicator of driving impairment than other driving performance variables and the results of the present study support its use as a performance outcome when examining alcohol-induced simulated driving impairment.

1. Introduction

Acute alcohol intoxication causes impaired driving [1-3] with relative risk increasing exponentially as blood alcohol concentration (BAC) rises [4]. Recent reports indicate the risk of motor vehicle crashes increases with BAC as low as 30 mg·dL⁻¹ [4]. Despite this, current legislation in many jurisdictions permits drivers to operate motor vehicles at intoxication levels above this, with typical enforceable limits being 50 – 80 mg·dL⁻¹ BAC. For example, countries such as Australia, New Zealand and South Africa have adopted a 50 mg·dL⁻¹ drink driving limit, while others such as the United States and the United Kingdom have a 80 mg·dL⁻¹ national maximum legal BAC limit [5]. The tolerance for some alcohol to be consumed prior to driving demonstrates a need for continued research into the effects of acute alcohol consumption on driving performance.

Driving a motor vehicle is a complex task, requiring a coordinated array of sensory, perceptual, cognitive, and motor control components [6, 7]. Clearly, there are obvious risks and ethical issues associated with using road-based vehicles to examine the effects of alcohol on driving performance [8]. Driving simulation provides an alternative experimental approach that allows researchers to examine all of the necessary skills required to operate a motor vehicle without the risk of injury [9]. In addition, driving simulation allows greater experimental control; eliminating environmental inconsistencies that would otherwise influence on-road study results [10]. Importantly, simulated driving has also demonstrated direct translation to on-road driving [10-13], making driving simulation a critical tool for the assessment of driving performance under the influence of alcohol.

Driving simulators have been used to test the effect of alcohol on driving performance for almost four decades [8]. In this time, significant technological advancements have seen driving simulators develop into complex multi-functional pieces of equipment, employing immersive scenarios and real-world driving experiences. As such, a large number of performance measures can be recorded during driving. However, there is no standard set of driving scenarios for testing alcohol impaired driving [8]. Despite this, a number of performance measures are commonly reported in simulator studies investigating the effects of alcohol, including lateral vehicle control parameters (standard deviation of lane position (SDLP) [14-20], number of lane marking crossings (LC) [20-23]), and longitudinal vehicle control parameters (standard deviation of speed (SDSP) [17, 20, 24-28], average driving speed (AVSP) [17, 20, 22, 25, 27, 29-31]). SDLP provides a method of assessing a driver's ability to maintain a given lane position and may also be regarded as a measure of driver safety [19]. That is, with increases in SDLP, the likelihood of crossing lane boundaries increases, which could result in the vehicle deviating into the lane of adjacent or oncoming traffic, increasing the chances of traffic crashes [19, 32].

However, many studies do not collect or report all of these driving performance measures and even when they do, some degree of variability is observed in performance outcomes. This may in part, be due to differences in the research question, study methodology (e.g. alcohol dose administered, timing of alcohol administration), simulator capabilities (e.g. low- or high- complexity systems) and the simulation scenarios (e.g. driving duration, driving environment) employed.

Therefore, the aim of the present study was to examine the impact of acute alcohol consumption on simulated driving performance by performing a systematic review and meta-analysis of the available evidence. Findings from this study will clarify the magnitude of effect that alcohol intoxication has on simulated driving performance measures. This information will provide researchers with greater confidence in simulated driving scenario design, the performance measures employed to test the effects of alcohol on simulated driving, and assist with the interpretation of driving performance data when conducting alcohol experiments using simulated driving tasks. It will also permit benchmarking of performance changes observed with alcohol impairment against other factors that may cause impaired driving (e.g., fatigue, distraction), allowing greater comparison to established driving impediments in future research studies.

2. Aim(s):

The purpose of this review is to investigate the effect of acute alcohol consumption on lateral and longitudinal vehicle control measures during simulated driving performance using meta-analytic procedures. More specifically, this study intends to:

- 1) Quantify the magnitude of simulated driving impairment at a given BAC using select indicators of driving performance;
- 2) Explore the dose-response effect of acute alcohol intoxication on simulated driving performance;
- 3) Determine the influence of methodological variables, such as the duration of the simulated driving task, the limb of the BAC curve (ascending vs. descending) and the type of driving simulator employed (i.e. car vs. PC-based), on the magnitude of the performance change.

3. Methods

The methodology of this review was devised in accordance with specifications outlined in the *Preferred Reporting Items for Systematic Reviews and Meta-Analysis Protocols PRISMA-P 2015 Statement* [33] and

registered at the International Prospective Register for Systematic Reviews (identification code: CRD 42015023802) ahead of the formal literature screening process.

3.1 Literature Search

Potential research studies were identified by searching the online databases PubMed (MEDLINE), Web of Science (via Thomas Reuters) and Scopus from inception until June 2016 using the terms alcohol and ethanol in combination with “driving simulat*” and “simulat* driving”. The star symbol was used to capture all words beginning with *simulat* (e.g. simulation, simulated, simulator) and the enclosed quotation marks were used to search for an exact phrase. Two investigators (D.M. and E.I.) independently screened the potential research studies to identify relevant texts. After records were identified and duplicates removed, titles were screened. All titles that indicated alignment to the topics of cognitive function/performance (as these papers may employ simulated driving tasks as a secondary measure when examining cognition) or psychoactive substances (any) were included for subsequent screening. All irrelevant titles were discarded. The remaining articles were systematically screened for eligibility by abstract and full text, respectively. The decision to include or discard potential research studies was made between three investigators (D.M., E.I. and C.I.). Any discrepancies were resolved in consultation with a fourth investigator (B.D.). The reference lists of all included studies were hand searched for missing publications. Full details of the screening process are displayed in Fig. 1.

Fig. 1. PRISMA Flow Chart (study selection methodology).

3.2 Inclusion and Exclusion Criteria

Research studies fulfilling the following criteria were eligible for inclusion:

- 1) Controlled trials using a repeated measures experimental design.
- 2) Human studies on adult (≥ 18 years of age) male or female participants with no known medical conditions (e.g. ADHD, sleep apnoea, diabetes, psychiatric disorders, or head/CNS injuries) or indication of recent psychoactive drug use
- 3) Simulated car driving performance (see *Primary and Secondary Research Outcomes* below for full details) was measured under control ('no alcohol' or 'placebo alcohol' ingestion) and intervention (acute alcohol ingestion) conditions (see *Control and Intervention Conditions* below for full details). For the purpose of this review, driving tests that were completed 'post-alcohol consumption', i.e. when BAC or

BrAC had returned to zero following a drinking episode, were not acceptable controls, due to evidence indicating drivers experiencing alcohol-hangover may be impaired [18].

- 4) BAC or BrAC is measured to indicate the level of intoxication attained at the onset of the simulated driving task.
- 5) Accessible full text research articles (including full conference proceedings) written in English. Other documents, e.g. review articles, meeting abstracts and research published in non-peer reviewed sources, were discarded.

Potential research studies containing at least one eligible comparison between an intervention and control condition were included in the present review. Other ineligible study-arms derived from the same investigation were excluded from the current analysis. For instance, where Weafer et al. [28] examined simulated driving performance amongst control vs. ADHD-affected subjects in the presence and absence of acute alcohol consumption, only those data pertaining to the control population were reviewed.

Studies were excluded from the review if: (1) alcohol was co-administered with another experimental or placebo treatment (e.g. cannabis, caffeine or sleep deprivation >8 h); (2) subjects completed a secondary task during the driving assessment (see *Secondary Tasks* for full details); (3) the ecological validity of the driving environment was compromised (e.g. driving tests completed in a MRI machine); (4) participants were offered monetary incentives for completing the driving task within a pre-defined timeframe (although rewarding of desirable driving behavior was accepted, e.g. Weafer and Fillmore [23]); or (5) performance data was not adequately reported, i.e. mean $\pm$ SD was not quantified and could not be computed using the data provided. In the event that data was not adequately reported and the study was published within the previous 10 years (2006 – 2016), the corresponding author was contacted via email in an attempt to retrieve missing data.

Several research studies identified in the literature search contained more than one intervention vs. control comparison that was eligible for inclusion. In these instances, the separate study-arms were treated as individual investigations, termed '*trials*' (e.g. Berthelon and Gineyt [29] tested the effects of three different alcohol doses against a placebo control, McCartney et al. [20] examined driving performance on two separate simulated scenarios, or Marcinski et al. [25] investigated alcohol effects amongst binge drinkers vs. non-binge drinkers). See Weighted Mean Effect Summary (3.5.1) the methodologies employed to minimize the risk data dependency.

3.2.1 Control and Intervention Conditions

Expectancies associated with the belief that one has ingested alcohol can negatively impact cognitive function [34]. To account for the influence of alcohol expectancy on simulated driving performance, included trials were grouped according to the comparator condition employed. Classification was as follows:

- (1) Under the 'placebo alcohol' condition, participants consumed a beverage that was said to contain alcohol, irrespective of the effectiveness of the placebo deception;
- (2) Under the 'no alcohol' condition participants were not given reason to believe that their driving performance would be impaired due to alcohol intoxication.

3.2.2 Secondary Tasks

Undertaking a cognitively demanding secondary task (e.g. using a mobile phone, eating, etc.) whilst operating a motor vehicle can impair driving performance [35]. Research studies were excluded from the present review if subjects completed a secondary task during the driving assessment, unless: (1) the manuscript gave sufficient evidence to indicate the task employed did not significantly alter simulated driving performance under the influence of alcohol; or (2) the manuscript indicated that performance data recorded at the time the task was administered were removed prior to computation of the mean performance score (i.e. where the task was intermittent in nature). A list of the eligible trials incorporating secondary tasks, including a full description of the secondary task and justification for inclusion or exclusion of the study on the basis of this criterion, is displayed in Appendix Table 3.

3.3 Methodological Quality Assessment

Included studies were examined for publication bias using the Rosendal Scale [36], where excellent methodological quality is indicated by a Rosendal Score $\geq 60\%$ [37]. Scoring was determined by dividing the number of 'yes' responses by the total number of applicable items. Studies with a Rosendal score $< 50\%$ were excluded from this review due to increased risk of bias.

3.4 Data Extraction and Synthesis

Data was extracted from relevant publications according to the Cochrane Handbook for Systematic Reviews of Interventions *Checklist of Items to Consider in Data Collection or Data Extraction* [38] and entered into a Microsoft Excel spreadsheet.

3.4.1 Primary and Secondary Research Outcomes

Primary research outcomes were SDLP and SDSP. These variables are among those most commonly reported as measures of simulated driving performance in the literature [8, 39]. Total number of LC and average speed were secondary research outcomes. All performance outcomes are defined in Table 1.

Where factors deemed *likely* to influence vehicle travelling speed or lateral movement were present in the simulated driving scenario, e.g. interactions with other road users (overtaking other vehicles, stopping for pedestrians etc.) or traffic signals (traffic lights, stop signs or changes to the speed limit etc.), driving performance data were ineligible for inclusion in the review (unless the manuscript clearly indicated that the affected data were removed prior to computation of the mean performance score). All ‘disruptors’ to usual driving behavior are summarized within the ‘*Driving Scenarios*’ section of the Results. Driving scenarios containing other vehicles and pedestrians that did not actively interact with the participant's vehicle were accepted.

Table 1. Simulated driving performance measures and definitions

Performance Outcome	Definition
Lateral Control Measures	
Standard deviation of lane position, SDLP	The standard deviation of the driver's average within-lane position (cm)
Lane crossings, LC	When the interactive vehicle moved outside the lane, either crossing the center line into the oncoming traffic lane or crossing the road shoulder. The total number of line crossings was recorded (<i>n</i>).
Longitudinal Control Measures	
Speed	The average travelling speed ($\text{km}\cdot\text{h}^{-1}$) of the vehicle
Standard deviation of speed, SDSP	The standard deviation of the driver's average travelling speed ($\text{km}\cdot\text{h}^{-1}$)

3.4.2 Other Relevant Data

Other information extracted from reviewed studies included: (1) standardized pre-trial conditions; (2) participant characteristics: sample size, age, weight, height, gender, ethnicity, drug use behaviors, drinking history (years, drinking frequency and standard drinks per drinking occasion) and driving history (years, driving frequency, driving duration); (3) driving simulator and scenario: simulator platform, software, scenario description, drive duration, lane width, concurrent secondary tasks; (4) alcohol administration and intoxication:

alcohol dosing method, effectiveness of the placebo deception, alcohol and placebo beverage composition, quantity of alcohol ingested, drinking time, time from commencing alcohol ingestion to beginning the simulated driving task and BAC/BrAC at the onset and completion of the simulated driving task. Data that were originally published as imperial units were converted to metric units.

3.5 Statistical Analyses

All statistical procedures were performed using IBM SPSS Statistical Software, Version 22.0 and Comprehensive Meta-Analysis, Version 3.0.

3.5.1 Weighted Mean Effect Summary

Meta-analyses were completed to determine the influence of acute alcohol ingestion on individual measures of simulated driving performance (SDLP, SDSP, LC and Speed), relative to 'no alcohol' and 'placebo alcohol' control conditions. Individual effect sizes were calculated as either the net difference in simulated driving performance (i.e. the difference in mean performance scores on control and intervention trials), or as the standardized difference in simulated driving performance (i.e. where the mean difference between each intervention and control performance score was standardized against the SD of the performance change). A positive effect estimate indicates an increase in SDLP, SDSP, LC or Speed under the alcohol condition, thus a detrimental effect of alcohol ingestion on simulated driving performance. Where meta-regression was used to explore the source of heterogeneity (i.e. following completion of a meta-analysis, See *Meta-Regression Analysis* below for full details), effect estimates were calculated as the difference in means; and where meta-regression was not performed, the standardized mean difference was used, since the purpose of standardizing the effect measure was to reduce variability between trials. The magnitude of standardized mean effect was defined in accordance with Cohen [40]: $ES \leq 0.2$ = small; $0.2 < ES \leq 0.5$ = medium; and $ES > 0.5$ = large. (Note: data sets that *appeared* eligible for meta-regression were initially analyzed using the difference in means effect measure. If heterogeneity was found to be low, i.e. exploration of the variability was not required; the original effect measure was retained).

Random-effect models were used to calculate weighted mean effects, where trials are weighted by the inverse variance of the performance change. Where the variance of the performance change was not reported, a *t*-statistic or associated *p* value was used to compute Cohen's d_z and subsequently the standard deviation of change using the following formulas [38, 41]:

$$(1) \text{Cohen's } d_z = \frac{t \text{ statistic}}{\sqrt{n}}$$

$$(2) SD_{\Delta} = \frac{|M_{\text{Alcohol}} - M_{\text{No Alcohol}}|}{\text{Cohen's } d_z}$$

Where SD_{Δ} is the missing standard deviation of change, n is the number of participants and M is the mean performance score.

If only $p > 0.05$ was reported, the missing variance was imputed using a correlation coefficient calculated using the following formula [38]:

$$SD_{\Delta} = \left(\frac{\sqrt{(SD_{\text{No Alcohol}}^2 + SD_{\text{Alcohol}}^2) - (2 \times R \times SD_{\text{No Alcohol}} \times SD_{\text{Alcohol}})}}{\sqrt{n}} \right)^2$$

Where SD_{Δ} is the missing standard deviation of change, n is the number of participants and R is the correlation coefficient. R was approximated as the mean correlation coefficient calculated using raw performance data. Sensitivity analysis was performed using $R = 0.800$ and 0.300 to test the robustness of the imputed correlation coefficient. Statistical significance was attained if the 95% CI did not include zero. Heterogeneity was assessed with Cochran's Q and the I^2 index. Low, moderate and high heterogeneity was indicated by an I^2 value of 25, 50 and 75%, respectively [42]. A p -value < 0.10 for Cochran's Q was used to indicate significant heterogeneity [38].

Sensitivity analyses were performed to determine the risk of bias due to data dependency (i.e. where multiple trials derived from a single publication bias a result). In this case, meta-analyses were performed using data derived from one or two trials per publication, only. Included trials were those that yielded the median effect estimate (i.e. compared to all other trials derived from the same investigation). Where the median effect estimate was calculated as the mean value for two trials (i.e. an even number of trials), both were included in the analysis. This structured approach was selected in preference to a random approach because certain variables (e.g. BAC level) are likely to influence the magnitude of the treatment effect, and random unequal distribution of these variables could alter the influence of alcohol ingestion by chance. Results are displayed in Appendix Table 6.

3.5.2 Meta-Regression Analysis

Restricted maximum likelihood random effects multiple meta-regression was used to quantify the dose-response effect of acute alcohol consumption on simulated driving performance and to determine the influence of methodological factors, such as: the driving simulator platform, the duration of the simulated driving task and the timing of the simulated driving task relative to the BAC curve (i.e. ascending vs. descending) on the magnitude of the performance change, where heterogeneity was present. The complexity of the driving environment and subject age were also initially proposed as potential moderator variables. However, the narrative description of each simulated driving scenario was inadequate to accurately predict the complexity of the driving task and subject age was relatively invariable (20 – 30 y) amongst the publications reviewed. Thus, insufficient data were available to examine the influence of these factors. Greater than 10 data points were required for a variable to qualify for meta-regression analysis. Regression analyses were examined for influential cases and outliers (studentized residuals, Cook's distance, and centered leverage values), normality of residuals (Shapiro-Wilk Test), multicollinearity (variance inflation factor), autocorrelation (Durbin-Watson statistic), homoscedasticity and linearity of the relationship between dependent and independent variables (plot of residuals vs. predicted values). Statistical significance was accepted as $p < 0.05$.

3.5.2.1 Alcohol Intoxication

All studies included in the present review used a breathalyzer instrument to approximate BAC. Therefore, BAC ($\text{mg} \cdot \text{dL}^{-1}$) is used to describe alcohol intoxication level. BAC measurements obtained at the onset and completion of the simulated driving task were averaged and used to indicate BAC at the time of driving (unless mean pre-/post-drive BAC was reported directly [24, 28, 43]). BAC level at the onset of driving was used if BAC at the completion of the driving task was not directly quantified [26, 27, 44]. BAC values are described as mean $\pm$ SD unless otherwise indicated; articles that reported SEM had their values multiplied by the square root of the sample size to convert to SD.

3.5.2.2 Driving Simulator Platform

The driving simulator platform employed in studies was defined as being either a PC-based system (PC) or a Car-based system (Car). Classification was determined by the described characteristics of the driving simulator and photographic evidence (where included) in the individual studies. A PC-based system was used in reference to a low level or low complexity driving simulator; typified by an unsophisticated visual display (usually a

single desktop computer monitor), rudimentary driver controls with little driver feedback (limited to some degree of torque feedback through a game-style steering wheel, e.g. Logitech® G25 or G27) [45, 46]. A car-based system was used in reference to mid-high level or moderate-high complexity driving simulators. These simulators typically have advanced visual displays (e.g. multiple screens) with a wide horizontal field of view, and drivers are usually seated in a vehicle cab (modified or full vehicle) that includes real instrument (dashboard) displays and driver controls. Kinesthetic functionality or feedback is also often applied in these systems though the steering wheel and/or foot pedals and motion cues may be represented (i.e. vehicle vibration, tilt cues, motion system) [45, 46]. Car-based systems were not sub-grouped by fidelity (e.g. fixed-base vs. motion based simulators), as too few studies employed high fidelity machines [22].

3.5.2.3 Drive Duration

The duration of the simulated driving task was taken as the total driving time (min), or when an approximate range was reported, e.g. 10 – 15 min, as the average value. Two research studies [22, 29] employed a driving scenario consisting of several distinct road types; each road yielded independent results, hence these were classified as separate trials. For these trials, drive duration was cumulative, initially taken as the time from commencing the driving test to completing each road type. For instance, the scenario by Kenntner-Mabiala et al. [22] comprised of two 24 km “vigilance” sections ($\sim 85 \text{ km}\cdot\text{h}^{-1}$) separated by rural roads, highways and urban environments. Given that the second vigilance section commences 50 – 60 min into the driving task [47], the total time to complete the vigilance roadway (i.e. both vigilance sections, as specified by the investigation) was approximated as 72 min (i.e. 55 min *plus* 17 min to drive 24 km at $\sim 85 \text{ km}\cdot\text{h}^{-1}$). The result was subsequently verified using a second analysis, where the drive duration was additive (i.e. 34 min, calculated as 17 min *plus* 17 min)

3.5.2.4 Limb of the BAC Curve

The timing of the simulated driving task relative to the BAC curve (ascending vs. descending limb) was determined for studies that: (1) reported BAC at the onset and conclusion of the simulated driving task, and at another time point to verify the direction of the BAC change [26]; (2) only reported BAC at the onset and conclusion of the simulated driving task, but yielded values indicating a clear shift ($>5 \text{ mg}\cdot\text{dL}^{-1}$) toward (i.e. ascending), or away from (i.e. descending) the target BAC level [25, 28, 29, 31]; or (3) specifically stated that driving occurred on a particular limb of the BAC curve [20, 23, 24]. Further, trials were only accepted in the analysis of this variable if alcohol administration was standardized, i.e. participants were administered a

standard quantity of alcohol (dosed per body water content or per body weight) that was ingested within a set timeframe before waiting a set period of time to commence the simulated driving test (or until a desired BAC level was attained). Three investigations failed to specify a set period of time for beverage consumption [17, 27, 30, 44] and two investigations [48, 49] administered some subjects ‘booster beverages’ to increase BAC (i.e. inconsistent alcohol dosing), and were therefore ineligible for this analysis.

4. Results

4.1 Overview of Included Studies and Study Quality

Thirty publications adhering to the eligibility criteria were retrieved via the literature search. However, three of the eligible studies [26, 50, 51] administered simulated wind gusts during the driving assessment, which pushed the vehicle away from the centre of the road and likely confounded measurements of lateral control. Therefore, SDLP and LC results from these investigations were removed from the data set before proceeding with the meta-analysis. A further 10 eligible publications issued within the previous decade [30, 31, 43, 44, 52-57] inadequately reported data (i.e. mean $\pm$ SD was not quantified and could not be computed) for at least one indicator of simulated driving performance. Unpublished data were retrieved for two studies [31, 44] and two studies [30, 43] were included having adequately documented findings for some outcomes (SDSP [43] and SDLP [30]), but not others. Six publications [52-57] were missing data on all outcomes relevant to the present review and were unable to be included. Of the remaining research studies, three [58-60] scored <50% on the Rosendal scale during the methodological quality assessment and were therefore ineligible for inclusion. Thus, a total of 50 repeated measures trials ($n = 962$ participants, 62% male) derived from 17 original publications were reviewed. Collectively, included studies yielded a median Rosendal Score of 60%. The highest Rosendal Score of 94% was calculated for Kenntner-Mabiala et al. [22]. Complete results of the quality assessment are displayed in Appendix Table 4.

4.2 Study Characteristics

The mean age of the participant population was 24 ± 2 y and mean sample size was approximately 19 ± 6 subjects per trial. Participants’ drinking history, driving history and ethnicity were described in too few studies for the data to accurately represent the reviewed sample and were therefore omitted from the present summary. In regards to standardization of pre-trial conditions, the majority of investigations prohibited consumption of

alcohol ≥ 24 h prior to experimental testing. However, approximately half of the studies reviewed did not report controlling caffeine intake [21, 22, 24-26, 28, 30, 61]. All of the investigations incorporated a familiarization, where participants practiced controlling the vehicle within the simulated driving environment prior to testing.

4.3 Driving Scenarios

The majority of simulated driving tasks involved driving, uninterrupted (i.e. no interaction with other road users or traffic signals that might alter driving behavior), along a roadway at a constant set-speed [17, 20, 23-26, 28, 30, 31]. Exceptions were:

- (1) Three driving scenarios [44, 48, 61] during which the speed limit changed on at least one occasion throughout the driving test. Longitudinal control was not recorded on any of these scenarios; therefore the removal of confounded data was not required. Performance data that was collected (i.e. lateral control measures) were included in the current analysis; and
- (2) Seven driving scenarios [17, 20-22, 27, 29, 44] required participants to interact with other vehicles or traffic signals present in the driving environment.

Interactions consisted of car following (i.e. tailing a lead vehicle) [20, 22, 29] (b & d; d – f; c & d, respectively), overtaking slower vehicles [17] (a – c), reacting to traffic lights [21], evading a stopped vehicle in the center of the roadway [29] (a – c) and braking upon detection of a slow moving truck along the roadway [44]. Performance data obtained during overtaking events and the evading of a stopped vehicle were reportedly removed from the recording prior to the computation of results, therefore lateral and longitudinal control parameters measured on these simulated driving tasks were included in the current analysis. Longitudinal control was not measured in the presence of any other vehicle disruptions. Car following, reacting to traffic lights and the presence of other vehicles (i.e. trucks) were deemed unlikely to influence lateral control performance, therefore the data obtained on these simulated driving tasks were retained in the analysis of SDLP and LC. Overall, no performance data were excluded from the current review due to the influence of vehicle ‘disruptions’. Only two studies [22, 43] provided a thorough description of the roadway to indicate the frequency and radii of curves present in the scenario.

4.4 Acute Alcohol Consumption vs. ‘Placebo Alcohol’ Control

Thirty-seven trials ($n = 721$ participants, 55% male) derived from 13 publications used a *placebo alcohol* control condition to determine the effect of acute alcohol consumption on SDLP (32/37), SDSP (22/37), LC (11/37) and average speed (20/37). Characteristics of included studies are summarized in Table 2a. The mean correlation coefficient (R) calculated for each driving performance outcome was determined using data from 9 trials investigating SDLP ($R = 0.550$) [20, 29], 5 trials [20, 26, 29] investigating SDSP ($R = 0.608$), 5 trials [20, 29, 31] investigating speed ($R = 0.428$) and 2 trials [20] investigating LC ($R = 0.700$). All values presented are mean $\pm$ SEM.

Under the placebo alcohol condition, techniques such as floating a small amount of alcohol on the surface of the beverage [21, 23-26, 30, 31], spraying an alcohol mist over the drink [20, 21, 23, 24, 27, 30, 31] and diffusing the odor of ethanol around the room where the beverages were administered [17, 22] were commonly used to provide sensory cues associated with alcoholic beverages and strengthen the illusion of alcohol administration. However, only two research studies evaluated the successfulness of the placebo deception [17, 20]. In one study [20], estimated BrAC was $35 \pm 10 \text{ mg}\cdot\text{dL}^{-1}$ ~30 min after ingesting an alcohol placebo, whilst approximately half of the alcohol dosages were guessed correctly in another study [17].

4.4.1 Meta-Analyses

The weighted mean effect summary indicates acute alcohol consumption (BAC range: 23 – 100 $\text{mg}\cdot\text{dL}^{-1}$) significantly increased SDLP $4.0 \pm 0.5 \text{ cm}$ (95% CI: 3.0, 5.1) (Fig. 2) and SDSP $0.38 \pm 0.10 \text{ km}\cdot\text{h}^{-1}$ (95% CI: 0.19, 0.57) (Fig. 3). The magnitude and statistical significance of the weighted mean effects were stable during sensitivity analysis where trials were sequentially removed, with the mean performance change ranging between 3.8 – 4.2 cm and $0.31 - 0.41 \text{ km}\cdot\text{h}^{-1}$ (95% CI's did not include zero) for SDLP and SDSP, respectively. Findings were also comparable across different levels of correlation; therefore the meta-analyses are robust to the imputed correlation coefficients (full details of the sensitivity analyses are presented in Appendix Tables 5a - d).

Analyses identified a trend towards significance for a small increase in average vehicle traveling speed after acute alcohol consumption (BAC range: 23 – 100 $\text{mg}\cdot\text{dL}^{-1}$) ($0.29 \pm 0.15 \text{ km}\cdot\text{h}^{-1}$, 95% CI: -0.005, 0.58) (Fig. 4). Whilst the magnitude of the weighted mean effect was relatively stable during sensitivity analysis where trials were sequentially removed (i.e. $0.27 - 0.46 \text{ km}\cdot\text{h}^{-1}$), the level of statistical significance of was inconsistent, likely due to the 95% CI's narrowly including zero. Similarly, the magnitude of the performance change was comparable across the different levels of correlation, whilst the significance of the performance change was

conflicting. Low heterogeneity was evident amongst trials ($I^2 = 0.00$), therefore meta-regression analyses to determine the circumstances where alcohol intake is likely to increase average speed could not be performed.

Three trials [29] reporting on LC were omitted from the meta-analysis on the basis of being extreme outliers, exceeding the mean effect estimate by >3 SD with studentized residual values ranging between 5.60 and 4.10. Analysis of the remaining trials revealed a trend towards significance for a small increase total number of LC (0.2 ± 0.1 ; 95% CI: 0.08, 0.41) (Fig. 5) with acute alcohol intake (BAC range: 62 – 87 mg·dL⁻¹). Yet prior research indicates that SDLP and LC variables are significantly correlated [62]. Visual inspection of the available data suggests that the current analysis may be confounded by differences in road lane-width employed within scenarios. However, exploration of this effect via meta-regression was not possible due to the limited number of trials ($n = 6$) reporting lane-width [20, 22], therefore LC was not subject to further analysis within the present review.

Fig. 2. Forest plot displaying the effect of acute alcohol intake (BAC range: 23 – 100 mg·dL⁻¹) on difference in mean SDLP. Size of the squares are proportional to the weight of the study.

Fig. 3. Forest plot displaying the effect of acute alcohol intake (BAC range: 23 – 100 mg·dL⁻¹) on difference in mean SDSP. Size of the squares are proportional to the weight of the study.

Fig. 4. Forest plot displaying the effect of acute alcohol intake (BAC range: 23 – 100 mg·dL⁻¹) on difference in mean speed. Size of the squares are proportional to the weight of the study.

Fig. 5. Forest plot displaying the effect of acute alcohol intake (BAC range: 62 – 87 mg·dL⁻¹) on difference in mean LC. Size of the squares are proportional to the weight of the study.

4.4.2 Meta-Regression Analyses

The effect of acute alcohol ingestion on SDLP ($I^2 = 75.8$, $p < 0.01$) and SDSP ($I^2 = 45.0$, $p < 0.01$) was heterogeneous. Therefore, multiple linear regression analyses were performed to determine the influence of methodological factors (i.e. BAC level, driving simulator platform, limb of the BAC curve and the duration of the driving task) on the magnitude of the performance change. All driving performance data were normally distributed (Shapiro-Wilk Test, $p > 0.05$).

4.4.2.1 Lateral Control Performance

Nineteen (out of the 32) trials [20, 23-25, 28, 29, 31] examining SDLP gave evidence to indicate ascending or descending BAC at the time of driving, whilst 25 trials [17, 20-25, 28, 29, 31] adequately described the duration of the simulated driving task. To maximize statistical power, these variables were investigated using two separate regression analyses; where BAC level and driving simulator platform effects were controlled in both models (Note: Including the limb of the BAC curve and the duration of the simulated driving task into the same model would reduce the data pool to 19 trials where all four of the explanatory variables were adequately reported; BAC level and driving simulator platform were known for all trials).

Exploring the relationship between limb of the BAC curve and the magnitude of the weighted mean effect, no significant effects were detected for ascending vs. descending BAC ($p = 0.960$) on lateral control performance. Modelling the remaining variables (Fig. 6) revealed significant effects of BAC ($p = 0.004$) and

driving simulator platform ($p < 0.001$) on change in SDLP, whereby higher BAC levels and the use of PC-based driving simulators were associated with a larger increase in SDLP following alcohol intake ($R^2 = 0.80$). Drive duration was omitted from the model as this variable did not significantly influence lateral control performance ($p = 0.247$ and $p = 0.171$, where ‘cumulative’ and ‘additive’ methods were used to approximate drive duration for Kenntner-Mabiala et al. [22], respectively) and would reduce the data pool to 25 trials. Using the equation generated, we can predict that SDLP begins to increase starting from $21 \text{ mg}\cdot\text{dL}^{-1}$ BAC with a 0.7 cm increase in SDLP for every $10 \text{ mg}\cdot\text{dL}^{-1}$ increase in BAC thereafter on a driving test involving a car-based simulator. Notably, regressing against the standardized difference in means removed any significant influence of driving simulator platform ($p = 0.942$). In this model, BAC alone ($p < 0.001$) accounted for 100% of the variation between trials (Fig. 7).

Fig. 6. Correlation between change in BAC level ($\text{mg}\cdot\text{dL}^{-1}$) and change in SDLP (difference in means) controlling for driving simulator platform. Circle diameter corresponds to the weight of each study. Difference in means = $3.748[\pm 2.004] + 0.069[\pm 0.022] \times \text{BAC} (\text{mg}\cdot\text{dL}^{-1}) - 5.184[\pm 1.273]$, if a car-based simulator was used. Alternatively, difference in means = $-1.436[\pm 1.408] + 0.069[\pm 0.022] \times \text{BAC} (\text{mg}\cdot\text{dL}^{-1}) + 5.184[\pm 1.273]$, if a PC-based simulator was used (model not shown). Square brackets are used to indicate the SEM of each regression co-efficient in the equation.

Fig. 7. Correlation between change in BAC level ($\text{mg}\cdot\text{dL}^{-1}$) and change in SDLP (standardized difference in means). Circle diameter corresponds to the weight of each study. Standardized difference in means = $-0.154[\pm 0.146] + 0.010[\pm 0.002] \times \text{BAC} (\text{mg}\cdot\text{dL}^{-1})$. Square brackets are used to indicate the SEM of each regression co-efficient in the equation.

4.4.2.2 Longitudinal Control Performance

Analyses were completed as per the procedures outlined in '*Lateral Control Performance*', where two of the explanatory variables (i.e. limb of the BAC curve and the duration of the driving task) were investigated using separate regression analyses. In this instance, 13 (out of the 22) trials [20, 24-26, 28, 29] indicated ascending or descending BAC at the time of driving and 16 trials [17, 20, 24-26, 28, 29] adequately described the duration of the simulated driving task.

The influence of BAC level and driving simulator platform were controlled in the modelling of the relationship between the limb of the BAC curve and the magnitude of the weighted mean effect. One trial [20] in this analysis yielded a larger Cook's Distance value than all others (i.e. Cook's $d = 1.4$, where all other analyses yielded values ≤ 0.18), it was therefore omitted from the current analysis due to potential confounding effects. Removing this trial revealed collinearity of driving simulator platform and the limb of the BAC curve (both yielded a VIF of 6.7). To account for this, these variables were investigated using separate regression analyses, where BAC level was controlled in both models. Findings indicate that the magnitude of the SDSP performance change is not significantly influenced by driving simulator platform ($p = 0.349$) or the limb of the BAC curve ($p = 0.610$).

Modelling of the remaining variables (Fig. 8) revealed a significant effect of BAC ($p = 0.019$) on the change in SDSP, whereby higher BAC levels were associated with a larger increase in SDSP following alcohol consumption ($R^2 = 0.96$). This model also accounted for drive duration effects. Whilst this variable did not significantly influence longitudinal performance ($p = 0.697$), it assisted to improve the precision of the present regression model (Note: improvement was relative to a simple meta-regression of BAC level vs. magnitude of the weighted mean effect, where trials failing to report drive duration were removed, i.e. indicating that the inclusion of drive duration was indeed beneficial, and confirming that the effect was not a consequence of omitting trials that failed to report drive duration).

Fig. 8. Correlation between change in BAC level ($\text{mg}\cdot\text{dL}^{-1}$) and change in SDSP (difference in means) controlling for drive duration (min). Circle diameter corresponds to the weight of each study. Difference in means = $-0.472[\pm 0.390] + 0.019[\pm 0.007] \times \text{BAC} (\text{mg}\cdot\text{dL}^{-1}) - 0.003[\pm 0.007] \times \text{Drive Duration (min)}$. Square brackets are used to indicate the SEM of each regression co-efficient in the equation.

4.5 Acute Alcohol Consumption vs. 'No Alcohol' Control

Thirteen trials ($n = 241$ participants, 81% male) derived from 7 publications used a *no alcohol* comparator condition to measure the influence of acute alcohol consumption on SDLP (12/13), SDSP (3/13), LC (2/13) and speed (4/13). Characteristics of included studies are summarized in Table 2b. Meta-analyses were not performed on the majority of research outcomes because: (1) too few trials measured LC and SDSP; and (2) the majority of

speed data were derived from a single publication (3/4), increasing the risk of data dependency. The mean correlation coefficient for SDLP was calculated using raw performance data from 5 trials ($R = 0.829$) [20, 44]. All values presented are mean $\pm$ SEM.

4.5.1 Meta-Analyses

Two trials [61] reporting on SDLP were excluded from the current analysis because the mean $\pm$ SD performance score lacked precision. In this study, mean SDLP was reported to the nearest one-tenth of a meter, however the analyses completed in the present review using a ‘placebo alcohol’ control (see ‘*Acute Alcohol Consumption vs. ‘Placebo Alcohol’ Control*’ above for full details) indicate data to the nearest centimeter may be required to detect a small change in SDLP. The small cohort of included trials were not appropriate for regression analysis, therefore the weighted mean effect was calculated as the standardized difference in means. The weighted mean effect summary indicates acute alcohol consumption (BAC range: 33 – 110 mg·dL⁻¹) has a small but significant effect to increase SDLP (standardized difference in means = 0.23, 95% CI: 0.06, 0.39) (Fig. 9). High heterogeneity was evident between trials ($I^2 = 74.7$, $p < 0.001$). The magnitude and statistical significance of the treatment effect was stable during sensitivity analysis where trials were sequentially removed, with the mean difference ranging between 0.17 – 0.26 (95% CI’s did not include zero). Findings are comparable across different levels of correlation, thus the meta-analysis is robust to the imputed correlation coefficient (full details of sensitivity analyses are presented in Appendix Table 5e).

Fig. 9. Forest plot displaying the effect of acute alcohol intake (BAC range: 33 – 110 mg·dL⁻¹) on the standardized difference in mean SDLP. Size of the squares are proportional to the weight of the study.

5. Discussion

This is the first meta-analysis to examine the effects of acute alcohol consumption on measures of simulated driving performance. Results demonstrate that lateral (SDLP and LC) and longitudinal (SDSP) vehicle control measures are sensitive to the effects of acute alcohol consumption. The results of this study clarify the magnitude of the effect that alcohol intoxication elicits on these specific simulated driving performance variables. This information will assist future researchers when interpreting performance changes observed with simulated driving, allowing direct comparison to an established driving impairment.

The present meta-analyses detected a significant increase in SDLP following acute alcohol consumption, compared to both placebo (+4.0 cm) and 'no alcohol' ($ES=0.23$) control conditions. In addition, a significant increase in number of LC and SDSP was also evident following acute alcohol consumption, compared to placebo conditions. In contrast to SDLP, the mean magnitude of effect for LC and SDSP was relatively small. Indeed, one could question the clinical significance of a +0.24 increase in number of lane boundary crossings and a +0.38 $\text{km}\cdot\text{h}^{-1}$ deviation in speed over the duration of a simulated drive. Average driving speed was unaffected by acute alcohol consumption. Collectively, these results are consistent with previous reports that SDLP is a more sensitive indicator of sedative drug-induced driving impairment than other driving performance variables [14, 17, 19], and support its use as a performance outcome when examining drug-induced simulated driving impairment.

Results from the meta-regression analyses in the present study demonstrate that, when using a car-based simulator, acute alcohol consumption causes an increase in SDLP, starting from as low as 21 $\text{mg}\cdot\text{dL}^{-1}$ BAC, with a 0.7 cm increase in SDLP for every 10 $\text{mg}\cdot\text{dL}^{-1}$ increase in BAC thereafter. Previous studies also indicate that impairment in driving-related skills can begin at very low BACs (10 – 20 $\text{mg}\cdot\text{dL}^{-1}$ BAC) [1, 3]. Likewise, a recent report states that the risk of motor vehicle crashes increases with a BAC as low as 30 $\text{mg}\cdot\text{dL}^{-1}$ [4]. Collectively, these results suggest that detectable alcohol-induced driving impairments are likely to commence at levels below that of many drink-driving enforceable limits (~50 – 80 $\text{mg}\cdot\text{dL}^{-1}$ BAC). While it is not possible from the current analysis to translate these small changes in SDLP (at the lowest BAC levels) into crash risk, the results appear to support a threshold BAC of 50 $\text{mg}\cdot\text{dL}^{-1}$ as the preferred enforceable drink-driving limit. This conclusion is based on the regression model suggesting that impairment at 50 $\text{mg}\cdot\text{dL}^{-1}$ BAC results in a mean difference in SDLP of 2.0 cm (a level below what might be clinically relevant [39]). It is important to acknowledge, however, that these results are based on a single outcome measure (SDLP), using simulated driving, which limit their translation to on-road driving.

The present analysis indicates that 80% of the total variance in SDLP results can be explained by the linear relationship between BAC level and the simulator platform used. Car-based systems are more complex simulators than PC-based systems and typically have a wider horizontal field of view, real instrument displays and driver controls with kinesthetic feedback [45, 46]. Complex simulators may allow greater vehicle control that assists with maintaining a constant lane position during a drive, thereby reducing lane deviation. These results suggest that low complexity simulators (PC-based systems) may exaggerate the effect of acute alcohol consumption on SDLP and care should be taken when interpreting raw data where a low complexity simulator has been employed. However, reporting results using standardized mean differences for SDLP appears to remove any significant influence of driving simulator platform. Thus, for researchers with a need to employ PC-based, low complexity systems, reporting SDLP measures as standardized mean differences will allow direct comparison of results to other studies, where the total variance can be explained by BAC level.

The duration of the driving task and the limb of the BAC curve (ascending or descending) did not significantly influence vehicle lateral control performance in the regression analyses. Intoxication effects produced by alcohol may change across the time course of the BAC curve due to alcohol's proposed biphasic actions [63, 64]. Previous reports suggest that some aspects of cognitive and motor performance (e.g. response speed and accuracy, exaggerated steering responses to hazards) exhibit greater impairment during the ascending (acute tolerance) or the descending (acute protracted error) phases of the BAC curve [63-65]. Likewise, it has been suggested that SDLP performance is likely to deteriorate as the duration of a driving task increases, resulting from lapses in vigilance [39]. However, results from the present study suggest that SDLP can indicate driving impairment following acute alcohol consumption when simulated driving tasks are shorter (~5 mins) or longer (60 mins) duration tasks. Thus, the effect of acute alcohol consumption on SDLP during simulated driving tasks appears to be independent of task duration and alcohol's proposed biphasic effects.

Whilst BAC level and simulator platform account for the majority of the total variance between studies in SDLP, at least 20% of the variance remains unexplained. Heterogeneous study methodologies likely contribute to this, particularly where driving scenario design (e.g. environmental stimuli, road type), duration of the driving tasks and alcohol administration procedures (e.g. timing of consumption, type of alcoholic beverage) differ. Unfortunately, few studies provide sufficient level of detail regarding the driving scenarios (e.g. the complexity of the virtual environment, the number and radii of roadway curves, and the width of the roadway) to allow replication or comparison. In particular, road width and curvature are likely to have a significant influence on lane positioning and lane keeping, defining permissible space in which the driver can travel and the need to

make steering adjustments to negotiate bends in the roadway. Simulated driving tasks should employ roadways that use class-appropriate design speeds, lane widths, shoulder and median treatments, markings and signage [66]. More importantly, standardized reporting of driving simulation scenarios and experimental procedures are required to allow a better understanding of the effect these factors have on SDLP.

Several other limitations of the literature examining the impact of acute alcohol consumption on simulated driving performance have been identified in this review. The majority of studies have been conducted with a younger driver demographic (aged <35 years), where factors such as driving experience and alcohol drinking history may influence results. Many studies also do not report all of the data captured by driving simulation software (e.g. many studies reporting SDLP do not provide LC data). There is a need for researchers to provide comprehensive performance data recorded in driving simulations to allow a better understanding of alcohol's effects on all aspects of driving performance. Few studies include a 'no alcohol' control (i.e. no placebo) condition as part of the methodological design, rather comparing alcohol's effects against an alcohol placebo. Evidence suggests that behavioral and cognitive responses to alcohol may be mediated by the expectancy of alcohol consumption [67]. Thus, it is possible that individuals compensate for alcohol induced deterioration in performance if they are aware of consuming or expect to consume alcohol [68]. Results that report differences between alcohol and placebo conditions may underestimate the true effect of alcohol on driving performance measures. Furthermore, individuals are acutely aware when they have consumed alcohol in real world settings, thus comparisons made against a placebo beverage reduce the ecological validity of driving based research. As such, future studies should include a 'no alcohol' control or baseline drive to allow direct comparison of alcohol induced deteriorations in driving performance against a known 'no alcohol' condition. Whilst the present study intended to measure the isolated effects of alcohol ingestion on simulated driving performance, increasingly drivers are engaging in secondary tasks (e.g. using a mobile phone, eating) [69], which may also occur under the influence of alcohol. The combined effects of alcohol and distracted driving are likely to exacerbate the impairment observed. Thus, future studies should examine these combined effects.

It is also important to recognize the overall limitations of driving simulators in research. While several studies have demonstrated direct translation of simulator performance to on-road driving [10-13], results of simulator studies may not generalize to driving in the real world if the simulator used lacks behavioral validity [70]. In fact, driving simulators appear to provide relative validity rather than absolute validity. That is, they approximate the effects observed in on-road driving, but with directional similarities [70]. Therefore, it is important to acknowledge that the driving performance values obtained in the present meta-analysis can not be

translated directly into on-road values to gauge crash risk. Thus, despite the present study identifying SDLP as a sensitive measure of acute alcohol impairment in simulated driving, this does not necessarily mean it is the most appropriate metric for driving safety. Clearly, there is a need for future work to advance driving simulation by exploring potential measures of crash risk within a simulated environment. Regardless, there is sufficient evidence supporting use of driving simulators as a valid tool to assess driving performance, particularly vehicle control parameters such as speed and lateral position [70].

Overall, this systematic review and meta-analysis supports the use of SDLP as a sensitive performance measure of alcohol-impaired driving in simulator studies. Furthermore, the results of this study clarify the magnitude of alcohol's effect on SDLP across various complexities of driving simulator, alcohol intoxication levels and driving duration. This information provides an established driving impairment value for SDLP that can be used as a direct comparator for future studies examining the effects of alcohol and other factors that impair simulated driving performance.

Table 2a. Characteristics of research studies examining the effect of alcohol consumption on simulated driving performance (relative to a placebo control)

Citation [ref]	Participants; Age (years, Mean ± SD)	Drive Duration, LW (m) and Driving Simulator Platform (Car vs. PC)	Timing on the BAC Curve	BAC (mg·dL ⁻¹) (Mean ± SD)			Performance Change (Mean ± SE) (Trials are listed in alphabetical order a – f downwards)			
				Target Level	Onset of Driving	Completion of Driving	SDLP (cm)	SDSP (km/h)	Speed (km/h)	LC (<i>n</i>)
McCartney et al. (2016c – d), Australia [20]	22 M; 23 ± 3 y	S1 (a), 5 min, LW = 4 m (Car) S2 (b), 5 min, LW = 4 m (Car)	Ascending	80	60 ± 10	63 ± 10	1.3 ± 1.5 3.3 ± 1.8	0.2 ± 0.15	0.3 ± 0.3	-0.2 ± 0.6 2.4 ± 1.0
Laude & Fillmore (2016), U.S. [31]	40 (19 M); 24 ± 4 y	15 min (PC)	Ascending	80	63 ± 14	71 ± 13	13.1 ± 2.9		0.9 ± 0.9	
Laude & Fillmore (2015), U.S. [30]	34 (14 M); 21 – 34 y	15 min, LW = 3.7 m (PC)	-	80	62 ± 16	72 ± 13			0.9 ± 0.9	
Kenntnre-Mabiala et al. (2015a – f), Germany [22]	24 (13 M); 30 ± 8.3 y	S1 (a – b), duration NS, LW = 1.75 m (Car) S2 (c – d), 72 min, LW = 3.5 m (Car) S3 (e – f), duration NS (Car)	-	50 (a, c, e) 80 (b, d, f)	58 ± 20 79 ± 20	44 ± 10 77 ± 10	0.0 ± 0.9		2.4 ± 1.2	3.5 ± 0.9
							1.0 ± 0.6		1.6 ± 1.1	3.8 ± 0.8
							3.0 ± 1.1			1.7 ± 0.1
							5.0 ± 1.1			2.2 ± 0.9
							2.0 ± 1.0 3.0 ± 0.9		0.1 ± 0.8 1.2 ± 1.5	0.0 ± 0.0 0.0 ± 0.0
Berthelon & Gineyt (2014a – f), France [29]	16 (8 M); 25 ± 3 y	S1 (a – c), 15 min (Car) S2 (d – f), 15 min (Car)	Descending	30 (a, d) 50 (b, e) 80 (c, f)	33 ± 7 55 ± 6 83 ± 7	23 ± 4 46 ± 8 77 ± 9	-0.5 ± 1.6	0.3 ± 0.23	-0.1 ± 0.5	
							2.0 ± 1.5	0.3 ± 0.24	0.2 ± 0.6	
							5.5 ± 2.0	0.8 ± 0.49	1.8 ± 1.1	
							0.8 ± 1.2			
							1.3 ± 1.5 4.9 ± 1.2			
Weafer & Fillmore (2012a – b), U.S. [23]	20 (10 M); 23 ± 3 y	7.5 min (PC)	(a) Ascending (b) Descending	90	(a) 72 ± 11 (pre-post mean) (b) 74 ± 12 (pre-post mean)		10.0 ± 2.2 5.2 ± 1.9			2.1 ± 1.3 1.5 ± 0.6
Veldstra et al. (2012a – f), Netherlands [27]	17 (9 M); 24 ± 4 y	S1 (a – c), duration NS (Car) S2 (d – f), duration NS (Car)	-	30 (a, d) 50 (b, e) 80 (c, f)	23 ± 5 49 ± 8 80 ± 10	NS	1.9 ± 0.9	0.2 ± 0.26	5.0 ± 2.7	
							2.1 ± 0.9	0.0 ± 0.26	-0.2 ± 2.4	
							4.1 ± 0.9	0.8 ± 0.41	-1.1 ± 2.6	
							0.0 ± 0.23	2.7 ± 4.2		
							-0.2 ± 0.17 0.1 ± 0.23	5.9 ± 4.3 1.3 ± 5.2		
Mets et al. (2011a – c), Netherlands [17]	27 (14 M); 23 ± 1 y	60 min, LW = 3.5 m (Car)	-	(a) 50 (b) 80 (c) 110	50 ± 3 81 ± 3 108 ± 7	33 ± 7 63 ± 9 92 ± 10	1.7 ± 1.2 5.8 ± 1.3 8.3 ± 1.6	0.0 ± 0.25 0.9 ± 0.32 1.4 ± 0.40	-0.3 ± 0.3 0.2 ± 0.5 0.4 ± 0.6	
Marczinski & Fillmore (2009a – d), U.S. [24]	(a – b) 10 (6 M) Non-Binge Drinkers; 23 ± 2 y (c – d) 18 (10 M) Binge Drinkers; 23 ± 3 y	20 min, LW = 3.7 m (PC)	Ascending (a, c) Descending (b, d)	80		(a) 82 ± 8 (pre-post mean) (b) 76 ± 15 (pre-post mean) (c) 75 ± 24 (pre-post mean) (d) 75 ± 22 (pre-post mean)	0.0 ± 3.3 15.3 ± 7.2 12.2 ± 3.0 15.2 ± 4.2	0.2 ± 1.3 4.3 ± 2.0 2.2 ± 0.84 -2.6 ± 1.5		
Marczinski et al. (2008a – b), U.S. [25]	(a) 24 (12 M) Non-Binge Drinkers; 23 ± 2 y (b) 16 (8 M) Binge Drinkers; 22 ± 2 y	20 min, LW = 3.7 m (PC)	Ascending	80	83 ± 15 75 ± 21	91 ± 19 87 ± 22	21.3 ± 4.7 12.2 ± 5.2	3.3 ± 1.5 2.2 ± 1.2	-0.9 ± 1.0 1.0 ± 1.1	
Weafer et al. (2008b – c), U.S. [28]	(a – b) 8 (6 M) 23± 1 y	20 min, LW = 3.7 m (PC)	Ascending	(b) 50 (c) 80	43 ± 19 48 ± 20	56 ± 10 79 ± 10	6.1 ± 3.7 6.1 ± 4.5	0.2 ± 0.30 0.4 ± 0.30		
Fillmore et al. (2008), U.S. [21]	14 (7 M) 24± 3 y	7.5 min (PC)	-	80	86 ± 19	87 ± 10	5.2 ± 3.3			1.5 ± 0.9
Rupp et al. (2007b), U.S. [26]	29 (9 M) 23± 1 y	15 min (PC)	Descending	50	41 ± 13	NS		1.0 ± 0.93		

Table 2b. Characteristics of research studies examining the effect of alcohol consumption on simulated driving performance (relative to a no alcohol control)

Citation [ref]	Participants; Age (years)	Drive Duration and Driving Simulator Platform (Car vs. PC)	Timing on the BAC Curve	BAC (mg·dL ⁻¹) (Mean ± SD)			Performance Change (Mean ± SE) (Trials are listed in alphabetical order a – f downwards)			
				Target Level	Onset of Driving	Completion of Driving	SDLP (cm)	*SDSP (km/h)	*Speed (km/h)	*LC (<i>n</i>)
McCartney et al. (2016a – b), Australia [20]	22 M; 23 ± 3 y	S1 (a), 5 min (Car) S2 (b), 5 min (Car)	Ascending	80	60 ± 10	63 ± 10	-0.8 ± 1.3	-0.1 ± 0.1	-0.1 ± 0.23	-0.7 ± 0.9
							2.7 ± 0.9			
Zhang et al. (2014a – c), China [43]	22 M; 25 ± 4 y	10 min, LW = 3.75 m (Car)	-	(a) 30	33 ± 10	NS	1.0 ± 2.4			7.3 ± 2.7
				(b) 60	64 ± 19		11.4 ± 4.4			6.0 ± 2.7
				(c) 90	92 ± 12		16.9 ± 5.3			4.9 ± 3.0
Leung et al. (2012a – c), Australia [44]	12 (2 M); 26 ± 3 y	20 min (PC)	-	(a) 40	40 ± 2	NS	-0.8 ± 2.4			
				(b) 70	70 ± 2		-0.7 ± 2.8			
				(c) 110	110 ± 2		0.3 ± 2.1			
Weafer et al. (2008a), U.S. [28]	23 (13 M); 22 ± 2 y	20 min, LW = 3.7 m (PC)	-	80	84 ± 18	87 ± 19	12.2 ± 3.3	2.4 ± 1.1		
Rupp et al. (2007a), U.S. [26]	29 (9 M); 23 ± 1 y	15 min (PC)	Descending	50	41 ± 13	NS		1.8 ± 2.7		
Arndt et al. (2000a – b), Canada [48]	22 M; 22 ± 3 y	30 min (PC)	-	80	83 ± 8	78 ± 11	32.0 ± 5.2			
Arndt et al. (2001a – b), Canada [61]	18 M; 20 ± 2 y	30 min (PC)	-	(a) 50 (b) 80						

BAC: blood alcohol concentration; LC: lane crossings; LW = lane width; M: male participants; S: scenario; SDLP: standard deviation of lane position; SDSP: standard deviation of speed.

* The SEM of the performance change was approximated using data from 2 trials [20] investigating LC (all *R*'s as calculated) , 1 trial [20] investigating speed (*R* = 0.759) and 2 trials [20, 26] investigating SDSP (*R* = 0.627). (Calculations for SDLP are described under '*Acute Alcohol Consumption vs. 'No Alcohol' Control*' in Results above.

Appendices

Table 3. Secondary tasks administered during the driving assessment.

Citation	Secondary Task Description	Decision	Explanation
Rupp et al. (2011)	<i>“A 3-digit number is displayed for 1 second in the center of the driving background on the computer screen, followed by a 1.4-second inter-stimulus interval. Participants respond to indicate whether the current 3-digit number displayed is 7 less than the preceding number by pressing the right-hand (correct) or left-hand (incorrect) button of the steering wheel as quickly as possible using their thumbs. The computer provides the correct result (previous number minus 7) on 75% of trials, and otherwise, an incorrect number either 6 or 8 less than the previous number.”</i>	Excluded	SDLP, SDSP and LC were calculated across all data points during the drive. Performance on a ‘drive only’ scenario (i.e. no dual task) was also measured within this study (see Rupp et al. (2011a – b) within the present review). A comparison of the ‘drive only’ and ‘dual drive’ scenarios reveals that SDLP, SDSP and LC measured at baseline (i.e. no alcohol) on the ‘dual task’ scenario were 8.5, 22 and 113% greater (respectively) than on the baseline ‘drive only’ scenario. (The statistical significance of this difference was not given). This suggests that this secondary task negatively influenced driving performance.
Liu & Ho (2010a – f)	Task 1: <i>“Upon detecting a change in the appearance of sign, subjects were asked to switch the indicator in the same direction as the sign on display. Four such shape-change detection tasks were programmed into each road section.”</i> Task 2: <i>“When the subjects perceived that the traffic sign was at a 5-s distance away, they said, “Here!” to complete the task. There were 4 such distance perception tasks in each road section. (Both tasks were included in all simulated driving tests).”</i>	Excluded	SDSP was calculated across all data points during the drive; no other parameters (relevant to this review) were assessed. The study did not provide evidence to indicate the task was, or was not, influential on simulated driving performance.
Freydier et al. (2014)	<i>“This number parity identification task required the participants to identify even and odd numbers and to activate the right control of the steering wheel if the target was even or the left control if the target was odd. A three-figure number appeared at 1.5 second to 2.5 second intervals with the duration of 400 ms, in either the central or peripheral visual field of the driver”</i>	Excluded	SDLP was calculated across all data points during the drive. Performance on a ‘drive only’ scenario (i.e. no dual task) was also measured within this study. A comparison of the ‘drive only’ and ‘dual drive’ scenarios reveals a significant main effect of task, whereby SDLP on the ‘dual task’ scenario was significantly greater than on the ‘drive only’ scenario. This suggests that this secondary task negatively influenced driving performance. Note: Data obtained during the ‘drive only’ test were ultimately excluded from the present review due to poor quality (Rosendal score <50%).
Lenné et al. (2003)	<i>“Two small grey squares on the upper left and right corners of the simulator screen contained a red diamond. At specific points along the drive, the diamond was momentarily replaced by a shape resembling a horn. This event occurred 24 times during each drive, and participants responded to this event by depressing the foot pedal.”</i>	Excluded	SDLP was calculated across all data points during the drive. The study did not provide evidence to indicate the task was, or was not, influential on simulated driving performance.

Table 4. Methodological quality assessment summary and Rosendal Score of included studies.

Citation	A clear description of the inclusion and exclusion criteria was provided	The trials were randomized	The method used to generate the random allocation sequence was described	Sample size was justified	Attempts were made to control and/or monitor pre-trial conditions	Design incorporated measures of important baseline variables	There was blinding of all subjects	There was blinding of all investigators involved in trials	Both the method of blinding and the evaluation of the successfulness of blinding were described	Details were provided regarding the inability of a subject to complete study requirements	Statistical methods used to compare groups for primary outcome measure(s), and methods for additional analyses were described	Both point measures and measures of variability for the primary outcome were provided	The results of between groups statistical comparisons were reported for the primary outcome measures	The method used to assess adverse effects was described	Reproducibility of the primary outcome measure(s) was reported	A familiarization was conducted (includes $\text{VO}_{2\text{ max}}$ tests, if comparable to the performance task)	Total Score (%)
McCartney et al. [20]	1	1	1	1	1	1	1	0	1	1	1	1	1	0	1	1	88
Laude and Fillmore [31]	1	0	-	0	1	1	1	0	0	-	1	0	1	0	0	1	50
Laude and Fillmore [30]	1	0	-	0	1	1	1	0	0	-	1	0	1	0	0	1	50
Kenntner-Mabiala et al. [22]	1	1	1	1	1	1	1	1	1	1	1	1	1	1	0	1	94
Berthelon and Gineyt [29]	1	1	1	0	1	1	1	0	0	-	1	1	1	0	0	1	67
Zhang et al. [43]	1	1	0	0	1	1	0	-	-	0	1	1	1	0	0	1	57
Weafer and Fillmore [23]	1	1	0	0	1	1	1	0	0	-	1	1	1	0	0	1	60
Veldstra et al. [27]	1	0	-	0	1	1	1	1	0	1	1	1	1	0	0	1	67
Mets et al. [17]	1	1	0	0	1	1	1	0	1	1	1	1	1	0	0	1	69
Marczinski and Fillmore [24]	1	1	0	0	1	1	1	0	0	-	1	1	1	0	0	1	60
Weafer et al. [28] Study 1	1	0	0	0	0	1	0	-	-	-	1	1	1	0	0	1	50
Weafer et al. [28] Study 2	1	1	0	0	0	1	1	0	0	-	1	1	1	0	0	1	53
Marczinski et al. [25]	1	1	0	0	1	1	1	1	0	-	1	1	1	0	0	1	67
Fillmore et al. [21]	1	0	-	0	1	1	1	0	0	-	1	1	1	0	0	1	57
Lenné et al. [71]	1	0	-	0	1	1	0	-	-	-	1	1	1	0	0	1	58
Rupp et al. [26]	1	0	-	0	1	1	1	0	0	-	1	1	1	0	0	1	57
Arnedt et al. [61]	1	1	1	0	1	1	0	-	-	-	1	1	1	0	0	1	69
Arnedt et al. [48]	1	1	1	0	1	1	0	-	-	1	1	1	1	0	0	1	71
Freydier et al. [60]	1	0	-	0	0	1	1	0	0	-	1	0	1	0	0	0	36
Fillmore and Harrison [58]	1	0	-	0	0	1	1	0	0	-	1	1	1	0	0	0	40
Barkley et al. [59]	1	1	0	0	1	1	1	0	0	-	1	0	1	0	0	0	47

Table 5a. Sensitivity analysis of SDLP (placebo control) using alternative levels of correlation to complete the meta-analysis, where $R = 0.800, 0.550$ (actual) and 0.300 .

R	Difference in means (95% CI)	p value	I^2 index
0.800	4.2 (3.2, 5.2)	<0.001	85.2
0.550	4.0 (3.0, 5.1)	<0.001	75.8
0.300	3.8 (2.8, 4.9)	<0.001	68.2

Table 5b. Sensitivity analysis of SDSP (placebo control) using alternative levels of correlation to complete the meta-analysis, where $R = 0.800, 0.608$ (actual) and 0.300 .

R	Difference in means (95% CI)	p value	I^2 index
0.800	0.43 (0.23, 0.62)	<0.001	64.7
0.608	0.38 (0.19, 0.57)	<0.001	45.0
0.300	0.33 (0.15, 0.50)	<0.001	16.2

Table 5c. Sensitivity analysis of speed (placebo control) using alternative levels of correlation to complete the meta-analysis, where $R = 800, 0.428$ (actual) and 0.300 .

R	Difference in means (95% CI)	p value	I^2 index
0.800	0.66 (0.25, 1.08)	0.002	55.5
0.428	0.29 (-0.005, 0.58)	0.054	0.0
0.300	0.29 (-0.017, 0.60)	0.064	0.0

Table 5d. Sensitivity analysis of LC (placebo control) using alternative levels of correlation to complete the meta-analysis, where $R = 800, 0.700$ (actual) and 0.300 .

R	Difference in means (95% CI)	p value	I^2 index
0.800	0.00 (-0.00, 0.00)	0.999	85.4
0.700	0.00 (-0.00, 0.00)	0.999	83.0
0.300	0.00 (-0.00, 0.00)	0.999	74.7

Table 5e. Sensitivity analysis of SDLP ('no alcohol' control) using alternative levels of correlation to complete the meta-analysis, where $R = 0.889$ (actual) and 0.300 .

R	Standardized difference in means (95% CI)	p value	I^2 index
0.889	0.23 (0.06, 0.39)	0.007	74.7
0.300	0.23 (0.03, 0.43)	0.022	60.6

Table 6. Sensitivity analyses of SDLP, SDSP, Speed and LC (placebo alcohol control) and SDLP (no alcohol control) to determine risk of data dependency. Analyses included trials that yielded the median effect of alcohol ingestion, compared to other trials derived from the same publication. All other trials were excluded from these analyses.

Outcome	Difference in means (95% CI)	p value	I^2 index
Placebo Alcohol Control			
SDLP	5.5 (3.75, 7.21)	<0.001	74.4
SDSP	0.37 (0.11, 0.62)	0.005	40.4
Speed	0.40 (-0.01, 0.81)	0.052	0.0
LC	1.4 (0.72, 2.00)	<0.001	6.8
No Alcohol Control			
SDLP	0.30 (0.07, 0.52)	0.010	76.6

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