

2020-08-12

The Effects of Cannabis and Alcohol on Driving Performance and Driver Behaviour: A Systematic Review and Meta-Analysis

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Simmons, S. M. (2020). The effects of cannabis and alcohol on driving performance and driver behaviour: a systematic review and meta-analysis (Doctoral thesis, University of Calgary, Calgary, Canada). Retrieved from <https://prism.ucalgary.ca>.

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The Effects of Cannabis and Alcohol on Driving Performance and Driver Behaviour:

A Systematic Review and Meta-Analysis

by

Sarah Michelle Simmons

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES

IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE

DEGREE OF DOCTOR OF PHILOSOPHY

GRADUATE PROGRAM IN PSYCHOLOGY

CALGARY, ALBERTA

AUGUST, 2020

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Abstract

Cannabis is the most frequently used drug in the world, and it is commonly detected in fatal crashes. Epidemiological research indicates that cannabis is associated with an increase in crash risk, but the mechanisms underlying this association remain unclear. The objective of the current systematic review and meta-analysis is to provide insight into these mechanisms by synthesizing experimental research focused on the effects of cannabis on driving performance and behaviour. Additionally, the experimental literature focused on the effects of alcohol on driving performance and behaviour is synthesized for comparative purposes. The four key aims of this dissertation are to (1) quantify the magnitude of the effect of cannabis on driving performance and behaviour; (2) compare the influence of cannabis to that of alcohol; (3) assess the effect of the combination of cannabis and alcohol on driving performance and behaviour; and, (4) identify knowledge gaps and quality limitations in the extant literature to direct the conduct of high quality research in the future. Academic Search Complete, CINAHL, Embase, Scopus, MEDLINE, PsycINFO, SportDISCUS and TRID were systematically searched in May 2018. Driving performance and behaviour data from experimental driving studies involving healthy participants of any age and sex collected in driving simulator, closed-course and on-road studies involving cannabis and/or alcohol administration, published in any language, were eligible for inclusion. Of 120 eligible studies, 81 were ultimately included in the meta-analysis. Most notably, cannabis was associated with impaired lateral control and decreased driving speed. Alcohol was associated with a variety of driving performance decrements and increased driving speed. The combination of drugs was associated with greater driving performance decrements than either drug in isolation. Finally, indirect comparisons indicated that the effects of cannabis on experimental driving measures were generally similar to low blood alcohol concentrations.

However, imprecision in effect size estimates limits interpretation, and more research in the area is needed. Future research directions and quality recommendations are identified and described to aid in this endeavour. Nonetheless, the meta-analysis indicates that cannabis, like alcohol, impairs driving, and the combination of the two drugs is more detrimental to driving performance than either in isolation.

Keywords: cannabis, marijuana, alcohol, impaired driving, driving under the influence, driving performance, driver behaviour, experimental driving studies, driving simulator, simulated driving, meta-analysis, systematic review, research synthesis

Acknowledgements

There are many individuals to thank for their contributions to this work. First, this research would not have been possible without the support of several individuals at the University of Calgary Library. Thank you to Associate Librarian Laura Koltutsky for guidance related to database selection, electronic search strategy development and study screening. I am also indebted to DDS Specialists Lorraine Baker, Kathleen James, Glenda Magallon, Kathy McDonnell, Lana Wong and Judy Zhao, who processed my (many) interlibrary loan requests.

Next, I am deeply grateful for the work of many volunteers. Thank you to Franci Sterzer, Dongzhao Shelley Liu, Gabby Janikian, Louisa Krile and Adam Turner, who volunteered their time to assist with abstract screening, full-text reviews and study quality and risk of bias assessments, and to Liz Suessenbach, Qing Li, Kari Smedstad, Caitlin Lang Faerevaag, Mélanie Paulin and Chelsea Hart, who volunteered their time to assist in reviewing the non-English studies for content.

I would like to thank the researchers who responded to my requests for clarification, elaboration and data related to their work. In particular, I want to recognize Thomas Arkell, Mark Fillmore, Arne Helland, Danielle McCartney, Nicola Starkey and Mark Vollrath, who provided missing data.

Many thanks to my supervisor, Dr. Jeff Caird, for providing mentorship, guidance and unparalleled support over the years, and to my supervisory committee, including Drs. Tom O'Neill, Piers Steel, Fiona Clement, Brent Hagel and Mark Asbridge, for providing guidance, advice and feedback on this project. Additionally, I would like to thank Dr. Michael Borenstein for volunteering time to provide technical advice.

Finally, I would like to acknowledge and thank my friends in the Department of Psychology at the University of Calgary including Devon Currie, Clark Amistad, Tim Wingate, Maria Pavlova, Kirsti Toivonen, Emilie Lacroix, Chelsea Hart and Alberto Umiltà – all of whom I credit with making my time in the doctoral program the most enjoyable and rewarding period of my life – for their friendship and support. I am also eternally grateful for the encouragement and support of my family at home in Saskatchewan.

This research received financial support from a URGC SSH Faculty Seed Grant. The funder did not have a role in the design of the study, its execution, its reported findings or publication.

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Chapter 1: Introduction

Cannabis is the most commonly-used drug in Canada (Statistics Canada, 2018) and in the world (United Nations Office on Drugs and Crime, 2019). Recently, cannabis has received increased attention as a contributor to motor vehicle crashes after efforts have been made to decriminalize or legalize the drug for medical and recreational use in countries across the world. Cannabis refers to both the cannabis plant (i.e., *cannabis sativa*) and to preparations made from it, including marijuana (or marihuana), hash, resin and oil (Ashton, 2001). The most prominent component of cannabis is delta-9-tetrahydrocannabinol (THC), which is the chief cannabinoid responsible for the cannabis high (Ashton, 2001; Huestis, 2007) and thus is most relevant to discussions and empirical work focused on cannabis-involved driving. As of October 2018, two common cannabis preparations – namely dried marijuana and cannabis oil – are legal for recreational use in Canada, and as of October 2019, edible cannabis products are also legal for recreational use (Government of Canada, n.d.-a). Given the characteristics of the typical cannabis high (e.g., changes in alertness, mood, perception, motor skill, memory and attention) (Ashton, 2001; Broyd et al., 2016; Grotenhermen, 2003; Hall & Solowij, 1998), and increased availability of the drug, there is a natural concern that these changes will have consequences for road safety.

Early evidence suggests that since the legalization of cannabis for recreational purposes in Canada, there have been no significant changes in rates of self-reported driving within two hours of using the drug (Rotermann, 2020). However, driving under the influence of cannabis (DUIC) is already relatively common. In 2019, 26% of respondents to the Canadian Cannabis Survey who had used cannabis in the previous 12 months indicated that they had ever driven a vehicle within two hours of smoking or vaporizing cannabis, and 16% indicated that they had ever driven a vehicle within four hours of ingesting cannabis (Government of Canada, n.d.-b). Of

those respondents, the prevalence of driving within two hours of smoking or vaporizing cannabis was 31%, and the prevalence of driving within two hours of ingesting cannabis was 39%, in the previous 12-month period (Government of Canada, n.d.-b). Additionally, cannabis is the most commonly implicated drug, with the exception of alcohol, in crashes (Compton & Berning, 2015). The bulk of the epidemiological evidence indicates that cannabis increases the risk of crashing by about two-fold (Asbridge et al., 2012; Li et al., 2012; Elvik, 2013; Rogeberg et al., 2018). However, as will be discussed in more detail later in this chapter, the epidemiological evidence does not allow inferences to be made about the precise mechanisms that link cannabis to an increased risk of crashing. The experimental literature, which can provide insight into that mechanism, suffers from a lack of standardization in operational definitions and measures of impairment. Unresolved discord in the scientific literature about operational definitions and measures needlessly generates mixed messages about the safety of cannabis with respect to driving. It is unsurprising that cannabis-using individuals in Canada have varying opinions on whether the drug is detrimental to driving ability (e.g., Government of Canada, n.d.-b).

The primary objective of the current systematic review and meta-analysis is to synthesize the available literature on the effects of cannabis on driving performance and behaviour as measured in experimental studies. Additionally, a synthesis of the experimental literature focused on the effects of alcohol on driving performance and behaviour is incorporated. Data from healthy participants of any age and sex collected in driving simulator, closed-course and on-road studies involving cannabis and/or alcohol administration were eligible for inclusion. There are four essential research goals of the current study: (1) to quantify the magnitude of the effect of cannabis on driving performance and behaviour; (2) to compare the influence of cannabis to that of alcohol; (3) to assess the effect of the combination of cannabis and alcohol on

driving performance and behaviour; and, (4) to identify knowledge gaps and quality limitations to direct the conduct of productive, high-quality scholarly inquiry focused on cannabis- and alcohol-involved driving in the future. In addition, experimental driving study measures in the current study are selected based on theoretical considerations, and an operational definition of impairment – based on those theoretical considerations – is offered. Thus, the overarching goal of the current study is not only to make sense of discordant findings within the literature, but to also rectify conceptual disagreements related to the measurement of impairment. Disagreements between the influence of cannabis on driving, stemming from ambiguity in the literature, must be resolved to the benefit of researchers interested in advancing the field focused on drug-involved driving and to the benefit of real-world, cannabis-using drivers seeking to make informed decisions about safe driving practices.

This chapter is organized as follows. First, the epidemiological evidence focused on the crash risk associated with cannabis is reviewed and critiqued. Second, the need to consult the experimental literature is discussed. Third, previous meta-analytic work focused on the experimental literature, and in particular the limitations of that work, are examined. Finally, the research questions of the current study are considered, and the hypotheses are listed.

Driving Under the Influence of Cannabis: A Theoretical Perspective

The bulk of the epidemiological evidence indicates that cannabis has a negative effect on traffic safety. A number of meta-analyses have examined the relationship between a positive test for cannabis and crash risk, and most (Asbridge et al., 2012; Li et al., 2012; Elvik, 2013; Rogeberg et al., 2018), but not all (Hostiuc et al., 2018) indicate that cannabis is associated with an increase in crash risk. Although estimates vary somewhat from analysis to analysis, increased

crash risk estimates range from a less than doubling of crash risk (Asbridge et al., 2012; Elvik, 2013; Rogeberg et al., 2018) to a more than doubled crash risk (Li et al., 2012).

The increase in crash risk associated with cannabis has important implications for public health. In addition to immediate injuries and fatalities, crashes can have long-term health consequences for survivors in the form of disability (Krug et al., 2000). Worldwide, the most common injuries that occur in crashes leading to disability are fractures of the patella, tibia, fibula, and ankle; the second leading cause of disability is traumatic brain injuries (James et al., 2020). However, even less severe crashes that do not result in traumatic brain injuries can have long-lasting consequences: Fitzharris and colleagues (2007) report that even “otherwise healthy people of working age involved in a traffic crash, with the absence of moderate-severe head injury and spinal cord injury” (p. 311) – who, they report, comprise a large portion of individuals admitted to hospitals in Victoria, Australia due to crashes – may experience long-term detriments to quality of life, including enduring pain, difficulties performing daily activities, and diminished physical and mental health. In Canada, cannabis has been estimated to have led to 75 deaths and 4,407 injuries in crashes in the year 2012 (Wettlaufer et al., 2017). An earlier study reported that road traffic injuries involving cannabis are estimated to have led to 94 deaths, 4,481 years of life lost due to premature mortality and 364 years of life lost due to disability in Canada in the same year (Imtiaz et al., 2015).

To understand the association between cannabis and increased crash risk, researchers must consult more than just the epidemiological evidence. The full scope of literature focused on the effects of cannabis and driving must be considered. However, different study methods and measures have different benefits and limitations, yield different types of information, and are of varying degrees of usefulness with respect to understanding the mechanisms underlying the

relationship between cannabis and increased crash risk. Decisions about which evidence to consult, and how to interpret that evidence – particularly when assessing the degree to which evidence converges – requires a theoretical foundation for analysis. In this dissertation, Fuller’s (2005) Task-Capability Interface (TCI) model of driving provides this foundation. The model is illustrated below.

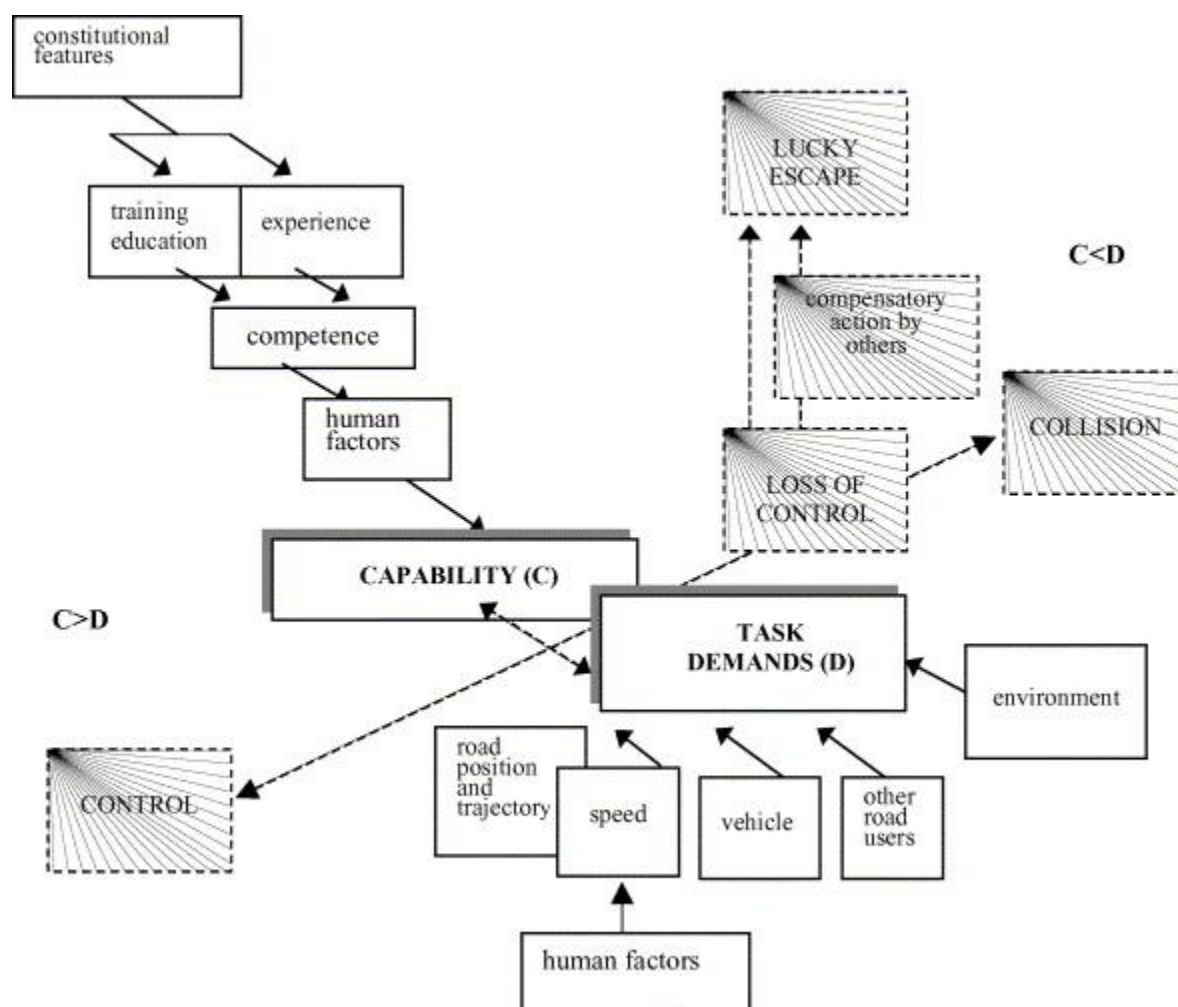


Figure 1. Fuller's (2005) Task-Capability Interface (TCI) model of driving¹.

¹ Reprinted from Accident Analysis & Prevention, Vol. 37, Fuller R., Towards a general theory of driver behaviour, Page No. 465, Copyright 2005, with permission from Elsevier.

First, the mechanisms by which cannabis increases crash risk can be predicted by consulting Fuller's (2005) model. Fuller (2005) suggests that drivers attempt to achieve a comfortable range of effort expenditure or workload while driving, and the degree of workload in a given situation is determined by the discrepancy between the *Capability* of the driver and driving *Task Demands*: the greater the discrepancy, the easier the driving task feels, and the lesser the discrepancy, the more difficult the driving task feels. According to Fuller (2005), *Capability* is "initially constrained by biological characteristics of the driver, such as information processing capacity and speed, reaction time, physical reach, motor coordination and perhaps flexibility and strength" (pp. 463-464) and enhanced with knowledge and skills attained through training, education and experience. However, *Capability* can be momentarily diminished by transient *Human Factors*, including "attitude, motivation, effort, fatigue, drowsiness, time-of-day, drugs, distraction, emotion and stress" (Fuller, 2005, p. 464). On the other side of the model are *Task Demands*, which are determined based on the driving *Environment*, *Other Road Users*, the "operational features" of the driven *Vehicle*, its *Road Position and Trajectory*, and its *Speed*. Fuller (2005) suggests that speed is the greatest determinant of task difficulty: "it is self-evident that the faster a driver travels, the less time is available to take information in, process it and respond to it" (p. 464). Thus, when *Task Demands* increase, drivers attempting to keep their workload within a comfortable range will likely slow down to reduce *Task Demands*; failing this, workload will increase, and the driving task will feel more effortful and more uncomfortable (Fuller, 2005). Left unchecked, at some point in time the driving task may start to feel dangerous (Fuller, 2005). Based on the model, crashes – including those that involve cannabis – occur when driving *Task Demands* overwhelm a driver's *Capability* (Fuller, 2005).

Thus, two mechanisms of action are readily apparent in Fuller's (2005) model that may prevent a driver from meeting the demands of the driving task while intoxicated by cannabis. First, cannabis may work to diminish the *Capability* of the driver through its influence as a transient *Human Factor*. Specifically, the state of acute cannabis intoxication may serve to diminish a driver's ability to retain control over the driving task and consequently increase crash risk. Based on the crash risk meta-analyses discussed above, this would suggest that cannabis intoxication is associated with an approximate doubling in crash risk. However, owing to the complicated pharmacokinetics and pharmacodynamics of cannabis, this may not necessarily be the case; although the increase in crash risk associated with testing positive for cannabis is commonly interpreted to mean the increase in crash risk associated with *driving while high* (e.g., Asbridge et al., 2012), a positive test for cannabis does not indicate that an individual was intoxicated or under its influence at the time of the test (Ashton, 2001; Huestis, 2007). The inability to know for certain whether a driver who tests positive for a drug was actually under the influence of the drug is a known issue in studies that seek to quantify crash risks associated with drugs (Compton & Berning, 2015; Gjerde et al., 2019).

Additionally, Fuller's (2005) model also points to a second contributor to losses in control – specifically, the *Constitutional Features* of the driver. Through this mechanism, individuals who use cannabis and drive may possess qualities that independently place them at an elevated risk of crashing. The existence of such a mechanism is known among alcohol-involved drivers. For example, Evans (2004) reports that increases in crash severity, which are a product of increased speed and risk-taking, occur with incrementally higher blood alcohol concentrations. Evans (2004) reasons that because of this, there must be an incrementally greater contribution of risk-taking (i.e., a *Constitutional Feature* of the driver) in crashes with

incrementally higher implicated blood alcohol concentrations (BAC). Just as with alcohol, it is possible and has been speculated that there are underlying characteristics of individuals who use cannabis and drive that increase their risk of crashing independently of acute cannabis intoxication (e.g., Walsh & Mann, 1999; Rogeberg & Elvik, 2016). However, although it is possible to stratify the crash risk and crash severity associated with alcohol across multiple levels of intoxication (i.e., multiple BAC levels) to glean insight into the contribution of risk-taking to alcohol-involved crashes, the same cannot be done with cannabis. This is because, as previously discussed, THC concentration is not a reliable indicator of cannabis intoxication.

Overall, epidemiological studies indicate that cannabis is associated with an increased risk of crashing, but they are limited in that they do not indicate why. Theoretically, two distinct but non-mutually exclusive mechanisms exist: (1) the acute state of cannabis intoxication diminishes driver *Capability*, leading to difficulties meeting driving *Task Demands*, and/or (2) drivers involved in cannabis-involved crashes belong to a special subpopulation of drivers predisposed to crashing. To investigate the contribution of acute cannabis intoxication to crashes, the experimental literature – wherein researchers attempt to place participants in a state of acute cannabis intoxication – must be consulted. The experimental literature focused on the cannabis is discussed next.

Cannabis, Driving Performance & Driver Behaviour

Experimental driving studies complement epidemiological studies focused on quantifying crash risk. They measure elements of drivers' abilities and behaviours, and their purpose is to provide insight into how drivers will perform in the real world (Caird & Horrey, 2011; Mullen et al., 2011). In surveying the literature, however, it is clear that some experimental measures offer more insight than others when it comes to evaluating the extent to which acute cannabis

intoxication affects driving. There are an abundance of tasks and measures used to make inferences about drivers' abilities and behaviours, many of which have ambiguous relationships with safety. The answer to the question, "does cannabis impair driving?" depends entirely on which measures are selected, so it is prudent to select the most defensible measures in answering the question. Given that crashes are of paramount importance to traffic safety, and experimental driving studies are limited in that they cannot measure them directly (Irwin et al., 2017; Gjerde et al., 2019), experimental driving study measures used to answer the impairment question should at the very least have a solid theoretical relationship with crashes. Fuller's (2005) model provides a useful theoretical framework for the selection of measures and the interpretation of the effects of cannabis on those measures with respect to traffic safety. Additionally, the framework allows for hypotheses to be generated to direct future study.

A number of measures commonly included in experimental driving studies focused on the effects of drugs, alcohol or secondary tasks involving mobile devices have theoretical relationships with safety based on Fuller's (2005) model. These measures, commonly referred to as *driving performance* measures, are related to elements such as hazard or target detection and response, lateral control of the vehicle, longitudinal control of the vehicle, speed and headway. Driving performance is commonly conceptualized as driving skill and ability (Evans, 2004). However, within the context of Fuller's (2005) model, these measures are perhaps more correctly conceptualized not as indicators of *Capability*, but rather – like crashes – as indicators of the difference between *Task Demands* and *Capability*. For example, the greater the degree of lane weaving, the greater the demands of the driving task relative to the ability of the driver to meet those demands. Although, as previously discussed, experimental driving study measures do not translate into crash risk directly, the ability to detect and respond to on-road hazards, keep the

vehicle in the centre of the lane, and maintain control over speed and following distance (i.e., longitudinal control) all have face validity as indicators of safe driving. For example, if a driver is unable to detect or respond to an on-road hazard in a timely manner, a collision with an obstacle, road user or other surface is likely to occur. If a driver is unable to effectively lane keep, a lane departure may occur, leading to a single-vehicle off-road collision, a lane departure into the path of an oncoming vehicle, or general susceptibility to loss of control given less than optimal road conditions. And, if a driver is unable to effectively maintain longitudinal control, a rear-end collision may occur. Thus, for the purposes of the current meta-analysis, experimental driving measures related to the detection and response to on-road hazards, to lane keeping and to longitudinal control are conceptualized as serving as objective, behavioural indicators of the discrepancy between *Task Demands* and *Capability*. Only these measures are referred to in this dissertation as *driving performance* measures. Speed and headway, in contrast, can be conceptualized as elements of what is known in the literature as *driver behaviour*. Whereas driving performance refers to a driver's abilities, driver behaviour refers to *how* individuals go about completing the driving task – or behave – given those abilities (Evans, 2004). Fuller (2005) posits that adjustments to driving speed are the primary means by which drivers regulate the difficulty of the driving task. Reductions in headway (i.e., following distance) may also, in certain circumstances, allow drivers to compensate for heightened task demands (Fuller, 2005). Both decreases of speed and increases in headway are commonly conceptualized as compensatory behaviours within the driving performance literature, even in studies that make no specific reference to any particular theory of driving. For the purposes of the current meta-analysis, speed and headway are conceptualized as measures of *driver behaviour* that reflect adjustments to driving task demands.

Finally, in answering the question, “does cannabis impair driving?” it is critical to consider what should serve as the operational definition of impairment. For the purposes of this meta-analysis, there is a distinction between *intoxication* and *impairment*. Here, intoxication refers to the state of experiencing the acute effects of a substance (i.e., cannabis or alcohol), with no specific reference to the effects of that substance on driving. In contrast, driving performance decrements – that is, changes in hazard detection and response, lateral control or longitudinal control that demonstrate a loss of control (i.e., an inability to meet the demands of the driving task) – are conceptualized as indicators of impairment and impaired driving. Changes in speed and headway, which indicate how drivers alter their behaviour to attempt to meet the demands of the driving task, are not conceptualized as indicators of impairment per se. For example, decrements in lateral control indicate impaired driving regardless of whether a driver also demonstrates slowed driving speed and increased following distance. Although changes in speed and headway do not indicate impairment, they are important because they indicate whether drivers perceive or are aware of increased task demands and whether they attempt to adjust their driving to meet those demands. This is an important consideration within the context of alcohol-involved driving where part of the danger associated with driving under the influence, in addition to temporary loss of skill, is thought to be increased risk-taking (i.e., acceptance of greater task demands) (e.g., Evans, 2004).

Previous Research

The experimental literature focused on the effects of cannabis on driving performance and behaviour is discordant not only in terms of findings, but also in terms of methodological approaches (see Sewell et al., 2009 for a review). This state of affairs indicates a clear need for a systematic review and meta-analysis, which serves to both rectify discordant research findings

and to lay the groundwork for future scholarly inquiry in the area. However, the current study is not the first research synthesis focused experimental driving studies involving cannabis (or alcohol). However, previous research syntheses are fundamentally limited, and the current meta-analysis presents a timely update to the field. Similarities and differences between the current meta-analysis and previous research syntheses are discussed next.

Berghaus, Krüger and colleagues. Some of the most notable and cited meta-analytic work focused on the effects of cannabis on driving was conducted by Berghaus, Krüger and colleagues. In these meta-analyses, eligible studies were required to be experimental, involve the measurement of at least one measure related to driving, involve at least five human participants, administrate only one drug at a time, be written in English or German, report the data needed to estimate the THC concentration associated with a specific effect, and report the data needed for vote-counting purposes (Berghaus et al., 1998a). Overall, cannabis was found to have negative effects on a variety of skills deemed relevant to the driving task (Berghaus et al., 1998a; Berghaus et al., 1998b). These findings were later incorporated into Grotenhermen and colleagues' (2005; 2007) analysis that focused on developing THC limits for applications in drugged driving legislation. Although Berghaus, Krüger and colleagues' meta-analyses were and continue to be influential, they have numerous limitations.

Before these major limitations can be discussed, an important caveat should be noted. A number of papers are linked to Berghaus, Krüger and colleagues' meta-analytic work, but it is unclear which paper contains the most authoritative report. Citations for papers linked with the project often point readers to several more meta-analyses, many of which cite each other. For example, Shinar (2007, pp. 661-663) discusses the results of Berghaus and colleagues' meta-analyses as they appear in a 1998 book (i.e., Berghaus et al., 1998a, 1998b) based on descriptions

provided by secondary sources – Ward and Dye (1999), and Ramaekers et al. (2004) – likely because the 1998 book is written in German. However, those two meta-analyses, after being electronically translated from German into English, were found to refer to several older reports, one of which (Krüger, 1993) is an English language summary that redundantly refers to two of the other cited reports (Krüger et al., 1990; Krüger, 1990). Krüger (1993) – but neither Krüger et al. (1990) nor Krüger (1990) – is referred to in a second English-language summary (Berghaus et al., 1995), which is the original version identified by this author while reviewing the literature. Of all the reports related to the Berghaus meta-analyses discussed thus far, Krüger et al. (1990) appears² to be the only one that lists citations for included studies. Subsequent meta-analyses do not list included (or excluded) research so it not possible to determine the overlap from meta-analysis to meta-analysis. This lack of transparency is concerning. During attempts to identify an original and complete report of their meta-analytic project, over a dozen citations potentially linked to the meta-analytic project were identified.

Given uncertainty about the most authoritative version of the work, and for simplicity's sake, Berghaus et al. (1998a) and Berghaus et al. (1998b), which are the two versions cited in Grotenhermen et al. (2005), are critiqued here. Grotenhermen et al. (2005) is an important policy paper that tries to establish a cutoff for THC blood concentration while driving that is based, in part, on these meta-analyses. The following critiques are based on electronic translations³ of Berghaus et al. (1998a) and Berghaus et al. (1998b), as well as descriptions from two secondary sources: Berghaus et al. (1995), and Grotenhermen et al. (2005). Given that none of these

² Based on a cursory scan of the paper. The report is over 400 pages long and written in German, and given time constraints, it was not translated for inspection.

³ Methods for electronically translating non-English studies are described in *Chapter 2: Method* of this dissertation.

sources offers a comprehensive description of meta-analytic methods, the following critique will be somewhat constrained.

Inclusion of driving-related skills. First, it is difficult to generalize Berghaus and colleagues' (1998a, 1998b) findings to real-world driver behaviour due to their incorporation of *driving-related skills* in their meta-analyses. Purported measures of driving-related skills, which appear to be implicitly operationalized as any laboratory experimental task that assesses any aspect of human information processing, frequently appear in the literature focused on the effects of alcohol and drugs on driving in addition to measures of driving performance and behaviour. Within the experimental literature focused on alcohol- and drug-involved driving, there does not appear to be a generally-accepted, comprehensive list of essential driving-related skills. Berghaus and colleagues focused on “published experimental investigations testing at least one effect of THC connected with the ability of safely driving a vehicle” (as described in Berghaus et al., 1995, p. 404), including tracking, psychomotor control, reaction time, “visual functions,” attention, divided attention, “encoding” and “decoding”, and “simulator driving.” Berghaus et al. (1998a, 1998b) provide examples of tasks that fall into these categories, but the specific criteria used to make judgements about the allocation of experimental tasks to categories are not reported in-text⁴.

Despite their frequent use in studies within the field of research focused on alcohol and drugs, “driving-related skills” do not have a clear relationship with traffic safety (Shinar, 2017, p. 659). Theoretically, there is no clear reason that they should. Within the context of Fuller's (2005) model, driving-related skills can be conceptualized as one of the elements of the driver's

⁴ Readers are instead referred to Krüger (1990), Krüger et al. (1990) and Krüger (1993).

Capability or ability to maintain control over the driving task. Specifically, they align with the *Constitutional Features* of the driver. However, it is generally accepted that drivers do not perform to the best of their abilities all the time; instead, they try to regulate the difficulty of the driving task so that task demands do not exceed their capability (Fuller, 2005). Consequently, driving-related skills and driver behaviour are inescapably inter-related, and it is not possible to infer the point at which a driver, suffering driving-related skill decrements, will be incapable of keeping driving task demands in check. The reality is that drivers will, to some extent, experience fluctuations in driving ability over hours or days (e.g., differences in arousal throughout the day, disruptions to sleeping schedules, etc.), which will require varying degrees of adjustment to driving task demands. Furthermore, given variations in driving task demands, certain driving-related skills will likely be more instrumental to a driver's capability than others at varying moments in time. Overall, it does not make sense to focus solely on "the upper limit of the competence of the driver" (i.e., *Capability*; Fuller, 2005, p. 464), and there are serious limitations to making inferences or generalizations about driver safety based on the results of Berghaus and colleagues' meta-analyses. The current meta-analysis differs from Berghaus and colleagues' in that only theoretically-defensible measures of driving performance and behaviour (i.e., products of the interface between *Task Demands* and *Capability*) are included as outcomes of interest, whereas driving-related skills (i.e., measures of *Capability*) are excluded.

Analytical approach. Berghaus et al. (1998a, 1998b) apply a meta-analytic method known as vote-counting. Within categories of driving-related skills, effects were categorized as "significantly deteriorated," "significantly improved" or unaffected (i.e., "no significant effect"; as described in Berghaus et al., 1995). The proportion of significantly deteriorated effects was calculated across multiple THC concentrations (i.e., Berghaus et al., 1998a). Additionally, the

pattern of task performance deterioration across a range of THC concentrations was compared to the pattern of task deterioration across a range of BAC concentrations based on the results of a second, methodologically-similar meta-analysis focused on alcohol (i.e., Berghaus et al., 1998b), which allows “equivalent levels of impairment” to be identified (Grotenhermen et al., 2005, p. 25). There are two important limitations to this approach.

First, vote-counting as a meta-analytic method is fundamentally flawed. Essentially, vote-counting involves making comparisons between the number of statistically significant effects and the number of statistically non-significant effects across studies in order to make judgements about the evidence for the existence of that effect (Borenstein et al., 2009, pp. 251-255).

Berghaus and colleagues’ (1998a, 1998b) took a vote-counting approach by counting and comparing the number of “significantly deteriorated” effects, “significantly improved” effects and “no significant effect[s]” in their included studies. According to Borenstein and colleagues (2009, pp. 251-255), the underlying assumption of the vote-counting approach is that statistically significant effects are evidence of the presence a genuine effect, and statistically non-significant effects are evidence of the absence of a genuine effect. However, these assumptions are not necessarily valid. Borenstein and colleagues (2009, pp. 251-255) criticize vote-counting primarily on the basis that the lack of a significant effect does not necessarily mean that no genuine effect exists. Alternatively, a study may simply have been incapable of detecting an effect due to a lack of statistical power. When underpowered studies that have failed to detect a genuine effect are submitted to a formal meta-analysis rather than a vote-count, the genuine effect may be elucidated, but a vote-count, in contrast, might leave the opposite impression – specifically, that the bulk of the evidence suggests no effect exists (Borenstein et al., 2009, pp. 251-255). For this reason, Borenstein and colleagues (2009) dismiss vote-counting as a method

that “has no validity whatsoever” (p. 325). Given the marginal and underpowered studies that have examined cannabis and driving, a number of studies are likely to fall into the no effect category, which would underestimate the true effect if vote counting is to be believed. In contrast, the inclusion of experimental studies where tasks have no relationship to driving, but may be sensitive to drugs and alcohol, would result in an overestimate of true effects if vote counting is relied upon.

Furthermore, in addition to the issues identified by Borenstein and colleagues (2009), recent work by Ioannidis (2005) and the Open Science Collaboration (2015) highlights the fact that statistical significance does not necessarily indicate the presence of a genuine effect. For example, the Open Science Collaboration (2015) recently reported that after a major collaborative effort to reproduce findings published in top-tier journals in psychology, a substantial proportion were irreproducible. Although spurious effects can be published in any scientific literature, the prevalence of potentially spurious effects in psychology may have implications for meta-analyses focused on driving-related skills because many driving-related skills studies fall within the domain of or borrow methods from cognitive psychology. In addition, there is an elevated risk of spurious effects in research domains with greater “flexibility” in measures and in studies focused on controversial or political topics (Ioannidis, 2005). Given the lack of standards as to what constitutes a driving-related skill or its measurement, and the political nature of cannabis, alcohol and driving under the influence, the potential for spurious effects in the extant literature is considerable. Finally, smaller effects – including null effects – may be less likely than larger, positive effects to be published in the first place, leading to further bias in the published literature available to researchers (Borenstein et al., 2009, pp. 277-280). To reiterate, the underlying assumptions of vote-counting methods – namely

that statistical significance indicates the presence of a genuine effect, and the absence of statistical significance indicates the absence of a genuine effect – are incorrect. As a result, the use of vote-counting is inappropriate for summarizing the effects of cannabis and/or alcohol on driving.

In addition, there are logical issues with attempting to identify “equivalent levels of impairment” based on an equivalent proportion of “significantly deteriorated” effects at a certain BAC concentration and at a certain THC concentration. Although the purpose of Berghaus and colleagues’ (1998b) analysis is to identify equivalent alcohol and THC concentrations with respect to driving ability, their approach is incompatible with that goal. To illustrate, Berghaus and colleagues (1998b) report that a BAC of 0.73% and a THC concentration of 11 ng/mL of plasma are both associated with the same proportion of significantly deteriorated effects – specifically, half of all effects included in their meta-analysis were significantly deteriorated at these concentrations. It is important to understand that the observation of the same proportion of significantly deteriorated effects at two different drug concentrations does not necessarily mean that the two concentrations are equally detrimental, as Berghaus and colleagues (1998b) and others (Grotenhermen et al., 2005) have suggested. Hypothetically, if 50% of the effects were found to be significantly deteriorated at both a BAC of 0.73% and at a THC concentration of 11 ng/mL, but each of the effects associated with a BAC of 0.73% were additionally reported to be twice as strong as the effects associated with a THC concentration of 11 ng/mL, then it would not make sense to conclude that the two are equally detrimental. Essentially, Berghaus and colleagues’ (1998b) analysis fails to incorporate practical significance, which is a fundamental aspect of meta-analysis (Borenstein et al., 2009).

Overall, the analytic approach taken by Berghaus, Krüger and colleagues is logically flawed. Vote-count approaches to meta-analyses lack validity, and equivalence between levels of impairment should be made on the basis of effect size rather than on the proportion of statistically-significant effects to non-statistically-significant effects within in a group of studies. The current meta-analysis differs from Berghaus and colleagues' in that a formal meta-analysis involving effect sizes is conducted. This allows for the influence of cannabis on driving performance and behaviour to be quantified relative to both sober driving and to driving under the influence of alcohol in experimental studies.

Reporting standards. It is unclear how a number of important decisions were made in Berghaus, Krüger and colleagues' meta-analyses. Firstly, the interpretation of statistically-significant effects with respect to safety requires consideration. The criteria for making judgements about whether an effect should be categorized as “significantly deteriorated,” “significantly improved” or “no significant effect” for the purposes of the vote count, is not reported in Berghaus and colleagues' (1998a, 1998b) meta-analyses. Such judgements are not necessarily intuitive because significant improvements or deteriorations in *scores* on laboratory tasks do not always correspond to meaningful improvements or detriments in driving safety. For example, “tracking” and “psychomotor skills” could potentially include experimental measures where a speed-accuracy trade-off could occur. It is unclear whether such trade-offs are implicated in measures included in these meta-analyses, and if so, how those effects would be extrapolated to safety. For example, if a participant under the influence of cannabis was significantly slower at performing a tracking task, but no less accurate, would this be considered “significantly deteriorated” performance or not? Additionally, “simulator driving” is not elaborated upon, and it is unclear which types of measures were included. As previously

discussed, not all measures should be conceptualized as indicators of impairment per se. Finally, Berghaus and colleagues' "simulator driving" category may have included measures from flight simulators, though it is not entirely clear whether this is the case from the descriptions provided (e.g., Berghaus et al., 1998a). Flying and driving involve different tasks and time constraints, and the extent to which changes in simulated flying measures predict changes in driving performance and behaviour is not clear.

Second, studies included in Berghaus' and colleagues' analyses may have each contributed multiple effects. If multiple effects from the same set of participants are deemed to measure the same construct and included a single analysis, an underlying assumption of meta-analysis is violated – namely, that included effects are independent of one another. It is not clear whether multiple effects from a single paper could contribute to the same driving-related skill category, so it is unclear whether the assumption of independence was violated in either of these meta-analyses.

Third, it should be noted that often, THC concentrations in included studies were not reported, so Berghaus and colleagues estimated THC concentration for experimental effects based on a method by Sticht and Käferstein (1998). Unfortunately, the validity of this method is not clearly described, and based on an inspection of the estimated THC concentrations reported over time in their paper, it does not appear to have a high degree of precision. It is unclear how specific estimated THC concentrations were linked with included effects in either of the two meta-analyses by Berghaus and colleagues (1998a, 1998b).

Finally, the reports discussed here would likely fail to meet the standards outlined in modern guidelines for conducting and reporting systematic reviews and meta-analyses, such as the Preferred Reporting Items for Systematic Review and Meta-Analyses (PRISMA; Moher et

al., 2009). However, as previously discussed, it is unknown whether there is an alternative version that completely describes study methodologies, and if so, whether that version would meet those standards. The current meta-analysis adheres to PRISMA guidelines throughout.

Summary. Previous meta-analyses conducted by Berghaus, Krüger and colleagues have a number of limitations that threaten the validity of the reported findings. Additionally, since the appearance of the meta-analyses by Berghaus, Krüger and colleagues, numerous empirical studies have been published. Overall, the literature focused on the effects of cannabis on driving performance and behaviour is in need of an update. The current meta-analysis seeks to update the literature and avoid some of the limitations associated with previous work.

Studies focused on alcohol. The current study is also not the first research synthesis of experimental driving studies involving alcohol. As previously discussed, Krüger and colleagues conducted a meta-analysis utilizing a vote-count methodology focused on alcohol, and its results were later incorporated into Berghaus and colleagues' comparative analysis (Berghaus et al., 1998b). Moskowitz and Fiorentino (2000) also conducted a meta-analysis focused on alcohol, and it shares many of the same limitations as those of Berghaus, Krüger and colleagues: the criteria used to make judgements about the allocation of experimental tasks to “driving-related behavior” categories, and the criteria used to make judgements about whether an effect constitutes impairment, are vague and limit replicability; a vote-count is conducted rather than a formal meta-analysis involving effect sizes; and, it is unclear how multiple measures from a single study eligible for a single category were handled, which affects the independence of multiple included measures from individual studies.

There are at least three more recent research syntheses focused exclusively on the effects of alcohol on driving performance and behaviour measures recorded in simulators, on closed

courses and in on-road studies. During the search for studies to be included in the current study, three systematic reviews and meta-analyses of the effects of alcohol on driving performance and behaviour were identified. All were published in the year 2017 and differ from the current analysis in important ways.

Irwin et al., 2017. In Irwin and colleagues' (2017) meta-analysis, data from healthy adults aged eighteen and older with no clinical conditions or recent drug use were eligible for inclusion. Experimental driving measures including standard deviation of lane position (SDLP), lane crossings, average speed and the standard deviation of speed were targeted. Overall, alcohol increased SDLP, lane crossings and standard deviation of speed, but there was no statistically significant change in speed.

The current meta-analysis is similar to Irwin et al. (2017) in that it involves comparisons of experimental driving measures while under the influence of alcohol to experimental driving measures either during a placebo treatment or a during non-alcohol control drive, in the absence of secondary task distraction or other experimental manipulations such as fatigue. However, the current study differs from Irwin et al. (2017) in several important ways. In general, Irwin et al. (2017) has a narrower scope than the current study. First, only experimental driving studies utilizing repeated measures designs and driving simulators, published in peer-reviewed sources such as journals and conference proceedings, were eligible for inclusion in Irwin et al. (2017). The current meta-analysis is broader in scope because both within-subjects and between-subjects designs, and studies involving simulators, closed courses or on-road were all eligible for inclusion. Grey literature that did not appear in peer-reviewed sources was also eligible for inclusion in the current analysis. Second, studies included in Irwin and colleagues' (2017) analysis were required to meet a minimum quality score. The practice of using a study quality

tool to make judgements about inclusion or exclusion in a systematic review and/or meta-analysis is generally discouraged (Siddaway et al., 2019). The current study included study quality assessments but not for the purposes of judging eligibility for inclusion. Finally, Irwin et al. (2017) targeted only four experimental driving measures. The current study targeted the same four measures, as well as seven additional measures, to more fully characterize the nature of the effect of alcohol on driving performance and behaviour.

Rezaee-Zavarah et al., 2017. Randomized controlled trials (i.e., between-subjects studies) involving participants of all ages and backgrounds were eligible for inclusion, and seven experimental driving measures related to lane position, speed and “accidents” were targeted. Unlike the current study, a meta-analysis was not conducted as part of Rezaee-Zavarah and colleagues’ (2017) review. Based on a qualitative review of 13 studies, Rezaee-Zavarah et al. (2017) reported that a majority of included studies reported significant effects of alcohol on driving performance and behaviour measures related to lane position and speed. However, they also identified “troublesome heterogeneity” among the included studies, resulting in “inconclusive” evidence that “can only generate the lowest grade of recommendations” (Rezaee-Zavarah et al., 2017, p. 170).

Again, there are some notable differences between the current study and Rezaee-Zavarah et al. (2017). Although the current study also targeted participants of all ages and backgrounds, the current study did not exclusively target between-subjects designs. Next, the current study synthesizes the literature by aggregating effect sizes generated for each study, rather than by calculating the proportion of included studies that reported statistically significant effects. Finally, the current analysis overlaps in terms of some targeted driving measures, but the current

meta-analysis also targets additional driving measures related to constructs other than lane position, speed and collisions.

Jongen et al., 2017. Finally, Jongen and colleagues (2017) conducted a study involving meta-analytic methods that applied a very narrow scope with respect to alcohol doses, study methods and experimental driving measures. Jongen et al. (2017) focused exclusively on the effect of alcohol on SDLP. Only nine studies, all from Maastricht University, where healthy participants were dosed to a target BAC of 0.05% prior to on-road driving – no lower, and no higher – in studies utilizing within-subjects designs, were included in the analysis. Alcohol was found to increase SDLP by 2.5 cm in these studies (Jongen et al., 2017). Jongen and colleagues (2017) suggested that an increase of 2.5 cm “can be reliably used to determine clinical relevance of drug-induced driving impairment in the standardized highway driving test” (p. 843).

Although Jongen et al. (2017) incorporated some meta-analytic methods, the analysis is limited. First, it does not appear that a systematic search for studies was conducted. Consequently, Jongen et al. (2017) shares many similarities with an informal literature review, but their results are presented quantitatively as in a typical meta-analysis. Second, although individual effect sizes were computed for each study, these effect sizes were not actually aggregated to yield an overall effect size estimate. Instead, the raw data from each of the individual studies were pooled, and an effect size representing the entirety of the raw data was generated. This approach is discouraged in meta-analysis because it can yield an inaccurate result via a phenomenon known as Simpson’s Paradox (Borenstein et al., 2009, pp. 303-309). The current study is fundamentally different than that of Jongen et al. (2017) in that a systematic search was conducted, and decisions about inclusion and exclusion of data were made based on systematic judgements against inclusion criteria with the goal of minimizing bias. Second, effect

sizes were aggregated across studies in the current study. Finally, a broader range of BAC levels, study settings and methods were targeted, and driving performance was not assessed solely based on SDLP.

Summary. Previous research syntheses focused on the effects of alcohol on driving in experimental studies, as with previous research syntheses focused on the effects of cannabis on driving in experimental studies, have important limitations. To date, Irwin and colleagues' (2017) analysis of alcohol-involved driving is the most rigorous within the existing literature and shares the most similarities with the current study. However, the scope of the current study is considerably more comprehensive than any research synthesis focused on the effects of cannabis or alcohol on driving in experimental studies to date. The aims of the current study are revisited next.

Current Study

The current systematic review and meta-analysis has four key objectives: (1) to quantify the magnitude of the effect of cannabis on driving performance and behaviour; (2) to compare the influence of cannabis to that of alcohol; (3) to assess the effect of the combination of cannabis and alcohol on driving performance and behaviour; and, (4) to identify knowledge gaps and quality limitations to direct the conduct of productive, high-quality scholarly inquiry focused on cannabis- and alcohol-involved driving in the future. More generally, the current study also seeks to outline a standardized approach to studying the effects of drugs on driving in experimental studies in the future. Although the dissertation is primarily exploratory in nature, objectives one through three test the robustness of some general tenets related to the influence of cannabis, alcohol and the combination of the two within the experimental driving literature. Each of these objectives is discussed in turn.

The first objective of the current study is to assess the extent to which cannabis impairs driving performance and changes driver behaviour. Within the literature, cannabis is generally reported to increase response times to targets and hazards, diminish lateral and longitudinal control, and lead to decreases in speed and increases in headway (Hartman et al., 2015, 2016; Lenné et al., 2010; Ramaekers et al., 2000a; Ronen et al., 2008). Consequently, these general effects were hypothesized to occur within the context of the current meta-analysis. To date, however, no meta-analyses focused on the effects of cannabis on driving in experimental studies have reported effects in terms of effect size (i.e., magnitude or degree of impairment). Nonetheless, it was additionally hypothesized that the effects would be small to moderate in magnitude because such effects are typically observed in experimental driving studies focused on other crash contributors such as cell phones and other technological distractions (Caird et al., 2018; Simmons et al., 2017).

The second objective of the current study is to assess the similarities and differences between the effects of acute cannabis intoxication and acute alcohol intoxication on driving performance and behaviour. Approaches to the understanding of and response to cannabis-involved driving often implicitly treat cannabis and alcohol as analogous, and the current study capitalizes on this analogy to offer an intuitive benchmark to judge the influence of cannabis on driving performance and behaviour against. In the existing literature, cannabis and alcohol are generally reported to have some similarities, but also some differences with respect to driving performance and behaviour. With respect to similarities, both cannabis and alcohol have been reported to slow response times and negatively affect lateral control (e.g., Smiley, 1986; Sewell et al., 2009). Thus, both cannabis and alcohol were hypothesized to exhibit these effects in the current meta-analysis. However, no specific hypotheses were made about similarities and

differences between cannabis and alcohol in terms of effect size magnitude. With respect to differences between cannabis and alcohol, cannabis is generally reported to slow driving speed and increase following distance, whereas alcohol is reported to lead to *faster* driving speeds (e.g., Smiley, 1986; Sewell et al., 2009). Again, these general effects are hypothesized to occur within the current meta-analysis.

The third aim of the current study is to assess how the combination of cannabis and alcohol on driving performance and behaviour compares to sober driving, as well as to either drug in isolation. Research indicates that it is not uncommon for drivers to operate vehicles while under the influence of both cannabis and alcohol simultaneously. For example, in Canada, 7.0% of individuals surveyed as part of the Road Safety Monitor reported driving within two hours of using cannabis, and 3.0% reported driving within two hours of using both alcohol and cannabis, in 2019 (Woods-Fry et al., 2019). Similarly, of the cannabis-using respondents of the Canadian Cannabis Survey who reported driving within two hours of using cannabis, 20% reported ever driving within two hours of combining cannabis and alcohol; of those, 34% reported doing so within the previous 12-month period (Government of Canada, n.d.-b). The combination of cannabis and alcohol is generally thought to be more detrimental to driving than either alone, but the literature is divided (e.g., Hartman & Huestis, 2013). Thus, with respect to the influence of the combination of cannabis and alcohol on driving performance relative to baseline or either drug in isolation, it is difficult to make any specific hypotheses. Potentially, the combination of cannabis and alcohol could lead to even greater performance decrements in terms of response time, lateral control and longitudinal control relative to sober driving and relative to driving under the influence of only cannabis or alcohol. Speculatively, this could be accompanied by greater speed reductions and longer following distances for drivers under the influence of the

combination of drugs relative to baseline or either drug in isolation. However, it is also possible that the speed-increasing effect of alcohol may counter-act the speed-reducing effect of cannabis, if those effects exist (Ronen et al., 2010; Hartman et al., 2016). Overall, the combination of cannabis and alcohol is anticipated to have more detrimental effects on response time, lateral control and longitudinal control relative to baseline or either drug in isolation, but no particular hypotheses are made with respect to speed or following distance.

The final objective of the current study is to assess the quality and validity of the extant literature and to make recommendations for future scientific inquiry in the area. Although the literature focused on the effects of cannabis on driving performance and behaviour is not new, it is small, and the legalization of cannabis for recreational use in Canada should allow for easier access to cannabis for research purposes in the future. In addition to providing recommendations related to conceptual issues in drug-involved driving research, including the measurement of impairment, the current study critically reviews the extant literature to make recommendations for quality research practices and future directions. Given that this aim is not associated with a specific research question, there are no hypotheses associated with this aim.

Chapter 2: Method

The following section describes the study protocol including eligibility criteria, information sources, the search strategy, study selection, data items, the data collection process, study quality and risk of bias assessments, summary measures, the synthesis of results, risk of bias across studies and additional analyses, in accordance with PRISMA guidelines (Moher et al., 2009). The study protocol is unregistered.

Eligibility Criteria

Eligibility for inclusion in the systematic review and meta-analysis was based on six inclusion criteria. Any studies that failed to meet any of the six inclusion criteria during the full-text review were excluded.

Study design. First, all papers eligible for inclusion were required to report on an original empirical study (*Criterion 1*). Papers that reported on qualitative reviews of previously published studies, as well as letters to editors, comments or opinion papers, lists and guidelines, and overviews of laboratory activities, were not eligible for inclusion. Secondly, all original empirical studies were required to report on an experimental driving study with human participants who drove in a simulated four-wheeled vehicle or in a real four-wheeled vehicle on a test track, closed course or actual road (*Criterion 2*). Observational studies based on police report or real-world crash data, which aim to quantify crash risk, were not eligible for inclusion. As previously discussed, numerous meta-analyses of observational studies focused on the effect of cannabis on crash risk have been published in the past.

Finally, quantitative research syntheses of experimental driving studies were not eligible for inclusion. However, relevant systematic reviews and meta-analyses (i.e., those focused on the effects of cannabis and/or alcohol on eligible measures of driving performance and behaviour)

were marked so that their included studies could be located, reviewed and considered for inclusion in the current systematic review and meta-analysis.

Participants. Experiments eligible for inclusion could not exclusively recruit and select participants with a clinical diagnosis that could interfere with the ability to operate a real or simulated vehicle (*Criterion 3*). However, participants with cannabis and/or alcohol use behaviours such as alcohol dependence or drug addiction were not excluded under this criterion. If a study included a healthy control group in addition to a non-eligible clinical sample, the study was included but only healthy control data was deemed eligible for inclusion.

There were no restrictions on demographics such as participant age, sex, or experience with cannabis or alcohol. This information was extracted and reported in Table 1 and Table B1 (Appendix B).

Experimental conditions. Eligible studies were required to include one or more of the following comparisons to be made with respect to driving performance and behaviour measurements (*Criterion 4*):

1. Cannabis v. control;
2. Alcohol v. control;
3. Cannabis and alcohol in combination v. control;
4. Cannabis and alcohol in combination v. cannabis;
5. Cannabis and alcohol in combination v. alcohol;
6. Cannabis v. alcohol.

To allow these comparisons to be made, the experiment had to include one or more of the following assignments:

1. The researchers allocated participants to an experimental condition wherein participants were administered cannabis and no other active drug;
2. The researchers allocated participants to an experimental condition wherein participants were administered alcohol and no other active drug;
3. The researchers allocated participants to an experimental condition wherein participants were administered a combination of cannabis and alcohol, and no other active drug;

And, one or more of the following elements:

1. The researchers allocated participants to an equivalent control condition wherein they were administered an equivalent placebo to an eligible experimental condition (e.g., placebo cannabis for the purposes of a cannabis v. control comparison);
2. The researchers allocated participants to a control condition where they received no treatment. This condition could include pre-test baseline driving.

Both within-subjects and between-subjects comparisons were eligible for inclusion.

Finally, it is important to note that in some studies, cannabis and alcohol were not the only drugs under study. In cases where participants received cannabis and/or alcohol with an additional non-cannabis or non-alcohol placebo drug, the “no other active drug” criterion was still met. However, the two conditions in an eligible comparison were still required to be equivalent when additional non-cannabis or non-alcohol placebo drugs were administered. For example, a non-equivalent (and thereby ineligible) comparison could involve comparing a condition wherein participants received only cannabis (and no additional placebo drug) to a condition wherein participants received only the placebo of some other drug. Additionally, when study conditions were suspected to also involve secondary, non-driving task distraction (e.g., cell

phone tasks) or other forms of impairment (e.g., sleep deprivation or fatigue), those conditions were not eligible for inclusion.

When a study reported both a placebo comparison and a non-treated comparison (e.g., a pre-drive baseline), the placebo comparison was targeted for inclusion. When between-subjects studies reported two non-treated comparisons (i.e., a time-matched control, and a pre-drive baseline), the between-subjects comparison was targeted for inclusion. The specific comparisons that were ultimately included in the meta-analysis are reported in Table 2.

Driving performance and behaviour measures. Eligible studies were required to include and report upon measures related to certain elements of driving performance and behaviour in association with the aforementioned experimental conditions (*Criterion 5*). When an otherwise eligible paper reported that relevant driving performance and/or behaviour data was collected but not reported on, and readers were instead directed to another source, the paper was excluded under this criterion. This occasionally occurred when studies used multiple types of measures and results were reported in multiple focused publications.

Hazard RT. Hazard RT was operationalized as the amount of time taken to respond to a tangible hazard. Responses might include braking or evading, and hazards might include situations such as slowing forward vehicles, on-road obstacles and intersecting pedestrians or vehicles. However, gain, coherence and phase shift, which are measures used to describe how well participants adjust their own speeds to match the speeds of forward vehicles (e.g., Veldstra et al., 2012), were not eligible for inclusion under hazard RT or any other category. Response times to artificial targets, such as flashing LED lights appearing in the periphery of the driver's view, were also ineligible for inclusion. Finally, distance travelled after responding to a hazard were not eligible for inclusion because the distance travelled would be influenced not only by

response time, but also by the participant's chosen speed up until that point, the force with which they applied the brakes, and the braking response of the vehicle.

Lateral position variability. Lateral position variability was operationalized as the amount of variability around either the participant's chosen lane position or around their deviation from a reference point, such as the centre of the lane. A common measure within the experimental driving literature that falls into this category is standard deviation of lane (or lateral) position (SDLP). The mean lane position or mean offset from a reference point was not eligible for inclusion.

Lane deviations or excursions. Lane deviations or excursions were operationalized as the number of times that participants' vehicles intersected with the boundaries of a driving lane.

Time out of lane. Time out of lane was operationalized as the amount of time that participants were engaged in a lane deviation or excursion.

Driving speed. Driving speed was operationalized as the velocity of the participant's vehicle while driving. Average speed collected across driving segments and at discrete moments within the drive were both eligible for inclusion under driving speed. However, speed measured at the moment of a collision was not eligible for inclusion given that the velocity of the vehicle would be affected not only by the participant's chosen speed up until that point, but also by their response time to the obstacle and the braking response of the vehicle. In some cases, a difference score (e.g., participants' average deviation from a posted speed limit or instructed driving speed) was used to make inferences about average driving speed differences between study conditions. Participants' average minimum speed and average maximum speeds were excluded.

Driving speed variability. Speed variability was operationalized as the amount of variability around the participant's average speed. A common measure within the experimental driving literature that falls into this category is standard deviation of speed.

Speed violations. Speed violations were operationalized as the number of times that participants' driving speed exceeded the posted speed limit. Subjective measures, such as cases of "driving too fast" based on the opinion of expert evaluators, were not eligible for inclusion under speed violations. Additionally, cases where the participant was deemed to be driving too slow were not eligible for inclusion.

Time speeding. Time speeding was operationalized as the amount of time that participants drove above the speed limit.

Headway. Headway was operationalized as the participants' following distance from a forward vehicle. Both time headway and distance headway were eligible for inclusion. However, participants' average minimum and average maximum headways were excluded. Additionally, time to collision (TTC), which refers to time headway at the moment that a participant brakes in response to a forward obstacle (e.g., Strayer et al., 2006) was excluded because it would be influenced not only by the participant's average following distance up until that point, but also by their response time to the hazard and the braking response of the vehicle.

Headway variability. Headway variability was operationalized as the amount of variability around the participant's average headway. A common measure within the experimental driving literature that falls into this category is standard deviation of headway.

Crashes. Crashes referred to collisions, crashes and accidents, generally with other road users or in the form of single-vehicle collisions resulting from lane departures. However, contact with pylons used to denote lane boundaries in closed-course studies were not generally eligible

for inclusion under crashes given that the situation could also be conceptualized as a lane deviation or excursion.

Original data. Eligible studies were required to report unique data (*Criterion 6*). When multiple studies that otherwise met inclusion criteria reported on the same set of data, only the most recently published, accurate and/or accessible paper was included. For example, peer-reviewed papers were usually preferred over earlier conference papers reporting on the same project. In some cases, data from papers excluded on this criterion were used to supplement any information missing in the included paper. When this occurred, the supplemental data was indicated with the appropriate citation. Additionally, when two publications reported both overlapping and unique data, both publications were retained to extract unique data from each publication.

Information Sources

A list of electronic databases to search for studies was developed with the assistance of a subject librarian. Academic Search Complete, CINAHL, Embase, Scopus, MEDLINE, PsycINFO, SportDISCUS and TRID were searched in May 2018. Later, a non-systematic search for new studies was conducted using Google Scholar in August 2019. Finally, lists of citations of studies included in previously published systematic reviews and meta-analyses located in the formal electronic search were generated. Specifically, titles appearing in the reference lists of Berghaus et al. (1995), Irwin et al. (2017), Jongen et al. (2017), Reimann et al. (2014) and Rezaee-Zavarah et al. (2017), which were identified as part of the electronic database search, were considered for follow-up.

In addition to these searches, studies were occasionally identified serendipitously, such as through informal internet searches or while reading through other papers.

Search Strategy

The electronic search strategy was also developed with the assistance of a subject librarian. First, informal preliminary searches were conducted to identify “model” studies that were judged to meet inclusion criteria. These model studies provided fundamental keywords and served as test items during the development of electronic search strategies. The search strategy ultimately included keywords associated with model studies, keywords related to cannabis, alcohol and driving, synonyms for other keywords, and indexing terms used by the databases of interest.

While developing the electronic search, it became apparent that the electronic indexing of this particular body of literature is imprecise. For example, we could not develop a search strategy that could reliably discriminate experimental driving studies from observational driving studies. We elected to adopt a more liberal search designed to maximize hits, with the trade-off that a large number of false positives would be returned. The electronic search strategies for each database are listed in Appendix A.

Study Selection

The present systematic review and meta-analysis is notable in that non-English studies were not excluded during study selection. The consideration of non-English studies required a unique approach, outlined below.

Screening. Two coders (SS, FS) independently screened the abstracts of identified studies based on general adherence to the inclusion criteria. The goal of abstract screening was to eliminate studies which were obviously unrelated to the research question and would clearly fail to satisfy inclusion criterion; thus, coders were liberal in passing studies on for full-text review. Although reviews were ineligible for inclusion, studies that appeared to be reviews of

experimental driving studies focused on the effects of cannabis and/or alcohol were flagged for advancement to full-text review. This allowed for the identification of systematic reviews and meta-analyses that could later be reviewed for the identification of additional relevant citations. Studies identified in the systematic search were not screened based on the content of their titles. Disagreements between coders were resolved through discussion.

Approximately 8% of the citations identified in the electronic search did not have abstracts. Rather than pass these on to full-text review directly, studies without abstracts were screened using a slightly different approach based on advice from a subject librarian. Full-texts were obtained and effectively skimmed to identify section heading content (which occasionally required electronic translations when the paper was not in English) and the overall structure of the paper. Two coders independently made judgements about whether the paper was likely to report on an original empirical study (*Criterion 1*). In cases where the paper clearly appeared to be a comment, letter to the editor, review or other non-empirical paper, the paper failed screening. In cases where it was unclear whether the paper followed an empirical formatting structure, the paper was flagged for advancement to full-text review. Disagreements between coders were resolved through discussion.

Full-text review. Studies that passed screening were advanced to full-text review. Two coders (SS, FS) independently judged the texts of studies that passed screening against the inclusion criteria. Studies that failed to satisfy all of the inclusion criteria were excluded. It was possible for a study to fail to meet inclusion criteria for several reasons. For example, an empirical paper that does not report on an experimental driving study (*Criterion 2*) is not likely to include a relevant driving performance or behaviour measure either (*Criterion 4*). The first criterion that a paper clearly failed to adhere to was marked as the reason for its exclusion.

Disagreements among coders were resolved through discussion. When a consensus could not be reached, a third coder (JKC) was consulted and a final consensus was made.

Non-English studies were subjected to additional processing. It was not financially feasible to commission professional translations of non-English full-texts. Instead, the method section (or the entire text, if the method section could not be reliably located or did not provide enough information) were electronically translated using Google Translate, which has shown promising utility for the purposes of systematic review (Jackson et al., 2019). However, in many cases, non-English papers were delivered in the form of photocopies, which precluded transcribing text directly from the PDF viewer to Google Translate using standard copy and paste functions. When this occurred, relevant sections of photocopied text were extracted from PDF files using the Microsoft Windows Snipping Tool and saved as JPEG image files. JPEG image files were then processed using Tesseract optical character recognition software, which yielded text files that could be read by Google Translate. Given that errors in optical character recognition often occurred, leading to imperfect translations, the translations were treated in a similar manner as were study abstracts. That is, coders adopted a liberal bias in their electronic translation screening judgements: the purpose of the translations was to eliminate studies which were obviously unrelated to the research question and would clearly fail to satisfy inclusion criterion. Disagreements between coders were resolved through discussion.

When translations passed screening, attempts were made to locate students and local volunteers who spoke and read the language of the study. In some cases (e.g., short papers), volunteers translated the study directly, and these translations were judged against inclusion criteria by the two coders. In other cases, it was more feasible to provide the native reader with an overview of the inclusion criteria and consult them on the contents of the paper using

informal, unstructured interviews. The interviewing coder (SS) then attempted to judge the study against inclusion criteria through consultation with the native reader.

Systematic review and meta-analysis. All studies that met inclusion criteria, except for the non-English studies, were subjected to study quality assessments, risk of bias assessments and qualitative data reporting as part of the systematic review. Additionally, all studies that met inclusion criteria, except for the non-English studies, were mined for statistical data relevant to the computation of effect sizes (see *Summary Measures*, below). However, insufficient or incompatible data reporting often precludes the computation of effect sizes. Only studies for which effect sizes could be computed could be included in the meta-analysis. Studies that were eligible for inclusion, but that did not contain enough data for effect size computation, are described in Appendix B.

Data Items

General descriptive data including study setting (i.e., simulator, on-road or closed-course), overall included sample size, participant age, eligible drug driving conditions and eligible driving performance and behaviour measures were collected (see Table 1). Additionally, information on inclusion criteria related to drug use, participant drug use frequency, specific drug driving conditions and methods of drug administration from individual studies was collected (see Table 2).

For the purposes of effect size computation, means, standard deviations, standard errors, 95% confidence intervals, sample sizes, measurement units, comparison types (i.e., between- or

within-subjects), t statistics and relevant F statistics⁵ were extracted, when available in tables or figures. Data was extracted from figures using Microsoft Paint (to identify co-ordinates of means and error bars) and Microsoft Excel (to transform those co-ordinates into useable statistics). Data was extracted to two decimal places further than the number of decimal places included in the axes of the figure. Additionally, for studies using repeated-measures designs, correlations between participants' scores in the drug driving condition and the comparison condition, known as the *pre-post correlation*, were required to compute effect sizes. Often, this correlation is unreported and cannot be derived from data reported in the paper. Initially, the plan was to estimate missing pre-post correlations from all available pre-post correlations collected from papers and from raw data supplied by study authors (Borenstein et al., 2009, p. 29). However, few pre-post correlations were recovered, and those that were recovered were highly variable in magnitude between comparisons, measures and studies. Consequently, the plan to estimate missing pre-post correlations was abandoned. Instead, for all comparisons where the correlation was missing, sensitivity analyses were conducted using a range of pre-post correlations (Borenstein et al., 2009, p. 29). Ultimately, pre-post correlations of zero, 0.5 and 0.9 were chosen to capture a wide range of possible correlations between pairs of scores in included studies. Attempts were made to retrieve data from studies published in the previous five years by contacting the authors of those studies.

In addition to effect size data, information related to the type of statistical analysis conducted and whether study conditions may have been contaminated due to the influence of

⁵ Only F statistics from analyses comparing two conditions, with no additional factor in the analysis, were eligible for inclusion. See Hullett and Levine (2003).

secondary driving-related tasks on driving performance and behaviour, such as target detection, was also collected. Finally, for the purposes of subgroup analysis, pre-drive BAC, post-drive BAC and average BAC throughout the drive were extracted when available for the purposes of sorting effect sizes into BAC bins (i.e., non-zero BAC levels up to 0.03%, 0.04% to 0.06% BAC, 0.07% to 0.09% BAC, and 0.10% to 0.12% BAC).

Data Collection Process

Most of the data, including the data appearing in Table 1 and Table 2, and the data extracted for effect size computation, was extracted by a single-coder (SS) and double-checked for accuracy. This data was entered into electronic spreadsheets and text files.

Study quality and risk of bias data was collected by multiple coders (SS, GJ, LK, AT, DSL) who completed study quality and risk of bias assessments independently. All studies were reviewed by SS as well as at least one other coder. All disagreements were resolved through discussion until a final judgement was agreed upon.

Study Quality & Risk of Bias

Study quality and risk of bias was assessed for two purposes: first, to contextualize the results of the meta-analysis; and second, to generate recommendations for good research practices in future studies. Study quality and risk of bias was assessed using an original dictionary based on the Quality Assessment Tool for Quantitative Studies (Effective Public Health Practice Project, 2007), as well as the Cochrane Risk of Bias Tool (Cochrane Collaboration, 2011a). The Quality Assessment Tool for Quantitative Studies was designed for health research and is recommended by the Cochrane Collaboration, and the Cochrane Risk of Bias tool is recommended for use in systematic reviews and meta-analyses of randomized controlled trials (Cochrane Collaboration, 2011b). The latter is designed to appraise risk of

selection bias, detection bias, performance bias, attrition bias and reporting bias (Cochrane Collaboration, 2011a). However, given the overlap with the Quality Assessment Tool for Quantitative Studies on four of these sources of bias, only the reporting bias section of the Cochrane Risk of Bias tool was used.

Initially, multiple coders independently judged included studies using the Quality Assessment Tool for Quantitative Studies and with the risk of reporting bias item from the Cochrane Risk of Bias Tool. However, it became apparent that the Quality Assessment Tool for Quantitative Studies tool had limited utility when used to appraise quality in experimental driving studies. For example, disagreements often arose due to differences in interpretation of dictionary items within the context of the included studies. For this reason, relevant items from the tool were adapted into a dictionary that used language specific to experimental driving studies involving alcohol and cannabis to guide coding decisions more effectively. Ultimately, only the judgements made by three of the five coders using this dictionary (SS, AT, DSL) were included in the final study quality and risk of bias assessment (see Table F1 in Appendix F). Specifically, coders evaluated whether the sample was likely to be representative (adapted from Component A, Question 1 of the Quality Assessment Tool for Quantitative Studies); what percentage of participants agreed to participate (adapted from Component A, Question 2); whether drug-driving conditions were randomized (adapted from Component B); whether counterbalancing or randomization of orders was utilized in repeated measures studies (adapted from Component B); whether there were important differences between groups prior to the driving assessment (adapted from Component C, Question 1); whether driving assessors were aware of the participants' drug treatment (adapted from Component D, Question 1); whether participants were aware of the study hypothesis (adapted from Component D, Question 2);

whether the driving data collection was reliable (adapted from Component E, Question 2); whether numbers and reasons for withdrawals and drop-outs within the context of relevant measures were clearly reported (adapted from Component F, Question 1); what percentage of participants completed the study within the context of relevant measures (adapted from Component F, Question 2); whether consistent drug administration across participants was reported (adapted from Component G, Question 2); and whether there was reason to believe that contamination may have occurred during drug administration (adapted from Component G, Question 3).

Additionally, an item related to risk of reporting bias, which was adapted from the risk of reporting bias section of the Cochrane Collaboration's Risk of Bias Tool, was also included in the dictionary. Coders (SS, AT, DSL) assessed whether all driving performance and behaviour measures reported in the method section were reported on in the results section. When there was a match, this was deemed low risk of bias. When a pre-specified measure was not reported on in the results, or a measure reported on in the results was not pre-specified (including cases where no measures were pre-specified), this was deemed high risk of bias. When it was unclear whether all driving measures reported in the method section were reported on in the results section (e.g., categories of measures were reported in the method, rather than specific measures), this was deemed unclear risk of bias.

Finally, an item related to sample size, which does not appear in the original Quality Assessment Tool for Quantitative Studies, was added to the adapted dictionary. Coders assessed whether the study reported enrolling a targeted sample size based on an a priori power assessment involving a hypothesized effect size.

Summary Measures

The principal summary measure was Hedge's g . Hedge's g is a bias-corrected form of Cohen's d (Borenstein et al., 2009, pp. 27-28). Like Cohen's d , Hedge's g quantifies differences between conditions in units of standard deviations, and the small (0.2), medium (0.5) and large (0.8) effect size conventions associated with Cohen's d (Cohen, 1992) also apply to Hedge's g . In addition to Hedge's g , average effects were also reported in the form of r . Effect sizes were generated automatically by entering extracted data into Comprehensive Meta Analysis (CMA) Version 3.3.070.

In some cases, the architecture of the CMA program required performing calculations on data used in effect size computation before the data could be entered into the program. Specifically, when a study reported data for one control group and multiple eligible drug driving conditions, data for the multiple drug driving conditions needed to be aggregated before they could be compared to the control group. Otherwise, data for the control group would be counted twice (for a discussion of this issue, see Borenstein et al., 2009, pp. 239 – 241). Charlton and Starkey (2015), Starkey and Charlton (2014), Beard (2012) and Chen et al. (2016) each contained three subgroups – two that received different levels of alcohol, and one control. For the primary meta-analysis, data for participants in low and high alcohol dose treatments were aggregated prior to entry into CMA to avoid counting the control group twice. The sample size for the aggregate alcohol group was computed using Equation 23.1 from Borenstein et al. (2009),

$$n_1 = n_{11} + n_{12}$$

the overall mean for the aggregate alcohol group was computed using Equation 23.2 from Borenstein et al. (2009),

$$\bar{X}_1 = \frac{n_{11}\bar{X}_{11} + n_{12}\bar{X}_{12}}{n_{11} + n_{12}}$$

and the overall standard deviation for the aggregate alcohol group was computed using Equation 23.3 from Borenstein et al. (2009).

$$S_1 = \sqrt{\frac{(n_{11} - 1)S_{11}^2 + (n_{12} - 1)S_{12}^2 + \frac{n_{11}n_{12}}{n_{11} + n_{12}}(\bar{X}_{11} - \bar{X}_{12})^2}{n_{11} + n_{12} - 1}}$$

A similar approach was taken with Sklar et al. (2014) and Price et al. (2018). These studies each contained six subgroups – two older groups that received different levels of alcohol, two younger groups that received different levels of alcohol, and two controls (one per age group). Low and high alcohol dose treatments were aggregated within young and older participant age groups.

Finally, when data loss occurred in within-subjects studies that resulted in unequal sample sizes represented in conditions (e.g., Sexton et al., 2002), the smaller sample size was used to compute the effect size (advice of Dr. Michael Borenstein, personal communication dated September 10, 2019).

Synthesis of Results

Random-effects meta-analyses were conducted in CMA with subgroup as the unit of analysis. For each analysis, the average effect, 95% confidence interval and 95% prediction interval were generated. Prediction intervals, which represent the plausible range of true effect sizes in a random-effects meta-analysis (Borenstein et al., pp. 127-133), were computed using the prediction interval worksheet available on the CMA website.

Risk of Bias Across Studies

The potential for publication bias was assessed by testing the presence of small study effects within each meta-analysis that included at least ten effect sizes (Cochrane Collaboration, 2011c). Specifically, the relationship between Hedge's g and its standard error was assessed. Tests of small study effects included visual inspection of funnel plots (i.e., Hedge's g by standard error) and Egger's regression tests. For Egger's regression test, the standard error of the effect size was set as the predictor and Hedge's g was set as the criterion in weighted least squares regression, wherein the inverse variances of the effect sizes served as weights (Sterne & Egger, 2005). Funnel plots are illustrated in Appendix D.

In post-hoc tests, significant Egger's regression tests were followed up by adding BAC as a second predictor to the regression. In all cases, these post-hoc tests were conducted on a smaller subset of the original effect sizes, occasionally with fewer than ten effect sizes, due to a lack of data required to verify the average BAC associated with each effect size. These post-hoc tests are described in more detail within *Chapter 3: Results*, below.

Additional Analyses

This dissertation sought to quantify the magnitude of the effect of different alcohol doses on driving performance and behaviour. Two approaches were considered: subgroup analysis, and meta-regression. It was noted that in the case of studies using within-subjects designs to assess the influence of multiple levels of alcohol on driving, the same participants may be represented in multiple effect sizes within the same analysis. Similarly, in the case of between-subjects designs with multiple BAC groups and a single control group, participants in the control group may be represented in multiple effect sizes in a comparison. For this reason, formal statistical tests – including meta-regression – were judged to be inappropriate.

Rather than conducting formal statistical tests, subgroup analyses were conducted to assess the magnitude of the effect of specific ranges of BAC on driving performance and behaviour. Effect sizes were parsed into bins as follows: Bin 1, any non-zero BAC up to 0.03%; Bin 2, BAC 0.04 – 0.06%; Bin 3, BAC 0.07 – 0.09%; Bin 4, BAC 0.10 – 0.12%. These bins were chosen for pragmatic reasons. Specifically, these bins most neatly captured the average BAC levels calculated for each effect size. The subgroup analyses only included comparisons for which there were ten or more effect sizes in the primary meta-analysis, and they only included effect sizes that could be reliably associated with an average BAC level. This required the reporting of an average BAC specifically associated with the driving component of a test battery, or an average pre-drive BAC and an average post-drive BAC (which allowed an average BAC for the driving component to be computed). Again, owing to participant overlap in multiple effect sizes included in a single analysis, differences between effect sizes associated with different BAC levels in the subgroup analysis were not subjected to formal statistical tests. Additionally, cannabis was not subjected to subgroup testing because unlike alcohol, there is no clear way to parse effect sizes by degree of intoxication.

Chapter 3: Results

Study Selection

The electronic search for studies, which was conducted in May 2018, yielded 5,923 citations. Additionally, studies were found via Google Scholar ($n = 7$; August 2019), in the references of research syntheses identified in the electronic search ($n = 12$), and through other informal means ($n = 18$). Altogether, 5,960 citations were identified. Of these, 2,266 were identified as duplicate citations, yielding 3,964 unique citations that were subjected to screening. Six-hundred sixteen citations passed screening and were subjected to full-text review. For an illustration of the study selection process, see Figure 1, below.

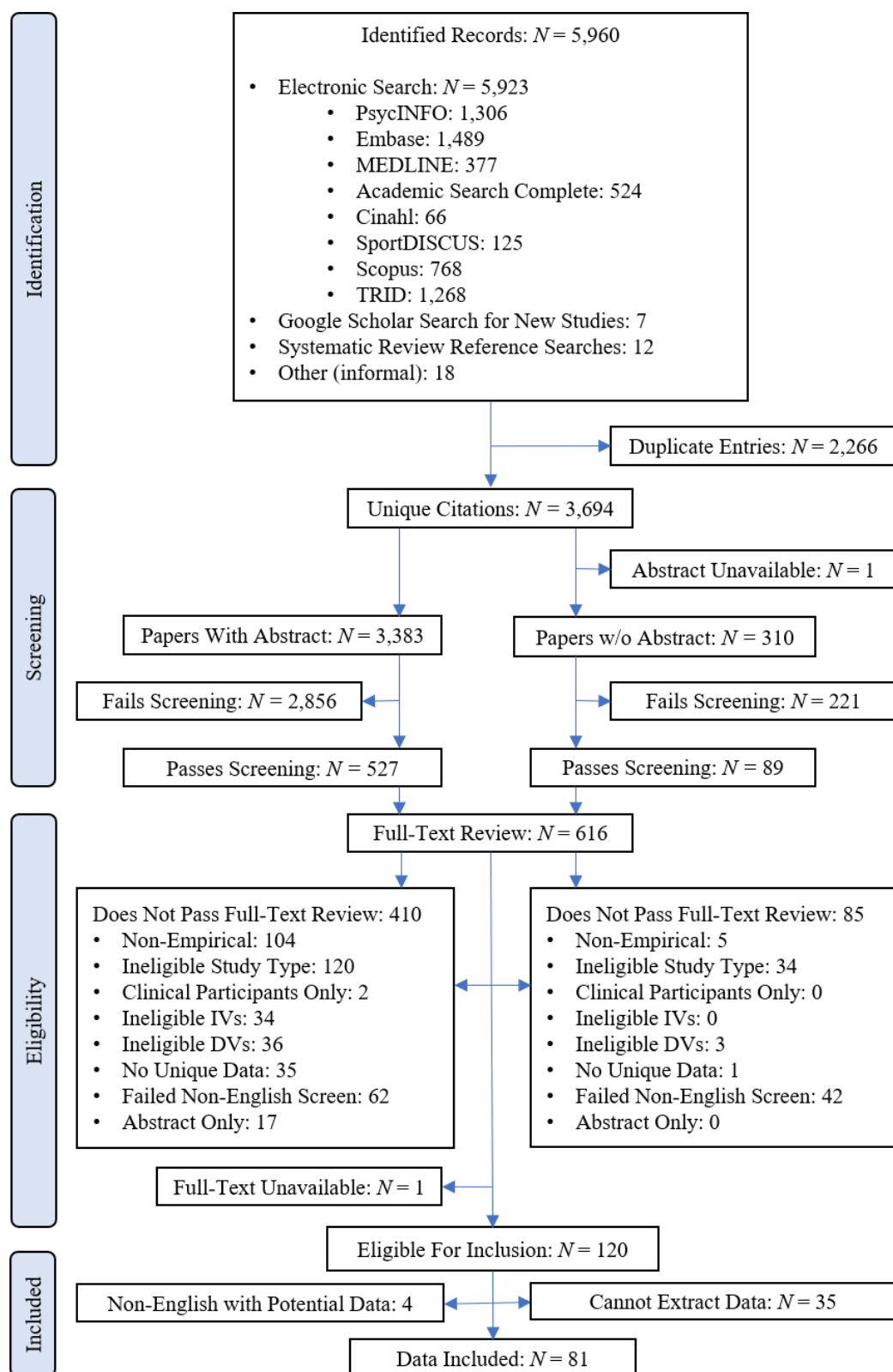


Figure 2. Study selection process.

At the end of the study selection process, 120 citations were judged to meet inclusion criteria. Of these, four were identified as non-English citations that were judged to possibly contain relevant data (Doenhoff, 1970; Bartl et al., 1998; Stephan et al., 2004; Schumacher, 2014 [Study 5]). However, due to the small number of identified non-English citations and some uncertainty in the accuracy of the electronic translations, these were not subjected to data extraction. Of the remaining 116 citations, 35 did not contain the necessary data for effect size computation (e.g., missing standard deviations, standard errors, etc.), or the data was not reported in a way to facilitate effect size computation (e.g., statistical data collapsed across eligible and ineligible conditions). Eighty-one citations were ultimately included in the meta-analysis.

Study Characteristics

The meta-analysis represents approximately 2,418 participants. For the slightly smaller subset of included studies where the number of female participants relative to all included participants could be identified ($n = 2,183$; see Table 1), the sample was approximately 43% female. Of the studies where the mean age of the included participants could be identified ($n = 1,724$; see Table 1), the sample had a mean age of 28.5 years. It should be noted that there is an overlap of four participants who participated in both Sexton et al. (2000) and Sexton et al. (2002). Additionally, participants from Ramaekers et al. (2000a) and from Study 1 of Ramaekers et al. (2000b) overlap and are counted only once in the meta-analysis. Both studies are included because they each contribute different measures. Additional study characteristics including setting, sample size, participant age, general drug conditions and driving performance and behaviour measures are reported in Table 1, below.

Table 1. Overview of studies included in the meta-analysis.

Study	Setting	Included N	M Age (SD)	Eligible IV's	Eligible DV's
Anderson et al., 2010	Simulator	73 (24 F)	M = 19.8 (2.1) for males in Placebo Cannabis condition (n = 25); M = 21.0 (2.6) for females in Placebo Cannabis condition (n = 15); M = 20.2 (2.6) for males in Active Cannabis condition (n = 24); M = 21.4 (3.6) for females in Active Cannabis condition (n = 9)	Cannabis, Placebo Control	RT: <i>Time to First Reaction [Hazard]</i> Speed: <i>Mean Speed in MPH</i> Long. Control: <i>SD of Mean Speed in MPH</i> Crashes: <i>Crash</i>
Arkell et al., 2019	Simulator	14 (3 F)	M = 27.5 (4.5), Range 21 – 38.	Cannabis, Placebo	Lat. Control: <i>SDLP</i> Speed: <i>Mean Speed</i> Headway: <i>Mean Headway</i> Long. Control: <i>SD of Headway, SD of Speed</i>
Arnedt et al., 2001	Simulator	18 (0 F)	M = 19.9 (2.3), Range 19 - 35	Alcohol, Non-Alcoholic Drink Control	Lat. Control: <i>Tracking Variability, Off-Road Incidents</i> Speed: <i>Speed Deviation</i> Long. Control: <i>Speed Variability</i>
Beard, 2012	Simulator	30 (16 F)	M = 40.03 (12.63), Range 20 – 64.	Alcohol, Placebo	RT: <i>Brake Reaction Time [Hazard]</i> Lat. Control: <i>Centerline Crossings</i> Collisions: <i>Crashes</i>

Study	Setting	Included N	M Age (SD)	Eligible IV's	Eligible DV's
Bernosky-Smith et al., 2011	Simulator	59 (59 F)	Unclear. For original $N = 60$, $M = 23.8$ (2.4), Range 21 – 29.	Alcohol, Placebo	Lat. Control: <i>Centerline Crossings, Road Edge Excursions</i> Speed: <i>Speed Exceedances</i> Crashes: <i>Collisions, Off-Road Accidents, Pedestrians Hit</i>
Bernosky-Smith et al., 2012	Simulator	40 (20 F)	Not reported.	Alcohol, Non-Treated Control	Speed: <i>Mean Driving Speed, Time Spent Speeding</i> Crashes: <i>Collisions</i>
Berthelon & Galy, 2018	Simulator	30 (Unclear F)	Age 18 (for $n = 15$ young novice drivers), 21 (for $n = 15$ young experienced drivers)	Alcohol, Non-Alcohol (unclear if placebo or untreated control)	Lat. Control: <i>SDLP</i> Speed: <i>Speed</i> Long. Control: <i>SD Speed</i>
Berthelon & Gineyt, 2014	Simulator	16 (8 F)	$M = 25.31$ (2.87), Range 21 – 29	Alcohol, Placebo	RT: <i>Highway Scenario, Urban Scenario [Hazards]</i> Lat. Control: <i>SDLP, Off-Lane Incidents</i> Speed: <i>Mean Speed</i> Headway: <i>Intervehicular Time</i> Long. Control: <i>SD of Speed</i> Crashes: <i>Collisions</i>
Bosker et al., 2012	On-Road (Highway)	24 (10 F)	$M = 23.6$ (SE = 0.6)	Dronabinol, Placebo Control	Lat. Control: <i>SDLP</i> Speed: <i>Mean Speed</i> Long. Control: <i>SD Speed</i>
Brands et al., 2019	Simulator	91 (26 F)	For $n = 30$ placebo group, $M = 21.9$ (2.2); for $n = 31$ Low THC group, $M = 22.2$ (1.8); for $n = 30$ High THC group, $M = 22.3$ (2.0).	Cannabis, Placebo	Lat. Control: <i>Lateral Control</i> Speed: <i>Mean Speed</i>

Study	Setting	Included N	M Age (SD)	Eligible IV's	Eligible DV's
Burns et al., 2002	Simulator	20 (10 F)	M = 32 (7.8), Range 21 – 45	Alcohol, Placebo	Lat. Control: <i>Lane Departures, SDLP, RMSE from Lane Centre</i> Speed: <i>Mean Speed</i> Long. Control: <i>SD of Speed, RMSE Speed, SD of Following Time Headway, RMSE of Time Headway</i>
Charlton & Starkey, 2015	Simulator	44 (23 F)	M = 32.84 (8.49), Range 20 – 47	Alcohol, Placebo	Lat. Control: <i>SDLP, Number of Centreline Crossings, Time Spent Over Centreline</i> Speed: <i>Mean Time Over 100 km/h</i>
Chen et al., 2016	Simulator	18 (Unclear F)	Range 18 – 24	Alcohol, Placebo	Speed: <i>Speed</i>
Christoforou et al., 2012	Simulator	49 (23 F)	M = 23.2 (2.7), Range 20 - 30	Alcohol, Untreated Control	RT: [Hazards] Lat. Control: <i>Variation in Within-Lane Position</i> Speed: <i>Average Traveling Speed</i> Long. Control: <i>Speed Variation</i>

Study	Setting	Included N	M Age (SD)	Eligible IV's	Eligible DV's
Downey et al., 2013	Simulator	80 (31 F)	M = 26.45 (5), Range 21 - 35	Cannabis, Alcohol, Combination, Placebo Control	RT: <i>Reaction Time to Emergencies [Hazard]</i> Lat. Control: <i>Steering Straddle Barrier Line, Violation Traffic Law Solid Line</i> Speed: <i>Violation Traffic Law Speed Limit, Initial Speed Freeway, Initial Speed City</i> Crashes: <i>Collisions</i>
Fillmore et al., 2008	Simulator	14 (7 F)	M = 23.5 (3.2), Range 21 – 30	Alcohol, Placebo	Lat. Control: <i>LPSD, Line Crossings</i> Speed: <i>Driving Speed</i> Crashes: <i>Off-Road and Other Vehicle Impacts</i>
Freydier et al., 2014	Simulator	32 (15 F)	Age 18 (for $n = 16$ novice drivers), 21 (for $n = 16$ experienced drivers)	Alcohol, Placebo	Lat. Control: <i>SDLP</i>
Harrison & Fillmore, 2005	Simulator	14 (7 F)	Unclear. For original $N = 24$, M = 23.8 (2.9), Range 21 – 31.	Alcohol, Baseline ¹	Lat. Control: <i>Within-Lane Deviation</i> Speed: <i>Speed</i> Crashes: <i>Crashes</i>
Harrison & Fillmore, 2011	Simulator	20 (Unclear F; for original $N = 40$, 20 F)	Unclear. For original $N = 40$, M = 24.0 (3.8), Range 21 – 35	Alcohol, Placebo	Lat. Control: <i>SDLP</i> Speed: <i>Drive Speed</i>
Harrison et al., 2007	Simulator	10 (5 F)	Unclear. For original $N = 30$, M = 22.5 (1.9).	Alcohol, Untreated Control	Lat. Control: <i>Within-Lane Deviation</i> Speed: <i>Speed</i> Crashes: <i>Crashes</i>

Study	Setting	Included N	M Age (SD)	Eligible IV's	Eligible DV's
Hartman et al., 2015	Simulator	18 (5 F)	M = 26.3 (4.2), Range 21 - 37	Cannabis, Alcohol, Combination, Placebo Control	Lat. Control: <i>SDLP, Lane Departures per Minute</i>
Helland et al., 2016	Simulator and Test Track	18 (0 F), simulator; 20 (0 F), test-track	Unclear. M = 28.7, Range 25 – 35 for original sample (N = 20).	Alcohol, Placebo	Lat. Control: <i>SDLP</i> Speed: <i>Average Speed</i> Long. Control: <i>SD of Speed</i> Crashes: <i>Collisions</i>
Horne & Baumber, 1991	Simulator	24 (24 F)	Range 20 - 25	Alcohol, Placebo	Lat. Control: <i>Position Variability</i> Long. Control: <i>Following Distance Variability</i> Headway: <i>Mean Following Distance</i>
Howard et al., 2007	Simulator	16 (Unclear F)	M = 46.2 (10.7)	Alcohol, Untreated Control	RT: <i>Braking Reaction Time [Hazard]</i> Lat. Control: <i>Variation in Lane Position</i> Crashes: <i>Crashes</i>
Howland et al., 2011	Simulator	67 (Unclear F; 47% F of unclear N)	Unclear; M = 22.9 (2.23), Range 21 – 30 for unclear N)	Alcohol ² , Placebo ²	Lat. Control: <i>Lane Position Variability</i> Speed: <i>Speed Deviation</i> Long. Control: <i>Speed Variability</i> Crashes: <i>Crashes</i>
Huemer & Vollrath, 2010	Simulator	23 (11 F)	M = 25.3 (5.9), Range 19 – 45.	Alcohol, Baseline ³	Lat. Control: <i>SDLP</i>
Jelen et al., 2011	Simulator	6 (0 F)	Not reported.	Alcohol, Untreated Control	RT: <i>Red Traffic Lights [Hazard]</i>

Study	Setting	Included N	M Age (SD)	Eligible IV's	Eligible DV's
Kay et al., 2013	Simulators	18 (Unclear F)	Range 21 – 34	Alcohol, Placebo	Lat. Control: <i>SD of Lane Position, Out of Lane</i> Long. Control: <i>SD of Speed</i>
Kenntner-Mabiala et al., 2015	Simulator	24 (11 F)	M = 30 (8.3), Range 23 – 53	Alcohol, Placebo	Lat. Control: <i>Lane Departures, SDLP</i> Speed: <i>Mean Speed</i> Crashes: <i>Collisions</i>
Kuypers et al., 2006	On-Road	18 (9 F)	M = 26.6 (5.4)	Alcohol, Untreated Control	RT: <i>BRT [Hazard]</i> Lat. Control: <i>SDLP</i> Speed: <i>Speed</i> Long. Control: <i>SD Speed</i>
Laude & Fillmore, 2015	Simulator	34 (20 F)	Range 21 – 34	Alcohol, Placebo	Lat. Control: <i>SDLP</i> Speed: <i>Average Drive Speed</i> Crashes: <i>Accident Frequency</i>
Laude & Fillmore, 2016	Simulator	40 (21 F)	M = 24.08 (4.03), Range 21 – 34	Alcohol, Placebo	Lat. Control: <i>SDLP</i> Speed: <i>Average Drive Speed</i> Crashes: <i>Accident Frequency</i>
Laude, 2016 (Study 3)	Simulator	12 (6 or 7 F)	M = 23.08 (6.35)	Alcohol, Placebo	Lat. Control: <i>SDLP</i> Speed: <i>Drive Speed</i>

Study	Setting	Included N	M Age (SD)	Eligible IV's	Eligible DV's
Lee et al., 2010	Simulator	108 (54 F)	For $n = 18$ males aged 21-34, $M = 26.56$; for $n = 18$ females aged 21-34, $M = 26.83$; for $n = 18$ males aged 38-51, $M = 43.22$; for $n = 18$ females aged 38-51, $M = 44.72$; for $n = 18$ males aged 55-68, $M = 59.56$; for $n = 18$ females aged 55-68, $M = 61.06$.	Alcohol, Placebo	Lat. Control: <i>Lane Deviation (SDLP)</i> Speed: <i>Average Speed</i> Long. Control: <i>Speed Deviation (SD of Speed)</i>
Lenne et al., 1999	Simulator	28 (14 F)	For $n = 14$ inexperienced drivers, $M = 18.9$ (0.7), Range 18 – 20; for $n = 14$ experienced drivers, $M = 27.4$ (1.8), Range 25 – 35.	Alcohol, Placebo	Lat. Control: <i>SDLP</i> Speed: <i>Mean Speed</i> Long. Control: <i>SD of Speed</i>
Lenne et al., 2003	Simulator	21 (12 or 13 F)	$M = 34.1$	Alcohol, Untreated Control	Lat. Control: <i>SD of Position</i> Speed: <i>Mean Speed</i> Long. Control: <i>SD of Speed</i>
Leung et al., 2012	Simulator	12 (10 F)	$M = 26.20$ (2.58), Range 23.5 – 30.8	Alcohol, Untreated Control	RT: <i>Braking Episodes [Hazard]</i> Speed: <i>Time Spent Speeding</i> Crashes: <i>Number of Crashes</i>
Liguori & Robinson, 2001	Simulator	15 (9 F)	$M = 32$, Range 21 – 45	Alcohol, Placebo	RT: <i>Brake Latency [Hazard]</i>
Liguori et al., 1998	Simulator	10 (3 F)	$M = 29$ (6)	Cannabis, Placebo Control	RT: <i>Brake Latency [Hazard]</i> Speed: <i>Mean Speed</i>

Study	Setting	Included N	M Age (SD)	Eligible IV's	Eligible DV's
Liguori et al., 1999	Simulator	18 (10 F)	M = 32 (6)	Alcohol, Placebo	RT: <i>Brake Latency [Hazard]</i>
Liguori et al., 2002	Simulator	12 (4 F)	M = 24 (3), Range 21 – 45	Cannabis, Alcohol, Combination, Placebo Control	RT: <i>Brake Latency [Hazard]</i>
Louwerens et al., 1987	On-Road	24 (12 F)	Range 22 – 45	Alcohol, Untreated Control	Lat. Control: <i>SD of Lateral Position</i> Long. Control: <i>Speed Variability</i>
Marczinski & Fillmore, 2009	Simulator	28 (12 F)	M = 22.6 (2.3), Range 21 – 28	Alcohol, Placebo	Lat. Control: <i>Within-Lane Deviation, Number of Center Line Crossings, Number of Edge Excursions</i> Long. Control: <i>Speed Deviation</i> Crashes: <i>Number of Accidents</i>
Marczinski et al., 2008	Simulator	40 (20 F)	M = 22.3 (2.0), Range 21 – 29	Alcohol, Placebo	Lat. Control: <i>Within-Lane Deviation, Number of Center Line Crossings, Number of Edge Excursions</i> Speed: <i>Speed, Number of Speed Limit Exceedances</i> Long. Control: <i>Speed Deviation</i> Crashes: <i>Number of Accidents</i>

Study	Setting	Included N	M Age (SD)	Eligible IV's	Eligible DV's
McCartney et al., 2017	Simulator	22 (0 F)	M = 23 (3)	Alcohol, Placebo	Lat. Control: <i>SDLP</i> , <i>Number of Lane Crossings</i> Speed: <i>Average Speed</i> Long. Control: <i>SD of Speed</i> Headway: <i>Distance Headway</i>
Mets et al., 2011	Simulator	27 (13 F)	M = 22.8 (1.4)	Alcohol, Placebo	Lat. Control: <i>SD Lateral Position</i> Speed: <i>Mean Speed</i> Long. Control: <i>SD of Speed</i> Crashes: <i>Collisions</i>
Price et al., 2018	Simulator	66 (28 F)	For $n = 33$ younger group, $M = 27.59$ (2.71). For $n = 33$ older group, $M = 60.06$ (3.76).	Alcohol, Placebo	Speed: <i>Average Speed</i>
Ramaekers et al., 1992	On-Road	16 (8 F)	Range 22 – 35	Alcohol, Placebo	Lat. Control: <i>SDLP</i> Long. Control: <i>SD Speed</i>
Ramaekers et al., 2000a ⁴	On-Road	18 (9 F)	Range 20 – 28.	Cannabis, Alcohol, Combination, Placebo	Lat. Control: <i>SDLP</i> , <i>Percentage Time Out of Lane</i> Long. Control: <i>SD of Headway</i>
Ramaekers et al., 2000b (Study 1) ⁴	On-Road (Highway)	18 (9 F)	Not reported.	Cannabis, Alcohol, Combination, Placebo Control	RT: <i>Decelerations in Car-Following Test [Hazard]</i>
Robbe, 1998 (Study 1)	On-Road (Closed Highway)	23 (12 F)	Not reported.	Cannabis, Placebo Control	Lat. Control: <i>SDLP</i> Speed: <i>Mean Speed</i> Long. Control: <i>SD Speed</i>

Study	Setting	Included N	M Age (SD)	Eligible IV's	Eligible DV's
Robbe, 1998 (Study 2)	On-Road (Open Highway)	15 (7 F)	Not reported.	Cannabis, Placebo Control	RT: <i>Movements of Preceding Vehicle</i> [Unclear] Lat. Control: <i>SDLP</i> Speed: <i>Speed</i> Headway: <i>Mean Distance</i> Long. Control: <i>SD Distance</i>
Roberts, 2016 (Exp. 2)	Simulator	40 (13 F)	For $n = 20$ controls, $M = 24.9$ (3.7); for $n = 20$ DUI offenders, $M = 23.4$ (2.7).	Alcohol, Placebo	Lat. Control: <i>LPSD, Line Crossings</i> Speed: <i>Average Speed</i> Long. Control: <i>Speed SD</i> Crashes: <i>Collisions</i>
Ronen et al., 2008	Simulator	14 (4 F)	$M = 26.1$ (1.3)	Cannabis, Alcohol, Placebo Control	Lat. Control: <i>RMS Lane Position</i> Speed: <i>Average Speed</i> Long. Control: <i>RMS Longitudinal Speed</i> Crashes: <i>Number of Collisions</i>
Ronen et al., 2010	Simulator	12 (5 F)	$M = 26.1$, Range 24 - 29	Cannabis, Alcohol, Combination, Placebo Control	Lat. Control: <i>RMS Lane Position</i> Speed: <i>Average Speed</i> Long. Control: <i>RMS Speed</i> Crashes: <i>Number of Collisions</i>
Rupp et al., 2007	Simulator	26 (18 F)	Unclear. For original $N = 29$, $M = 22.6$ (1.2), Range 21 - 25.	Alcohol, Placebo	Lat. Control: <i>Lane Variability, Off-Road Events</i> Long. Control: <i>Speed Variability</i>

Study	Setting	Included N	M Age (SD)	Eligible IV's	Eligible DV's
Schumacher et al., 2017 ⁵	On-Road	17 (Unclear F; 7 F for original 19)	Unclear. For original $N = 19$, $M = 43$ (11.07), Range 23 – 58.	Alcohol, Placebo	RT: <i>Brake RT</i> [Hazard] Speed: <i>Mean Speed</i> Lat. Control: <i>SDLP</i> Long. Control: <i>SD Speed</i>
Sexton, 1997	On-Road, Simulator	18 (18 F)	Not reported.	Alcohol, Placebo	RT: <i>Pulling-In Events, Pulling-Out Events</i> [Hazards] Lat. Control: <i>SD of Lateral Deviation</i> Long. Control: <i>SD of Following Distance</i> Headway: <i>Mean Following Distance</i>
Sexton et al., 2000	Simulator	15 (0 F)	$M = 27.0$ (7.52)	Cannabis, Placebo Control	RT: <i>Pulling Out RT</i> [Hazard], <i>Braking RT</i> [Hazard] Lat. Control: <i>SDLP</i> Speed: <i>Average Speed</i>
Sexton et al., 2002	Simulator	21 (0 F)	$M = 24.9$ (3.51)	Cannabis, Alcohol, Combination, Placebo Control	RT: <i>Pulling-Out Events</i> [Hazard], <i>Braking Events</i> [Hazard] Lat. Control: <i>SDLP</i> Speed: <i>Average Speed</i>

Study	Setting	Included N	M Age (SD)	Eligible IV's	Eligible DV's
Simons et al., 2012	Simulator	16 (4 F)	M = 25.7, Range 21 – 37.	Alcohol, Non-Alcohol ⁵	Lat. Control: <i>SDLP, Number of Line Crossings</i> Speed: <i>Average Speed, Violating Speed Limit, Ramp Entry Velocity, Velocity When Merging</i> Headway: <i>Time Headway, Distance Headway</i> Long. Control: <i>SD of Speed</i> Crashes: <i>Accidents</i>
Sklar et al., 2014	Simulator	72 (31 F)	For $n = 12$ younger adults in placebo condition, $M = 27.75$ (2.1); for $n = 13$ younger adults in 0.04% BAC condition, $M = 28.69$ (3.3); for $n = 11$ younger adults in 0.065% BAC condition, $M = 27.18$ (2.0); for $n = 12$ older adults in placebo condition, $M = 62.25$ (4.5); for $n = 13$ older adults in 0.04% BAC condition, $M = 58.54$ (2.8); for $n = 11$ older adults in 0.065% BAC condition, $M = 60.55$ (4.1).	Alcohol, Placebo	Speed: <i>Average Speed</i> Lat. Control: <i>LPSD</i> Long. Control: <i>SD of Average Speed</i>

Study	Setting	Included N	M Age (SD)	Eligible IV's	Eligible DV's
Starkey & Charlton, 2014	Simulator	49 (Unclear F; for original $N = 61$, 28 F).	Unclear. For original $N = 61$, $M = 31.11$ (8.34), Range 20 – 50.	Alcohol, Placebo	Speed: <i>Seconds Over 100 km/hr</i> Lat. Control: <i>Edge Line Crossings, Seconds Over Edge Line, SD of Lane Position, Centre Line Crossings, Seconds Over Centre Line</i>
Strayer et al., 2006	Simulator	40 (15 F)	$M = 25$, Range 22 – 34	Alcohol, Untreated Control	RT: <i>Brake RT [Hazard]</i> Speed: <i>Speed</i> Long. Control: <i>SD Following Distance</i> Headway: <i>Mean Following Distance</i> Crashes: <i>Total Accidents</i>
Subramaniyam et al., 2018	Simulator	8 (0 F)	$M = 29.63$ (3.16)	Alcohol, Untreated Control	Speed: <i>Over Speed Rate</i> Collisions: <i>Accident Rate/Crash Rate</i>
Tremblay et al., 2015	Simulator	16 (6 F)	For $n = 8$ experimental group, $M = 21.6$ (2.32); for $n = 8$ control group, $M = 20.9$ (2.35).	Alcohol, Time-Matched Untreated Control	Speed: <i>Percentage of Time Spent Over Speed Limit</i>
van der Sluiszen et al., 2016	On-Road (Highway)	25 (13 F)	$M = 33.4$ (8.9)	Alcohol, Untreated Control	Lat. Control: <i>SDLP</i> Long. Control: <i>SD of Speed</i>
Van Dyke & Fillmore, 2014	Simulator	50 (14 F)	For $n = 25$ DUI offenders, $M = 25.95$ (4.11); for $n = 25$ controls, $M = 24.65$ (3.41); for all ($N = 50$), Range 21 – 34.	Alcohol, Placebo	Speed: <i>Drive Speed</i> Lat. Control: <i>LPSD, Centerline and Road Edge Crossings</i>

Study	Setting	Included N	M Age (SD)	Eligible IV's	Eligible DV's
Van Dyke & Fillmore, 2015	Simulator	50 (14 F)	Range 21 – 34	Alcohol, Placebo	Lat. Control: <i>SDLP, Lane Exceedances</i> Speed: <i>Average Speed</i> Crashes: <i>Traffic Accidents</i>
Van Dyke & Fillmore, 2017	Simulator	20 (10 F)	M = 24.0 (3.0), Range 21 – 35	Alcohol, Placebo	Speed: <i>Average Drive Speed</i> Crashes: <i>Accident Frequency</i>
Veldstra et al., 2012 (Study 1)	Simulator	17 (8 F)	M = 23.6 (3.8)	Alcohol, Placebo	Lat. Control: <i>SDLP</i> Speed: <i>Average Speed</i> Long. Control: <i>SD of Speed</i> Crashes: <i>Crashes</i>
Veldstra et al., 2012 (Study 2)	Simulator	19 (9 F)	M = 30.8 (5.65), Range 21 - 40	Alcohol, Placebo	Lat. Control: <i>SDLP</i> Speed: <i>Average Speed</i> Long. Control: <i>SD of Speed</i> Crashes: <i>Crashes</i>
Veldstra et al., 2015 ⁶	Simulator	24 (10 F)	M = 23.6 (3.0)	Dronabinol, Placebo Control	Lat. Control: <i>SDLP</i>
Vermeeren & O'Hanlon, 1998	On-Road	24 (12 F)	M = 31.5 (8.5)	Alcohol, Untreated Control	Lat. Control: <i>SDLP</i>
Vermeeren et al., 2002a	On-Road	19 (10 F)	M = 34.4 (7.5)	Alcohol, Untreated Control	Lat. Control: <i>SDLP</i>
Vermeeren et al., 2002b (Part 1)	On-Road (Highway)	30 (15 F)	M = 31.6 (6.9), Range 21 – 45	Alcohol, Placebo	Lat. Control: <i>SDLP</i> Long. Control: <i>SD of Speed</i>
Verster et al., 2002 (Part 1)	On-Road (Highway)	30 (15 F)	M = 24.0 (2.4)	Alcohol, Placebo	Lat. Control: <i>SDLP</i> Long. Control: <i>SD Speed</i>

Study	Setting	Included N	M Age (SD)	Eligible IV's	Eligible DV's
Vollrath & Fischer, 2017 (Study 1)	Simulator	48 (0 F)	Unclear. For all $N = 48$, $M = 23.2$ (2.0), Range 20 – 29	Alcohol, Placebo	RT: <i>Parking Car, Pedestrian [Hazard]</i> Speed: <i>Speed</i> Crashes: <i>Number of Accidents</i>
Vollrath & Fischer, 2017 (Study 2)	Simulator	42 (0 F)	Unclear. For $N = 63$ (includes one additional non-eligible group ³), $M = 23$ (2.3).	Alcohol, Placebo ⁷	RT: <i>Parking Car, Pedestrian [Hazard]</i> Speed: <i>Speed</i> Crashes: <i>Number of Accidents</i>
Wan et al., 2017	Simulator	28 (14 F)	$M = 23.43$ (3.12), Range 21 – 36	Alcohol, Placebo	RT: <i>Yellow Lights [Hazard]</i> Lat. Control: <i>SDLP</i> Long. Control: <i>SD of Driving Speed</i> Crashes: <i>Accidents</i>
Weafer & Fillmore, 2012	Simulator	20 (10 F)	$M = 23.2$ (2.6), Range 21 – 31	Alcohol, Placebo	Lat. Control: <i>LPSD, Number of Line Crossings</i>
Weafer et al., 2008 (Study 1)	Simulator	23 (10 F)	$M = 22.0$ (1.7)	Alcohol, Placebo	Lat. Control: <i>Deviation of Lane Position</i> Speed: <i>Average Driving Speed</i> Long. Control: <i>SD of Average Speed</i> Crashes: <i>Off-Road Crashes/Impacts</i>

Study	Setting	Included N	M Age (SD)	Eligible IV's	Eligible DV's
Weafer et al., 2008 (Study 2)	Simulator	8 (3 F)	M = 23.1 (1.2)	Alcohol, Placebo	Lat. Control: <i>Deviation of Lane Position</i> Speed: <i>Average Driving Speed</i> Long. Control: <i>SD of Average Speed</i> Crashes: <i>Off-Road Crashes/Impacts</i>
Weiler et al., 2000	Simulator	40 (25 F)	M = 31, Range 25 – 44	Alcohol, Placebo	RT: <i>Blocking Vehicle [Hazard]</i> Lat. Control: <i>Root Mean Square Deviation, Left-Lane Excursions</i> Crashes: <i>Collisions</i>
Zhang et al., 2014	Simulator	22 (0 F)	Unclear. For original $N = 25$, $M = 25$ (4.1), Range 20 – 35.	Alcohol, Placebo	Lat. Control: <i>LPSD</i> Speed: <i>Average Speed</i> Long. Control: <i>SD of Speed</i>

Note that this table describes all studies meeting inclusion criteria for the meta-analysis prior to data extraction attempts. Likewise, *Eligible IV's* describes relevant drug driving conditions included in a study (i.e., whether a study includes cannabis, alcohol or both; for more specific details on drug driving conditions, see Table 2), and *Eligible DV's* describes all relevant driving performance and behaviour measures included in a study (i.e., in the method section, results section, or both). It should be noted that any given DV that was identified as contributing only duplicate data with a DV from another study was ineligible for inclusion under *Criterion 6* and was therefore omitted from this table. Due to incomplete or incompatible reporting, not all relevant study data is included in the meta-analysis. Additionally, *Included N* describes the maximum number of participants represented in the meta-analysis per study, but does not necessarily correspond to the number of participants represented in each *Eligible IV* and/or *Eligible DV* (e.g., as in between-subjects designs, or as a consequence of attrition or data loss). Please refer to individual forest plots for specific information on data included in the meta-analysis.

1. This is a between-subjects study where one group of participants received alcohol and one received placebo; and, both groups completed baseline testing. Data was reported in terms of change scores, which complicated effect size computation. Ultimately, the change score comparing alcohol to baseline was used to compute the effect size included in the meta-analysis.
2. Caffeinated beverage conditions excluded from meta-analysis.
3. This is a totally within-subjects study where participants received both alcohol and placebo in a counterbalanced order; and, they completed baseline testing prior to consuming beverages. However, data was reported in terms of change scores, which complicated effect size computation. Ultimately, the change score comparing alcohol to baseline was used to compute the effect size included in the meta-analysis.
4. Studies report upon a common participant dataset.

5. In the alcohol condition, alcohol, along with a placebo drug, was administered within orange juice. In the non-alcohol condition, participants received a placebo drug with orange juice. It is unclear if the orange juice in the non-alcohol condition was meant to act as placebo for alcohol.

5. Data for this study was extracted and included from Schumacher et al. (2011). However, Schumacher et al. (2017) was ultimately deemed an *included* study because it contained more useable information (other than statistical data) than the 2011 poster.

6. Veldstra et al. (2015) and Bosker et al. (2012) report on a common dataset. Specifically, Bosker et al. (2012) reports driving data collected on-road, and Veldstra et al. (2015) re-reports the same on-road data; thus, the on-road data reported in Bosker et al. (2012) is eligible for inclusion, but the same data reported in Veldstra et al. (2015) is not eligible for inclusion. However, Veldstra et al. (2015) also reports on driving data collected during driving simulation; this data is not reported in Bosker et al. (2012). Thus, only the driving data collected during driving simulation is eligible for inclusion from the Veldstra et al. (2015) paper.

7. This study had both a placebo and a sober group. Participants in the sober group received a beverage but were informed that it did not contain alcohol. The placebo group was included, but the sober group was excluded from the meta-analysis.

Studies that were eligible for inclusion, but which were not included due to the inability to compute standardized mean difference effect sizes, are reported in Appendix B. Notable recent studies that investigated the effects of cannabis on driving performance and behaviour, but which did not report data needed for effect size computation, included Hartley et al. (2019), Micallef et al. (2018), Hartman et al. (2016) and Lenne et al. (2010). Attempts were made to recover the original data from study authors, but as of the time of this writing, the data have not been received. Additionally, older studies that investigated the effects of cannabis on driving performance and behaviour, but which did not report data needed for effect size computation, included Smiley et al. (1987), Smiley et al. (1985), Stein (1985), Sutton (1983), Attwood et al. (1981), Moskowitz et al. (1976a) and Rafaelsen et al. (1973a, 1973b). Finally, some notable studies that investigated the effects of cannabis on driving, but which were not deemed eligible for inclusion, included Crancer et al. (1969), Moskowitz et al. (1976b) and Ménétrety et al. (2005), which involved tasks that were not deemed sufficiently similar to simulated or on-road driving (*Criterion 2*); Krueger and Vollrath (2000), in which researchers did not experimentally control the administration of cannabis (*Criterion 4*); and Biasotti et al., (1986), in which driving measures were combined using factor analysis to yield composite measures (*Criterion 5*).

In addition to study characteristics, participant drug use inclusion criteria, reported drug use frequency, specific drug driving conditions and drug administration details are reported in Table 2, below. Participants were typically occasional, non-dependent users of the drugs administered in the studies. Alcohol was administered in doses up to 0.12% BAC, and cannabis was typically low-strength in terms of THC, with concentrations increasing in more recent studies.

Table 2. Overview of participant drug use inclusion criteria and reported frequency, and drug driving conditions.

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Anderson et al. 2010	<p><i>For all eligible participants:</i></p> <p>Cannabis: “Occasional marijuana smokers” who use 1 – 10 times per month.</p> <p>Alcohol: No current or previous alcohol dependence.</p>	<p>Cannabis: For $n = 25$ males in placebo group, average 4.6 (2.8) times using marijuana per month. For $n = 15$ females in placebo group, average 4.5 (2.9) times per month. For $n = 24$ males in Active THC group, average 4.9 (2.8) times per month. For $n = 9$ females in Active THC group, average 4.1 (3.0) times per month.</p> <p>Alcohol: For $n = 25$ males in placebo group, average 11.0 (6.3) drinks per week. For $n = 15$ females in placebo group, average 7.4 (8.7) drinks per week. For $n = 24$ males in Active THC group, average 12.6 (8.1) drinks per week. For $n = 9$ females in Active THC group, average 9.1 (7.4) drinks per week.</p>	<p>Comparison:</p> <p>1. 0% (~0 mg) THC cannabis</p> <p>Cannabis:</p> <p>1. 2.9% (~22.9 mg) THC cannabis</p>	<p>Cannabis: Cannabis cigarettes (average weight 0.790 grams), including placebo, smoked in a structured smoking paradigm until totally consumed.</p> <p>Alcohol: N/A</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Arkell et al., 2019	<p data-bbox="373 253 688 282"><i>For all eligible participants:</i></p> <p data-bbox="373 315 699 925">Cannabis: Ten or more previous experiences using cannabis; less than twice weekly use of cannabis in the past three months; no history of “clinically significant adverse response” during cannabis use; no “moderate or severe substance use disorder as assessed by an addiction medicine specialist” (p. 2714); and no interest in treatments to reduce cannabis consumption. Required to abstain from illicit drugs (assumed to include cannabis) throughout the entirety of the study. No positive oral fluid screen for cannabis prior to start of study sessions.</p> <p data-bbox="373 958 699 1136">Alcohol: Not reported. Required to abstain from alcohol from the night prior to testing. No positive test for breath alcohol prior to start of study sessions.</p>	<p data-bbox="726 253 1031 282"><i>For all eligible participants:</i></p> <p data-bbox="726 315 1031 464">Cannabis: Participants used cannabis on M = 4.5 (4.8) days in the past 28 days and M = 11.2 (8) days in the last three months.</p> <p data-bbox="726 496 1031 587">Alcohol: M = 7.1 (5.3) drinking occasions per month.</p>	<p data-bbox="1073 253 1230 282">Comparison:</p> <ol data-bbox="1073 282 1514 341" style="list-style-type: none"> 1. Placebo THC (from <1% THC, <1% CBD cannabis) <p data-bbox="1073 341 1199 370">Cannabis:</p> <ol data-bbox="1073 370 1514 496" style="list-style-type: none"> 1. 125 mg THC (from 11% THC, <1% CBD cannabis) 2. 125 mg THC (from 11% THC, 11% CBD cannabis) 	<p data-bbox="1541 253 1866 464">Cannabis: Vapor from cannabis, including placebo, inhaled in a structured vaporization paradigm for 5 minutes or until no vapor was visible during vaporization (whichever was later).</p> <p data-bbox="1541 496 1698 526">Alcohol: N/A</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Arnedt et al., 2001	<p><i>For all eligible participants:</i></p> <p>Cannabis: Score <5 on Drug Abuse Screening Test (no evidence of drug abuse; assumed to include cannabis). Required to abstain from drugs (assumed to include cannabis) from 48 hours prior to start of first study session to end of study.</p> <p>Alcohol: Score <9 on Alcohol Dependence Scale (no evidence of alcohol abuse). Required to abstain from 48 hours prior to start of first study session to end of study.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Not reported.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Non-alcoholic drink control¹ <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BAC 0.05% 2. Target BAC 0.08% 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose consisted of 100% ethanol in tonic water, divided into two beverages. Non-alcoholic drink control consisted of tonic water.</p>
Beard, 2012	<p><i>For all eligible participants:</i></p> <p>Cannabis: No substance use disorder (assumed to include cannabis).</p> <p>Alcohol: No alcohol use disorder. Required to abstain from alcohol for 24 hours prior to testing.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: All “had a history” of drinking alcohol at least weekly; M = 5.90 (6.31) alcoholic drinks weekly.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BAC 0.02% 2. Target BAC 0.05% 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose consisted of vodka in orange juice. Placebo consisted of pure orange juice.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Bernosky-Smith et al., 2011	<p><i>For all eligible participants:</i></p> <p>Cannabis: No illicit or psychoactive drug use currently or in the previous six months (assumed to include cannabis); no positive urine test for illicit drugs (assumed to include cannabis).</p> <p>Alcohol: Drink at least once monthly, with an Alcohol Use Disorders Identification Test (AUDIT) score of 12 or less.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: For $n = 30$ high-frequency binge drinker group, $M = 8.4$ (4.2) drinks per week. For $n = 30$ low-frequency binge drinker group, $M = 6.1$ (5.0) drinks per week.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. 0.2 g/kg alcohol 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose administered as 95% alcohol in tonic water, served in an opaque foam cup with a lid to be consumed through a straw. Placebo administered as tonic water, served in an opaque foam cup with a lid covered with alcohol, to be consumed through a straw also covered with alcohol.</p>
Bernosky-Smith et al., 2012	<p><i>For all eligible participants:</i></p> <p>Cannabis: No positive urine test for illicit drugs (assumed to include cannabis).</p> <p>Alcohol: No “hazardous drinkers” (i.e., no score >12 on AUDIT).</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: “Moderate drinkers.” Also referred to as “binge drinkers.”</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Baseline driving <p>Alcohol:</p> <ol style="list-style-type: none"> 1. 0.8 g/kg 95% alcohol for males, and 8% less for females (unclear Target BAC; $M = 0.06\%$ [0.02%] at start of drive) 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose consisted of 95% alcohol in lemonade, divided into ten 50 mL drinks to be consumed over two hours.</p>
Berthelon & Galy, 2018	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Not reported.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Not reported.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Target BAC 0.00% <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BAC 0.02% 2. Target BAC 0.05% 	<p>Cannabis: N/A</p> <p>Alcohol: Not reported.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Berthelon & Gineyt, 2014	<p><i>For all eligible participants:</i></p> <p>Cannabis: No previous drug abuse (assumed to include cannabis).</p> <p>Alcohol: Previous alcohol consumption, but no “excessive drinkers” or previous alcohol abuse. Required to abstain the day before the study session.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Not reported.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BAC 0.03% 2. Target BAC 0.05% 3. Target BAC 0.08% 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose consisted of vodka and orange juice. Placebo consisted of pure orange juice.</p>
Bosker et al. 2012	<p>Cannabis: For $n = 12$ “occasional” user group, 5 – 36 times using cannabis yearly, abstention from “any drugs” from one week prior to medical exam to end of study, and no positive test for THC at start of experiment. For $n = 12$ “heavy” user group, >160 times using cannabis yearly and positive test for THC at start of experiment.</p> <p><i>For all eligible participants (i.e., both “occasional” and “heavy” user groups):</i></p> <p>Alcohol: No “excessive drinking;” no history of addiction to non-cannabinoids (assumed to include alcohol); no alcohol during 24 hours prior to testing.</p>	<p>Cannabis: “Occasional” users ($n = 12$) reported $M = 274.1$ ($SE = 89.6$) times using cannabis across lifetime. “Heavy” users ($n = 12$) reported $M = 2444.2$ ($SE = 708.8$) times using cannabis across lifetime, and a range of 4.7 – 23.1 joints per week.</p> <p>Alcohol: Not reported for either user group.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo dronabinol <p>Cannabis:</p> <ol style="list-style-type: none"> 1. 10 mg dronabinol 2. 20 mg dronabinol 	<p>Cannabis: Dronabinol, including placebo, administered orally in capsule form.</p> <p>Alcohol: N/A</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Brands et al., 2019	<p><i>For all eligible participants:</i></p> <p>Cannabis: “Regular recreational,” non-medical users with “recent cannabis use” (based on drug test), but who did not have a current or past cannabis dependency (based on DSM-IV). Required to abstain for 48 hours prior to practice session and throughout study (verified with drug test).</p> <p>Alcohol: No prior substance dependency (based on DSM-IV; assumed to include alcohol). Required to abstain for 48 hours prior to practice session and throughout study (verified with breathalyzer).</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: “Regular” use of cannabis (i.e., 1 to 4 days per week). For $n = 30$ placebo participants, $M = 2.8$ (1.1) days using per week; for $n = 31$ low THC participants, $M = 2.4$ (0.9) days using per week; for $n = 30$ high THC participants, $M = 2.6$ (0.8) days using per week.</p> <p>Alcohol: Not reported.</p>	<p>Comparison:</p> <p>1. 0.009% (~0.07 mg) THC, <0.5% CBD cannabis</p> <p>Cannabis:</p> <p>1. 12.5% (~93.75 mg) THC, <0.5% CBD cannabis</p> <p>Note that a median split was used to divide participants who received active cannabis into High THC and Low THC groups.</p>	<p>Cannabis: Cannabis cigarettes (approximate weight 750 mg), including placebo, smoked ad libitum for ten minutes.</p> <p>Alcohol: N/A.</p>
Burns et al., 2002	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Not reported. Participants provided breath samples on arrival, likely to verify that they had not been drinking.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Not reported.</p>	<p>Comparison:</p> <p>1. Placebo alcohol</p> <p>Alcohol:</p> <p>1. Target BAC 0.08%</p>	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose consisted of vodka in cream soda. Placebo consisted of pure cream soda.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Charlton & Starkey, 2015	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: “Occasional” but not “excessive” alcohol use, with a score of <8 on AUDIT. Required to abstain for 24 hours prior to study sessions and have zero BAC at the start of study sessions.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: “Moderate but not excessive” alcohol consumers, $M = 5.0$ (1.74), range 2 – 8 on AUDIT.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BAC 0.03% (ascending section of curve, i.e., Block 2) 2. Target BAC 0.05% (ascending section of curve, i.e., Block 2) 3. Target BAC 0.05% (peak, i.e., Blocks 3 and 4) 4. Target BAC 0.08% (peak, i.e., Blocks 3 and 4) 5. Target BAC 0.03% (descending section of curve, i.e., Block 5) 6. Target BAC 0.05% (descending section of curve, i.e., Block 5) 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose administered as vodka in orange juice, divided into four beverages. Placebo consisted of orange juice, divided into four beverages, topped with 5 mL of vodka each.</p>
Chen et al., 2016	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Not reported. Required to abstain the day before study sessions and have zero BAC at the start of study sessions.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Not reported.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BAC 0.05% 2. Target BAC 0.08% 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose administered as absolute alcohol in orange juice. Placebo administered as pure orange juice topped with 3 mL of white wine.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Christoforou et al., 2012	<p data-bbox="373 256 688 289"><i>For all eligible participants:</i></p> <p data-bbox="373 315 701 467">Cannabis: Unclear. Required to abstain from drugs (assumed to include cannabis) for 18 hours prior to study sessions.</p> <p data-bbox="373 493 680 651">Alcohol: Unclear. Required to abstain for 18 hours prior to study sessions and test negative for alcohol at the start of study sessions.</p>	<p data-bbox="724 256 1031 289"><i>For all eligible participants:</i></p> <p data-bbox="724 315 995 347">Cannabis: Not reported.</p> <p data-bbox="724 373 1052 773">Alcohol: Of sample, 32.7% were “heavy drinkers” (i.e., >3 times weekly alcohol consumption), 12.0% were “moderate drinkers” (i.e., 2 or 3 times weekly alcohol consumption), 47.0% “light drinkers” (i.e., <2 times weekly alcohol consumption) and 8.2% were “occasional-drinkers” (i.e., <2 times monthly alcohol consumption).</p>	<p data-bbox="1073 256 1226 289">Comparison:</p> <ol data-bbox="1073 289 1310 321" style="list-style-type: none"> 1. Baseline driving <p data-bbox="1073 321 1178 354">Alcohol:</p> <ol data-bbox="1073 354 1310 380" style="list-style-type: none"> 1. 40 mL of ethanol 	<p data-bbox="1541 256 1717 289">Cannabis: N/A</p> <p data-bbox="1541 315 1877 500">Alcohol: Alcohol consisted of 100 mL of vodka, whisky or gin containing approximately 40 mL of ethanol, consumed either straight or in a mix such as fruit juice.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Downey et al. 2013	<p><i>For all eligible participants:</i></p> <p>Cannabis: No current or previous substance abuse (assumed to include cannabis). No positive blood test for cannabinoids.</p> <p>Alcohol: No current or previous substance abuse (assumed to include alcohol).</p>	<p>Cannabis: 48 “regular” cannabis users, 32 “non-regular” cannabis users based on a “Frequency of Cannabis Use” questionnaire.</p> <p>Alcohol: Not reported for either user group.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 0% THC (placebo) cannabis + placebo alcohol <p>Cannabis:</p> <ol style="list-style-type: none"> 1.78% THC cannabis + placebo alcohol 3.42% THC cannabis + placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 0% THC cannabis + 0.03% Target BAC 0% THC cannabis + 0.05% Target BAC <p>Combination:</p> <ol style="list-style-type: none"> 1.78% cannabis + 0.03% Target BAC 1.78% cannabis + 0.05% Target BAC 3.42% THC cannabis + 0.03% Target BAC 3.42% cannabis + 0.05% Target BAC 	<p>Cannabis: Cannabis cigarettes, including placebo, smoked in a structured smoking procedure with ten inhalations.</p> <p>Alcohol: Alcohol dose consisted of vodka in orange juice. Placebo alcohol consisted of pure orange juice.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Fillmore et al., 2008	<p><i>For all eligible participants:</i></p> <p>Cannabis: No substance abuse disorder (i.e., did not meet DSM-IV criteria for dependence or withdrawal; assumed to include cannabis); no positive urine test for THC.</p> <p>Alcohol: No substance abuse disorder (i.e., did not meet DSM-IV criteria for dependence or withdrawal; assumed to include alcohol); scored <5 on Short-Michigan Alcoholism Screening Test [S-MAST]). Required to abstain for 24 hours prior to study sessions.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: $M = 1.7$ (0.7) drinking occasions per week, with “typical dose” of $M = 1.3$ (0.5) mL/kg (approx. 5 bottles of 5% alcohol beer for a 75 kg person per week).</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BAC 0.08% 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose administered as 94.6% alcohol in carbonated mix, divided into two beverages. Placebo administered as carbonated mix, divided into two beverages each topped with 3 mL of alcohol, served in glasses sprayed with alcohol mist.</p>
Freydier et al., 2014	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: “Social drinkers” (i.e., approx. 2 servings of alcohol (denominator unclear), “not every day” and “chiefly in a social context” [p. 14]).</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Not reported.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BAC 0.02% 2. Target BAC 0.05% 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose administered as vodka in orange juice. Placebo administration not described.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Harrison & Fillmore, 2005	<p data-bbox="373 251 688 284"><i>For all eligible participants:</i></p> <p data-bbox="373 316 699 560">Alcohol: No abstinence from alcohol, no substance use disorder (assumed to include alcohol), and no treatment for issues associated with alcohol use. Required to abstain for 24 hours prior to study sessions</p> <p data-bbox="373 592 699 771">Cannabis: No substance use disorder (assumed to include cannabis), and no treatment for issues associated with drug use (assumed to include cannabis).</p>	<p data-bbox="724 251 1035 284"><i>For all eligible participants:</i></p> <p data-bbox="724 316 997 349">Cannabis: Not reported.</p> <p data-bbox="724 381 1035 462">Alcohol: Average weekly alcohol dose of 3.2 (2.7) mL/kg.</p>	<p data-bbox="1073 251 1228 284">Comparison:</p> <p data-bbox="1073 284 1302 316">1. Baseline driving</p> <p data-bbox="1073 316 1176 349">Alcohol:</p> <p data-bbox="1073 349 1333 381">1. Target BAC 0.08%</p>	<p data-bbox="1541 251 1711 284">Cannabis: N/A</p> <p data-bbox="1541 316 1848 430">Alcohol: Alcohol dose administered as absolute alcohol in lemon-lime soda, divided into two beverages.</p>
Harrison & Fillmore, 2011	<p data-bbox="373 771 688 803"><i>For all eligible participants:</i></p> <p data-bbox="373 836 699 950">Cannabis: No substance abuse disorder (assumed to include cannabis); no positive urine test for THC.</p> <p data-bbox="373 982 699 1138">Alcohol: No substance abuse disorder (assumed to include alcohol); score <5 on Short-Michigan Alcoholism Screening Test (S-MAST).</p>	<p data-bbox="724 771 1035 803"><i>For all eligible participants:</i></p> <p data-bbox="724 836 997 868">Cannabis: Not reported.</p> <p data-bbox="724 901 1035 1015">Alcohol: $M = 1.88$ (1.29) drinking occasions per week, with “typical dose” of $M = 0.99$ (0.43) mL/kg.</p>	<p data-bbox="1073 771 1228 803">Comparison:</p> <p data-bbox="1073 803 1291 836">1. Placebo alcohol</p> <p data-bbox="1073 836 1176 868">Alcohol:</p> <p data-bbox="1073 868 1333 901">1. Target BAC 0.08%</p>	<p data-bbox="1541 771 1711 803">Cannabis: N/A</p> <p data-bbox="1541 836 1848 1138">Alcohol: Alcohol dose administered as absolute alcohol in lemon-lime soda, divided into two beverages. Placebo administered as lemon-lime soda divided into two beverages, each topped with 5 mL of alcohol and served glasses sprayed with alcohol mist.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Harrison et al., 2007	<p data-bbox="373 256 688 285"><i>For all eligible participants:</i></p> <p data-bbox="373 318 688 557">Cannabis: No substance use disorder (assumed to include cannabis); no positive urine test for marijuana. Required to abstain for 24 hours prior to study sessions and have zero BAC at start of study sessions.</p> <p data-bbox="373 589 688 740">Alcohol: No substance use disorder (assumed to include alcohol), score <5 on Short Michigan Alcoholism Screening Test (SMAST).</p>	<p data-bbox="724 256 1045 285"><i>For all eligible participants:</i></p> <p data-bbox="724 318 1045 347">Cannabis: Not reported.</p> <p data-bbox="724 380 1045 557">Alcohol: Unclear. Average weekly alcohol dose of 1.2 (0.7) mL/kg (approx. 5 bottles of 5% beer per week for a 75 kg person), for whole $N = 30$ sample.</p>	<p data-bbox="1073 256 1507 285">Comparison:</p> <ol data-bbox="1073 285 1507 315" style="list-style-type: none"> Baseline (control group)² <p data-bbox="1073 315 1507 344">Alcohol:</p> <ol data-bbox="1073 344 1507 380" style="list-style-type: none"> Target BAC 0.08% (control group)² 	<p data-bbox="1541 256 1843 285">Cannabis: N/A</p> <p data-bbox="1541 318 1843 407">Alcohol: Alcohol dose administered as absolute alcohol in lemon-lime soda.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Hartman et al. 2015	<p data-bbox="357 253 714 285"><i>For all eligible participants:</i></p> <p data-bbox="357 318 714 659">Cannabis: At least one or more times using cannabis per month, but no more than three days per week using over previous three months. No previous “clinically significant adverse event” with cannabis, and no “interest in drug abuse treatment” (p. 26) over past 60 days.</p> <p data-bbox="357 691 714 984">Alcohol: “Light,” “moderate” or “heavy” alcohol use (based on Quantity-Frequency-Variability [QFV] scale). In cases of “heavy” use, 3-4 alcohol servings maximum per “typical” occasion. No previous “clinically significant adverse event” with alcohol.</p>	<p data-bbox="714 253 1050 285"><i>For all eligible participants:</i></p> <p data-bbox="714 318 1050 496">Cannabis: “Most” used cannabis two or more times per month, but three or fewer times per week, and had last used cannabis within the week prior to the study.</p> <p data-bbox="714 529 1050 561">Alcohol: Not reported.</p>	<p data-bbox="1050 253 1512 285">Comparison:</p> <ol data-bbox="1050 285 1512 350" style="list-style-type: none"> 0.008% (placebo) THC cannabis + placebo alcohol <p data-bbox="1050 350 1512 383">Cannabis:</p> <ol data-bbox="1050 383 1512 496" style="list-style-type: none"> 2.9% THC cannabis + placebo alcohol 6.7% THC cannabis + placebo alcohol <p data-bbox="1050 496 1512 529">Alcohol:</p> <ol data-bbox="1050 529 1512 594" style="list-style-type: none"> 0.008% (placebo) THC cannabis + 0.065% Target BrAC <p data-bbox="1050 594 1512 626">Combination</p> <ol data-bbox="1050 626 1512 740" style="list-style-type: none"> 2.9% THC cannabis + 0.065% Target BrAC 6.7% THC cannabis + 0.065% Target BrAC 	<p data-bbox="1512 253 1890 383">Cannabis: Vapor from 500 mg cannabis, including placebo, inhaled ad libitum for 10 minutes.</p> <p data-bbox="1512 415 1890 626">Alcohol: Alcohol dose consisted of 90% grain alcohol in fruit juice. Placebo alcohol consisted of fruit juice topped with 1 mL alcohol, served in a glass with an alcohol-wiped rim.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Helland et al., 2016	<p><i>For all eligible participants:</i></p> <p>Cannabis: No current or past drug abuse (assumed to include cannabis); no daily use of any drug (assumed to include cannabis).</p> <p>Alcohol: “Recreational drinkers” with no current or past alcohol abuse; no history of “deviant,” “violent” or “aggressive” reactions to alcohol; no history of driving under the influence of alcohol (DUIA).</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Not reported.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Target BAC 0% (placebo alcohol) <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BAC 0.05% 2. Target BAC 0.09% <p>In each of the three conditions, participants also received a placebo pill and “were told [it] may or may not contain a sedative drug” (p. 247).</p>	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose consisted of vodka in fruit juices. Placebo alcohol consisted of ethanol-free vodka extract in fruit juices.</p>
Horne & Baumber, 1991	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Unclear. Required to abstain from alcohol on study session days.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Drank an average of 0.5 to 2.0 units of alcohol per day.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. 94.8 mL of 40% alcohol vodka (no specific target BAC) 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose administered as vodka in tonic water. Placebo consisted of tonic water served in a glass with a vodka-wiped rim.</p>
Howard et al., 2007	<p><i>For all eligible participants:</i></p> <p>Cannabis: Could not be users of “illicit drugs that might affect performance” (p. 1335) (assumed to include cannabis).</p> <p>Alcohol: Not reported. Required to have zero BAC at start of study sessions.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Not reported.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Baseline driving <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BAC “approximately” 0.03% 2. Target BAC “over” 0.05% 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose administered as vodka in orange juice or soft drink.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Howland et al., 2011	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported. Required to abstain from recreational drugs (assumed to include cannabis) for 24 hours prior to study sessions.</p> <p>Alcohol: No “drinking problems” (i.e., score <5 on SMAST); no history of treatment/counseling for “chronic alcohol problems;” consumed 5 or more drinks (or 4+ for females) on a single drinking occasion one or more times in 30 days prior to study screening. Required to abstain for 24 hours prior to study sessions and pass a breath alcohol test prior to start of testing.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Average Daily Volume (ADV) score $M = 1.60$ (2.23), range 0.10 – 4.74 alcoholic drinks (based on past 30 days).</p>	<p>Comparison:</p> <ol style="list-style-type: none"> Target BrAC 0% (i.e., non-caffeinated non-alcoholic beer) <p>Alcohol:</p> <ol style="list-style-type: none"> Target BrAC 0.12% (i.e., non-caffeinated beer) 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose administered as 8.1% beer. Placebo administered as non-alcoholic beer (i.e., <0.01% alcohol).</p>
Huemer & Vollrath, 2010	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: No risk of alcoholism; consumption of alcohol at least weekly, but no more than 300 mL of pure alcohol on any one occasion, and no more than 150 mL of pure alcohol on multiple occasions per week. Required to be sober prior to testing.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Not reported.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> Baseline driving <p>Alcohol:</p> <ol style="list-style-type: none"> Target BAC 0.08% 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose consisted of vodka in fruit juice with ice, divided into three beverages.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Jelen et al., 2011	<i>For all eligible participants:</i> Cannabis: Not reported. Alcohol: Not reported.	<i>For all eligible participants:</i> Cannabis: Not reported. Alcohol: Not reported.	Comparison: 1. Baseline driving Alcohol: 1. Target BAC 0.10%	Cannabis: N/A Alcohol: Alcohol dose consisted of 40% liquor. No further details reported.
Kay et al., 2013	<i>For all eligible participants:</i> Cannabis: Not reported. Alcohol: Not reported.	<i>For all eligible participants:</i> Cannabis: Not reported. Alcohol: Not reported.	Comparison: 1. Placebo alcohol Alcohol: 1. Target BAC 0.10%	Cannabis: N/A Alcohol: Alcohol dose consisted of vodka in orange juice. Placebo consisted of water in orange juice, topped with a “small quantity” of alcohol, served in a glass with an alcohol-wiped rim.
Kenntner-Mabiala et al., 2015	<i>For all eligible participants:</i> Cannabis: Not reported. Alcohol: At least one alcoholic drink consumed per month, but no more than 14 drinks per week; 6 or fewer points on the Short Questionnaire for Alcohol-Related Problems. Required to have zero BAC at start of study sessions.	<i>For all eligible participants:</i> Cannabis: Not reported. Alcohol: Not reported.	Comparison: 1. Placebo alcohol Alcohol: 1. Target BAC 0.05% 2. Target BAC 0.08%	Cannabis: N/A Alcohol: Alcohol dose administered as vodka in caffeine-free soft drinks (with flavor chosen by participant), divided into four beverages. Composition of placebo beverage not described, but it was consumed by participants in a room with diffused alcohol odor (created by placing hidden vodka-scented tissues near participants).

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Kuypers et al., 2006	<p data-bbox="357 256 714 289"><i>For all eligible participants:</i></p> <p data-bbox="357 321 714 714">Cannabis: Unclear. No history of drug abuse except for MDMA use or drug addiction (assumed to include cannabis). Required to abstain from drugs (assumed to include cannabis) for one week prior to screening until two weeks following last study session and to pass a drug screen prior to testing (assumed to include cannabis).</p> <p data-bbox="357 747 714 985">Alcohol: No “excessive drinking” (i.e., no more than 20 alcoholic drinks per week). Required to abstain for the day prior to testing and to have a negative breath test for alcohol prior to testing.</p>	<p data-bbox="714 256 1029 289"><i>For all eligible participants:</i></p> <p data-bbox="714 321 1029 354">Cannabis: Not reported.</p> <p data-bbox="714 386 1029 418">Alcohol: Not reported.</p>	<p data-bbox="1029 256 1512 289">Comparison:</p> <ol data-bbox="1029 289 1512 321" style="list-style-type: none"> 1. Baseline driving <p data-bbox="1029 321 1512 354">Alcohol:</p> <ol data-bbox="1029 354 1512 386" style="list-style-type: none"> 1. Target BAC 0.05%³ <p data-bbox="1029 418 1512 500">In both the baseline driving and alcohol condition, participants also consumed placebo MDMA.</p>	<p data-bbox="1512 256 1890 289">Cannabis: N/A</p> <p data-bbox="1512 321 1890 406">Alcohol: Alcohol beverage composition and administration not described.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Laude & Fillmore, 2015	<p><i>For all eligible participants:</i></p> <p>Cannabis: No use of cannabis in the 24 hours prior to start of study sessions.</p> <p>Alcohol: Self-reported consumption of alcohol two or more times per month, with two or more drinks per occasion; no current dependence or withdrawal (based on DSM-IV criteria). Required to abstain for 24 hours prior to study sessions and have zero BAC prior to start of testing.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Eleven participants reported cannabis use, and seven tested positive for THC at the start of the study, but none reported not using within the past 24 hours. No participants reported daily use of any drug except caffeine (assumed to include cannabis).</p> <p>Alcohol: $M = 30.29$ (18.75) drinking days, and $M = 106.78$ (85.31) total drinks consumed, in the previous three months.</p>	<p>Comparison:</p> <p>1. Placebo alcohol</p> <p>Alcohol:</p> <p>1. Target BAC 0.08%</p>	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol administered as 95% alcohol in carbonated mix. Placebo administered as carbonated mix topped with 3 mL of alcohol, served in a glass sprayed with alcohol mist.</p>
Laude & Fillmore, 2016	<p><i>For all eligible participants:</i></p> <p>Cannabis: No use of cannabis in the 24 hours prior to start of study sessions.</p> <p>Alcohol: Self-reported consumption of alcohol two or more times per month, with two or more drinks per occasion, in previous 90 days; no current alcohol dependence or withdrawal (based on DSM-IV criteria). Required to abstain for 24 hours prior to study sessions and pass a breath alcohol test prior to start of testing.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported. Ten participants tested positive for THC but reported not using within the past 24 hours.</p> <p>Alcohol: $M = 2.49$ (1.47) drinking occasions per week, with $M = 3.34$ (1.53) drinks per occasion.</p>	<p>Comparison:</p> <p>1. Placebo alcohol</p> <p>Alcohol:</p> <p>1. Target BrAC 0.08%</p>	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose administered as 95% alcohol in carbonated mix. Placebo administered as carbonated mix topped with 3 mL of alcohol, served in a glass sprayed with alcohol mist.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Laude, 2016 (Study 3)	<p data-bbox="375 256 684 285"><i>For all eligible participants:</i></p> <p data-bbox="375 318 695 376">Cannabis: No use of cannabis in the past 24 hours.</p> <p data-bbox="375 409 695 649">Alcohol: Alcohol consumers with score <5 on Short Michigan Alcoholism Screening Test (S-MAST). Required to abstain for 24 hours prior to study sessions and have zero BAC prior to start of testing.</p>	<p data-bbox="726 256 1035 285"><i>For all eligible participants:</i></p> <p data-bbox="726 318 1052 435">Cannabis: Nine participants reported cannabis use, and six tested positive for THC at the start of the study.</p> <p data-bbox="726 467 1052 591">Alcohol: $M = 2.63$ (1.07) drinking occasions per week, with $M = 4.50$ (2.50) drinks per occasion.</p>	<p data-bbox="1073 256 1226 285">Comparison:</p> <ol data-bbox="1073 285 1499 344" style="list-style-type: none"> 1. Target BrAC 0% (placebo alcohol, control group⁴) <p data-bbox="1073 344 1178 373">Alcohol:</p> <ol data-bbox="1073 373 1520 409" style="list-style-type: none"> 1. Target BrAC 0.08% (control group⁴) 	<p data-bbox="1541 256 1717 285">Cannabis: N/A</p> <p data-bbox="1541 318 1881 526">Alcohol: Alcohol administered as 95% alcohol in carbonated mix. Placebo administered as carbonated mix topped with 3 mL of alcohol, served in a glass sprayed with alcohol mist.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Lee et al., 2010	<p><i>For all eligible participants:</i></p> <p>Cannabis: No current illegal drug use including cannabis (verified with urine screen). Participants could not show evidence of substance abuse (assumed to include cannabis). Required to abstain from recreational drugs (assumed to include cannabis) for 30 days prior to sessions.</p> <p>Alcohol: Moderate to heavy alcohol use (based on QFV scale). No “chronic alcohol abusers” (based on AUDIT). Participants could not show evidence of substance abuse (assumed to include alcohol). Required to abstain from alcohol for 24 hours prior to testing. Participants completed breath test at start of study (presumably to verify zero BAC).</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: For n = 18 males aged 21-34, n = 2 moderate drinkers and n = 16 heavy drinkers; for n = 18 females aged 21-34, n = 7 moderate drinkers and n = 11 heavy drinkers; for n = 18 males aged 38-51, n = 4 moderate drinkers and n = 14 heavy drinkers; for n = 18 females aged 38-51, n = 9 moderate drinkers and n = 9 heavy drinkers; for n = 18 males aged 55-68, n = 6 moderate drinkers and n = 12 heavy drinkers; for n = 18 females aged 55-68, n = 7 moderate drinkers and n = 11 heavy drinkers.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BAC 0.05% 2. Target BAC 0.10% 	<p>Cannabis: N/A.</p> <p>Alcohol: Alcohol dose administered as vodka in orange juice, divided into three beverages to be consumed over three ten-minute periods. Placebo consisted of water in orange juice, topped with 10 mL of vodka and served in glasses with alcohol-wiped rims.</p>
Lenne et al., 1999	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: “Inexperienced drinkers” (i.e., <6 drinks consumed, on average, per week). Required to abstain for 24 hours prior to study sessions.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Not reported.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BAC 0.05% 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose consisted of vodka in orange juice. Placebo consisted of pure orange juice.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Lenne et al., 2003	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Required to abstain from alcohol for 24 hours prior to testing.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Not reported.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> Baseline driving <p>Alcohol:</p> <ol style="list-style-type: none"> Target BAC 0.05% 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose consisted of vodka in orange juice.</p>
Leung et al., 2012	<p><i>For all eligible participants:</i></p> <p>Cannabis: If using illicit drugs (assumed to include cannabis), no more than five times per week.</p> <p>Alcohol: No first-time users of alcohol; average four (men; two, women) or fewer standard drinks consumed per day; average six (men; four, women) or fewer drinks consumed per occasion. Required to abstain for 24 hours prior to study sessions.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Not reported.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> Baseline driving⁵ <p>Alcohol:</p> <ol style="list-style-type: none"> Target BAC 0.04% / Range 0.03-0.05% BAC Target BAC 0.07% / Range 0.06-0.08% BAC Target BAC 0.10% / Range 0.09-0.11% BAC 	<p>Cannabis: N/A</p> <p>Alcohol: “Measured amounts of alcohol” administered based on total body water. No further details provided.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Liguori & Robinson, 2001	<p data-bbox="373 251 688 284"><i>For all eligible participants:</i></p> <p data-bbox="373 316 695 584">Alcohol: No self-reported history of substance dependence (assumed to include alcohol); 3-14 alcoholic drinks consumed per week. Required to abstain for 24 hours prior to study sessions (verified with breath alcohol measurement).</p> <p data-bbox="373 617 695 984">Cannabis: No self-reported history of substance dependence (assumed to include cannabis); no illicit psychoactive drug use (assumed to include cannabis). Required to abstain from psychoactive drugs (assumed to include cannabis) for duration of study (verified with negative drug screen).</p>	<p data-bbox="726 251 1031 284"><i>For all eligible participants:</i></p> <p data-bbox="726 316 993 349">Cannabis: Not reported.</p> <p data-bbox="726 381 1045 771">Alcohol: “Most subjects drank two to three times per week (53% of subjects), had one or two drinks on a typical day when drinking (67%), and either never had six or more drinks on one occasion (40%) or did so less than monthly (40%)” (p. 124) (based on AUDIT scores; all scored 7 or less). Overall, average 5 standard drinks consumed per week.</p>	<p data-bbox="1073 251 1226 284">Comparison:</p> <ol data-bbox="1073 284 1297 316" style="list-style-type: none"> 1. Placebo alcohol <p data-bbox="1073 316 1178 349">Alcohol:</p> <ol data-bbox="1073 349 1444 381" style="list-style-type: none"> 1. 0.6g/kg (unclear Target BAC) <p data-bbox="1073 406 1499 527">In both the placebo and alcohol condition, participants also consumed a methylcellulose (placebo caffeine) capsule.</p>	<p data-bbox="1541 251 1717 284">Cannabis: N/A</p> <p data-bbox="1541 316 1871 560">Alcohol: Alcohol dose administered as vodka in orange juice with 5 mL of vodka applied to the top and sides of the cup. Placebo administered as orange juiced with 5 mL of vodka applied to the top and sides of the cup.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Liguori et al., 1998	<p data-bbox="375 253 688 285"><i>For all eligible participants:</i></p> <p data-bbox="375 318 695 651">Cannabis: At least weekly use of marijuana, but not daily, with 4 to 28 uses in the past 30 days; no history of drug dependence except nicotine (assumed to include cannabis); no history of drug counseling (assumed to include cannabis). Required to abstain for 48 hours prior to each study session.</p> <p data-bbox="375 683 695 831">Alcohol: No more than 14 standard alcoholic drinks per week. Required to abstain for 36 hours prior to each study session.</p>	<p data-bbox="726 253 1041 285"><i>For all eligible participants:</i></p> <p data-bbox="726 318 1041 440">Cannabis: Average 12 (7) uses of marijuana in previous 30 days; all reported at least 40 uses within their lifetime.</p> <p data-bbox="726 472 1041 529">Alcohol: Average 4 (2) alcoholic drinks per week.</p>	<p data-bbox="1073 253 1461 285">Comparison:</p> <ol data-bbox="1073 285 1461 350" style="list-style-type: none"> 0.00% (placebo) THC cannabis cigarette <p data-bbox="1073 350 1461 383">Cannabis:</p> <ol data-bbox="1073 383 1461 440" style="list-style-type: none"> 1.77% THC cannabis cigarette 3.95% THC cannabis cigarette 	<p data-bbox="1541 253 1860 407">Cannabis: Cannabis cigarettes, including placebo, smoked in a structured smoking paradigm with 10 inhalations.</p> <p data-bbox="1541 440 1860 464">Alcohol: N/A</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Liguori et al., 1999	<p data-bbox="373 256 688 586"><i>For all eligible participants:</i></p> <p data-bbox="373 318 688 586">Cannabis: No self-reported substance use history (assumed to include cannabis). Required to abstain from psychoactive drugs (assumed to include cannabis) for duration of study (verified with negative drug screen).</p> <p data-bbox="373 626 688 954">Alcohol: No self-reported substance use history (assumed to include alcohol); no “alcohol-related problems” (i.e., no TWEAK scores >2, no Short Alcohol Dependence Data [SADD] scores >8). Required to abstain for 36 hours prior to study sessions (verified with breath alcohol measurement).</p>	<p data-bbox="724 256 1037 285"><i>For all eligible participants:</i></p> <p data-bbox="724 318 1037 347">Cannabis: Not reported.</p> <p data-bbox="724 380 1037 435">Alcohol: $M = 7$ (3) alcoholic drinks per week</p>	<p data-bbox="1073 256 1472 285">Comparison:</p> <ol data-bbox="1073 289 1472 467" style="list-style-type: none"> 1. Placebo alcohol <p data-bbox="1073 318 1472 347">Alcohol:</p> <ol data-bbox="1073 350 1472 467" style="list-style-type: none"> 1. 0.5 g/kg alcohol (unclear Target BAC) 2. 0.8 g/kg alcohol (unclear Target BAC) 	<p data-bbox="1541 256 1877 285">Cannabis: N/A</p> <p data-bbox="1541 318 1877 618">Alcohol: Alcohol dose administered as vodka in orange juice, divided into two beverages, with 5 mL of vodka applied to the top and sides of the cup. Placebo administered as orange juice, divided into two beverages, with 5 mL of vodka applied to the top and sides of the cup.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Liguori et al., 2002	<p><i>For all eligible participants:</i></p> <p>Cannabis: Used cannabis for 2 – 21 days of previous 30 days; no drug abuse or dependence in the past year (excluding nicotine; assumed to include cannabis). Required to abstain for 48 hours prior to study sessions. Urine tested for “illicit drug content,” including cannabis. One participant tested positive on each visit but was not excluded.</p> <p>Alcohol: Not currently attempting to stop or reduce alcohol consumption; score of 10 or less on AUDIT; no alcohol abuse or dependence in the past year. Required to abstain for 12 hours prior to study sessions.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Participants reported cannabis use on average 10 (range 2 – 19) out of 30 days prior to study.</p> <p>Alcohol: Consumed on average 12 (range 4 – 24) standard alcoholic drinks per week.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 0.003% THC (placebo) cannabis cigarette + placebo alcohol <p>Cannabis:</p> <ol style="list-style-type: none"> 1.75% THC cannabis cigarette + placebo alcohol 3.33% THC cannabis cigarette + placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 0.003% THC (placebo) cannabis cigarette + 0.25 g/kg alcohol (unclear Target BAC) 0.003% THC (placebo) cigarette + 0.5 g/kg alcohol (unclear Target BAC) <p>Combination:</p> <ol style="list-style-type: none"> 1.75% THC cannabis cigarette + 0.25 g/kg alcohol (unclear Target BAC) 1.75% THC cannabis cigarette + 0.5 g/kg alcohol (unclear Target BAC) 3.33% THC cannabis cigarette + 0.25 g/kg alcohol (unclear Target BAC) 3.33% THC cannabis cigarette + 0.5 g/kg alcohol (unclear Target BAC) 	<p>Cannabis: Cannabis cigarettes, including placebo, smoked in a structured smoking paradigm with 10 inhalations.</p> <p>Alcohol: Alcohol dose consisted of 95% alcohol in diet tonic water with 4 mL of lime juice, divided into three beverages. Placebo alcohol consisted of diet tonic water with 4 mL of lime juice, divided into three beverages, topped with 1 mL of alcohol each.</p>
Louwerens et al., 1987	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported. Abstained from drugs (assumed to include cannabis) during study.</p> <p>Alcohol: At least four standard alcoholic drinks per week, but fewer than four per day. Required to be sober on arrival for testing.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Not reported.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> Baseline driving <p>Alcohol:</p> <ol style="list-style-type: none"> 0.5 g/kg alcohol 1.0 g/kg alcohol 1.5 g/kg alcohol 2.0 g/kg alcohol 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose consisted of vodka in orange juice.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Marczinski & Fillmore, 2009	<p data-bbox="375 256 688 285"><i>For all eligible participants:</i></p> <p data-bbox="375 318 699 526">Cannabis: No substance abuse disorder (assumed to include cannabis). Participants' urine tested for THC on arrival, but unclear if positive test was an exclusion criterion.</p> <p data-bbox="375 558 699 863">Alcohol: Two or more drinks per month; no substance abuse disorder (assumed to include alcohol); no risk of alcohol dependence (i.e., no score 5+ on SMAST). Required to abstain for 24 hours prior to study sessions and have zero BAC prior to testing.</p>	<p data-bbox="726 256 1037 285"><i>For all eligible participants:</i></p> <p data-bbox="726 318 995 347">Cannabis: Not reported.</p> <p data-bbox="726 380 1037 711">Alcohol: For $n = 18$ "binge" group (i.e., 5+ drinks on a single occasion for males, 4+ for females), $M = 2.5$ (1.1) drinking occasions per week, with $M = 7.3$ (2.7) drinks per occasion. For $n = 10$ "nonbinge" group, $M = 2.1$ (2.1) drinking occasions per week, with $M = 2.9$ (0.5) drinks per occasion.</p>	<p data-bbox="1075 256 1226 285">Comparison:</p> <p data-bbox="1075 285 1293 315">1. Placebo alcohol</p> <p data-bbox="1075 318 1176 347">Alcohol:</p> <p data-bbox="1075 347 1331 376">1. Target BAC 0.08%</p>	<p data-bbox="1543 256 1717 285">Cannabis: N/A</p> <p data-bbox="1543 318 1881 587">Alcohol: Alcohol dose consisted of alcohol in carbonated mix, divided into two beverages. Placebo consisted of carbonated mix, divided into two drinks, topped with 3 mL of alcohol each and served in glasses sprayed with alcohol mist.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Marczinski et al., 2008	<p data-bbox="375 251 699 462"><i>For all eligible participants:</i></p> <p data-bbox="375 316 699 462">Cannabis: No current substance abuse disorder (assumed to include cannabis; no positive urine test for THC).</p> <p data-bbox="375 495 699 954">Alcohol: No “extremely infrequent drinkers” (i.e., <2 standard alcoholic drinks per month); no “drinkers with a potential risk of alcohol dependence” (p. 1330) (i.e., score of 5+ on Short-Michigan Alcoholism Screen Test [S-MAST]); no current substance abuse disorder (assumed to include alcohol). Required to abstain for 24 hours prior to study sessions (verified with BrAC measurement).</p>	<p data-bbox="726 251 1050 284"><i>For all eligible participants:</i></p> <p data-bbox="726 316 1050 349">Cannabis: Not reported.</p> <p data-bbox="726 381 1050 836">Alcohol: “Typical social drinking college students” (p. 1330). Participants divided into binge ($n = 24$; 5+ drinks for men, 4+ drinks for women, within a single drinking occasion) and nonbinge ($n = 16$) groups. For binge group, $M = 2.5$ (1.3) drinking occasions per week with $M = 5.9$ (1.6) drinks per occasion. For nonbinge group, $M = 1.3$ (0.8) drinking occasions per week with $M = 3.4$ (1.1) drinks per occasion.</p>	<p data-bbox="1073 251 1480 284">Comparison:</p> <ol data-bbox="1073 284 1480 316" style="list-style-type: none"> <li data-bbox="1073 284 1480 316">Placebo alcohol <p data-bbox="1073 316 1480 349">Alcohol:</p> <ol data-bbox="1073 349 1480 406" style="list-style-type: none"> <li data-bbox="1073 349 1480 406">0.65 g/kg alcohol (unclear Target BAC) 	<p data-bbox="1541 251 1881 284">Cannabis: N/A</p> <p data-bbox="1541 316 1881 617">Alcohol: Alcohol dose administered as 95% alcohol per volume in lemon lime soda, divided into two alcoholic beverages. Placebo consisted of lemon lime soda topped with 3 mL of alcohol, divided into two beverages, served in glasses sprayed with alcohol mist.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
McCartney et al., 2017	<p><i>For all eligible participants:</i></p> <p>Cannabis: No current use of “recreational or psychoactive” drugs in the previous six months (assumed to include cannabis).</p> <p>Alcohol: Score of 5 or less on the Self-Administered Short Michigan Alcoholism Screening Test. Required to abstain for 24 hours prior to study sessions and have zero BrAC at the start of study sessions.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: $M = 1.0$ (0.80) drinking occasions per week, with $M = 5.4$ (3.5) standard drinks per occasion.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BrAC 0.08% 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose administered as vodka in diet ginger beer, ginger beer cordial and diet lime cordial. Placebo administered as water in diet ginger beer, ginger beer cordial and diet lime cordial, served in a glass sprayed with alcohol mist.</p>
Mets et al., 2011	<p><i>For all eligible participants:</i></p> <p>Cannabis: No current or previous drug use (assumed to include cannabis); no positive urine test for cannabinoids prior to testing.</p> <p>Alcohol: Consumed 7 – 21 alcoholic beverages per week. Required to abstain for 24 hours and have a negative breath alcohol test prior to testing.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: $M = 14.1$ (3.9) standard alcoholic drinks per week.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BAC 0.05% 2. Target BAC 0.08% 3. Target BAC 0.11% 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose consisted of 99.9% ethanol in orange juice, flavored with cognac aroma and consumed while wearing a nose clip. Placebo consisted of orange juice, flavored with cognac aroma and consumed while wearing a nose clip.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Price et al., 2018	<p><i>For all eligible participants:</i></p> <p>Cannabis: No current or past diagnosis of substance dependence (assumed to include cannabis). Urine tested for drugs, but unclear whether this included cannabis, and if so, whether a positive test was an exclusion criterion.</p> <p>Alcohol: “Social drinkers” (i.e., two or fewer drinks per day for males under 65; one or fewer for men over 65 and women); no current or past diagnosis of alcohol dependence. Required to abstain for 24 hours prior to testing (verified with breath alcohol test).</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: For n = 33 younger group, $M = 0.39$ (0.27) QFI scores. For n = 33 older group, $M = 0.36$ (0.33) QFI score. All reported drinking at least monthly, with an overall average of one or fewer drinks per day.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BrAC 0.04% 2. Target BrAC 0.065% 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose consisted of alcohol in sugar-free, caffeine-free lemon-lime soda, divided into two drinks, served in a glass sprayed with alcohol mist. Placebo consisted of sugar-free, caffeine-free lemon-lime soda, divided into two drinks, served in a glass sprayed with alcohol mist.</p>
Ramaekers et al., 1992	<p><i>For all eligible participants:</i></p> <p>Cannabis: No history of drug abuse (assumed to include cannabis).</p> <p>Alcohol: No history of alcohol abuse.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Not reported.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. 0.72 g/kg lean body mass <p>In both the comparison and alcohol condition, participants also consumed placebo drug.</p>	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose consisted of 99.8% ethanol in orange juice. Placebo consisted of pure orange juice.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Ramaekers et al., 2000a (see also Study 1, Ramaekers et al. [2000b], below)	<i>For all eligible participants:</i> Cannabis: Cannabis use at least once per month but not daily. Required to abstain from smoking marijuana or hashish, or any other illicit drug, from seven days prior to the first day of testing to the end of the study (verified with urine test). Alcohol: Alcohol use at least once per week but not daily; no history of alcohol abuse or dependency. Required to abstain for 24 hours prior to testing (verified with breath test).	<i>For all eligible participants:</i> Cannabis: Not reported. Alcohol: Not reported.	Comparison: 1. Placebo alcohol + placebo cannabis Cannabis: 1. Placebo alcohol + 100 ug/kg THC (from 2.2% THC cannabis) 2. Placebo alcohol + 200 ug/kg THC (from 3.95% THC cannabis) Alcohol: 1. Target BAC 0.04-0.05% + placebo cannabis Combination: 1. Target BAC 0.04-0.05% + 100 ug/kg THC (from 2.2% THC cannabis) 2. Target BAC 0.04-0.05% + 200 ug/kg THC (from 3.95% THC cannabis)	Cannabis: Cannabis cigarettes (approximately 0.8 g), including placebo, cut to lengths based on each participant's weight and smoked through a plastic holder in participants' "customary fashion." Alcohol: Alcohol dose administered as 99.8% ethanol in orange juice and flavoured with Grand Marnier essence. Placebo consisted of orange juice flavoured with Grand Marnier essence.
Ramaekers et al., 2000b (Study 1) (see also Ramaekers et al. [2000a] above)	<i>For all eligible participants:</i> Cannabis: Unclear. Participants' urine tested for cannabinoids on arrival, but unclear if positive test was an inclusion or exclusion criterion. Alcohol: Unclear. Participants provided breath samples on arrival, likely to verify that they had not been drinking. No prior charges or convictions for DUIA.	<i>For all eligible participants:</i> Cannabis: "Recreational" users who used more than once per month, but not daily. Alcohol: "Used to consum[ing] alcohol at least once a week" (<i>General procedures</i> , para. 1).	Comparison: 1. Placebo cannabis + placebo alcohol Cannabis: 1. 100 ug/kg ⁶ dose of THC (from 2.2% THC cannabis) + placebo alcohol 2. 200 ug/kg ⁶ dose of THC (from 3.95% THC cannabis) + placebo alcohol Alcohol: 1. Placebo cannabis + 0.04 – 0.07% Target BAC ⁷ Combination: 1. 100 ug/kg ⁶ dose of THC (from 2.2% THC cannabis) + 0.04 – 0.07% Target BAC ⁷ 2. 200 ug/kg ⁶ dose of THC (from 3.95% THC cannabis) + 0.04 – 0.07% Target BAC ⁷	Cannabis: Cannabis cigarettes, including placebo, cut according to participants' body weight and smoked "as completely as possible through a plastic holder in their customary fashion" (<i>General procedures</i> , para. 3). Alcohol: Administration not described.

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Robbe, 1998 (Study 1)	<p><i>For all eligible participants:</i></p> <p>Cannabis: Unclear. Urine tested for cannabinoids, but unclear if positive test was an inclusion or exclusion criterion.</p> <p>Alcohol: Unclear. Participants provided breath samples on arrival, likely to verify that they had not been drinking.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Used cannabis at least monthly, but no more than daily.</p> <p>Alcohol: Not reported.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 0 ug/kg dose of THC (placebo cannabis) <p>Cannabis:</p> <ol style="list-style-type: none"> 100 ug/kg dose of THC (from 1.75% THC cannabis) 200 ug/kg dose of THC (from 1.75% THC cannabis) 300 ug/kg dose of THC (from 2.57% THC) 	<p>Cannabis: Cannabis cigarettes, including placebo, cut according to participants' body weight and smoked "as completely as possible through a plastic holder in their customary fashion" (p. S71).</p> <p>Alcohol: N/A</p>
Robbe, 1998 (Study 2)	<p><i>For all eligible participants:</i></p> <p>Cannabis: Unclear. Urine tested for cannabinoids, but unclear if positive test was an inclusion or exclusion criterion.</p> <p>Alcohol: Unclear. Participants provided breath samples on arrival, likely to verify that they had not been drinking.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Used cannabis at least monthly, but no more than daily.</p> <p>Alcohol: Not reported.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 0 ug/kg dose of THC (placebo cannabis) <p>Cannabis:</p> <ol style="list-style-type: none"> 100 ug/kg dose of THC (from 1.77% THC cannabis) 200 ug/kg dose of THC (from 2.64% THC cannabis) 300 ug/kg dose of THC (from 3.58% THC) 	<p>Cannabis: Cannabis cigarettes, including placebo, cut according to participants' body weight and smoked "as completely as possible through a plastic holder in their customary fashion" (p. S71).</p> <p>Alcohol: N/A</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Roberts, 2016 (Study 2)	<p data-bbox="375 251 688 284"><i>For all eligible participants:</i></p> <p data-bbox="375 316 695 495">Cannabis: Unclear. Urine tested for drugs, but unclear whether this included cannabis, and if so, whether a positive test was an exclusion criterion.</p> <p data-bbox="375 527 680 625">Alcohol: Unclear. Required to abstain for 24 hours prior to study sessions.</p> <p data-bbox="375 649 646 682"><i>For DUI offender group:</i></p> <p data-bbox="375 706 663 803">Alcohol: At least one DUI conviction in the past five years.</p> <p data-bbox="375 828 621 860"><i>For the control group:</i></p> <p data-bbox="375 885 669 954">Alcohol: No previous DUI convictions.</p>	<p data-bbox="726 251 1031 284"><i>For all eligible participants:</i></p> <p data-bbox="726 316 993 349">Cannabis: Not reported.</p> <p data-bbox="726 373 1041 868">Alcohol: For $n = 20$ controls, $M = 29.4$ (12.7) drinking occasions with $M = 111.7$ (92.6) total drinks in past 90 days; $M = 0.8$ (1.1) SCID score for alcohol abuse; $M = 1.1$ (1.4) SCID score for alcohol dependence. For $n = 20$ DUI group, $M = 34.2$ (14.5) drinking occasions with $M = 152.2$ (63.1) total drinks in past 90 days; $M = 3.5$ (0.9) SCID score for alcohol abuse; $M = 2.8$ (1.3) SCID score for alcohol dependence.</p>	<p data-bbox="1073 251 1226 284">Comparison:</p> <p data-bbox="1073 284 1293 316">1. Placebo alcohol</p> <p data-bbox="1073 316 1178 349">Alcohol:</p> <p data-bbox="1073 349 1339 381">1. Target BAC 0.08%⁸</p>	<p data-bbox="1541 251 1717 284">Cannabis: N/A</p> <p data-bbox="1541 316 1875 592">Alcohol: Alcohol dose administered as alcohol in carbonated mix, divided into two glasses. Placebo consisted of carbonated mix, divided into two beverages, each topped with 5 mL of alcohol and served in glasses sprayed with alcohol mist.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Ronen et al. 2008	<p data-bbox="373 253 688 285"><i>For all eligible participants:</i></p> <p data-bbox="373 318 594 350">Cannabis: Unclear.</p> <p data-bbox="373 375 573 407">Alcohol: Unclear.</p>	<p data-bbox="726 253 1031 285"><i>For all eligible participants:</i></p> <p data-bbox="726 318 1052 683">Cannabis: “Low” to “moderate” cannabis use (i.e., 1 – 4 cannabis uses per month). “Most” reported using primarily in social situations or on the weekend. Required to abstain from cannabis from a week prior to the study to the end of the study. However, all had a positive urine test for THC metabolites before study.</p> <p data-bbox="726 716 1052 924">Alcohol: “Recreational” alcohol use. Required to restrict their consumption to no more than a serving of alcohol a day for at least a week prior to the start of the study.</p>	<p data-bbox="1073 253 1230 285">Comparison:</p> <ol data-bbox="1073 285 1482 342" style="list-style-type: none"> <li data-bbox="1073 285 1482 342">1. Placebo cannabis + 0% placebo alcohol⁹ <p data-bbox="1073 350 1199 383">Cannabis:</p> <ol data-bbox="1073 383 1482 496" style="list-style-type: none"> <li data-bbox="1073 383 1482 440">1. Active (13 mg) THC cannabis + placebo alcohol <li data-bbox="1073 440 1482 496">2. Active (17 mg) THC cannabis + placebo alcohol <p data-bbox="1073 505 1178 537">Alcohol:</p> <ol data-bbox="1073 537 1482 586" style="list-style-type: none"> <li data-bbox="1073 537 1482 586">1. Placebo cannabis + 0.05% Target BAC 	<p data-bbox="1541 253 1877 561">Cannabis: For active cannabis, THC in ethanol vehicle injected into 0.5 g tobacco cigarette. For placebo cannabis, ethanol vehicle (i.e., no THC) injected into 0.5 g tobacco cigarette. Cigarettes smoked in a structured smoking paradigm until totally consumed.</p> <p data-bbox="1541 594 1877 708">Alcohol: Alcohol dose consisted of vodka in orange drink. Placebo alcohol consisted of pure orange drink.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Ronen et al. 2010	<i>For all eligible participants:</i> Cannabis: Unclear. Alcohol: Unclear.	<i>For all eligible participants:</i> Cannabis: “Low” to “moderate” “recreational” cannabis use (i.e., 1 – 4 uses per month). “Most” reported using cannabis primarily in social situations or on the weekend. Required to abstain from cannabis from a week prior to the study to the end of the study. However, all had a positive urine test for THC metabolites before study. Alcohol: “Recreational” alcohol use. “Most” reported using alcohol primarily in social situations or on the weekend. Required to restrict their consumption to no more than a serving of alcohol a day for at least a week prior to the start of the study.	Comparison: 1. Placebo cannabis + placebo alcohol ¹⁰ Cannabis: 1. Active (13 mg) THC cannabis + placebo alcohol Alcohol: 1. Placebo cannabis + 0.05% Target BAC Combination: 1. Active (13 mg) THC cannabis + 0.05% Target BAC	See Ronen et al. (2008), above.
Rupp et al., 2007	<i>For all eligible participants:</i> Cannabis: Not reported. Alcohol: Not reported.	<i>For all eligible participants:</i> Cannabis: Not reported. Alcohol: Not reported.	Comparison: 1. Placebo alcohol Alcohol: 1. 0.54 g/kg alcohol (males) or 0.49 g/kg alcohol (females); no specific target BAC	Cannabis: N/A Alcohol: Alcohol dose consisted of vodka in tonic. Placebo beverage composition not described.

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Schumacher et al., 2017	<p><i>For all eligible participants:</i></p> <p>Cannabis: No illicit drug use including cannabis (verified with urine screen).</p> <p>Alcohol: No previous alcohol abuse or addiction (i.e., no more than 20 units of alcohol weekly). Required to have zero BAC prior to testing (verified with a breath test).</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Not reported.</p>	<p>Comparison:</p> <p>1. Placebo alcohol</p> <p>Alcohol:</p> <p>1. Target BAC 0.05%</p>	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose consisted of alcohol in orange juice. Placebo consisted of orange juice topped with 3 mL of alcohol.</p>
Sexton, 1997	<p><i>For all eligible participants:</i></p> <p>Cannabis: Unclear. No “adverse drug reactions” (unclear if this includes cannabis).</p> <p>Alcohol: Five to fifteen units of alcohol per week.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Not reported.</p>	<p>Comparison:</p> <p>1. Placebo alcohol</p> <p>Alcohol:</p> <p>1. Target BAC 0.04%¹¹</p> <p>2. Target BAC 0.08%¹¹</p>	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose administered as vodka in American Cream Soda. Placebo consisted of cream soda served in a vodka-wiped glass.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Sexton et al., 2000	<p><i>For all eligible participants:</i></p> <p>Cannabis: Cannabis use (at least once per week) for more than 12 months, with positive urine test for THC metabolites at start of study; no previous substance abuse except nicotine (assumed to include cannabis).</p> <p>Alcohol: No previous substance abuse except nicotine (assumed to include alcohol); no drinking prior to start of study sessions, verified with breath alcohol measurement.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: All participants were alcohol consumers with an average 18.7 (7.89) units of alcohol consumed per week.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 0.005% (placebo) THC cannabis cigarette <p>Cannabis:</p> <ol style="list-style-type: none"> 1.70% THC cannabis cigarette 2.67% THC cannabis cigarette 1.7% THC resin 	<p>Cannabis: Cannabis cigarettes, including placebo, smoked in a structured smoking paradigm until totally consumed. Cannabis resin prepared by the participant and smoked “in his customary fashion” (i.e., ad libitum).</p> <p>Alcohol: N/A</p>
Sexton et al., 2002	<p><i>For all eligible participants:</i></p> <p>Cannabis: Cannabis use at least weekly for more than 12 months, with positive test for THC metabolites at start of study. No history of substance abuse except nicotine (assumed to include cannabis); had driven under the influence of cannabis in the past.</p> <p>Alcohol: No previous substance abuse except nicotine (assumed to include alcohol); alcohol use at least weekly for more than 12 months; 5 to 25 units of alcohol consumed per week.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: No specific frequency of cannabis use reported.</p> <p>Alcohol: All participants were alcohol consumers with an average 24.5 (19.22) units of alcohol consumed per week.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 0.005% (placebo) THC cannabis cigarette + placebo alcohol <p>Cannabis:</p> <ol style="list-style-type: none"> 1.70% THC cannabis cigarette + placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 0.005% (placebo) THC cannabis cigarette + 0.05% Target BAC <p>Combination:</p> <ol style="list-style-type: none"> 1.70% THC cannabis cigarette + 0.05% Target BAC 	<p>Cannabis: Cannabis cigarettes, including placebo, smoked in a structured smoking paradigm until totally consumed.</p> <p>Alcohol: Alcohol dose consisted of vodka in tonic water with Angostura bitters, served in a glass with a vodka-dipped rim. Placebo alcohol consisted of tonic water with Angostura bitters, served in a glass with a vodka-dipped rim.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Simons et al., 2012	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported. Required to abstain from psychoactive drugs (assumed to include cannabis) for 24 hours prior to test days (verified with a urine screen).</p> <p>Alcohol: Unclear. Required to abstain for 24 hours prior to test days (verified with breath alcohol test).</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: All were “infrequent recreational” alcohol consumers.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Non-alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. 0.8 g/kg alcohol <p>In both the comparison and alcohol condition, participants also consumed placebo drug.</p>	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose administered in orange juice. In non-alcohol condition, placebo drug administered with orange juice as well.</p>
Sklar et al., 2014	<p><i>For all eligible participants:</i></p> <p>Cannabis: No positive urine test for THC.</p> <p>Alcohol: “Moderate drinkers” (based on USDA dietary guidelines).</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: For n = 12 younger adults in placebo condition, M = 00.34 (0.2) QFI score; for n = 13 younger adults in 0.04% BAC condition, M = 0.44 (0.3) QFI score; for n = 11 younger adults in 0.065% BAC condition, M = 00.35 (0.2) QFI score; for n = 12 older adults in placebo condition, M = 00.44 (0.4) QFI score; for n = 13 older adults in 0.04% BAC condition, M = 00.26 (0.2) QFI score; for n = 11 older adults in 0.065% BAC condition, M = 00.21 (0.2) QFI score.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BrAC 0.04% 2. Target BrAC 0.065% 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose administered in sugar-free, noncaffeinated lemon-lime soda. Placebo consisted of sugar-free, noncaffeinated lemon-lime soda sprayed with a “negligible amount” of alcohol.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Starkey & Charlton, 2014	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Drank alcohol “occasionally but not excessively” (i.e., score <8 on AUDIT). Required to abstain the evening prior to sessions and have zero BAC prior to testing.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Not reported.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BAC 0.05%¹² 2. Target BAC 0.08%¹² 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose consisted of vodka in orange juice, divided into three beverages. Placebo consisted of orange juice, divided into three beverages, topped with 5 mL of vodka per beverage.</p>
Strayer et al., 2006	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: “Social drinkers” (i.e., 3 – 5 alcoholic drinks per week)</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Not reported.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Baseline driving <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BAC 0.08% 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose consisted of vodka in orange juice.</p>
Subramaniyam et al., 2018	<p><i>For all eligible participants:</i></p> <p>Cannabis: Unclear.</p> <p>Alcohol: Not reported.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: “Free from drug use” (assumed to include cannabis).</p> <p>Alcohol: Not reported.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Baseline driving <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BAC 0.03% 2. Target BAC 0.05% 3. Target BAC 0.1% 	<p>Cannabis: N/A.</p> <p>Alcohol: Alcohol dose administered as 50 mL of soju (unclear if consumed neat or in a mix).</p>
Tremblay et al., 2015	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: At least weekly alcohol consumption, but no “issues” associated with consuming alcohol.</p> <p><i>For alcohol group:</i></p> <p>Alcohol: Required to abstain for 24 hours prior to testing.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p><i>For alcohol group:</i></p> <p>Alcohol: M = 7 (4.6) drinks per week.</p> <p><i>For control group:</i></p> <p>Alcohol: Not reported.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Matched time-point baseline driving <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BAC 0.05 – 0.07% 2. Target BAC 0.01 – 0.04% 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose administered as vodka (unclear if served alone or in a mix).</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
van der Sluiszen et al., 2016	<p data-bbox="375 253 688 285"><i>For all eligible participants:</i></p> <p data-bbox="375 318 701 529">Cannabis: No history of drug abuse (assumed to include cannabis); no use of “drugs of abuse” (assumed to include cannabis) from two weeks prior to study treatments, to end of study treatments.</p> <p data-bbox="375 561 701 833">Alcohol: No history of alcoholism; up to 21 standard units of alcohol per week; no alcohol abstainers. Required to abstain for 24 hours prior to study sessions and consume no alcohol from time of arrival to end of study sessions.</p>	<p data-bbox="726 253 1031 285"><i>For all eligible participants:</i></p> <p data-bbox="726 318 989 350">Cannabis: Not reported.</p> <p data-bbox="726 383 968 407">Alcohol: Not reported.</p>	<p data-bbox="1073 253 1230 285">Comparison:</p> <ol data-bbox="1073 285 1304 318" style="list-style-type: none"> 1. Baseline driving <p data-bbox="1073 318 1178 342">Alcohol:</p> <ol data-bbox="1073 342 1346 375" style="list-style-type: none"> 1. Target BAC 0.045% <p data-bbox="1073 407 1503 496">In both the placebo and alcohol condition, participants also consumed a placebo capsule.</p>	<p data-bbox="1541 253 1713 285">Cannabis: N/A</p> <p data-bbox="1541 318 1860 407">Alcohol: Alcohol dose consisted of ethyl alcohol in orange juice.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Van Dyke & Fillmore, 2014	<p data-bbox="373 251 688 284"><i>For all eligible participants:</i></p> <p data-bbox="373 316 688 560">Alcohol: At least two standard alcoholic drinks consumed monthly, but no alcohol dependence or withdrawal (based on DSM-IV criteria). Required to abstain for 24 hours prior to test sessions.</p> <p data-bbox="373 592 688 738">Cannabis: No substance use disorder except alcohol (assumed to include cannabis); no positive urine test for THC.</p> <p data-bbox="373 771 646 803"><i>For DUI offender group:</i></p> <p data-bbox="373 836 695 917">Alcohol: One or more alcohol-related convictions in the previous five years.</p> <p data-bbox="373 950 621 982"><i>For the control group:</i></p> <p data-bbox="373 1015 667 1105">Alcohol: No previous DUI convictions or license revocations.</p>	<p data-bbox="726 251 1035 284"><i>For all eligible participants:</i></p> <p data-bbox="726 316 1052 495">Cannabis: Four participants in the DUI group and five participants in the control group reported using cannabis on average two days in the previous month.</p> <p data-bbox="726 527 1052 917">Alcohol: For $n = 25$ DUI offenders, $M = 29.76$ (19.82) drinking occasions in previous three months, with $M = 142.86$ (109.68) drinks consumed. $M = 11.40$ (6.34) AUDIT score. For $n = 25$ controls, $M = 29.96$ (14.53) drinking occasions in previous three months, with $M = 129.96$ (100.55) drinks consumed. $M = 7.80$ (5.07) AUDIT score.</p>	<p data-bbox="1073 251 1228 284">Comparison:</p> <p data-bbox="1073 284 1297 316">1. Placebo alcohol</p> <p data-bbox="1073 316 1178 349">Alcohol:</p> <p data-bbox="1073 349 1333 381">1. Target BAC 0.08%</p>	<p data-bbox="1541 251 1717 284">Cannabis: N/A</p> <p data-bbox="1541 316 1871 527">Alcohol: Alcohol dose consisted of absolute alcohol in carbonated soda. Placebo consisted of carbonated soda topped with 3 mL of alcohol, served in a glass sprayed with alcohol mist.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Van Dyke & Fillmore, 2015	<p><i>For all eligible participants:</i></p> <p>Cannabis: No substance abuse disorder (assumed to include cannabis); no positive urine test for THC.</p> <p>Alcohol: No substance abuse disorder (assumed to include alcohol); alcohol use one or more times per week, but no dependence or withdrawal (based on DSM-IV criteria). Required to abstain for 24 hours prior to study sessions.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Nine participants reported marijuana use in the past month, with an average of two times using in the past month.</p> <p>Alcohol: $M = 29.9$ (17.2) drinking occasions, and $M = 136.4$ (104.3) drinks, in the previous 3 months.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BrAC 0.07-0.08% 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose consisted of absolute alcohol in carbonated soda. Placebo consisted of carbonated mix, topped with 3 mL of alcohol, served in a glass sprayed with an alcohol mist.</p>
Van Dyke & Fillmore, 2017	<p><i>For all eligible participants:</i></p> <p>Cannabis: No substance abuse disorder (assumed to include cannabis). Required to abstain for 24 hours prior to study sessions.</p> <p>Alcohol: No substance abuse disorder (assumed to include alcohol); alcohol use one or more times per week, but no dependence or withdrawal (based on DSM-IV criteria). Required to abstain for 24 hours prior to study sessions and have zero BAC prior to study sessions.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Five participants reported marijuana use in the past month; two tested positive but reported no use in over a week.</p> <p>Alcohol: $M = 8.80$ (4.35) AUDIT score; $M = 27.26$ (17.92) drinking occasions in past three months, with $M = 126.32$ (86.68) drinks consumed in past three months.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BAC 0.05% 2. Target BAC 0.08% 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose consisted of absolute alcohol in carbonated lemon-lime soda. Placebo consisted of carbonated mix, topped with 3 mL of alcohol, served in a glass sprayed with an alcohol mist.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Veldstra et al., 2012 (Study 1)	<p><i>For all eligible participants:</i></p> <p>Cannabis: Unclear.</p> <p>Alcohol: Unclear. Required to abstain for 24 hours prior to study sessions.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Self-reported no prior problems with drug abuse (assumed to include cannabis).</p> <p>Alcohol: Self-reported no prior problems with alcohol abuse.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BAC 0.03% 2. Target BAC 0.05% 3. Target BAC 0.08% 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose consisted of vodka in orange juice. Placebo consisted of orange juice sprayed with alcohol.</p>
Veldstra et al., 2012 (Study 2)	<p><i>For all eligible participants:</i></p> <p>Cannabis: No previous drug abuse or addiction (assumed to include cannabis). Required to abstain from “any drugs” from one week prior to study screening to end of study.</p> <p>Alcohol: Consumed 2 – 20 drinks per week. Required to abstain on the day prior to study sessions.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: $M = 7.8$ (5.8) drinks per week.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BAC 0.05% <p>In both the placebo and alcohol condition, participants also consumed a placebo capsule.</p>	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose consisted of vodka in orange juice. Placebo consisted of orange juice sprayed with alcohol.</p>
Veldstra et al., 2015	See Bosker et al., 2012, this table.	See Bosker et al., 2012, this table.	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo dronabinol <p>Cannabis:</p> <ol style="list-style-type: none"> 1. 10 mg dronabinol 2. 20 mg dronabinol 	<p>Cannabis: Dronabinol, including placebo, administered orally in capsule form.</p> <p>Alcohol: N/A</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Vermeeren & O'Hanlon, 1998	<p data-bbox="375 256 688 285"><i>For all eligible participants:</i></p> <p data-bbox="375 318 701 558">Cannabis: No current or previous drug abuse (assumed to include cannabis). Required to abstain from “drugs of abuse” (assumed to include cannabis) for one week prior to and during study sessions.</p> <p data-bbox="375 591 701 862">Alcohol: No current or previous alcohol abuse; consumption of no more than 28 alcoholic beverages weekly. Required to limit alcohol consumption to “two glasses of wine or beer with a meal” (p. 307) for duration of study.</p>	<p data-bbox="726 256 1031 285"><i>For all eligible participants:</i></p> <p data-bbox="726 318 995 347">Cannabis: Not reported.</p> <p data-bbox="726 380 1041 496">Alcohol: For $n = 12$ men, $M = 9.5$ (5.8) drinks weekly; for $n = 12$ women, $M = 6.0$ (6.0) drinks weekly.</p>	<p data-bbox="1073 256 1230 285">Comparison:</p> <ol data-bbox="1073 285 1304 315" style="list-style-type: none">1. Baseline driving <p data-bbox="1073 318 1178 347">Alcohol:</p> <ol data-bbox="1073 347 1331 376" style="list-style-type: none">1. Target BAC 0.05% <p data-bbox="1073 409 1482 496">In both the comparison and alcohol condition, participants also consumed placebo drug.</p>	<p data-bbox="1543 256 1717 285">Cannabis: N/A</p> <p data-bbox="1543 318 1864 406">Alcohol: Alcohol dose consisted of 99.8% alcohol in orange juice.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Vermeeren et al., 2002a	<p data-bbox="373 256 701 678"><i>For all eligible participants:</i></p> <p data-bbox="373 321 701 678">Cannabis: No history of drug abuse (assumed to include cannabis). Required to abstain from “drugs of abuse” (assumed to include cannabis) for two weeks prior to and during study sessions. Urine tested for drugs, but unclear whether this included cannabis, and if so, whether a positive test was an exclusion criterion.</p> <p data-bbox="373 716 701 894">Alcohol: Consumption of alcohol that does not exceed 40 g per day; no history of alcoholism. Required to abstain for 24 hours prior to and during study sessions.</p>	<p data-bbox="724 256 1031 289"><i>For all eligible participants:</i></p> <p data-bbox="724 321 1031 354">Cannabis: Not reported.</p> <p data-bbox="724 386 1031 407">Alcohol: Not reported.</p>	<p data-bbox="1073 256 1478 289">Comparison:</p> <ol data-bbox="1073 289 1478 321" style="list-style-type: none"> 1. Baseline driving <p data-bbox="1073 321 1478 354">Alcohol:</p> <ol data-bbox="1073 354 1478 386" style="list-style-type: none"> 1. Target BAC 0.05% <p data-bbox="1073 440 1478 532">In both the comparison and alcohol condition, participants also consumed placebo drug.</p>	<p data-bbox="1541 256 1877 289">Cannabis: N/A</p> <p data-bbox="1541 321 1877 407">Alcohol: Alcohol dose consisted of 99.8% alcohol in orange juice.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Vermeeren et al., 2002b (Part 1)	<p><i>For all eligible participants:</i></p> <p>Cannabis: No current or past drug abuse (assumed to include cannabis). Urine tested for cannabis, but unclear if positive test for cannabinoids was an exclusion criterion. Required to abstain from drugs (assumed to include cannabis) for two weeks prior to study sessions until end of study.</p> <p>Alcohol: No current or past alcoholism. Required to abstain from 24 hours prior to study sessions to end of study sessions.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: $M = 5.9$ (5.6) units of alcohol consumed per week.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BAC “just under” 0.05% 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose consisted of pure ethanol in orange juice and Grand Marnier essence, consumed while wearing a nose clip. Placebo consisted of orange juice and Grand Marnier essence, consumed while wearing a nose clip.</p>
Verster et al., 2002 (Part 1)	<p><i>For all eligible participants:</i></p> <p>Cannabis: Unclear.</p> <p>Alcohol: Unclear.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: No prior drug dependence (assumed to include cannabis). All had negative drug test for cannabinoids at start of testing.</p> <p>Alcohol: No prior alcohol dependence. All had negative breath alcohol test at start of testing.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BAC 0.05% 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose consisted of pure ethanol in orange juice and Grand Marnier essence, consumed while wearing a nose clip. Composition and administration of placebo alcohol not described.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Vollrath & Fischer, 2017 (Study 1)	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: No “alcohol problems” as indicated in the LAST; at least weekly alcohol consumption.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Not reported.</p>	<p>Comparison:</p> <p>1. Placebo alcohol</p> <p>Alcohol:</p> <p>1. Target BAC 0.05%</p>	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose consisted of vodka in passion fruit juice, orange juice and grenadine, divided into two beverages. Placebo consisted of passion fruit juice, orange juice and grenadine, divided into two beverages, each topped with drops of vodka.</p>
Vollrath & Fischer, 2017 (Study 2)	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: No “alcohol problems” as indicated in the LAST; at least weekly alcohol consumption.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Not reported.</p>	<p>Comparison:</p> <p>1. Placebo alcohol</p> <p>Alcohol:</p> <p>1. Target BAC 0.05%</p>	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose consisted of vodka in passion fruit juice, orange juice and grenadine, divided into two beverages. Placebo consisted of passion fruit juice, orange juice and grenadine, divided into two beverages, each topped with drops of vodka.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Wan et al., 2017	<p data-bbox="375 251 688 284"><i>For all eligible participants:</i></p> <p data-bbox="375 316 701 771">Cannabis: No current or previous drug use (including cannabis), no current or previous-year involvement in substance abuse treatment (assumed to include cannabis). Urine tested for marijuana, but unclear if positive test was an inclusion or exclusion criterion. Required to abstain from drugs except tobacco (assumed to include cannabis) for 72 hours prior to study sessions.</p> <p data-bbox="375 803 701 1045">Alcohol: No current or previous-year involvement in substance abuse treatment (assumed to include alcohol). Required to abstain for 24 hours prior to study sessions and pass a breath alcohol test prior to start of testing.</p>	<p data-bbox="726 251 1041 284"><i>For all eligible participants:</i></p> <p data-bbox="726 316 995 349">Cannabis: Not reported.</p> <p data-bbox="726 381 1041 682">Alcohol: For $n = 14$ “binge” drinkers, $M = 1.5$ (1.0) drinking occasions per week in past 3 months, with $M = 4.6$ (1.6) drinks per occasion; for $n = 14$ “non-binge” drinkers, $M = 0.8$ (0.5) drinking occasions per week in past 3 months, with $M = 2.0$ (0.5) drinks per occasion.</p>	<p data-bbox="1073 251 1226 284">Comparison:</p> <p data-bbox="1073 284 1293 316">1. Placebo alcohol</p> <p data-bbox="1073 316 1178 349">Alcohol:</p> <p data-bbox="1073 349 1331 381">1. Target BAC 0.08%</p>	<p data-bbox="1541 251 1717 284">Cannabis: N/A</p> <p data-bbox="1541 316 1881 592">Alcohol: Alcohol dose administered as 95% alcohol in tonic water, divided into three beverages. Placebo administered as tonic water, divided into three beverages, topped with 1 mL alcohol per beverage, served in glasses with alcohol-wiped rims.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Weafer & Fillmore, 2012	<p><i>For all eligible participants:</i></p> <p>Cannabis: No substance abuse disorder (assumed to include cannabis); urine tested for THC, but unclear if positive test was an exclusion criterion. Required to abstain from psychoactive drugs (assumed to include cannabis) for 24 hours prior to study sessions.</p> <p>Alcohol: No substance abuse disorder (assumed to include alcohol), and no potential risk for alcohol dependence (i.e., no score of 5+ on Short-Michigan Alcoholism Screening Test [SMAST]). Required to abstain for 24 hours prior to study sessions.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: $M = 1.7$ (0.9) drinking occasions per week, with $M = 4.6$ (2.3) drinks per occasion.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BAC 0.09% 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose consisted of absolute alcohol in carbonated soda. Placebo consisted of carbonated soda, topped with 3 mL of alcohol, served in a glass sprayed with alcohol mist.</p>
Weafer et al., 2008 (Study 1)	<p><i>For all eligible participants:</i></p> <p>Cannabis: No history of substance abuse (assumed to include cannabis).</p> <p>Alcohol: No history of substance abuse (assumed to include alcohol).</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: $M = 5.0$ (1.8) drinks per occasion (but number of occasions per unit of time not reported).</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BAC 0.08% 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose consisted of absolute alcohol in lemon soda. Placebo consisted of pure lemon soda.</p>

Study	Drug Use Inclusion Criteria*	Reported Drug Use Frequency	Drug Driving Conditions	Drug Administration
Weafer et al., 2008 (Study 2)	<p><i>For all eligible participants:</i></p> <p>Cannabis: Unclear; assumed to be similar to or the same as Study 1, above.</p> <p>Alcohol: Unclear; assumed to be similar to or the same as Study 1, above.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: $M = 3.4$ (1.9) drinks per occasion (but number of occasions per unit of time not reported).</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BAC 0.05% 2. Target BAC 0.08% 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose consisted of absolute alcohol in lemon soda. Placebo consisted of pure lemon soda.</p>
Weiler et al., 2000	<p><i>For all eligible participants:</i></p> <p>Cannabis: Unclear. No positive result on a drug screen, but unclear if this includes cannabinoids.</p> <p>Alcohol: Experience with alcohol, but not “excessive” alcohol use.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Not reported.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BAC 0.1% <p>In both the placebo and alcohol condition, participants also consumed a placebo capsule.</p>	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol administered as absolute alcohol in noncaffeinated carbonated soda, served in a glass with an alcohol-wiped rim. Placebo administered as “placebo alcohol” in noncaffeinated carbonated soda, served in a glass with an alcohol-wiped rim.</p>
Zhang et al., 2014	<p><i>For all eligible participants:</i></p> <p>Cannabis: No drug use (assumed to include cannabis).</p> <p>Alcohol: Experience with drinking a sufficient quantity of alcohol to reach BrAC 0.1%.</p>	<p><i>For all eligible participants:</i></p> <p>Cannabis: Not reported.</p> <p>Alcohol: Not reported.</p>	<p>Comparison:</p> <ol style="list-style-type: none"> 1. Placebo alcohol <p>Alcohol:</p> <ol style="list-style-type: none"> 1. Target BrAC 0.03% 2. Target BrAC 0.06% 3. Target BrAC 0.09% 	<p>Cannabis: N/A</p> <p>Alcohol: Alcohol dose administered as “Chinese liquor” in water. Placebo administered as pure water.</p>

*Note that “drug” specifically refers to cannabis and alcohol. Inclusion and exclusion criteria, drug use frequency and drug driving conditions specifically related to or specifically involving other drugs are not included in this table.

1. Subjects received an alcohol-free version of the alcoholic drink, but they were aware that it did not contain alcohol.

2. Participants in “challenge” and “repeated exposure” groups excluded. Only control group deemed eligible for inclusion.

3. The dose was reported as a target blood alcohol level of 0.05 mg/ml of alcohol during the driving tests. This is assumed to be reported in error because it would correspond to a BAC of 0.005%.

4. Participants in “training” group excluded. Only control group deemed eligible for inclusion.
5. This study had both a “BAC = 0.00” condition for the “alcohol session” and a “no phone usage” condition for the “mobile phone session.” Both are theoretically equivalent in that no alcohol was administered and therefore eligible for inclusion. For the sake of simplicity, only the former “BAC = 0.00” condition was used as a comparison for the purposes of this meta-analysis.
6. Doses reported as “.g/kg” in report, assumed to refer to micrograms per kilogram.
7. From the paper: “The initial alcohol dose was sufficient for achieving a peak BAC of about 0.07 g/dl. Booster doses were later given to sustain BAC around 0.04 g/dl during testing” (*Methods*, para. 1).
8. This study included two 0.64 g/kg alcohol conditions, but the condition involving sham feedback was excluded to avoid confounding experimental driving measures.
9. Three comparison conditions were reported in this paper: “Control” (i.e., alcohol placebo, no smoking), “Con +” (i.e., alcohol placebo, cannabis placebo) and “After” (i.e., “a session 24h after smoking the high dose THC cigarette, and drinking orange juice without smoking” [p. 928]). “Con +” was selected for inclusion, and both “Control” and “After”
10. Two comparison conditions were reported in the paper: placebo, and “24.” According to the paper, “24” is “identical to the placebo but was always used twenty-four hours after the combination of THC and alcohol” (p. 1858). For the purposes of the meta-analysis, the placebo condition was deemed eligible for inclusion, and “24” was excluded.
11. From the paper: “The treatments were either a placebo or a low (40 mg/l) or high (80 mg/l) dose of alcohol” (p. 4). These are assumed to be reported in error because these would correspond to 0.004% and 0.008% BAC, respectively. It is assumed that the intended doses are actually 40 mg/dL and 80 mg/dL which correspond to BAC 0.04% and 0.08%, respectively.
12. The medium alcohol dose was reported as “a goal of 0.05 mg/ml BAC” (p. 374) and the high alcohol dose was reported as “a goal of 0.08 mg/ml BAC” (p. 374). This is assumed to be reported in error because these would correspond to 0.005% BAC and 0.008% BAC, respectively.

Finally, recall that study conditions that involved driving while engaged in secondary tasks, other than embedded target detection and response tasks, were not eligible for inclusion in the meta-analysis. For example, data collected during a drive wherein participants used a cell phone while under the influence of alcohol would not be eligible for inclusion, but data for eligible measures collected during a drive wherein participants responded to a peripheral target, such as a light or other non-hazard (i.e., for the purposes of measuring detection rates and/or response times to those targets) would be eligible. Nonetheless, during data extraction, attempts were made to extract driving performance and behaviour data that was not contaminated by embedded target detection and response tasks. However, this was not possible in all cases. Specifically, effect sizes from Burns et al. (2002), Kay et al. (2013), Lenne et al. (1999), Lenne et al. (2003), Ronen et al. (2008) and Starkey and Charlton (2014) are contaminated by embedded target detection and response tasks.

Primary Meta-Analysis

Each meta-analysis was conducted in CMA. Although most studies contributed a single effect size, studies that included multiple independent subgroups (e.g., men and women, occasional users and heavy users, etc.) contributed more than one effect size. In a few cases, however, multiple independent groups needed to be aggregated into a single composite due to the constraints of the meta-analytic software (i.e., to avoid counting the control group twice, as discussed in *Chapter 2: Method*). However, when studies utilized multiple relevant comparisons (e.g., different levels of alcohol) or different comparisons (e.g., multiple measures relevant to a category in the meta-analysis), data were aggregated such that each study included in the meta-analysis contributed a single effect size per unique group of participants. Summary statistics were computed using random-effects meta-analysis.

Insufficient reporting of statistical data necessary for effect size computation was encountered frequently, particularly within older studies. Additionally, correlations between pairs of scores in studies were rarely reported in original studies that utilized repeated measures designs. Unless otherwise specified, the meta-analysis was conducted three times: once with pre-post correlations of zero, once with pre-post correlations of 0.5, and once with pre-post correlations of 0.9 substituted in cases where no pre-post correlation could be recovered.

In all of the following descriptions of study findings, asterisks indicate the number of statistically significant effects observed among the three meta-analyses.

Cannabis v. Baseline. Meta-analyses were conducted for crashes, hazard RT, headway, headway variability, lateral position variability, lane excursions, time out of lane, speed, speed variability and speed exceedances. Due to lack of data, time speeding was not meta-analyzed. Many of the analyses have a small number of included effect sizes, which limits precision and interpretation.

**Crashes.* This meta-analysis included one effect size representing 80 participants. Although several other studies measured collisions and were therefore eligible for inclusion, collisions often occurred so infrequently that statistical analyses could not be conducted. Consequently, means and standard deviations for crashes were often unreported.

With a pre-post correlation of zero, cannabis was not associated with a reliable change in crashes relative to baseline (Hedge's $g = 0.158$; 95% CI = -0.152, 0.467; Figure C1). Results were similar with a pre-post correlation of 0.5 (Hedge's $g = 0.155$; 95% CI = -0.063, 0.374; Figure C2). However, a statistically significant effect was observed with a pre-post correlation of 0.9 (Hedge's $g = 0.140$; 95% CI = 0.043, 0.238; Figure C3). Results, including a conversion to r effect size and prediction intervals, appear in Table 3, below.

Table 3. Effect of cannabis on crashes (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	0.079	0.158	-0.152	0.467	N/A	N/A
0.5	0.078	0.155	-0.063	0.374	N/A	N/A
0.9*	0.071	0.140	0.043	0.238	N/A	N/A

The pattern of results indicates a lack of compelling evidence that cannabis increases rates of simulated crashes relative to baseline. The statistically significant effect observed with a pre-post correlation of 0.9 is trivial in magnitude, and the pre-post correlation itself may or may not be as high as 0.9. Additionally, the 95% confidence intervals indicate a lack of precision; consequently, the average effects reported here may or may not be reliable. Finally, there is not enough data to compute prediction intervals or for the meaningful exploration of small study effects and potential moderating factors that might influence the magnitude of the summary statistic.

***Hazard RT.** This meta-analysis includes nine effect sizes representing approximately 242 participants. These effects represent the time taken to respond to obstacles on the shoulder of the road (Anderson et al., 2010), obstacles within the roadway (Liguori et al., 1998; Liguori et al., 2002), slowing forward vehicles (Ramaekers et al., 2000b [Study 1]; Robbe, 1998 [Study 2]; Sexton et al., 2000; Sexton et al., 2002), vehicles pulling out in front of the participant's vehicle (Sexton et al., 2000; Sexton et al., 2002) and general "emergencies"⁶ (Downey et al., 2013). Although measures relevant to hazard RT were conceptualized as relating to circumstances

⁶ The nature of the events to which participants responded is not entirely clear. However, based on their conceptualization as "emergencies," and based on the description of the simulator and the other experimental driving measures included in the study, they do not appear to be secondary peripheral targets (i.e., Target RT).

wherein participants' failure to respond would lead to a collision, it should be noted that the data included from Robbe (1998; Study 2) may have included responses not only to decelerations of the forward vehicle, but also to accelerations of the forward vehicle.

In addition, it should be noted that Sexton et al. (2000) did not appear to state the number of participants represented in the means and standard deviations reported for hazard RT measures; instead, it appears that the number of individual data points for all participants are reported. Sample sizes for the four conditions were assumed to be the same as those reported for other measures – specifically, $n = 14$ for the low THC dose, $n = 15$ for the high THC dose, $n = 13$ for resin, and $n = 14$ for the placebo dose.

With a pre-post correlation of zero, cannabis was not associated with a reliable change in hazard RT relative to baseline (Hedge's $g = 0.115$; 95% CI = -0.077, 0.307; Figure C4). A pre-post correlation of 0.5 yielded similar results (Hedge's $g = 0.148$, 95% CI = -0.013, 0.309; Figure C5). However, results became statistically significant with a pre-post correlation of 0.9 (Hedge's $g = 0.164$; 95% CI = 0.037, 0.290; Figure C6). Results, including a conversion to r effect size and prediction intervals, appear in Table 4, below.

Table 4. Effect of cannabis on hazard RT (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	0.057	0.115	-0.077	0.307	-0.117	0.347
0.5	0.080	0.148	-0.013	0.309	-0.138	0.434
0.9*	0.086	0.164	0.037	0.290	-0.206	0.534

As with crashes, the pattern of findings indicate that there is little evidence that cannabis changes hazard RT relative to baseline. Again, this is due to a lack of evidence, and lack of evidence is not evidence of a null effect. The statistically-significant increase observed with a

pre-post correlation of 0.9 is small in magnitude, and it is unknown if the actual pre-post correlation is high as 0.9. The 95% confidence intervals indicate a lack of precision. Partly due to imprecision, the 95% prediction intervals are also wide and indicate that the true effect probably ranges from a small to trivial decrease in hazard RT, to a small to moderate increase in hazard RT (depending on the pre-post correlation used). There are not enough studies included in the meta-analysis to meaningfully explore small study effects or the influence of potential moderating factors.

Headway. This meta-analysis includes one effect size representing 14 participants. Pre-post correlations were recovered from raw data provided by the study author of Arkell et al. (2019) (Thomas Arkell, personal communication dated September 13, 2019). Thus, the meta-analysis only needed to be conducted once.

Cannabis was not associated with a reliable change in headway relative to baseline (Hedge's $g = 0.304$; 95% CI = -0.171, 0.780; Figure C7). Results, including a conversion to r effect size and prediction intervals, appear in Table 5, below.

Table 5. Effect of cannabis on headway (compared to baseline).

r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.160	0.304	-0.171	0.780	N/A	N/A

Once again, the 95% confidence intervals indicate a lack of measurement precision, and there is not enough data to compute prediction intervals or for the meaningful exploration of small study effects or potential moderating factors.

Headway Variability. This meta-analysis includes one effect size representing 14 participants. As with headway, pre-post correlations were recovered from raw data for one study

(Thomas Arkell, personal communication dated September 13, 2019), so the meta-analysis only needed to be conducted once.

As with headway, cannabis was not associated with a change in headway variability compared to baseline (Hedge's $g = 0.319$; 95% CI = -0.313, 0.951; Figure C8). Results, including a conversion to r effect size and prediction intervals, appear in Table 6, below.

Table 6. Effect of cannabis on headway variability (compared to baseline).

r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.166	0.319	-0.313	0.951	N/A	N/A

The 95% confidence intervals indicate a lack of precision. Due to the inclusion of only a single effect size in the meta-analysis, prediction intervals cannot be computed, and small study effects and potential moderators cannot be explored meaningfully.

*****Lateral Position Variability.** This meta-analysis included 14 effect sizes representing approximately 257 participants. Pre-post correlations were only recovered for one of the included studies; specifically, raw data was provided by the author of Arkell et al. (2019) (Thomas Arkell, personal communication dated September 13, 2019).

With pre-post correlations of zero, cannabis was associated, on average, with a small increase in lateral position variability (Hedge's $g = 0.366$; 95% CI = 0.205, 0.528; Figure C9). Pre-post correlations of 0.5 yielded similar results (Hedge's $g = 0.331$; 95% CI = 0.212, 0.451; Figure C10), as did pre-post correlations of 0.9 (Hedge's $g = 0.270$; 95% CI = 0.175, 0.365; Figure C11). Results, including a conversion to r effect size and prediction intervals, appear in Table 7, below.

Table 7. Effect of cannabis on lateral position variability (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0*	0.191	0.366	0.205	0.528	0.186	0.546
0.5*	0.173	0.331	0.212	0.451	0.199	0.464
0.9*	0.141	0.270	0.175	0.365	-0.061	0.602

The results indicate that cannabis increases lateral position variability relative to baseline.

The effect is quite consistent; the 95% prediction intervals are not much wider than the 95% confidence intervals, which indicates that the influence of unknown moderating factors is minimal (except in the case where the pre-post correlation is set to 0.9). According to the 95% prediction intervals, the true effect lies somewhere between a small and moderate increase in lateral position variability for pre-post correlations of zero and 0.5. For a pre-post correlation of 0.9, the effect may range from a trivial decrease in lateral position variability to a moderate increase in lateral position variability. Nonetheless, it appears that cannabis will, more often than not, lead to an increase in lateral position variability within experimental studies.

Next, small study effects were explored. There was no compelling evidence for publication bias with this particular measure and comparison: funnel plots (i.e., Hedge's g by standard error) appeared somewhat ambiguous in all three meta-analyses (see Figures E1 to E3), but Egger's test did not indicate the presence of small study effects for analyses with a pre-post correlation of zero [$t(12) = 1.382, p = 0.192$, two-tailed test], with a pre-post correlation of 0.5 [$t(12) = 2.077, p = 0.060$, two-tailed test] or with a pre-post correlation of 0.9 [$t(12) = 1.922, p = 0.079$, two-tailed test].

****Lane Excursions.** This meta-analysis included two effect sizes representing 98 participants. With a pre-post correlation of zero, cannabis was not associated with a reliable

change in lane excursions (Hedge's $g = 0.201$; 95% CI = -0.078, 0.480; Figure C12). However, results became statistically significant with a pre-post correlation of 0.5 (Hedge's $g = 0.198$; 95% CI = 0.001, 0.395; Figure C13) and a pre-post correlation of 0.9 (Hedge's $g = 0.180$; 95% CI = 0.092, 0.268; Figure C14). Results, including a conversion to r effect size and prediction intervals, appear in Table 8, below.

Table 8. Effect of cannabis on lane excursions (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	0.102	0.201	-0.078	0.480	N/A	N/A
0.5*	0.100	0.198	0.001	0.395	N/A	N/A
0.9*	0.091	0.180	0.092	0.268	N/A	N/A

Although the 95% confidence intervals indicate a lack of precision, the pattern of results suggests that cannabis may, on average, increase lane excursions relative to baseline. However, this rests on the assumption that there is at least a small correlation between pairs of measures in the included studies utilizing repeated-measures designs. Due to the small number of studies included in the meta-analysis, prediction intervals cannot be generated, and small study effects and potential moderating factors cannot be explored meaningfully.

Time Out of Lane. This meta-analysis included one effect size representing 18 participants. With a pre-post correlation of zero, cannabis was not associated with a reliable change in time out of lane compared to baseline (Hedge's $g = 0.219$; 95% CI = -0.417, 0.856; Figure C15). Similar results were obtained with a pre-post correlation of 0.5 (Hedge's $g = 0.212$; 95% CI = -0.237, 0.661; Figure C16) and a pre-post correlation of 0.9. (Hedge's $g = 0.180$; 95% CI = -0.020, 0.380; Figure C17). Results, including a conversion to r effect size and prediction intervals, appear in Table 9, below.

Table 9. Effect of cannabis on time out of lane (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	0.114	0.219	-0.417	0.856	N/A	N/A
0.5	0.110	0.212	-0.237	0.661	N/A	N/A
0.9	0.093	0.180	-0.020	0.380	N/A	N/A

Overall, cannabis is not associated with a reliable change in time out of lane relative to baseline. The 95% confidence intervals indicate an appreciable lack of precision, and the results are limited by the inclusion of only one study in the meta-analysis which precludes the generation of prediction intervals and the meaningful exploration of small study effects and potential moderators.

****Speed*. This meta-analysis included 12 effect sizes representing 312 participants. Pre-post correlations could not be recovered for any studies utilizing repeated measures designs except for the one associated with Arkell et al. (2019; Thomas Arkell, personal communication dated September 13, 2019).

With a pre-post correlation of zero, cannabis was associated a decrease in speed compared to baseline (Hedge's $g = -0.182$; 95% CI = -0.348, -0.017; Figure C18). Results were similar with a pre-post correlation of 0.5 (Hedge's $g = -0.176$; 95% CI = -0.298, -0.053; Figure C19) and a pre-post correlation of 0.9 (Hedge's $g = -0.205$; 95% CI = -0.336, -0.074; Figure C20). Results, including a conversion to r effect size and prediction intervals, appear in Table 10, below.

Table 10. Effect of cannabis on speed (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0*	-0.095	-0.182	-0.348	-0.017	-0.371	0.006
0.5*	-0.092	-0.176	-0.298	-0.053	-0.315	-0.036
0.9*	-0.107	-0.205	-0.336	-0.074	-0.639	0.230

Overall, cannabis is associated with, on average, a small decrease in speed. However, the 95% confidence intervals indicate a lack of precision. Owing in part to this, as well as the influence of unknown moderating factors, the 95% prediction intervals indicate that the true effect lies somewhere between a trivial to moderate decrease in speed, to a trivial to small increase in speed (depending on the pre-post correlation used)..

Small study effects. There was no compelling evidence for publication bias with this particular measure and comparison: funnel plots (i.e., Hedge's g by standard error) were somewhat ambiguous in all three meta-analyses (see Figures E4 to E6), likely due in part to the small number of included datapoints, but Egger's test did not indicate the presence of small study effects for analyses with a pre-post correlation of zero [$t(10) = 1.066$, $p = 0.312$, two-tailed test], with a pre-post correlation of 0.5 [$t(10) = 1.075$, $p = 0.308$, two-tailed test] or with a pre-post correlation of 0.9 [$t(10) = 0.953$, $p = 0.363$, two-tailed test].

**Speed Variability.* This meta-analysis included seven effect sizes representing 137 participants. Pre-post correlations for one of the included studies were recovered from raw data provided by the study author (Thomas Arkell, personal communication dated September 13, 2019).

With a pre-post correlation of zero, cannabis was not associated with a change in speed variability compared to baseline (Hedge's $g = 0.047$; 95% CI = -0.220, 0.314; Figure C21).

Results were similar with a pre-post correlation of 0.5 (Hedge's $g = 0.104$; 95% CI = -0.113, 0.321; Figure C22) but became statistically significant with a pre-post correlation of 0.9 (Hedge's $g = 0.166$; 95% CI = 0.048, 0.284; Figure C23). Results, including a conversion to r effect size and prediction intervals, appear in Table 11, below.

Table 11. Effect of cannabis on speed variability (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	0.023	0.047	-0.220	0.314	-0.303	0.397
0.5	0.054	0.104	-0.113	0.321	-0.180	0.388
0.9*	0.088	0.166	0.048	0.284	-0.020	0.352

As with crashes and hazard RT, the pattern of results suggest that cannabis is not associated with a reliable change in speed variability relative to baseline. The 95% confidence intervals indicate a lack of precision. Even in the case of the statistically-significant increase with a pre-post correlation of 0.9, the increase is small in magnitude, and the 95% prediction intervals indicate that the true effect lies somewhere between a trivial to small decrease in speed variability to a small increase in speed variability.

***Speed Exceedances.** This meta-analysis included one effect size representing 80 participants. With a pre-post correlation of zero, cannabis was not associated with a change in speed exceedances compared to baseline (Hedge's $g = -0.206$; 95% CI = -0.516, 0.104; Figure C24). Results were similar with a pre-post correlation of 0.5 (Hedge's $g = -0.205$; 95% CI = -0.425, 0.014; Figure C25), but statistical significance was achieved with a pre-post correlation of 0.9 (Hedge's $g = -0.202$; 95% CI = -0.300, -0.104; Figure C26). Results, including a conversion to r effect size and prediction intervals, appear in Table 12, below.

Table 12. Effect of cannabis on speed exceedances (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	-0.103	-0.206	-0.516	0.104	N/A	N/A
0.5	-0.103	-0.205	-0.425	0.014	N/A	N/A
0.9*	-0.101	-0.202	-0.300	-0.104	N/A	N/A

As with crashes, hazard RT and speed exceedances, there is little evidence to suggest that cannabis reliably changes rates of speed exceedances relative to baseline. A statistically significant decrease in speed exceedances is only observed with a pre-post correlation of 0.9, which may be optimistically high, and the 95% confidence intervals indicate a lack of measurement precision. The results are limited by the inclusion of only one effect size in the meta-analysis, which precludes the generation of prediction intervals and meaningful exploration of small study effects and potential moderators.

Summary of the effects of cannabis. Little data is available to quantify the effect of cannabis on measures of driving performance and behaviour. Most of the effects considered here suffer from measurement imprecision due to a small number of studies reporting the statistical data needed to calculate effect size. However, cannabis had a consistent effect on lateral control and speed. Lateral position variability and rates of lane excursions were generally increased by cannabis, and speed was generally decreased by cannabis. More research is needed to reliably quantify effect size magnitude and identify circumstances in which they may vary.

Alcohol v. Baseline. Meta-analyses were conducted for crashes, hazard RT, headway, headway variability, lateral position variability, lane excursions, time out of lane, speed, speed variability, speed exceedances and time speeding. In contrast to *Cannabis v. Baseline* comparisons (above), there was much more data available for analyses.

****Crashes*. This meta-analysis included 14 effect sizes representing 441 participants. It should be noted that for Bernosky-Smith et al. (2011), a dropout was reported, yielding an overall sample size of 59; however, it could not be ascertained which group the dropout occurred in. Thus, all sample sizes were set to 15, yielding an overall sample size of 60 in the meta-analysis, which conflicts with the number of eligible participants stated in Table 1. Additionally, both Marczinski et al. (2008) and Study 2 from Roberts et al. (2016) contained data that posed technical issues to the meta-analytic software. Specifically, these data reported standard deviations of zero (likely as a result of rounding) for baseline means which precluded the computation of Hedge's g effect sizes. Thus, Marczinski et al. (2008) only contributed data from 24 participants (out of 40 total), and Roberts et al. (2016) contributed data from only one of the two eligible drives in their study. Finally, pre-post correlations could only be recovered for one of the included studies (Bernosky-Smith et al., 2012).

With a pre-post correlation of zero, alcohol increased crashes relative to baseline (Hedge's $g = 0.374$; 95% CI = 0.106, 0.643; Figure C27). Results were similar with a pre-post correlation of 0.5 (Hedge's $g = 0.376$; 95% CI = 0.150, 0.603; Figure C28) and a pre-post correlation of 0.9 (Hedge's $g = 0.352$; 95% CI = 0.187, 0.517; Figure C29). Results, including a conversion to r effect size and prediction intervals, appear in Table 13, below.

Table 13. Effect of alcohol on crashes (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0*	0.204	0.374	0.106	0.643	-0.541	1.289
0.5*	0.203	0.376	0.150	0.603	-0.424	1.177
0.9*	0.181	0.352	0.187	0.517	-0.269	0.973

Overall, alcohol increases crashes to a small extent relative to baseline. However, the 95% confidence intervals indicate a lack of measurement precision. Additionally, in part due to a lack of precision (but also due to the influence of unknown moderating factors), the prediction intervals are wide. The true effect of alcohol lies somewhere between a small to moderate decrease in crashes, to a large increase in crashes, depending on the pre-post correlation used. Thus, although the average effect is a small increase in crashes, the effect of alcohol does not appear consistent.

Small study effects. Small study effects were explored with funnel plots and Egger's test across all three meta-analyses (i.e., pre-post correlations of zero, 0.5 and 0.9). With a pre-post correlation of zero, funnel plots (Hedge's g by standard error) appeared asymmetrical (Figure E7), but Egger's test was not statistically significant, $t(12) = 1.759, p = 0.104$ (two-tailed test). A similar funnel plot asymmetry was observed with pre-post correlations of 0.5 (Figure E8), as well as a lack of statistical significance in Egger's test, $t(12) = 1.319, p = 0.212$. Funnel plots became more ambiguous with a pre-post correlation of 0.9 (Figure E9), but Egger's test remained non-statistically significant, $t(12) = 1.020, p = 0.328$ (two-tailed test).

The negatively-skewed effect size from Bernosky-Smith et al. (2012) appeared to offset the positive bias in the funnel plot and render Egger's test statistically non-significant. Given that this was the only study associated with a negative effect size in the analysis, it was reviewed for accuracy in data extraction and effect size computation. In reviewing the data, the means and standard deviations reported in the original paper appeared to be rounded to the nearest whole number. If this is the case, then the effect size calculated based on these values may be imprecise due to rounding error. Thus, the study was removed from the analysis to test for its sensitivity to inclusion.

Re-analysis. When removed, the effect size increased with a pre-post correlation of zero (Hedge's $g = 0.419$; 95% CI = 0.264, 0.574; Figure C30), with a pre-post correlation of 0.5 (Hedge's $g = 0.431$; 95% CI = 0.275, 0.587; Figure C31) and with a pre-post correlation of 0.9 (Hedge's $g = 0.414$; 95% CI = 0.263, 0.564; Figure C32). Results, including a conversion to r effect size and prediction intervals, appear in Table 14, below.

Table 14. Re-analysis of the effect of alcohol on crashes (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0*	0.227	0.419	0.264	0.574	0.245	0.593
0.5*	0.236	0.431	0.275	0.587	0.027	0.835
0.9*	0.217	0.414	0.263	0.564	-0.130	0.958

In all three cases, funnel plot asymmetries became more apparent (Figures E10 to E12). Egger's regression test became statistically significant, [$t(11) = 4.481, p < 0.001$, pre-post correlation = zero; $t(11) = 3.929, p = 0.002$, pre-post correlation = 0.5; $t(11) = 2.310, p = 0.041$, pre-post correlation = 0.9; all two-tailed tests].

Given that tests for publication bias tend to be underpowered (Borenstein et al., 2009, p. 284), the observation of statistically significant Egger's regression tests with a small number of included effect sizes was surprising. Borenstein and colleagues (2009, p. 291) warn that positive results in tests for small study effects are not necessarily evidence of publication bias; it is possible that the relationship between study precision and effect size is genuine. Because effect sizes were theorized to moderate effect size magnitude (see *Subgroup Analyses*, below), a post-hoc analysis was conducted wherein Egger's regression test was repeated with the addition of average BAC as a second predictor. Unfortunately, average BAC could not be verified to all

included studies, which led to some data loss. Specifically, eight of the original 13 effect sizes (following the removal of Bernosky-Smith et al., 2012) were retained.

The post-hoc analysis was conducted in IBM SPSS Statistics Version 24 rather than CMA in order to accommodate the inclusion of a second predictor. Specifically, standard error of the effect size was set as the predictor (first step), average BAC was set as a second predictor (second step), Hedge's g was set as criterion, and inverse variance of the effect size was set as the weight in a weighted least squares regression. When standard error is found to be a significant predictor of effect size, small study effects are indicated. The results of this post-hoc test are reported in Table 15, below.

Table 15. The relationship between Hedge's g and SE, with and without BAC.

Pre-Post r	k	Without BAC (Step 1)	With BAC (Step 2)
0.0	8	$t = 3.608, p = 0.011$	$t = 1.580, p = 0.175$
0.5	8	$t = 3.299, p = 0.016$	$t = 1.813, p = 0.130$
0.9	8	$t = 1.830, p = 0.117$	$t = 1.591, p = 0.172$

The post-hoc analyses indicate the despite the smaller sample of included effect sizes, Egger's regression test (i.e., Step 1) remained statistically significant with pre-post correlations of zero and 0.5. However, the statistically significant relationship between standard error and effect size disappeared with the addition of BAC to the regression model (i.e., Step 2). Thus, the relationship between effect size and its standard error may not be due to small study effects but possibly due to differences in BAC. In sum, there is no compelling evidence for publication bias in this set of analyses, and overall, alcohol is associated with a small increase in crashes.

*****Hazard RT.** This meta-analysis included 18 effect sizes representing approximately 451 participants. Participants responded to hazards including forward vehicles (Howard et al.,

2007; Kuypers et al., 2006; Ramaekers et al., 2000b [Study 1]; Sexton et al., 2002; Leung et al., 2012; Schumacher et al., 2017; Strayer et al., 2006) yellow and red traffic lights (Jelen et al., 2011; Wan et al., 2017), on-road obstacles (Liguori et al., 1999; Liguori & Robinson, 2001; Liguori et al., 2002) approaching pedestrians and vehicles (Berthelon & Gineyt, 2014; Vollrath & Fischer, 2017 [Studies 1 and 2]; Sexton, 1997; Sexton et al., 2002; Beard, 2012) and general “emergencies” (Downey et al., 2013). Pre-post correlations were recovered for Jelen et al. (2011) and Liguori et al. (1999).

With a pre-post correlation of zero, alcohol was associated with an increase (i.e., slowing) in hazard RT (Hedge’s $g = 0.283$; 95% CI = 0.100, 0.466; Figure C33). Results were similar with a pre-post correlation of 0.5 (Hedge’s $g = 0.288$; 95% CI = 0.115, 0.462; Figure C34) and pre-post correlations of 0.9 (Hedge’s $g = 0.280$; 95% CI = 0.131, 0.429; Figure C35). Results, including a conversion to r effect size and prediction intervals, appear in Table 16, below.

Table 16. Effect of alcohol on hazard RT (compared to baseline).

Pre-Post r	r	Hedge’s g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0*	0.148	0.283	0.100	0.466	-0.278	0.844
0.5*	0.150	0.288	0.115	0.462	-0.342	0.918
0.9*	0.144	0.280	0.131	0.429	-0.343	0.903

On average, alcohol is associated with a small increase (i.e., slowing) in hazard RT.

However, the 95% confidence intervals indicate some measurement imprecision. Due to this, and also due to the influence of unknown moderating factors, the prediction intervals indicate that the true effect of alcohol lies somewhere between a small decrease in hazard RT to a moderate

increase in hazard RT. Thus, although the average effect of alcohol is a small increase in hazard RT, this average obfuscates substantial variability in effects.

Small study effects. Small study effects were explored with funnel plots and Egger's test across all three meta-analyses (i.e., pre-post correlations of zero, 0.5 and 0.9). With a pre-post correlation of zero, the funnel plot was somewhat ambiguous (Figure E13), but Egger's regression test was not statistically significant: $t(16) = 0.696, p = 0.497$ (two-tailed test). There was no obvious asymmetry with a pre-post correlation (Figure E14) and no statistically significant effect with Egger's regression test, $t(16) = 0.034, p = 0.973$ (two-tailed test). The funnel plot was again somewhat ambiguous with a pre-post correlation of 0.9 (Figure E15); however, Egger's regression test remained non-statistically significant, $t(16) = 0.715, p = 0.485$ (two-tailed test). Thus, there was no compelling evidence of small study effects in this meta-analysis.

Headway. This meta-analysis included six effect sizes representing approximately 120 participants. Pre-post correlations were recovered for only one of the included studies (McCartney et al., 2017; Danielle McCartney, personal communication dated October 10, 2019).

With a pre-post correlation of zero, alcohol did not reliably change headway relative to baseline (Hedge's $g = 0.071$; 95% CI = -0.319, 0.461; Figure C36). Results were similar with a pre-post correlation of 0.5 (Hedge's $g = 0.140$; 95% CI = -0.247, 0.528; Figure C37) and a pre-post correlation of 0.9 (Hedge's $g = 0.166$; 95% CI = -0.192, 0.524; Figure C38). Results, including a conversion to r effect size and prediction intervals, appear in Table 17, below.

Table 17. Effect of alcohol on headway (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	0.066	0.071	-0.319	0.461	-1.083	1.225
0.5	0.090	0.140	-0.247	0.528	-1.140	1.421
0.9	0.087	0.166	-0.192	0.524	-1.128	1.460

The results indicate that alcohol and baseline may not differ in terms of headway.

However, the 95% confidence intervals indicate a lack of precision, and the prediction intervals indicate the influence of unknown moderating factors. According to the prediction intervals, the true effect lies somewhere between a very large decrease in headway and a very large increase in headway. There are not enough studies included in this meta-analysis for meaningful exploration of potential moderating factors or small study effects.

****Headway Variability.** This meta-analysis included four effect sizes representing 82 participants. With a pre-post correlation of zero, there was no reliable difference between alcohol and baseline for headway variability (Hedge's $g = 0.561$; 95% CI = -0.022, 1.143; Figure C39). However, results became statistically significant with a pre-post correlation of 0.5 (Hedge's $g = 0.634$; 95% CI = 0.061, 1.207; Figure C40) and a pre-post correlation of 0.9 (Hedge's $g = 0.674$; 95% CI = 0.141, 1.207; Figure C41). Results, including a conversion to r effect size and prediction intervals, appear in Table 18, below.

Table 18. Effect of alcohol on headway variability (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	0.327	0.561	-0.022	1.143	-1.735	2.857
0.5*	0.340	0.634	0.061	1.207	-1.871	3.138
0.9*	0.326	0.674	0.141	1.207	-1.874	3.221

The results indicate that headway variability generally increases with alcohol. The average increase is moderate in magnitude; however, the 95% confidence intervals indicate a lack of precision, and the prediction intervals indicate the influence of unknown moderating factors. The true effect lies somewhere between a very large decrease in headway variability and a very large increase in headway variability. The small number of studies included in the meta-analysis precludes meaningful exploration of small study effects and potential moderators.

*****Lateral Position Variability.** This meta-analysis included 63 effect sizes representing approximately 1,573 participants. Pre-post correlations were recovered from Harrison et al. (2005) and from raw data associated with McCartney et al. (2017) (Danielle McCartney, personal communication dated October 10, 2019) and Helland et al. (2016) (Arne Helland, personal communication dated March 9, 2020).

With a pre-post correlation of zero, alcohol was associated with an increase in lateral position variability (Hedge's $g = 0.498$, 95% CI = 0.411, 0.585; Figure C42). Results were similar with pre-post correlations of 0.5 (Hedge's $g = 0.495$, 95% CI = 0.413, 0.578; Figure C43) and pre-post correlations of 0.9 (Hedge's $g = 0.428$; 95% CI = 0.353, 0.502; Figure C44). Results, including a conversion to r effect size and prediction intervals, appear in Table 19, below.

Table 19. Effect of alcohol on lateral position variability (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0*	0.310	0.498	0.411	0.585	0.170	0.826
0.5*	0.307	0.495	0.413	0.578	0.027	0.964
0.9*	0.277	0.428	0.353	0.502	-0.114	0.969

On average, alcohol is associated with a small to moderate increase in lateral position variability (depending on the pre-post correlation used). However, the 95% confidence intervals indicate a degree of imprecision, and the prediction intervals indicate the appreciable influence of unknown moderating factors. Again, although the average effect of alcohol is a small to moderate increase in lateral position variability, this average obfuscates inconsistency in the effect.

Small study effects. Interestingly, the funnel plots (Hedge's g by standard error) generated for inspection of small study effects were uninterpretable due to the presence of an extreme outlier (Veldstra et al., 2012, Study 1) which skewed the scale of the plot such that all other studies were tightly packed at the top of the funnel plot (see Figures E16 to E18). Egger's regression was statistically significant with a pre-post correlation of zero [$t(61) = 4.391, p < .001$, two-tailed test], with a pre-post correlation of 0.5 [$t(61) = 3.993, p < .001$, two-tailed test] and with a pre-post correlation of 0.9 [$t(61) = 2.952, p = 0.004$, two-tailed test]. Due to the massive difference in effect size magnitude between Study 1 from Veldstra et al. (2012) and rest of the included effect sizes, it was reviewed for accuracy in data extraction and effect size computation. Although no specific issues were identified, this study was omitted due to suspicion that the statistical data reported in the paper, from which the effect size was computed, were erroneous.

Re-analysis. With a pre-post correlation of zero, the resulting meta-analysis still indicated that alcohol was associated with an increase in lateral position variability (Hedge's $g = 0.486$; 95% CI = 0.409, 0.564; Figure C45). Results were similar with a pre-post correlation of 0.5 (Hedge's $g = 0.489$, 95% CI = 0.417, 0.562; Figure C46) and 0.9 (Hedge's $g = 0.422$, 95% CI = 0.360, 0.485; Figure C47). Notably, the prediction intervals narrowed appreciably with the

omission of Study 1 from Veldstra et al. (2012). Results, including a conversion to r effect size and prediction intervals, appear in Table 20, below.

Table 20. Re-analysis of the effect of alcohol on lateral position variability (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0*	0.260	0.486	0.409	0.564	0.336	0.637
0.5*	0.254	0.489	0.417	0.562	0.136	0.842
0.9*	0.216	0.422	0.360	0.485	-0.016	0.861

On average, alcohol increased lateral position variability to a small to moderate degree.

The prediction intervals still indicated the influence of unknown moderating factors in all three cases, but not to the same degree as the original meta-analyses that included Study 1 from Veldstra et al. (2012).

Finally, small study effects were investigated once again. With a pre-post correlation of zero, the funnel plot appeared asymmetrical due to the presence of a few right-skewed datapoints (Figure E19), and Egger's regression test was statistically significant, $t(60) = 3.096$, $p = .003$ (two-tailed test). Similarly, with a pre-post correlation of 0.5, the funnel plot was somewhat asymmetrical (Figure E20), and Egger's regression test was again statistically significant, $t(60) = 2.573$, $p = 0.013$. With a pre-post correlation of 0.9, the funnel plot was more ambiguous (Figure E21); however, Egger's regression was not statistically significant, $t(60) = 1.671$, $p = 0.100$ (two-tailed test). Again, statistically significant effects with Egger's regression tests were somewhat surprising, so a post-hoc test was conducted wherein average BAC level was added as a second predictor to Egger's regression test. However, as with crashes, average BAC could not be verified to all included effect sizes. Of the original 62 effect sizes (following the removal of

Study 1 from Veldstra et al. [2012]), 45 effect sizes were retained. The results of this post-hoc test are reported in Table 21, below.

Table 21. The relationship between Hedge's g and SE, with and without BAC.

Pre-Post r	k	Without BAC (Step 1)	With BAC (Step 2)
0.0	45	$t = 3.292, p = .002$	$t = 3.242, p = .002$
0.5	45	$t = 2.831, p = .007$	$t = 2.755, p = .009$
0.9	45	$t = 2.119, p = .040$	$t = 2.252, p = .030$

Interestingly, the relationship between Hedge's g and its standard error persisted even after the inclusion of average BAC as a second predictor in the weighted least squares regression. Consequently, small study effects are apparent. However, it remains unclear whether these effects are due to publication bias or some other legitimate relationship between effect size and its standard error. Thus, it is also unclear whether the effect sizes reported in the meta-analysis are spuriously high, and if so, the degree to which they need to be adjusted.

*****Lane Excursions.** This meta-analysis included 25 effect sizes representing approximately 686 participants. As noted above, Bernosky-Smith et al. (2011) reported a dropout, yielding an overall sample size of 59; however, because it was not known which group the dropout occurred in, all sample sizes were set to 15, yielding an overall sample size of 60 in the meta-analysis. Pre-post correlations were recovered from raw data associated with McCartney et al. (2017) (Danielle McCartney, personal communication dated October 10, 2019). Additionally, Kenntner-Mabiala et al. (2015) contained data that posed technical issues to the meta-analytic software. These data reported standard deviations of zero for some comparisons which precluded the computation of Hedge's g effect sizes. However, all 24 participants are still represented in the analysis.

With a pre-post correlation of zero, alcohol was associated with an increase in lane excursions (Hedge's $g = 0.504$; 95% CI = 0.334, 0.674; Figure C48). Results were similar with a pre-post correlation of 0.5 (Hedge's $g = 0.502$; 95% CI = 0.337, 0.667; Figure C49) and a pre-post correlation of 0.9 (Hedge's $g = 0.439$; 95% CI = 0.297, 0.580; Figure C50). Results, including a conversion to r effect size and prediction intervals, appear in Table 22, below.

Table 22. Effect of alcohol on lane excursions (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0*	0.300	0.504	0.334	0.674	-0.120	1.128
0.5*	0.288	0.502	0.337	0.667	-0.210	1.213
0.9*	0.225	0.439	0.297	0.580	-0.248	1.125

On average, alcohol increased lane excursions to a small degree. However, the 95% confidence intervals indicate a lack of measurement precision. Partly due to this, and partly due to the influence of unknown moderating factors, the prediction intervals indicate the true effect of alcohol lies somewhere between a small decrease in lane excursions and a large increase in lane excursions. Thus, although the average effect was a small increase, the effect of alcohol is not consistent.

Small study effects. With a pre-post correlation of zero, the funnel plot (i.e., Hedge's g by standard error) appeared asymmetrical (Figure E22), and Egger's regression was statistically significant, $t(23) = 4.616$, $p < 0.001$ (two-tailed test). A similar asymmetry and statistically significant Egger's regression was observed with a pre-post correlation of 0.5 [$t(23) = 4.073$, $p < 0.001$, two-tailed test] and with a pre-post correlation of 0.9 [$t(23) = 2.164$, $p = 0.041$, two-tailed test; see Figures E23 and E24].

The funnel plot asymmetries and significant Egger's regression tests may have been driven in part by the large effect sizes associated with Berthelon and Gineyt's (2014) study and with the study by Weiler and colleagues (2000). Notably, Berthelon and Gineyt (2014) was flagged for subjecting lane excursion data to nonparametric tests, and Weiler et al. (2000) was flagged for transforming lane excursion data. It is possible that the standardized mean differences calculated with these data are not reliable. None of the other included studies were flagged for using non-parametric tests or transformed data. Thus, the analyses were re-run without these two studies to test sensitivity to their inclusion.

Re-analysis. When these studies were removed from the analysis, the effect sizes decreased with a pre-post correlation of zero (Hedge's $g = 0.387$; 95% CI = 0.269, 0.506; Figure C51), 0.5 (Hedge's $g = 0.383$; 95% CI = 0.278, 0.489; Figure C52) and 0.9 (Hedge's $g = 0.278$; 95% CI = 0.217, 0.339; Figure C53). Results, including a conversion to r effect size and prediction intervals, appear in Table 23, below.

Table 23. Re-analysis of the effect of alcohol on lane excursions (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0*	0.209	0.387	0.269	0.506	0.233	0.542
0.5*	0.201	0.383	0.278	0.489	0.103	0.663
0.9*	0.144	0.278	0.217	0.339	0.073	0.482

However, a funnel plot asymmetry was still evident in all three cases (see Figures E25 to E27), and Egger's regression test remained statistically significant with a pre-post correlation of zero [$t(21) = 3.932$, $p < .001$, two-tailed test], 0.5 [$t(21) = 3.921$, $p < .001$, two-tailed test] and 0.9 [$t(21) = 2.936$, $p = 0.008$, two-tailed test]. As with crashes and lateral position variability, a post-hoc test was conducted wherein average BAC level was added as a second predictor to Egger's

regression test. Of the original 23 effect sizes (following the removal of Berthelon et al. [2014] and Weiler et al. [2000]), 19 effect sizes were retained. The results of this post-hoc test are reported in Table 24, below.

Table 24. The relationship between Hedge's g and SE, with and without BAC.

Pre-Post r	k	Without BAC (Step 1)	With BAC (Step 2)
0.0	19	$t = 4.279, p = .001$	$t = 2.513, p = .023$
0.5	19	$t = 4.387, p < .001$	$t = 2.878, p = .011$
0.9	19	$t = 3.174, p = .006$	$t = 2.500, p = .024$

Again, the relationship between Hedge's g and its standard error persisted even after the inclusion of average BAC as a second predictor in the weighted least squares regression. Small study effects are apparent, but it is unclear whether this is due to publication bias.

*****Time Out of Lane.** This meta-analysis included three effect sizes representing 111 participants. With a pre-post correlation of zero, alcohol was associated with an increase in time out of lane (Hedge's $g = 0.694$; 95% CI = 0.232, 1.155; Figure C54). Results were similar with a pre-post correlation of 0.5 (Hedge's $g = 0.648$, 95% CI = 0.140, 1.156; Figure C55) and a pre-post correlation of 0.9 (Hedge's $g = 0.621$; 95% CI = 0.048, 1.194; Figure C56). Results, including a conversion to r effect size and prediction intervals, appear in Table 25, below.

Table 25. Effect of alcohol on time out of lane (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0*	0.333	0.694	0.232	1.155	-3.715	5.102
0.5*	0.307	0.648	0.140	1.156	-4.849	6.146
0.9*	0.291	0.621	0.048	1.194	-6.089	7.331

On average, alcohol is associated with a moderate increase in time out of lane. However, the 95% confidence intervals indicate imprecision. Due in part to imprecision, and due in part to the influence of unknown moderating factors, the prediction intervals are wide. The true effect of alcohol on time out of lane lies somewhere between a very large decrease in time out of lane to a very large increase in time out of lane. Thus, although the average effect of alcohol is moderate increase in time out of lane, the effect of alcohol is not always consistent. Due to the small number of studies included in the meta-analysis, there is not enough data for meaningful exploration of moderating factors, or additionally, for small study effects.

****Speed*. This meta-analysis included 43 effect sizes representing approximately 1,226 participants. Though most studies reported this measure in the form of a mean representing driving speed, two studies reported measures representing the mean difference between the participant's driving speed and the posted speed limit (Arnedt et al., 2001; Howland et al., 2011). Pre-post correlations were recovered for Bernosky-Smith et al. (2012) and from raw data associated with McCartney et al. (2017) (Danielle McCartney, personal communication dated October 10, 2019) and Helland et al. (2016) (Arne Helland, personal communication dated March 9, 2020).

With a pre-post correlation of zero, alcohol was associated with an increase in speed (Hedge's $g = 0.164$; 95% CI = 0.086, 0.241; Figure C57). Results were similar with a pre-post correlation of 0.5 (Hedge's $g = 0.143$; 95% CI = 0.072, 0.214; Figure C58) and a pre-post correlation of 0.9 (Hedge's $g = 0.126$; 95% CI = 0.042, 0.188; Figure C59). Results, including a conversion to r effect size and prediction intervals, appear in Table 26, below.

Table 26. Effect of alcohol on speed (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0*	0.085	0.164	0.086	0.241	0.084	0.244
0.5*	0.075	0.143	0.072	0.214	-0.081	0.367
0.9*	0.066	0.126	0.064	0.188	-0.207	0.458

On average, alcohol is associated with only a trivial increase in speed relative to baseline.

Although the 95% confidence intervals are generally narrow, the prediction intervals are wide.

Thus, unknown moderating factors appear to influence the relationship between alcohol and speed. The potential influence of BAC on effect size magnitude is explored in *Subgroup Analysis*, below.

Small study effects. There was no obvious asymmetry in the funnel plot and no statistically significant Egger's regression with a pre-post correlation of zero [$t(41) = 0.070$, $p = 0.944$, two-tailed test; Figure E28], The funnel plot was asymmetrical with pre-post correlations of 0.5 and 0.9 (see Figures E29 and E30), but Egger's regression remained non-statistically significant in both cases [$t(41) = 0.952$, $p = 0.347$, two-tailed test, pre-post correlation = 0.5; $t(41) = 1.467$, $p = 0.150$, two-tailed test, pre-post correlation = 0.9]. Thus, there is no compelling evidence of small study effects in this meta-analysis.

*****Speed Variability.** This meta-analysis included 32 effect sizes representing approximately 806 participants. Pre-post correlations were recovered from raw data associated with McCartney et al. (2017) (Danielle McCartney, personal communication dated October 10, 2019) and Helland et al. (2016) (Arne Helland, personal communication dated March 9, 2020).

With a pre-post correlation of zero, alcohol was associated with an increase in speed variability (Hedge's $g = 0.266$; 95% CI = 0.170, 0.362; Figure C60). Results were similar with a

pre-post correlation of 0.5 (Hedge's $g = 0.264$; 95% CI = 0.184, 0.344; Figure C61) and a pre-post correlation of 0.9 (Hedge's $g = 0.233$; 95% CI = 0.163, 0.302; Figure C62). Results, including a conversion to r effect size and prediction intervals, appear in Table 27, below.

Table 27. Effect of alcohol on speed variability (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0*	0.138	0.266	0.170	0.362	0.166	0.366
0.5*	0.138	0.264	0.184	0.344	0.074	0.454
0.9*	0.120	0.233	0.163	0.302	-0.108	0.573

On average, alcohol is associated with a small increase in speed variability. According to the prediction intervals, the effects are generally consistent; it is only in the case of a pre-post correlation of 0.9 that the 95% prediction interval includes a negative effect (albeit of trivial magnitude). The prediction intervals are not much wider than the confidence intervals, which indicates that the influence of moderating factors is minimal. Still, the potential influence of BAC on effect size magnitude is investigated in *Subgroup Analyses*, later. Next, small study effects were explored.

Small study effects. There did not appear to be any obvious asymmetry in the funnel plots with pre-post correlations of zero or 0.5; however, the plot was ambiguous with a pre-post correlation of 0.9 (see Figures E31 to E33). However, Egger's regression was not statistically significant with a pre-post correlation of zero [$t(30) = 1.054$, $p = 0.300$ (two-tailed test)], a pre-post correlation of 0.5 [$t(30) = 1.634$, $p = 0.113$ (two-tailed test)] or a pre-post correlation of 0.9 [$t(30) = 1.613$, $p = 0.117$ (two-tailed test)]. Thus, there is no compelling evidence of publication bias in this analysis.

***Speed Exceedances.** This meta-analysis included four effect sizes representing 128 participants. With a pre-post correlation of zero, alcohol was not associated with a reliable change in speed exceedances relative to baseline (Hedge's $g = 0.194$; 95% CI = -0.258, 0.645; Figure C63). Results were similar with a pre-post correlation of 0.5 (Hedge's $g = 0.271$; 95% CI = -0.193, 0.735; Figure C64), but they became statistically significant with a pre-post correlation of 0.9 (Hedge's $g = 0.516$; 95% CI = 0.093, 0.938; Figure C65). Results, including a conversion to r effect size and prediction intervals, appear in Table 28, below.

Table 28. Effect of alcohol on speed exceedances (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	0.282	0.194	-0.258	0.645	-1.473	1.860
0.5	0.309	0.271	-0.193	0.735	-3.555	4.097
0.9*	0.292	0.516	0.093	0.938	-1.432	2.464

The pattern of results indicates and absence of evidence that alcohol reliably changes rates of speed exceedances relative to baseline. An average increase, of moderate magnitude, is only achieved with a pre-post correlation of 0.9, which may be optimistically high. The 95% confidence intervals indicate major imprecision. Consequently, the prediction intervals are also wide and vary from a very large decrease in speed exceedances to a very large increase in speed exceedances with alcohol. There are not enough studies included in the meta-analysis for meaningful exploration of small study effects and potential moderators.

*****Time Speeding.** This meta-analysis includes five effect sizes representing 161 participants. A pre-post correlation was retrieved from Bernosky-Smith et al. (2012). With a pre-post correlation of zero, alcohol was associated with an increase in time speeding (Hedge's $g = 0.512$; 95% CI = 0.042, 0.982; Figure C66). Results were similar with a pre-post correlation of

0.5 (Hedge's $g = 0.496$; 95% CI = 0.054, 0.938; Figure C67) and a pre-post correlation of 0.9 (Hedge's $g = 0.388$; 95% CI = 0.054, 0.721; Figure C68). Results, including a conversion to r effect size and prediction intervals, appear in Table 29, below.

Table 29. Effect of alcohol on time speeding (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0*	0.255	0.512	0.042	0.982	-1.023	2.047
0.5*	0.249	0.496	0.054	0.938	-0.965	1.958
0.9*	0.203	0.388	0.054	0.721	-0.669	1.445

On average, alcohol is associated with a small to moderate increase in time speeding.

However, the 95% confidence intervals indicate a lack of precision. Partly due to this, and partly due to the influence of unknown moderating factors, the prediction intervals are wide.

Specifically, the true effect of alcohol lies somewhere between a large decrease in time speeding to a very large increase in speeding. Thus, although the prevailing effect is on average a small increase in speeding, the effect is inconsistent. Unfortunately, the small number of included studies precludes the exploration of potential moderating factors, as well as the exploration of small study effects.

Summary of the effects of alcohol. The meta-analyses reported here indicate a clearly detrimental effect of alcohol on driving performance and changes in driver behaviour. Alcohol was consistently associated with statistically significant average increases in crashes, hazard RT, lateral position variability, lane excursions, time out of lane, speed, speed variability and time speeding. Significant effects were small to moderate in magnitude.

Although alcohol tended to reliably influence the measures studied here, measurement imprecision was a common theme. Additionally, many of the measures were associated with

wide prediction intervals, indicating that the influence of alcohol is not necessarily consistent from circumstance to circumstance. In part, this may be due to wide confidence intervals, as well as the influence of BAC level (which is investigated in the *Subgroup Analysis*, below). Finally, small study effects were evident in some measures, but it is unclear whether this is due to publication bias (in which case, effect sizes are overestimated) or a legitimate relationship between effect size and standard error.

Cannabis v. Alcohol. Meta-analyses were conducted for crashes, hazard RT, lateral position variability, lane excursions, time out of lane, speed, speed variability and speed exceedances. As with *Cannabis v. Baseline* comparisons (above), there is a limited amount of data available for analyses. There was not enough data available to meta-analyze headway, headway variability or time speeding.

Crashes. This meta-analysis includes one effect size representing 80 participants. With a pre-post correlation of zero, there was no reliable difference between cannabis and alcohol for crashes (Hedge's $g = -0.020$; 95% CI = -0.327, 0.287; Figure C69). Similar results were obtained with a pre-post correlation of 0.5 (Hedge's $g = -0.020$; 95% CI = -0.237, 0.197; Figure C70) and a pre-post correlation of 0.9 (Hedge's $g = -0.021$; 95% CI = -0.119, 0.076; Figure C71). Results, including a conversion to r effect size and prediction intervals, appear in Table 30, below.

Table 30. Effect of cannabis on crashes (compared to alcohol).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	-0.010	-0.020	-0.327	0.287	N/A	N/A
0.5	-0.010	-0.020	-0.237	0.197	N/A	N/A
0.9	-0.011	-0.021	-0.119	0.076	N/A	N/A

The results indicate that cannabis and alcohol may not differ in terms of simulated crash rates. However, this result is based on only a single effect size. The 95% confidence intervals indicate a lack of precision, and there is not enough data to generate prediction intervals. The inclusion of one effect further precludes the meaningful exploration of small study effects and the role of moderating factors.

Hazard RT. This meta-analysis included four effect sizes representing 128 participants. Hazards included on-road obstacles (Liguori et al., 2002), slowing forward vehicles (Ramaekers et al., 2000b [Study 1]; Sexton et al., 2002), other vehicles (Sexton et al., 2002) and general “emergencies” (Downey et al., 2013). With a pre-post correlation of zero, there was no reliable difference between cannabis and alcohol for hazard RT (Hedge’s $g = 0.131$; 95% CI = -0.289, 0.550; Figure C72). Results were similar with a pre-post correlation of 0.5 (Hedge’s $g = 0.148$; 95% CI = -0.243, 0.540; Figure C73) and a pre-post correlation of 0.9. (Hedge’s $g = 0.117$; 95% CI = -0.161, 0.395; Figure C74). Results, including a conversion to r effect size and prediction intervals, appear in Table 31, below.

Table 31. Effect of cannabis on hazard RT (compared to alcohol).

Pre-Post r	r	Hedge’s g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	0.073	0.131	-0.289	0.550	-1.471	1.733
0.5	0.079	0.148	-0.243	0.540	-1.526	1.823
0.9	0.061	0.117	-0.161	0.395	-1.181	1.415

Overall, cannabis and alcohol do not appear to differ in terms of hazard RT. However, the 95% confidence intervals indicate a lack of precision. Due in part to this, as well as the influence of unknown moderating factors, the 95% prediction intervals are wide. According to these intervals, the true effect lies somewhere between cannabis having a very large decrease in hazard

RT (i.e., faster reaction time) relative to alcohol, to cannabis having a very large increase in hazard RT (i.e., slower reaction time) relative to alcohol. However, there is not enough data for meaningful exploration of potential moderating factors, or additionally, of small study effects.

***Lateral Position Variability.** This meta-analysis included five effect sizes representing approximately 81 participants. With a pre-post correlation of zero, cannabis was not associated with a reliable change in lateral position variability compared to alcohol (Hedge's $g = 0.170$; 95% CI = -0.127, 0.467; Figure C75). Results were similar with a pre-post correlation of 0.5 (Hedge's $g = 0.166$; 95% CI = -0.044, 0.376; Figure C76), but they became statistically significant with a pre-post correlation of 0.9 (Hedge's $g = 0.146$; 95% CI = 0.041, 0.251; Figure C77). Results, including a conversion to r effect size and prediction intervals, appear in Table 32, below.

Table 32. Effect of cannabis on lateral position variability (compared to alcohol).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	0.089	0.170	-0.127	0.467	-0.312	0.653
0.5	0.087	0.166	-0.044	0.376	-0.175	0.507
0.9*	0.077	0.146	0.041	0.251	-0.093	0.385

Overall, the pattern of results suggests that cannabis and alcohol do not differ in terms of lateral position variability. Although a statistically significant effect was observed with a pre-post correlation of 0.9, it is unknown whether the actual pre-post correlation had this magnitude in reality, and even if it did, the increase associated with cannabis is trivial in magnitude. Finally, the 95% prediction intervals, which reflect both sampling variability and the influence of unknown moderators, indicate that the true effect ranges from a trivial to small decrease in lateral position variability, to a small to moderate increase in lateral position variability, with cannabis

(depending on the pre-post correlation). However, there are not enough studies included in the meta-analysis for meaningful exploration of potential moderators. Additionally, there are not enough studies included in the meta-analysis for meaningful exploration of small study effects.

Lane Excursions. This meta-analysis includes two effect sizes representing 98 participants. With a pre-post correlation of zero, there was no reliable difference between cannabis and alcohol for lane excursions (Hedge's $g = 0.054$; 95% CI = -0.224, 0.331; Figure C78). Results were similar with a pre-post correlation of 0.5 (Hedge's $g = 0.052$; 95% CI = -0.145, 0.248; Figure C79) and a pre-post correlation of 0.9 (Hedge's $g = 0.002$; 95% CI = -0.184, 0.188; Figure C80). Results, including a conversion to r effect size and prediction intervals, appear in Table 33, below.

Table 33. Effect of cannabis on lane excursions (compared to alcohol).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	0.028	0.054	-0.224	0.331	N/A	N/A
0.5	0.027	0.052	-0.145	0.248	N/A	N/A
0.9	0.001	0.002	-0.184	0.188	N/A	N/A

Overall, the results suggest that cannabis and alcohol do not differ in terms of their influence on lane excursions. Once again, there are not enough studies included in the meta-analysis to generate prediction intervals or to meaningfully explore small study effects or potential moderating factors.

Time Out of Lane. This meta-analysis included one effect size representing 18 participants. With a pre-post correlation of zero, there was no reliable change between cannabis and alcohol for time out of lane (Hedge's $g = 0.005$; 95% CI = -0.627, 0.637; Figure C81). Results were similar with a pre-post correlation of 0.5 (Hedge's $g = 0.003$; 95% CI = -0.444,

0.449; Figure C82) and a pre-post correlation of 0.9 (Hedge's $g = -0.006$; 95% CI = -0.204, 0.193; Figure C83). Results, including a conversion to r effect size and prediction intervals, appear in Table 34, below.

Table 34. Effect of cannabis on time out of lane (compared to alcohol).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	0.003	0.005	-0.627	0.637	N/A	N/A
0.5	0.001	0.003	-0.444	0.449	N/A	N/A
0.9	-0.003	-0.006	-0.204	0.193	N/A	N/A

Overall, cannabis and alcohol do not appear to differ in terms of their effects on time out of lane. However, results are limited by the inclusion of only one effect size in the meta-analysis. The 95% confidence intervals indicate a lack of precision, and there is not enough data to generate prediction intervals, or to meaningfully explore small study effects and potential moderators.

*****Speed.** This meta-analysis included four effect sizes representing 125 participants. With a pre-post correlation of zero, cannabis was associated with a decrease in speed compared to alcohol (Hedge's $g = -0.314$; 95% CI = -0.613, -0.015; Figure C84). Results were similar with a pre-post correlation of 0.5 (Hedge's $g = -0.392$; 95% CI = -0.710, -0.074; Figure C85) and 0.9. (Hedge's $g = -0.371$; -0.633, -0.108; Figure C86). Results, including a conversion to r effect size and prediction intervals, appear in Table 35, below.

Table 35. Effect of cannabis on speed (compared to alcohol).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0*	-0.172	-0.314	-0.613	-0.015	-1.167	0.539
0.5*	-0.205	-0.392	-0.710	-0.074	-1.627	0.842
0.9*	-0.192	-0.371	-0.633	-0.108	-1.581	0.839

On average, cannabis decreases driving speed to a small degree relative to alcohol.

However, the 95% confidence intervals indicate a lack of precision, which is reflected – in addition to the influence of unknown moderating factors – in the 95% prediction intervals.

According to these intervals, the true effect lies somewhere between cannabis being associated with a very large decrease in speed relative to alcohol, to cannabis being associated with a moderate to large *increase* in speed relative to alcohol. Thus, although driving is, on average, slower with cannabis, this is not always the case. Unfortunately, there are not enough studies included in the meta-analysis for a meaningful exploration of factors that contribute to the variability in effects observed in the prediction intervals. The small number of included studies also precludes the meaningful exploration of small study effects.

Speed Variability. This meta-analysis included two effect sizes representing 26 participants. Overall, there was no reliable change between cannabis and alcohol for speed variability (Hedge's $g = 0.134$; 95% CI = -0.383, 0.652; Figure C87). Results were similar with a pre-post correlation of 0.5 (Hedge's $g = 0.131$; 95% CI = -0.239, 0.501; Figure C88). However, with a pre-post correlation of 0.9 (Hedge's $g = 0.116$; 95% CI = -0.188, 0.421; Figure C89), the results became statistically significant. Results, including a conversion to r effect size and prediction intervals, appear in Table 36, below.

Table 36. Effect of cannabis on speed variability (compared to alcohol).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	0.072	0.134	-0.383	0.652	N/A	N/A
0.5	0.070	0.131	-0.239	0.501	N/A	N/A
0.9	0.062	0.116	-0.188	0.421	N/A	N/A

The pattern of results suggest that cannabis and alcohol do not differ in terms of speed variability. A reliable difference of trivial magnitude is only observed with a pre-post correlation of 0.9, which may be optimistically high. The 95% confidence intervals indicate a lack of precision, and there is not enough data to compute prediction intervals. The small number of included studies also precludes meaningful exploration of small study effects and potential moderators.

****Speed Exceedances.** This meta-analysis included one effect size representing 80 participants. Overall, cannabis was not associated with a reliable change in speed exceedances compared to alcohol (Hedge's $g = -0.235$; 95% CI = -0.546, 0.077; Figure C90). However, results became statistically significant with a pre-post correlation of 0.5 (Hedge's $g = -0.231$; 95% CI = -0.451, -0.011; Figure C91) and a pre-post correlation of 0.9 (Hedge's $g = -0.205$, 95% CI = -0.303, -0.107; Figure C92). Results, including a conversion to r effect size and prediction intervals, appear in Table 37, below.

Table 37. Effect of cannabis on speed exceedances (compared to alcohol).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	-0.118	-0.235	-0.546	0.077	N/A	N/A
0.5*	-0.116	-0.231	-0.451	-0.011	N/A	N/A
0.9*	-0.103	-0.205	-0.303	-0.107	N/A	N/A

The pattern of results suggest that cannabis may be associated, on average, with fewer speed exceedances compared to alcohol. The average decrease is small in magnitude. However, this rests on the assumption that there is at least a small correlation between pairs of measurements in the included studies utilizing repeated-measures designs. However, the 95% confidence intervals indicate a lack of precision, and there is not enough data to generate prediction intervals. Furthermore, there are not enough included studies for a meaningful exploration of small study effects and potential moderators.

Summary of the effect of cannabis compared to alcohol. The number of studies that directly compare cannabis to alcohol on measures of driving performance and behaviour is small. As with studies focused on cannabis, many of the effects reported here are imprecise. More data is needed to improve precision and allow for the exploration of moderating factors.

For most measures, there were no statistically significant differences between cannabis and alcohol. However, cannabis was consistently associated with a statistically significant small average decrease in speed compared to alcohol. Cannabis was also generally associated with a statistically significant small average decrease in speed exceedances compared to alcohol.

Combination v. Baseline. Meta-analyses were conducted for crashes, hazard RT, lateral position variability, lane excursions, speed, speed variability, speed exceedances and time out of lane. There was not enough data to meta-analyze headway, headway variability or time speeding.

****Crashes.** This meta-analysis includes one effect size representing 80 participants. With a pre-post correlation of zero, the combination of cannabis and alcohol was not associated with a reliable change in crashes compared to baseline (Hedge's $g = 0.226$; 95% CI = -0.088, 0.540; Figure C93). Results became statistically significant and indicated a small increase in crashes with a pre-post correlation of 0.5 (Hedge's $g = 0.223$; 95% CI = 0.000, 0.445; Figure C94) and

0.9 (Hedge's $g = 0.201$; 95% CI = 0.102, 0.300; Figure C95). Results, including a conversion to r effect size and prediction intervals, appear in Table 38, below.

Table 38. Effect of cannabis combined with alcohol on crashes (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	0.113	0.226	-0.088	0.540	N/A	N/A
0.5*	0.111	0.223	0.000	0.445	N/A	N/A
0.9*	0.101	0.201	0.102	0.300	N/A	N/A

Overall, the pattern of results suggest that the combination of cannabis and alcohol may be associated, on average, with a small increase crashes relative to baseline. However, this conclusion assumes that there is at least a small correlation between pairs of measurements in the included study. The results are limited by the inclusion of only one effect size in the meta-analysis, which precludes the generation of prediction intervals and the meaningful exploration of small study effects and potential moderators. The 95% confidence intervals indicate issues with measurement precision. More research should be conducted to verify that the combination of cannabis and alcohol increases crashes relative to baseline.

*****Hazard RT.** This meta-analysis included four effect sizes representing 129 participants. Hazards included slowing forward vehicles (Ramaekers et al., 2000b [Study 1]; Sexton et al., 2002), on-road obstacles (Liguori et al., 2002), approaching vehicles (Sexton et al., 2002) and general “emergencies” (Downey et al., 2013). With a pre-post correlation of zero, the combination of cannabis and alcohol was associated, on average, with a small increase (i.e., slowing) in hazard RT relative to baseline (Hedge's $g = 0.275$; 95% CI = 0.028, 0.523; Figure C96). Results were similar with a pre-post correlation of 0.5 (Hedge's $g = 0.352$; 95% CI = 0.074, 0.630; Figure C97) and a pre-post correlation of 0.9 (Hedge's $g = 0.382$; 95% CI = 0.131,

0.632; Figure C98). Results, including a conversion to r effect size and prediction intervals, appear in Table 39, below.

Table 39. Effect of cannabis combined with alcohol on Hazard RT (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0*	0.147	0.275	0.028	0.523	-0.268	0.819
0.5*	0.186	0.352	0.074	0.630	-0.668	1.373
0.9*	0.196	0.382	0.131	0.632	-0.769	1.532

Overall, the pattern of results indicate that the combination of cannabis and alcohol increases hazard RT relative to baseline. On average, the effect is small in magnitude, and the 95% confidence intervals indicate limited precision. The prediction intervals, due in part to a lack of measurement prediction but also due to the influence of unknown moderating factors, indicate that the true effect lies somewhere between a small to large decrease in hazard RT, to a large increase in hazard RT (depending on the pre-post correlation used). Unfortunately, due to the low number of included studies, small study effects and potential moderating factors cannot be explored in a meaningful way. In sum, the prevailing effect of the combination of cannabis and alcohol is a minor slowing of hazard RT, but the effect is not consistent from circumstance to circumstance.

*****Lateral Position Variability.** This meta-analysis includes four effect sizes representing 68 participants. With a pre-post correlation of zero, the combination of cannabis and alcohol was associated, on average, with an increase in lateral position variability (Hedge's $g = 0.502$; 95% CI = 0.080, 0.925; Figure C99). Results were similar with a pre-post correlation of 0.5 (Hedge's $g = 0.531$; 95% CI = 0.107, 0.954; Figure C100) and a pre-post correlation of 0.9

(Hedge's $g = 0.531$; 95% CI = 0.116, 0.945; Figure C101). Results, including a conversion to r effect size and prediction intervals, appear in Table 40, below.

Table 40. Effect of cannabis combined with alcohol on lateral position variability (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0*	0.270	0.502	0.080	0.925	-0.839	1.843
0.5*	0.273	0.531	0.107	0.954	-1.213	2.274
0.9*	0.263	0.531	0.116	0.945	-1.441	2.502

As with lane excursions, the results are consistent across all three meta-analyses.

Regardless of the pre-post correlation used, the combination of cannabis and alcohol, on average, increases lateral position variability relative to baseline. On average, the increase is moderate in magnitude. However, the 95% confidence intervals indicate a lack of precision. As a result of this, and also due to unknown moderating factors, the prediction intervals are wide. According to the prediction intervals, the true effect appears to lie somewhere between a large decrease and a large increase in lateral position variability. Unfortunately, the small number of included studies precludes the meaningful exploration of small study effects and moderating factors that might explain the variance in effects represented in the prediction intervals.

*****Lane Excursions.** This meta-analysis includes two effect sizes representing 98 participants. With a pre-post correlation of zero, the combination of cannabis and alcohol was associated with a small increase in lane excursions relative to baseline (Hedge's $g = 0.297$; 95% CI = 0.014, 0.579; Figure C102). Results were similar with a pre-post correlation of 0.5 (Hedge's $g = 0.284$; 95% CI = 0.084, 0.483; Figure C103) and a pre-post correlation of 0.9 (Hedge's $g =$

0.228; 95% CI = 0.139, 0.316; Figure C104). Results, including a conversion to r effect size and prediction intervals, appear in Table 41, below.

Table 41. Effect of cannabis combined with alcohol on lane excursions (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0*	0.149	0.297	0.014	0.579	N/A	N/A
0.5*	0.143	0.284	0.084	0.483	N/A	N/A
0.9*	0.115	0.228	0.139	0.316	N/A	N/A

Overall, the results indicate that the combination of cannabis and alcohol, on average, increases rates of lane excursions relative to baseline. However, due to the small number of included studies, prediction intervals cannot be computed, and meaningful exploration of small study effects and moderating factors is not feasible.

Speed. This meta-analysis includes three effect sizes representing 112 participants. With a pre-post correlation of zero, the combination of cannabis and alcohol was not associated with a reliable change in speed compared to baseline (Hedge's $g = -0.279$; 95% CI = -0.674, 0.117; Figure C105). The results were similar with a pre-post correlation of 0.5 (Hedge's $g = -0.315$; 95% CI = -0.727, 0.098; Figure C106) and a pre-post correlation of 0.9 (Hedge's $g = -0.311$; 95% CI = -0.709, 0.087; Figure C107). Results, including a conversion to r effect size and prediction intervals, appear in Table 42, below.

Table 42. Effect of cannabis combined with alcohol on speed (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	-0.150	-0.279	-0.674	0.117	-4.068	3.511
0.5	-0.164	-0.315	-0.727	0.098	-4.959	4.330
0.9	-0.159	-0.311	-0.709	0.087	-5.323	4.701

Overall, the pattern of results suggest that the combination of cannabis and alcohol may not change speed relative to baseline. However, the 95% confidence intervals indicate a lack of measurement precision. The 95% prediction intervals are also wide and indicate that the true effect lies somewhere between a very large decrease in speed and a very large increase in speed. There are not enough studies included in the meta-analysis for meaningful exploration of small study effects and potential moderators.

***Speed Variability.** This meta-analysis included one effect size representing 12 participants. With a pre-post correlation of zero, the combination of cannabis and alcohol was not associated with a reliable change in speed variability relative to baseline (Hedge's $g = 0.249$; 95% CI = -0.508, 1.007; Figure C108). Results were similar with a pre-post correlation of 0.5 (Hedge's $g = 0.248$; 95% CI = -0.287, 0.784; Figure C109). However, a small, statistically significant increase in speed variability was observed with a pre-post correlation of 0.9 (Hedge's $g = 0.239$; 95% CI = 0.000, 0.479; Figure C110). Results, including a conversion to r effect size and prediction intervals, appear in Table 43, below.

Table 43. Effect of cannabis combined with alcohol on speed variability (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	0.133	0.249	-0.508	1.007	N/A	N/A
0.5	0.132	0.248	-0.287	0.784	N/A	N/A
0.9*	0.128	0.239	0.000	0.479	N/A	N/A

Overall, the evidence for a change in speed variability between the combination of drugs and baseline is lacking. The 95% confidence intervals indicate a lack of precision. The results are limited by the inclusion of only one study, which precludes the generation of prediction intervals and does not allow for a meaningful exploration of small study effects and potential moderators.

Speed Exceedances. One study was eligible and included in the meta-analysis. The effect size represents 80 participants. With a pre-post correlation of zero, the combination of cannabis and alcohol was not associated with a reliable change in speed exceedances compared to baseline (Hedge's $g = 0.010$; 95% CI = -0.297, 0.317; Figure C111). Results were similar with a pre-post correlation of 0.5 (Hedge's $g = 0.010$; 95% CI = -0.207, 0.227; Figure 112) and a pre-post correlation of 0.9 (Hedge's $g = 0.009$; 95% CI = -0.088, 0.107; Figure 113). Results, including a conversion to r effect size and prediction intervals, appear in Table 44, below.

Table 44. Effect of cannabis combined with alcohol on speed exceedances (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	0.005	0.010	-0.297	0.317	N/A	N/A
0.5	0.005	0.010	-0.207	0.227	N/A	N/A
0.9	0.005	0.009	-0.088	0.107	N/A	N/A

As with speed, the pattern of results suggest that the combination of cannabis and alcohol does not change rates of speed exceedances relative to baseline. However, the 95% confidence intervals indicate a lack of precision. The results are limited by the inclusion of only a single study in the meta-analysis, which precludes the generation of prediction intervals as well as the meaningful exploration of small study effects and potential moderating factors.

*****Time Out of Lane.** This meta-analysis included one effect size representing 18 participants. With a pre-post correlation of zero, the combination of cannabis and alcohol was associated with, on average, a small increase in time out of lane relative to alcohol (Hedge's $g = 0.715$; 95% CI = 0.005, 1.426; Figure C114). The results were similar with a pre-post correlation of 0.5 (Hedge's $g = 0.673$; 95% CI = 0.178, 1.168; Figure C115) and a pre-post correlation of 0.9

(Hedge's $g = 0.496$; 95% CI = 0.285, 0.706; Figure C116). Results, including a conversion to r effect size and prediction intervals, appear in Table 45, below.

Table 45. Effect of cannabis combined with alcohol on time out of lane (compared to baseline).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0*	0.350	0.715	0.005	1.426	N/A	N/A
0.5*	0.331	0.673	0.178	1.168	N/A	N/A
0.9*	0.251	0.496	0.285	0.706	N/A	N/A

The results indicate that on average, the combination of cannabis and alcohol increases time out of lane relative to baseline. However, the 95% confidence intervals indicate a lack of precision, and there is not enough data to generate prediction intervals or to meaningfully explore small study effects and potential moderators.

Summary of the effects of the combination of drugs on driving. Only a small number of studies compare the combination of cannabis and alcohol to baseline. Based on a small sample of studies, the combination of cannabis and alcohol is associated with average increases in hazard RT, lateral position variability, lane excursions and time out of lane, relative to baseline. However, more data would be beneficial.

Combination v. Alcohol. Meta-analyses were conducted for crashes, hazard RT, lateral position variability, lane excursions, time out of lane, speed, speed variability, speed exceedances. There was not enough data to meta-analyze headway, headway variability or time speeding.

Crashes. Only one study reported the statistical data necessary for effect size computation. The resulting meta-analysis includes one effect size representing 80 participants. With a pre-post correlation of 0, the combination of cannabis and alcohol was not associated

with a reliable change in crash rates compared to alcohol alone (Hedge's $g = 0.066$; 95% CI = -0.243, 0.376; Figure C117). A pre-post correlation of 0.5 yielded similar effects (Hedge's $g = 0.067$; 95% CI = -0.152, 0.286; Figure C118), as did a pre-post correlation of 0.09 (Hedge's $g = 0.067$; 95% CI = -0.031, 0.164; Figure C119). Results, including a conversion to r effect size and prediction intervals, appear in Table 46, below.

Table 46. Effect of cannabis combined with alcohol on crashes (compared to alcohol).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	0.033	0.066	-0.243	0.376	N/A	N/A
0.5	0.034	0.067	-0.152	0.286	N/A	N/A
0.9	0.034	0.067	-0.031	0.164	N/A	N/A

Overall, the pattern of results suggest that the combination of cannabis and alcohol does not change rates of crashes reliably compared to alcohol alone. However, the confidence intervals, which include zero in all three analyses, indicate a lack of measurement precision, and the results are limited by the inclusion of only a single study in the meta-analysis. There is not enough data to compute prediction intervals to estimate the range of plausible effects, and tests for small study effects and potential moderating factors can not be conducted.

Hazard RT. This meta-analysis includes four effect sizes representing 128 participants. Hazards included slowing forward vehicles (Ramaekers et al., 2000b [Study 1]; Sexton et al., 2002), approaching vehicles (Sexton et al., 2002), on-road obstacles (Liguori et al., 2002) and general “emergencies” (Downey et al., 2013).

Using a pre-post correlation of zero, the combination of cannabis and alcohol was not associated with a reliable change in hazard RT compared to alcohol alone (Hedge's $g = 0.344$; 95% CI = -0.127, 0.814; Figure C120). The results were similar with a pre-post correlation of 0.5

(Hedge's $g = 0.360$; 95% CI = -0.087, 0.808; Figure C121) and a pre-post correlation of 0.9 (Hedge's $g = 0.287$; 95% CI = -0.044, 0.619; Figure C122). Results, including a conversion to r effect size and prediction intervals, appear in Table 47, below.

Table 47. Effect of cannabis combined with alcohol on hazard RT (compared to alcohol).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	0.184	0.344	-0.127	0.814	-1.520	2.208
0.5	0.186	0.360	-0.087	0.808	-1.598	2.319
0.9	0.148	0.287	-0.044	0.619	-1.281	1.856

As with crashes, the results suggest that hazard RT associated the combination of cannabis and alcohol is not different than hazard RT associated with alcohol alone. On average, the effect is small, but the 95% confidence intervals, which include zero, indicate a lack of precision. Consequently, the prediction intervals are wide and indicate that the true effect of the combination of cannabis and alcohol lies somewhere between a large decrease and a large increase in hazard RT relative to alcohol alone. The small number of studies included in the meta-analysis precludes meaningful exploration of small study effects, as well as the influence of potential moderating factors such as alcohol dose or study setting that might influence the magnitude of the effect.

*****Lateral Position Variability.** This meta-analysis includes four effect sizes representing 67 participants. With a pre-post correlation of zero, the combination of cannabis and alcohol was associated, on average, with a small increase in lateral position variability compared to alcohol alone (Hedge's $g = 0.457$; 95% CI = 0.068, 0.847; Figure C123). The results were similar with a pre-post correlation of 0.5 (Hedge's $g = 0.480$; 95% CI = 0.096, 0.865; Figure C124) and a pre-post correlation of 0.9 (Hedge's $g = 0.462$; 95% CI = 0.124, 0.799; Figure

C125). Results, including a conversion to r effect size and prediction intervals, appear in Table 48, below.

Table 48. Effect of cannabis combined with alcohol on lateral position variability (compared to alcohol).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0*	0.242	0.457	0.068	0.847	-0.656	1.571
0.5*	0.247	0.480	0.096	0.865	-1.045	2.006
0.9*	0.234	0.462	0.124	0.799	-1.119	2.043

Overall, the results indicate that the combination of cannabis and alcohol increases lateral position variability relative to alcohol alone. On average, the increase is small; however, the 95% prediction intervals indicate that the true effect may range from a moderate to large decrease in lateral position variability (depending on the pre-post correlation used) to a large increase in lateral position variability. The wide prediction intervals represent both a lack of measurement precision (as indicated in the 95% confidence intervals) as well as the presence of moderating factors. However, with so few studies, small study effects and the potential influence of moderating factors cannot be explored. Thus, although the average effect of the combination of cannabis and alcohol is an increase in lateral position variability relative to alcohol alone, the effect is not consistent from circumstance to circumstance.

***Lane Excursions.** This meta-analysis includes two effect sizes representing 98 participants. With a pre-post correlation of zero, the combination of cannabis and alcohol was not associated with a reliable change in lane excursions compared to alcohol alone (Hedge's $g = 0.147$; 95% CI = -0.133, 0.427; Figure C126). Results were similar with a pre-post correlation of 0.5 (Hedge's $g = 0.138$; 95% CI = -0.060, 0.335; Figure C127). However, the summary statistic

became statistically significant with a pre-post correlation of 0.9 (Hedge's $g = 0.099$; 95% CI = 0.012, 0.187; Figure C128). Results, including a conversion to r effect size and prediction intervals, appear in Table 49, below.

Table 49. Effect of cannabis combined with alcohol on lane excursions (compared to alcohol).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	0.075	0.147	-0.133	0.427	N/A	N/A
0.5	0.070	0.138	-0.060	0.335	N/A	N/A
0.9*	0.051	0.099	0.012	0.187	N/A	N/A

As with other measures discussed thus far, the confidence intervals indicate a lack of precision. In all cases except for where a correlation of 0.9 was used, the confidence intervals include zero. With a pre-post correlation of 0.9, the combination of cannabis and alcohol is associated with an increase in lane excursions relative to alcohol alone, but only to a very small degree. The small number of studies included in the meta-analysis precludes the generation of prediction intervals and the meaningful exploration of small study effects and potential moderating factors.

****Time Out of Lane.** This meta-analysis includes one effect size representing 18 participants. With a pre-post correlation of zero, the combination of cannabis and alcohol was not associated with a reliable change in time out of lane compared to alcohol alone (Hedge's $g = 0.577$; 95% CI = -0.108, 1.261; Figure C129). The results became statistically significant with a pre-post correlation of 0.5 (Hedge's $g = 0.525$; 95% CI = 0.049, 1.002; Figure C130) and a pre-post correlation of 0.9 (Hedge's $g = 0.354$; 95% CI = 0.150, 0.559; Figure C131). Results, including a conversion to r effect size and prediction intervals, appear in Table 50, below.

Table 50. Effect of cannabis combined with alcohol on time out of lane (compared to alcohol).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	0.288	0.577	-0.108	1.261	N/A	N/A
0.5*	0.264	0.525	0.049	1.002	N/A	N/A
0.9*	0.182	0.354	0.150	0.559	N/A	N/A

Overall, the pattern of results suggest that the combination of cannabis and alcohol may be associated, on average, with a small to moderate increase in time out of lane. However, this conclusion rests on the assumption that there is at least a small correlation between pairs of measurements in the included study. However, the analysis is limited by the inclusion of only a single study and the inability to generate prediction intervals or meaningfully explore small study effects and potential moderators.

***Speed.** This meta-analysis includes three effect sizes representing 111 participants. With a pre-post correlation of zero, the combination of cannabis and alcohol was not associated with a reliable change in speed compared to alcohol alone (Hedge's $g = -0.239$; 95% CI = -0.513, 0.036; Figure C132). The results were similar with a pre-post correlation of 0.5 (Hedge's $g = -0.318$; 95% CI = -0.641, 0.006; Figure C133). The results became statistically significant and indicated a small decrease in speed with a pre-post correlation of 0.9 (Hedge's $g = -0.322$; 95% CI = -0.613, -0.031; Figure C134). Results, including a conversion to r effect size and prediction intervals, appear in Table 51, below.

Table 51. Effect of cannabis combined with alcohol on speed (compared to alcohol).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	-0.130	-0.239	-0.513	0.036	-2.137	1.659
0.5	-0.166	-0.318	-0.641	0.006	-3.655	3.020
0.9*	-0.166	-0.322	-0.613	-0.031	-3.887	3.242

Overall, there is a lack of compelling evidence that the combination of cannabis and alcohol changes speed compared to alcohol alone. However, the 95% confidence intervals indicate a lack of precision. Even in the case where the combination of drugs is associated with a small decrease in speed relative to alcohol alone (i.e., when a pre-post correlation of 0.9 is used), the prediction intervals indicate that the true effect varies from an very large decrease in speed to a very large increase in speed. Given the small number of included studies, small study effects and the potential influence of moderating factors cannot be explored meaningfully.

***Speed Variability.** This meta-analysis includes one effect size representing 12 participants. With a pre-post correlation of zero, the combination of cannabis and alcohol was not associated with a change in speed variability compared to alcohol alone (Hedge's $g = 0.320$; 95% CI = -0.446, 1.086; Figure C135). The results were similar with a pre-post correlation of 0.5 (Hedge's $g = 0.315$; 95% CI = -0.226, 0.856; Figure C136). Results became statistically significant and indicated a small increase in speed variability with a pre-post correlation of 0.9 (Hedge's $g = 0.282$; 95% CI = 0.041, 0.523; Figure C137). Results, including a conversion to r effect size and prediction intervals, appear in Table 52, below.

Table 52. Effect of cannabis combined with alcohol on speed variability (compared to alcohol).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	0.169	0.320	-0.446	1.086	N/A	N/A
0.5	0.167	0.315	-0.226	0.856	N/A	N/A
0.9*	0.150	0.282	0.041	0.523	N/A	N/A

As with speed, there is a lack of compelling evidence that the combination of cannabis and alcohol changes speed variability reliably compared to alcohol alone. The 95% confidence intervals indicate a lack of measurement precision. In the case of the statistically significant increase in speed variability (i.e., when a pre-post correlation of 0.9 is used), the average effect is small. However, the true effect would be best reflected within the 95% prediction interval, but the inclusion of only a single study precludes the generation of prediction intervals (as well as the meaningful exploration of small study effects and potential moderators).

Speed Exceedances. Only one study was eligible and included in the meta-analysis. The single effect size represents 80 participants. With a pre-post correlation of zero, the combination of cannabis and alcohol was not associated with a change in speed exceedances compared to alcohol alone (Hedge's $g = -0.037$; 95% CI = -0.345, 0.270; Figure C138). The results were similar with a pre-post correlation of 0.5 (Hedge's $g = -0.037$; 95% CI = -0.254, 0.181; Figure C139) and 0.9 (Hedge's $g = -0.033$; 95% CI = -0.131, 0.064; Figure C140). Results, including a conversion to r effect size and prediction intervals, appear in Table 53, below.

Table 53. Effect of cannabis combined with alcohol on speed exceedances (compared to alcohol).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	-0.019	-0.037	-0.345	0.270	N/A	N/A
0.5	-0.019	-0.037	-0.254	0.181	N/A	N/A
0.9	-0.017	-0.033	-0.131	0.064	N/A	N/A

Overall, the pattern of results suggests that the combination of cannabis and alcohol does not reliably change speed exceedances relative to alcohol alone. The 95% confidence intervals indicate a lack of measurement precision. Again, prediction intervals cannot be generated, and small study effects and the potential influence of moderating factors cannot be explored meaningfully due to the inclusion of only a single study in the analysis.

Summary of the combination of drugs compared to alcohol. Lateral position variability appears to be the only measure reliably associated with a statistically significant average increased by the combination of cannabis and alcohol, relative to alcohol alone. For other measures, imprecision is a general issue, and statistical significance varies depending on the pre-post correlation utilized in within-subjects studies.

Combination v. Cannabis. Meta-analyses were conducted for crashes, hazard RT, lateral position variability, lane excursions, time out of lane, speed, speed variability and speed exceedances. There was not enough data available to meta-analyze headway, headway variability or time speeding.

Crashes. Of the studies eligible for inclusion in the meta-analysis, only one reported the statistical data needed to compute effect sizes. The resulting meta-analysis included one effect size representing 80 participants. With a pre-post correlation of zero, the combination of

cannabis and crashes was not associated with a reliable change in crashes compared to cannabis alone (Hedge's $g = 0.051$; 95% CI = -0.259, 0.360; Figure C141). A pre-post correlation of 0.5 yielded similar effects (Hedge's $g = 0.053$; 95% CI = -0.166, 0.272; Figure C142), as did a pre-post correlation of 0.9 (Hedge's $g = 0.061$; 95% CI = -0.037; 0.159; Figure C143). Results, including a conversion to r effect size and prediction intervals, appear in Table 54, below.

Table 54. Effect of cannabis combined with alcohol on crashes (compared to cannabis).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	0.026	0.051	-0.259	0.360	N/A	N/A
0.5	0.026	0.053	-0.166	0.272	N/A	N/A
0.9	0.031	0.061	-0.037	0.159	N/A	N/A

Overall, the combination of cannabis and alcohol is not associated with a reliable change in crashes relative to cannabis alone. However, the 95% confidence intervals indicate a lack of measurement prediction. The results are also limited by the inclusion of only a single study in the analysis, which precludes both the generation of prediction intervals and the meaningful exploration of small study effects and the potential moderating factors.

***Hazard RT.** This meta-analysis includes four effect sizes representing 129 participants. Hazards included slowing forward vehicles (Ramaekers et al., 2000b [Study 1]; Sexton et al., 2002), approaching vehicles (Sexton et al., 2002), on-road obstacles (Liguori et al., 2002) and general “emergencies” (Downey et al., 2013).

With a pre-post correlation of zero, the combination of cannabis and alcohol is not associated with a change in hazard RT compared to cannabis alone (Hedge's $g = 0.171$; 95% CI = -0.070, 0.412; Figure C144). Similar results were obtained with a pre-post correlation of 0.5 (Hedge's $g = 0.166$; 95% CI = -0.004, 0.336; Figure C145). However, the summary statistic

became statistically significant with a pre-post correlation of 0.9 (Hedge's $g = 0.145$; 95% CI = 0.069, 0.221; Figure C146). Results, including a conversion to r effect size and prediction intervals, appear in Table 55, below.

Table 55. Effect of cannabis combined with alcohol on hazard RT (compared to cannabis).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	0.087	0.171	-0.070	0.412	-0.358	0.700
0.5	0.084	0.166	-0.004	0.336	-0.208	0.540
0.9*	0.074	0.145	0.069	0.221	-0.021	0.312

Overall, the pattern of results suggests that there is weak evidence for a difference in hazard RT between cannabis alone and the combination of drugs. The 95% confidence intervals indicate a lack of precision. Even in the case of the statistically significant increase in hazard RT, the effect is trivial in magnitude, and a pre-post correlation of 0.9 may be optimistically high. Furthermore, the 95% prediction intervals indicate that the true effect probably lies somewhere between a trivial to small decrease in hazard RT, to a small to moderate increases in hazard RT (depending on the pre-post correlation used). Due to the low number of included studies, small study effects and the potential influence of moderating factors cannot be explored meaningfully.

****Lateral Position Variability.** This meta-analysis includes four effect sizes representing 68 participants. With a pre-post correlation of zero, the combination of cannabis and alcohol was not associated, on average, with an increase in lateral position variability (Hedge's $g = 0.332$; 95% CI = -0.008, 0.672; Figure C147). The results were similar, but statistically significant, with a pre-post correlation of 0.5 (Hedge's $g = 0.336$; 95% CI = 0.036, 0.636; Figure C148) and a pre-post correlation of 0.9 (Hedge's $g = 0.286$; 95% CI = 0.047, 0.525; Figure C149). Results, including a conversion to r effect size and prediction intervals, appear in Table 56, below.

Table 56. Effect of cannabis combined with alcohol on lateral position variability (compared to cannabis).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	0.178	0.332	-0.008	0.672	-0.414	1.078
0.5*	0.177	0.336	0.036	0.636	-0.687	1.359
0.9*	0.148	0.286	0.047	0.525	-0.789	1.362

Overall, the combination of cannabis and alcohol generally increases lateral position variability relative to cannabis alone. On average, the increase is small in magnitude, but the 95% confidence intervals indicate a lack of precision. Due in part to this, as well as unknown moderating factors, the prediction intervals are wide: they range from small to large decreases in lateral position variability with the combination of drugs (depending on the pre-post correlation used) to a very large increase in lateral position variability, relative to cannabis alone. Unfortunately, the low number of included studies precludes exploration of potential moderating factors, as well as small study effects.

***Lane Excursions.** This meta-analysis included two effect sizes representing 98 participants. With a pre-post correlation of zero, the combination of cannabis and alcohol was not associated with a reliable change in lane excursions relative to cannabis alone (Hedge's $g = 0.108$; 95% CI = -0.169, 0.385; Figure C150). The results were similar with a pre-post correlation of 0.5 (Hedge's $g = 0.106$; 95% CI = -0.090, 0.302; Figure C151). However, a statistically significant result was achieved with a pre-post correlation of 0.9 (Hedge's $g = 0.095$; 95% CI = 0.007, 0.182; Figure C152). Results, including a conversion to r effect size and prediction intervals, appear in Table 57, below.

Table 57. Effect of cannabis combined with alcohol on lane excursions (compared to cannabis).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	0.055	0.108	-0.169	0.385	N/A	N/A
0.5	0.054	0.106	-0.090	0.302	N/A	N/A
0.9*	0.048	0.095	0.007	0.182	N/A	N/A

As with hazard RT, the pattern of results suggests a lack of evidence for a difference in rates of lane excursions between the combination of drugs and cannabis alone. In the case of the statistically significant increase in lane excursions with the combination of drugs, the effect size is trivial in magnitude, and a pre-post correlation is probably optimistically high. Furthermore, due to the low number of included studies, prediction intervals cannot be generated, and small study effects and the potential influence of moderating factors cannot be explored meaningfully.

****Time Out of Lane.** This meta-analysis included one effect size representing 18 participants. With a pre-post correlation of zero, the combination of cannabis and alcohol was not associated with a reliable change in time out of lane relative to cannabis alone (Hedge's $g = 0.531$; 95% CI = -0.152, 1.213; Figure C153). Results became statistically significant with a pre-post correlation of 0.5 (Hedge's $g = 0.475$; 95% CI = 0.002, 0.949; Figure C154) and 0.9 (Hedge's $g = 0.328$; 95% CI = 0.124, 0.532; Figure C155). Results, including a conversion to r effect size and prediction intervals, appear in Table 58, below.

Table 58. Effect of cannabis combined with alcohol on time out of lane (compared to cannabis).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	0.265	0.531	-0.152	1.213	N/A	N/A
0.5*	0.240	0.475	0.002	0.949	N/A	N/A
0.9*	0.169	0.328	0.124	0.532	N/A	N/A

Overall, the pattern of results suggest that the combination of cannabis and alcohol, relative to cannabis alone, increases time out of lane to a small to moderate degree. However, this rests on the assumption that there is at least a small correlation between pairs of measurements in the included study. However, the 95% confidence interval indicates a lack of measurement precision, and there is not enough data to compute prediction intervals. The inclusion of only a single effect size in the meta-analysis also precludes the meaningful exploration of small study effects and potential moderators.

Speed. This resulting meta-analysis includes three effect sizes representing 112 participants. With a pre-post correlation of zero, the combination of cannabis and alcohol was not associated with a reliable change in speed relative to cannabis alone (Hedge's $g = -0.037$; 95% CI = -0.294, 0.221; Figure C156). The results were similar with a pre-post correlation of 0.5 (Hedge's $g = -0.036$; 95% CI = -0.219, 0.146; Figure C157) and a pre-post correlation of 0.9 (Hedge's $g = -0.038$; 95% CI = -0.161, 0.085; Figure C158). Results, including a conversion to r effect size and prediction intervals, appear in Table 59, below.

Table 59. Effect of cannabis combined with alcohol on speed (compared to cannabis).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	-0.019	-0.037	-0.294	0.221	-1.707	1.633
0.5	-0.019	-0.036	-0.219	0.146	-1.217	1.144
0.9	-0.020	-0.038	-0.161	0.085	-1.240	1.163

Overall, the results indicate that the combination of cannabis and alcohol may not differ from alcohol alone in terms of speed. However, the 95% confidence intervals indicate a lack of measurement precision. The 95% prediction intervals indicate that the true effect lies somewhere between a very large decrease in speed and a very large increase in speed with the combination

of drugs relative to cannabis alone. However, the low number of included studies precludes the meaningful exploration of potential moderating factors, as well as small study effects.

Speed Variability. This meta-analysis included one effect size representing 12 participants. With a pre-post correlation of zero, the combination of cannabis and alcohol was not associated with a reliable change in speed variability relative to cannabis alone (Hedge's $g = -0.049$; 95% CI = -0.794, 0.696; Figure C159). Similar results were obtained with a pre-post correlation of 0.5 (Hedge's $g = -0.049$; 95% CI = -0.575, 0.478; Figure C160) and a pre-post correlation of 0.9 (Hedge's $g = -0.047$; 95% CI = -0.283, 0.188; Figure C161). Results, including a conversion to r effect size and prediction intervals, appear in Table 60, below.

Table 60. Effect of cannabis combined with alcohol on speed variability (compared to cannabis).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	-0.026	-0.049	-0.794	0.696	N/A	N/A
0.5	-0.026	-0.049	-0.575	0.478	N/A	N/A
0.9	-0.025	-0.047	-0.283	0.188	N/A	N/A

The pattern of results suggest that the combination of drugs may not differ from cannabis alone in terms of speed variability. However, the 95% confidence intervals indicate a lack of precision. The meta-analysis is limited by the inclusion of only a single effect. For this reason, it is not possible to generate prediction intervals, nor is there sufficient data for the meaningful exploration of small study effects and potential moderators.

***Speed Exceedances.** This meta-analysis included one effect size representing 80 participants. With a pre-post correlation of zero, the combination of cannabis and alcohol was not associated with a reliable change in speed exceedances relative to alcohol alone (Hedge's $g = 0.209$; 95% CI = -0.101, 0.520; Figure C162). The results were similar with a pre-post

correlation of 0.5 (Hedge's $g = 0.208$; 95% CI = -0.011, 0.428; Figure C163). However, the results became statistically significant with a pre-post correlation of 0.9 (Hedge's $g = 0.199$; 95% CI = 0.100, 0.297; Figure C164). Results, including a conversion to r effect size and prediction intervals, appear in Table 61, below.

Table 61. Effect of cannabis combined with alcohol on speed exceedances (compared to cannabis).

Pre-Post r	r	Hedge's g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
0.0	0.105	0.209	-0.101	0.520	N/A	N/A
0.5	0.104	0.208	-0.011	0.428	N/A	N/A
0.9*	0.100	0.199	0.100	0.297	N/A	N/A

As with hazard RT and lane exceedances, the pattern of results suggests there is little evidence that the combination of drugs changes rates of speed exceedances compared to cannabis alone. A statistically significant increase in speed exceedances only occurs with a pre-post correlation of 0.9. The results are limited by the inclusion of only a single effect size in the meta-analysis, and the 95% confidence intervals indicate a lack of measurement precision. Additionally, there are not enough studies included in this meta-analysis to compute prediction intervals and to meaningfully explore small study effects and potential moderators.

Summary of the combination of drugs compared to cannabis. Statistically significant changes between the combination of cannabis and alcohol, and cannabis alone, on experimental driving measures tended to depend on the pre-post correlation used in within-subjects studies. Again, measurement imprecision limits interpretation.

Subgroup Analyses

The effect of varying levels of alcohol on measures of driving performance and behaviour, relative to baseline, is presented here. Only driving performance and behaviour measures with ten or more effect sizes included in the primary analysis were parsed by BAC level and subjected to subgroup analysis. The BAC groups are as follows: Bin 1, any non-zero BAC up to 0.03%; Bin 2, BAC 0.04 – 0.06%; Bin 3, BAC 0.07 – 0.09%; Bin 4, BAC 0.10 – 0.12%. Note that the number of effect sizes in the tables (i.e., column *k*) and the number of omitted studies will not necessarily sum to the number of effect sizes in the primary meta-analyses. This is because some studies involve multiple BAC levels which were collapsed to generate a single effect size per study in the primary meta-analyses; in contrast, a single study may contribute multiple effect sizes (i.e., to multiple bins) in the subgroup analyses presented here. Additionally, the subgroup analysis only includes effect sizes that could be reliably associated with an average BAC level. This required the reporting of a pre-drive BAC, a post-drive BAC, and/or an average BAC specifically associated with the driving component of a test battery.

Crashes. In total, nine effect sizes that compared alcohol to baseline were included. No effect sizes were associated with Bin 1 or Bin 4. Additionally, results from the primary analyses comparing cannabis to baseline are re-reported here. Results are presented in Table 62, below, and in Appendix D (Figures D1 to D3).

Table 62. The effects of varying levels of alcohol on crashes (relative to baseline).

<i>Pre-Post Correlation = zero (see also Fig. D1)</i>						
	k	g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
Cannabis						
N/A	1	0.158	-0.152	0.467	N/A	N/A
Alcohol						
1	0	N/A	N/A	N/A	N/A	N/A
2	3	0.238	-0.015	0.490	-1.399	1.875
3*	6	0.507	0.262	0.752	0.161	0.854
4	0	N/A	N/A	N/A	N/A	N/A
<i>Pre-Post Correlation = 0.5 (see also Fig. D2)</i>						
	k	g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
Cannabis						
N/A	1	0.155	-0.063	0.374	N/A	N/A
Alcohol						
1	0	N/A	N/A	N/A	N/A	N/A
2*	3	0.228	0.050	0.406	-0.927	1.383
3*	6	0.438	0.256	0.620	0.180	0.696
4	0	N/A	N/A	N/A	N/A	N/A
<i>Pre-Post Correlation = 0.9 (see also Fig. D3)</i>						
	k	g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
Cannabis						
N/A*	1	0.140	0.043	0.238	N/A	N/A
Alcohol						
1	0	N/A	N/A	N/A	N/A	N/A
2*	3	0.190	0.110	0.269	-0.324	0.703
3*	6	0.276	0.174	0.378	0.055	0.497
4	0	N/A	N/A	N/A	N/A	N/A

As indicated in the primary analysis, there is insufficient evidence to conclude that cannabis reliably increases crashes relative to baseline in experimental studies, except when a

pre-post correlation of 0.9 is used. However, in all cases, the meta-analysis includes only a single effect size of uncertain generalizability, and more data is needed to increase statistical precision. In contrast, BAC levels ranging from 0.04% to 0.06% (i.e., Bin 2) are generally associated, to a small degree, with an increase in crashes relative to baseline. BAC levels ranging from 0.07% to 0.09% (i.e., Bin 3) consistently increase crashes relative to baseline to a small to moderate degree. Still, despite the average increase in crashes, prediction intervals indicate a wide range of values, particularly in the case of Bin 2. The results suggest that at BAC levels ranging from 0.04% to 0.06% (i.e., Bin 2), rates of crashes may *decrease* in certain cases. It is unknown whether this is a statistical artifact stemming from imprecision, or whether this reflects some drivers' compensation attempts. More data is needed to clarify the issue.

Next, effect sizes are considered in relation to each other. The average effect of alcohol on crashes appears to increase from Bin 2 to Bin 3, which suggests a dose-response relationship. However, data for the lowest levels and highest levels of alcohol are missing. There is insufficient evidence to conclude that the dose-response relationship is linear. Additionally, cannabis appears to have a uniformly weaker effect on crashes than does either level of alcohol, but it is unclear how the influence of cannabis compares to the lowest and highest levels of alcohol (i.e., Bins 1 and 4).

Hazard RT. In total, 15 effect sizes that compare alcohol to baseline were included. No effect sizes were sorted into Bin 4. Additionally, results from the primary analyses comparing cannabis to baseline are re-reported here. Results are presented in Table 63, below, and in Appendix D (see Figures D4 to D6).

Table 63. The effects of varying levels of alcohol on hazard RT (relative to baseline).

<i>Pre-Post Correlation = zero (see also Fig. D4)</i>						
	<i>k</i>	<i>g</i>	95% CI LL	95% CI UL	95% PI LL	95% PI UL
Cannabis						
N/A	9	0.115	-0.077	0.307	-0.117	0.347
Alcohol						
1	3	0.115	-0.282	0.511	-2.456	2.686
2*	7	0.404	0.217	0.592	0.068	0.741
3*	5	0.543	0.077	1.009	-1.030	2.116
4	N/A	N/A	N/A	N/A	N/A	N/A
<i>Pre-Post Correlation = 0.5 (see also Fig. D5)</i>						
	<i>k</i>	<i>g</i>	95% CI LL	95% CI UL	95% PI LL	95% PI UL
Cannabis						
N/A	9	0.148	-0.013	0.309	-0.138	0.434
Alcohol						
1	3	0.110	-0.170	0.390	-1.707	1.926
2*	7	0.373	0.178	0.568	-0.133	0.878
3*	5	0.523	0.081	0.966	-1.075	2.121
4	N/A	N/A	N/A	N/A	N/A	N/A
<i>Pre-Post Correlation = 0.9 (see also Fig. D6)</i>						
	<i>k</i>	<i>r</i>	95% CI LL	95% CI UL	95% PI LL	95% PI UL
Cannabis						
N/A*	9	0.164	0.037	0.290	-0.206	0.534
Alcohol						
1	3	0.085	-0.067	0.238	-1.304	1.474
2*	7	0.329	0.169	0.490	-0.201	0.860
3*	5	0.455	0.076	0.834	-1.000	1.910
4	N/A	N/A	N/A	N/A	N/A	N/A

As reported in the primary analyses, there is a lack of association between cannabis and increases in hazard RT relative to baseline. Significant increases are only observed with a pre-

post correlation of 0.9; in this case, prediction intervals indicate that the effect is inconsistent. Similarly, Bin 1 (i.e., BAC up to 0.03%) was not found to increase hazard RT in any case. Bins 2 and 3 (i.e., BAC levels ranging from 0.04% to 0.09%) were consistently associated with increases in hazard RT, to a small to moderate degree. However, prediction intervals generally indicated a wide degree of variability around these small increases.

When effect sizes from Bins 1 through 3 are compared to one another, a dose-response relationship between BAC level and hazard RT is apparent. However, data for BAC levels ranging from 0.10% to 0.12% (i.e., Bin 4) are missing. Interestingly, the influence of cannabis appears to fall somewhere between Bin 1 and Bin 2. That is, cannabis appears to slow hazard RT slightly more than BAC levels up to 0.03%, but it does not slow hazard RT to quite the same extent as BAC levels ranging from 0.04% to 0.06%.

Lateral Position Variability. In total, the subgroup analysis included 60 effect sizes that compared alcohol to baseline. Additionally, results from the primary analyses comparing cannabis to baseline are re-reported here. Results are presented in Table 64, below, and in Appendix D (see Figures D7 to D9).

Table 64. The effects of varying levels of alcohol on lateral position variability (relative to baseline).

<i>Pre-Post Correlation = zero (see also Fig. D7)</i>						
	<i>k</i>	<i>g</i>	95% CI LL	95% CI UL	95% PI LL	95% PI UL
Cannabis						
N/A*	14	0.366	0.205	0.528	0.186	0.546
Alcohol						
1*	8	0.304	0.040	0.569	-0.239	0.847
2*	27	0.310	0.217	0.403	0.212	0.408
3*	24	0.621	0.489	0.753	0.377	0.865
4*	1	0.969	0.335	1.603	N/A	N/A
<i>Pre-Post Correlation = 0.5 (see also Fig. D8)</i>						
	<i>k</i>	<i>g</i>	95% CI LL	95% CI UL	95% PI LL	95% PI UL
Cannabis						
N/A*	14	0.331	0.212	0.451	0.199	0.464
Alcohol						
1*	8	0.336	0.090	0.582	-0.328	0.999
2*	27	0.377	0.283	0.471	0.100	0.654
3*	24	0.599	0.493	0.706	0.325	0.873
4*	1	0.933	0.490	1.376	N/A	N/A
<i>Pre-Post Correlation = 0.9 (see also Fig. D9)</i>						
	<i>k</i>	<i>g</i>	95% CI LL	95% CI UL	95% PI LL	95% PI UL
Cannabis						
N/A*	14	0.270	0.175	0.365	-0.061	0.602
Alcohol						
1*	8	0.353	0.132	0.574	-0.372	1.078
2*	27	0.354	0.278	0.429	0.011	0.696
3*	24	0.478	0.407	0.549	0.201	0.755
4*	1	0.741	0.555	0.927	N/A	N/A

As reported in the primary analyses, cannabis consistently increased lateral position variability to a small degree. All three BAC bins were also consistently associated with small to moderate increases in lateral position variability. In all cases, the prediction intervals associated with Bin 1 (i.e., BAC levels up to 0.03%) indicated a possible *decrease* in lateral position variability in some cases.

A visual inspection of average effect sizes suggests a possible dose-response relationship between BAC level and lateral position variability, such that higher BAC levels lead to more variability. Similarities between cannabis and specific levels appear to depend on the pre-post correlation utilized. With a pre-post correlation of zero, cannabis appears to fall somewhere between Bin 2 (i.e., BAC 0.04% to 0.06%) and Bin 3 (i.e., BAC 0.07% to 0.08%). With pre-post correlations of 0.5 and 0.9, it appears to exert a weaker effect on lateral position variability than BAC levels up to 0.03% (i.e., Bin 1). However, interpretation is complicated by the presence of small study effects. As discussed in the primary meta-analysis, it is unclear whether the relationship between Hedge's g and its standard error observed within this sample of studies is legitimate, or whether it is due to publication bias. If the relationship is due to publication bias, then some or all of the effect sizes associated with Bins 1 through 4 are spuriously high, which makes it difficult to compare them with the effect size associated with cannabis.

Lane Excursions. Ultimately, the subgroup analysis contains 22 effect sizes that compared alcohol to baseline. No effect sizes were associated with Bin 1 or Bin 4. Additionally, results from the primary analyses comparing cannabis to baseline are re-reported here. Results are presented in Table 65, below, and in Appendix D (Figures D10 to D12).

Table 65. The effects of varying levels of alcohol on lane excursions (relative to baseline).

<i>Pre-Post Correlation = zero (see also Fig. D10)</i>						
	k	g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
Cannabis						
N/A	2	0.201	-0.078	0.480	N/A	N/A
Alcohol						
1	0	N/A	N/A	N/A	N/A	N/A
2*	9	0.317	0.134	0.500	-0.029	0.663
3*	13	0.626	0.445	0.808	0.422	0.830
4	N/A	N/A	N/A	N/A	N/A	N/A
<i>Pre-Post Correlation = 0.5 (see also Fig. D11)</i>						
	k	g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
Cannabis						
N/A*	2	0.198	0.001	0.395	N/A	N/A
Alcohol						
1	0	N/A	N/A	N/A	N/A	N/A
2*	9	0.326	0.154	0.498	-0.119	0.771
3*	13	0.568	0.417	0.719	0.256	0.881
4	N/A	N/A	N/A	N/A	N/A	N/A
<i>Pre-Post Correlation = 0.9 (see also Fig. D12)</i>						
	k	g	95% CI LL	95% CI UL	95% PI LL	95% PI UL
Cannabis						
N/A*	2	0.180	0.092	0.268	N/A	N/A
Alcohol						
1	0	N/A	N/A	N/A	N/A	N/A
2*	9	0.246	0.138	0.354	-0.079	0.572
3*	13	0.367	0.291	0.442	0.183	0.550
4	N/A	N/A	N/A	N/A	N/A	N/A

As previously reported in the primary analyses, cannabis is generally associated with a small increase in lane excursions relative to baseline, assuming a pre-post correlation of at least

0.5 in included studies utilizing repeated-measures designs. In contrast, BAC levels up to 0.03% were not associated with a reliable change in lane excursions. It should be noted that this observation is based on only a single study of unknown generalizability, which did not utilize a repeated measures design. Finally, Bins 2 and 3 (i.e., BAC levels ranging from 0.04% to 0.09%) were associated with small to moderate increases in lane excursions in all cases. As with crashes, prediction intervals consistently indicated that BAC levels ranging from 0.04% to 0.06% (i.e., Bin 2) may be associated in some cases with *decreases* in lane excursions. Again, it is unknown whether this is a statistical artifact or indicative of compensatory behaviours among drivers.

Next, effect sizes are considered in relation to each other. First, there appears to be a dose-response relationship between BAC level and lane excursions, such that higher BAC levels lead to more lane excursions. Second, the effect of cannabis appears to fall somewhere between Bin 1 (i.e., BAC up to 0.03%) and Bin 2 (i.e., BAC 0.04% to 0.06%) in terms of its effect on lane excursions. Cannabis appears to affect lane excursions to a weaker degree than higher doses of alcohol. However, as with lateral position variability, small study effects were observed among this sample of effect sizes within the primary meta-analysis. It is unclear whether the relationship between Hedge's *g* and its standard error observed within this sample of studies is due to publication bias. If it is, then some or all of the effect sizes associated with Bins 1 through 4 are spuriously high, which makes it difficult to compare them with the effect size associated with cannabis.

Speed. Overall, the subgroup analysis included 50 effect sizes that compared alcohol to baseline. Additionally, results from the primary analyses comparing cannabis to baseline are re-reported here. Results are presented in Table 66, below, and in Appendix D (Figures D13 to D15).

Table 66. The effects of varying levels of alcohol on speed (relative to baseline).

<i>Pre-Post Correlation = zero (see also Fig. D13)</i>						
	<i>k</i>	<i>g</i>	95% CI LL	95% CI UL	95% PI LL	95% PI UL
Cannabis						
N/A*	12	-0.182	-0.348	-0.017	-0.371	0.006
Alcohol						
1	9	0.110	-0.116	0.336	-0.202	0.421
2*	21	0.113	0.014	0.212	0.008	0.219
3*	19	0.188	0.076	0.299	0.067	0.308
4	1	0.144	-0.376	0.665	N/A	N/A
<i>Pre-Post Correlation = 0.5 (see also Fig. D14)</i>						
	<i>k</i>	<i>g</i>	95% CI LL	95% CI UL	95% PI LL	95% PI UL
Cannabis						
N/A*	12	-0.176	-0.298	-0.053	-0.315	0.036
Alcohol						
1	9	0.086	-0.117	0.289	-0.347	0.519
2*	21	0.102	0.025	0.180	0.019	0.186
3*	19	0.171	0.058	0.285	-0.158	0.500
4	1	0.132	-0.236	0.500	N/A	N/A
<i>Pre-Post Correlation = 0.9 (see also Fig. D15)</i>						
	<i>k</i>	<i>g</i>	95% CI LL	95% CI UL	95% PI LL	95% PI UL
Cannabis						
N/A*	12	-0.205	-0.336	-0.074	-0.639	0.230
Alcohol						
1	9	0.047	-0.108	0.202	-0.379	0.474
2*	21	0.102	0.029	0.175	-0.170	0.374
3*	19	0.147	0.047	0.246	-0.276	0.569
4	1	0.086	-0.078	0.250	N/A	N/A

As reported in the primary analyses, cannabis was consistently associated with a small decrease in speed. In contrast, Bins 2 and 3 (i.e., BAC 0.04% to 0.09%) were consistently

associated with a small *increase* in speed. Interestingly, prediction intervals associated with these two significant effects sometimes spanned ranges in the opposite direction. Specifically, the prediction intervals indicated that cannabis may *increase* speed in some cases, and in most cases, Bin 3 (i.e., BAC 0.06% to 0.08%) may be associated with *decreases* in speed as well. Bins 1 (i.e., BAC up to 0.03%) and 4 (i.e., BAC 0.07% to 0.09%), in all cases, had confidence intervals that spanned zero, indicating imprecision.

Next, effect sizes were considered in relation to each other. Unlike the other measures, a linear dose-response relationship between alcohol and effect size magnitude is not apparent. However, the sudden decrease in effect size magnitude associated with Bin 4 is based on only a single effect size of uncertain generalizability. It is unknown whether the effect is spuriously low, or whether the decreased propensity to speed at Bin 4 compared to Bin 3 represents deliberate compensatory strategies among drivers. More data is needed to verify driver behaviour at BAC levels ranging from 0.10% to 0.12% and to increase precision to facilitate comparisons between varying levels of alcohol in terms of their effects on speed. However, cannabis clearly appeared to have the opposite effect on speed as alcohol.

Speed Variability. Ultimately, the subgroup analysis included 36 effect sizes that compared alcohol to baseline. Additionally, results from the primary analyses comparing cannabis to baseline are re-reported here. Results are presented in Table 67, below, and in Appendix D (Figures D16 to D18).

Table 67. The effects of varying levels of alcohol on speed variability (relative to baseline).

<i>Pre-Post Correlation = zero (see also Fig. D16)</i>						
	<i>k</i>	<i>g</i>	95% CI LL	95% CI UL	95% PI LL	95% PI UL
Cannabis						
N/A	7	0.047	-0.220	0.314	-0.303	0.397
Alcohol						
1	7	0.135	-0.109	0.379	-0.185	0.455
2*	16	0.187	0.065	0.309	0.054	0.321
3*	12	0.289	0.145	0.433	0.125	0.453
4*	1	0.640	0.069	1.212	N/A	N/A
<i>Pre-Post Correlation = 0.5 (see also Fig. D17)</i>						
	<i>k</i>	<i>g</i>	95% CI LL	95% CI UL	95% PI LL	95% PI UL
Cannabis						
N/A	7	0.104	-0.113	0.321	-0.180	0.388
Alcohol						
1	7	0.158	-0.024	0.340	-0.081	0.397
2*	16	0.220	0.093	0.347	-0.123	0.564
3*	12	0.273	0.158	0.388	0.087	0.459
4*	1	0.601	0.201	1.001	N/A	N/A
<i>Pre-Post Correlation = 0.9 (see also Fig. D18)</i>						
	<i>k</i>	<i>g</i>	95% CI LL	95% CI UL	95% PI LL	95% PI UL
Cannabis						
N/A*	7	0.166	0.048	0.284	-0.020	0.352
Alcohol						
1*	7	0.166	0.054	0.278	-0.096	0.427
2*	16	0.212	0.095	0.329	-0.235	0.660
3*	12	0.229	0.142	0.317	-0.051	0.510
4*	1	0.431	0.259	0.603	N/A	N/A

As reported in the primary analyses, cannabis was not associated with a reliable increase in speed variability except for the case where a pre-post correlation of 0.9 was used. In contrast,

Bins 2 through 4 (i.e., BAC ranging from 0.04% to 0.12%) were consistently associated with increases in speed variability. Bin 4 (i.e., BAC 0.10% to 0.12%), which included only a single effect size of unknown generalizability, had the least precise estimate. Bin 1 (i.e., BAC up to 0.03%) was associated with a reliable increase in speed variability with a pre-post correlation of 0.9.

Visual inspection of average effects suggests a possible dose-response relationship, such that speed variability increases with higher BAC levels. Additionally, the effect of cannabis appeared to be weaker than the effect of Bin 1 (i.e., BAC up to 0.03%).

Study Quality & Risk of Bias

The results of the study quality and risk of bias assessment are reported in Appendix F. Here, notable study quality issues and risk of bias issues related to selection bias, blinding, participant attrition, reporting bias and sample size are discussed.

Selection bias. Many, if not most, of the included studies are at risk of selection bias. Of the studies that described their recruitment method, most relied on self-referral (e.g., potential participants responded to posters or advertisements), and a small number involved recruiting individuals known to the research team. Although researchers are largely limited to these methods out of necessity, participants recruited via these methods do not necessarily represent the broader population of drivers who use cannabis and/or alcohol. Possibly, participants who are self-referred may be interested in demonstrating their perceived efficacy (or inefficacy) in driving under the influence of cannabis or alcohol. Participants may be motivated to prove that they can drive safely under the influence of cannabis (Hartman et al., 2015). Notably, Brands et al. (2019) indicated that a participant was removed from their analysis for attempting to “skew” the data. Participants’ attempts to bias driving data, however, can only succeed to the extent that

participants are unblinded. Unfortunately, debinding to experimental conditions appears to be a pervasive issue in this research domain (see *Deblinding and nonblinding*, below).

In addition to the possible risk of selection bias arising from recruitment methods, study inclusion and exclusion criteria also limit the generalizability of findings. Typically, studies exclude participants with substance dependency, including cannabis and alcohol (see Table 2). Although research ethics may preclude researchers from administering cannabis or alcohol to individuals with substance dependencies, the literature indicates that driving under the influence of cannabis and alcohol in real life is often associated with problematic use or dependency (Cook et al., 2017; Swift et al., 2010; Jones et al., 2007; Begg et al., 2003; Evans, 2004).

Deblinding and nonblinding. It was often difficult to assess whether research participants were aware of the researchers' hypotheses based on methodological descriptions, which led to more disagreements and discussions among coders. These disagreements are reflected in lower Kappa scores⁷ (see Table F2 in Appendix F). Also, it was not uncommon for studies to report that ratings of subjective drug high varied between active and placebo cannabis conditions (e.g., Anderson et al., 2010; Bosker et al., 2012; Liguori, 1998; Liguori et al. 2002) or that participants could tell the difference between active and placebo cannabis conditions (e.g., Arkell et al., 2019; Ronen et al., 2010; Sexton et al., 2000; Sexton et al., 2002; Stein, 1985). Concerns about the utility of placebo cannabis are not new (Sutton, 1983).

Although individuals who drive while under the influence of cannabis and/or alcohol in the real world are not typically blind to their state, limitations in recruitment methods could lead

⁷ As previously discussed, all disagreements were followed up with discussions between coders until a consensus was reached.

to the enrolment of participants who may not drive as they normally would in real life, but would instead exaggerate their driving behaviour in an attempt to “prove” that they can drive safely while under the influence. The extent of this issue in biasing research findings across the literature, however, is unknown. However, the lack of blinding among participants in experimental driving studies focused on cannabis and alcohol is not exceptional among the broader impaired driving literature. Notably, experimental driving studies that investigate the effects of cell phones or other technological distractions compared to baseline driving cannot blind participants to study conditions either. On the other hand, systematic changes in driver behaviour toward safety in experimental driving studies has interesting real-world implications. Findings from experimental driving studies can be thought of as demonstrative of participants’ driving abilities while they are driving as well as they possibly can (Evans, 2004). Driving performance decrements observed in experimental driving studies (i.e., slowing of hazard RT, impaired lane keeping and longitudinal control) may theoretically be worse in real life.

In addition to participant debinding, non-blinding of researchers to participant drug conditions was common. Although non-blinding of researchers is unlikely to influence the *measurement* of driving performance and behaviour data (i.e., detection bias), which is usually captured automatically (and objectively) in simulators or with instrumented vehicles, the non-blinding of researchers or study personnel to participant drug conditions could theoretically lead to changes in the way that researchers or study personnel interact with research participants and consequently lead to changes in the way that participants behave in study conditions.

Attrition reporting. In many cases, it was difficult to tell how many of the enrolled participants completed the entirety of the study. Even when no dropouts or withdrawals were reported, it was unclear whether all enrolled participants had actually completed the study or

whether non-completing participants went without mention. Notably, one study that reported one withdrawal did not report a substantial number of participant drop-outs that were identified only after reviewing the study's online registration (Hartman et al., 2015). Rates and reasons for withdrawals and dropouts help readers understand whether research findings reported in a study may be biased due to attrition. Reporting rates of study dropouts and withdrawals also helps future researchers anticipate and prepare for rates of attrition in their own future studies.

Reporting bias. Most of the included studies were deemed to be at low risk of reporting bias. Specifically, the measures reported in the method section matched the measures reported in the results section. When measures indicated in the method were not reported in the results, or the results section contained measures that were not pre-specified (including cases where no measures were pre-specified at all), the study was typically deemed to be at high risk of reporting bias. In cases where it was unclear whether all measures were reported upon, such as cases where categories of measures were pre-specified instead of specific measures, or cases where it was unclear whether measures were listed because they were actually variables of interest (e.g., crashes, or automatically-captured driving performance data), the study was typically deemed to be at an unclear risk of reporting bias. This item often had more disagreements and discussions among coders (see Table F2 in Appendix F).

However, in assessing risk of bias, it became clear that the criteria for judging risk of bias was fundamentally limited. Publishing multiple studies with different measures collected from the same participant dataset, known as “salami-slicing,” is not an uncommon practice in this body of literature. Clearly, there is little utility in judging whether measures reported in the method and results match when additional undeclared measures may be reported in additional publications. Researchers are strongly encouraged to be transparent in reporting non-

independence between data appearing in multiple publications. Transparency is particularly important when measures collected from a common participant dataset that appear in multiple publications tap into a common construct (e.g., lane keeping ability). Without context, readers may believe that the literature of evidence for an effect is larger and more consistent than it really is. Similarly, when similar measures from multiple studies involving common participant datasets are inadvertently pooled in a meta-analysis, the precision of the effect is over-estimated, leading to bias in the summary statistic (Borenstein et al., 2009, pp. 225-238).

Sample size. Finally, very few of the included studies reported targeting a specific sample size based on hypothesized effect size and power. Sample sizes that are too small lead to not only decreased power, but also the increased probability that statistically-significant effects are spurious (Ioannidis, 2005). Researchers who conduct experimental driving studies focused on the effects of cannabis in the future should ensure that studies are adequately powered by enrolling an appropriate number of participants. The summary statistics reported in this meta-analysis may be consulted for this purpose.

Chapter 4: Discussion

The following section is composed of five key subsections. First, the results of the meta-analysis are summarized. Second, theoretical implications are discussed with reference to Fuller's (2005) Task-Capability Interface model. Third, practical implications for policy-makers and real world drivers are described. Fourth, limitations of the meta-analysis are listed. Finally, future research directions are outlined.

Results of the Meta-Analysis

The body of experimental literature focused on the effects of cannabis on driving performance and behaviour, for which effect sizes can be computed, is relatively small. Within this body of literature, there is clear evidence that cannabis impairs lateral control (i.e., increases in lateral position variability, possible increases in lane excursions) and causes reductions in speed relative to baseline driving. In contrast, there was no compelling evidence that cannabis reliably changes rates of crashes, hazard RT, headway, headway variability, time out of lane, speed variability, speed exceedances or time speeding. Results are summarized in Table 68, below.

Table 68. Summary of the effects of cannabis on driving performance and behaviour relative to baseline.

Measure	k	N	Results
Crashes	1	80	No compelling evidence that cannabis reliably changes crash rates relative to baseline in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Hazard RT	9	242	No compelling evidence that cannabis reliably changes hazard RT relative to baseline in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Headway	1	14	No compelling evidence that cannabis reliably changes headway relative to baseline in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Headway Variability	1	14	No compelling evidence that cannabis reliably changes headway variability relative to baseline in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Lateral Position Variability	14	257	On average, cannabis increases lateral position variability by approximately 0.3 to 0.4 standard deviations relative to baseline in experimental studies.
Lane Excursions	2	98	Based on limited evidence, cannabis generally increases lane excursions by approximately 0.2 standard deviations, on average, relative to baseline in experimental studies.
Time Out of Lane	1	18	No compelling evidence that cannabis reliably changes time out of lane relative to baseline in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Speed	12	312	On average, cannabis decreases speed by approximately 0.2 standard deviations relative to baseline in experimental studies.
Speed Variability	7	137	No compelling evidence that cannabis reliably changes speed variability relative to baseline in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Speed Exceedances	1	80	No compelling evidence that cannabis reliably changes rates of speed exceedances relative to baseline in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Time Speeding	0	0	Insufficient data for meta-analysis.

It is critical to understand that for all the above variables where there is a lack of evidence for an effect of cannabis on an experimental driving measure, it is not necessarily the case that the measure is wholly unaffected by cannabis. Likewise, it is incorrect to conclude that lateral control and speed are more strongly affected by cannabis than are crashes, hazard RT, headway, headway variability, time out of lane, speed variability and speed exceedances. Very few studies have studied the influence of cannabis on these measures and reported data necessary for effect size computation. Consequently, the meta-analyses conducted to assess the influence of cannabis on these measures lack precision. Additional data is sorely needed to understand how cannabis

affects response time to hazards, following distance, longitudinal control and crash involvement in experimental studies.

Compared to the body of experimental literature focused on the effects of cannabis on driving performance and behaviour relative to sober driving, the literature focused on directly comparing the effects of cannabis and alcohol on driving performance and behaviour, for which effect sizes can be computed, is even smaller. However, this report also incorporates indirect evidence by way of comparing the effects of cannabis on driving performance and behaviour relative to baseline driving, to the effects of alcohol on driving performance and behaviour relative to baseline driving, via subgroup analyses. Direct comparisons are summarized in Table 69, below.

Table 69. Summary of the effects of cannabis on driving performance and behaviour relative to alcohol.

Measure	k	N	Results
Crashes	1	80	No compelling evidence that cannabis reliably changes crash rates relative to alcohol in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Hazard RT	4	128	No compelling evidence that cannabis reliably changes hazard RT relative to alcohol in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Headway	0	0	Not enough data for meta-analysis.
Headway Variability	0	0	Insufficient data for meta-analysis.
Lateral Position Variability	5	81	No compelling evidence that cannabis reliably changes lateral position variability relative to alcohol in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Lane Excursions	2	98	No compelling evidence that cannabis reliably changes rates of lane excursions relative to alcohol in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Time Out of Lane	1	18	No compelling evidence that cannabis reliably changes time out of lane relative to alcohol in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Speed	4	125	Based on limited evidence, cannabis decreases speed by approximately 0.3 to 0.4 standard deviations, on average, relative to alcohol in experimental studies. However, both the strength and direction of the effect vary substantially based on unknown moderating factors. More research is needed to fully characterize how cannabis and alcohol differ in terms of their effects on speed.
Speed Variability	2	26	No compelling evidence that cannabis reliably changes speed variability relative to alcohol in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Speed Exceedances	1	80	Based on limited evidence, cannabis generally decreases rates of speed exceedances by approximately 0.2 standard deviations, on average, relative to alcohol in experimental studies.
Time Speeding	0	0	Insufficient data for meta-analysis.

Again, it is critical to understand that for all cases where there is a lack of evidence for a difference between cannabis and alcohol on an experimental driving measure, it is not necessarily the case that cannabis and alcohol have the same effect on that measure. Few effect sizes were included in these analyses, leading to measurement imprecision.

There are limitations to interpreting the similarities and differences between cannabis and alcohol without accounting for differences in dose. Obviously, higher doses of alcohol are more intoxicating than lower doses of alcohol, so the natural question to ask is how cannabis differs from different levels of alcohol. For this reason, subgroup analyses based on BAC were

conducted. However, given the paucity of literature that directly compares the influence of cannabis to the influence of alcohol on driving performance and behaviour, only indirect comparisons were included in the subgroup analyses. Specifically, the effects of alcohol on driving performance and behaviour relative to baseline were stratified within subgroup analyses for measures that had ten or more included studies. The effects of each range of blood alcohol concentrations on driving performance and behaviour relative to baseline were then compared to the effect of cannabis on driving performance and behaviour relative to baseline. Indirect comparisons based on subgroup analyses are summarized in Table 70, below.

Table 70. Summary of the effects of cannabis on driving performance and behaviour relative to baseline, compared to the effects of alcohol on driving performance and behaviour relative to baseline.

Measure	Results
Crashes	Based on limited data, cannabis is not associated with an increase in crashes in experimental studies. However, crashes increase at BAC levels of to 0.03%, and to an even greater extent at BAC levels of 0.04% to 0.06%. Thus, cannabis affects crashes to a lesser extent than BAC levels ranging from 0.04% to 0.06%, and to an even lesser extent than BAC levels ranging from 0.07% to 0.09%. However, there is not enough data to compare cannabis to BAC levels up to 0.03%, or from 0.10% to 0.12%.
Hazard RT	Based on limited data, cannabis is not associated with an increase in hazard RT. However, hazard RT slows with increasing BAC levels starting at a BAC of 0.04%. Thus, cannabis affects hazard RT to a similar or greater extent than BAC levels up to 0.03%, but to a lesser extent than BAC levels of 0.04% and higher.
Lateral Position Variability	Based on limited data, lateral position variability increases with increasing BAC levels. Cannabis increases lateral position variability to a similar, greater or lesser extent than BAC levels up to 0.03% (depending on the pre-post correlation used), but it increases lateral position variability to a lesser extent than BAC levels of 0.07% and higher.
Lane Excursions	Based on limited data, cannabis increases lane excursions to a lesser extent than BAC levels ranging from 0.04% to 0.06%, and to an even lesser extent than BAC levels ranging from 0.07% to 0.09%. However, there is not enough data to compare cannabis to BAC levels up to 0.03%, or from 0.10% to 0.12%.
Speed	Based on limited data, speed increases with increasing BAC levels up to 0.09%. Only one effect size is included with a BAC level of 0.10% to 0.12%. Cannabis decreases speed relative to all BAC levels. Up to a BAC level of 0.09%, greater differences in speed between cannabis and alcohol are observed with increasing BAC levels.
Speed Variability	Based on limited data, cannabis is not associated with an increase in speed variability. However, speed variability increases with increasing BAC levels starting at a BAC of 0.04%. Thus, cannabis affects speed variability to a similar or lesser extent than BAC levels up to 0.03%.

To the extent that greater blood alcohol concentrations lead to greater driving performance decrements, cannabis appears to affect driving performance to a similar extent as low levels of alcohol. Specifically, for the measures reported here, there are no instances where the average effect of cannabis is equal to or greater than the driving performance decrements associated with a BAC concentration ranging from 0.04% to 0.06%. With respect to speed, cannabis and alcohol had opposite effects. Cannabis led to decreases in speed, whereas alcohol led to increases in speed, with generally greater increases in speed at higher BAC levels.

The body of literature that compares the effect of the combination of cannabis and alcohol to baseline or either in isolation is, like the body of literature focused on comparisons of cannabis to alcohol on driving performance and behaviour, small and in need of further study. Results of the effect of the combination of drugs on driving performance and behaviour, relative to baseline, are summarized in Table 71, below. Results of the effect of the combination of drugs on driving performance and behaviour, relative to either in isolation, are also summarized and appear in subsequent tables in this section, below.

Table 71. Summary of the effects of the combination of cannabis and alcohol on driving performance and behaviour, relative to baseline.

Measure	k	N	Results
Crashes	1	80	Based on limited evidence, the combination of drugs generally increases crash rates by approximately 0.2 standard deviations, on average, relative to baseline in experimental studies.
Hazard RT	4	129	Based on limited evidence, the combination of drugs slows hazard RT by approximately 0.3 to 0.4 standard deviations, on average, relative to baseline in experimental studies. However, both the strength and direction of the effect vary substantially based on unknown moderating factors. More research is needed to fully characterize the relationship between the combination of drugs and hazard RT.
Headway	0	0	Insufficient data for meta-analysis.
Headway Variability	0	0	Insufficient data for meta-analysis.
Lateral Position Variability	4	68	Based on limited evidence, the combination of drugs increases lateral position variability by approximately 0.5 standard deviations, on average, relative to baseline in experimental studies. However, both the strength and direction of the effect vary substantially based on unknown moderating factors. More research is needed to fully characterize the relationship between the combination of drugs and headway.
Lane Excursions	2	98	Based on limited evidence, the combination of drugs increases rates of lane excursions by approximately 0.2 to 0.3 standard deviations, on average, relative to baseline in experimental studies.
Time Out of Lane	1	18	Based on limited evidence, the combination of drugs increases time out of lane to approximately 0.5 to 0.7 standard deviations, on average, relative to baseline in experimental studies.
Speed	3	112	No compelling evidence that the combination of drugs reliably changes speed relative to baseline in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Speed Variability	1	12	No compelling evidence that the combination of drugs reliably changes speed variability relative to baseline in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Speed Exceedances	1	80	No compelling evidence that the combination of drugs reliably changes rates of speed exceedances relative to baseline in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Time Speeding	0	0	Insufficient data for meta-analysis.

Generally, the combination of cannabis and alcohol is detrimental to driving performance relative to baseline. However, the literature is small, and the meta-analyses suffer from imprecision.

Next, the effects of the combination of cannabis and alcohol on driving performance and behaviour relative to alcohol are considered. Results are summarized in Table 72, below.

Table 72. Summary of the effects of the combination of cannabis and alcohol on driving performance and behaviour, relative to alcohol.

Measure	k	N	Results
Crashes	1	80	No compelling evidence that the combination of drugs reliably changes crash rates relative to alcohol in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Hazard RT	4	128	No compelling evidence that the combination of drugs reliably changes hazard RT relative to alcohol in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Headway	0	0	Insufficient data for meta-analysis.
Headway Variability	0	0	Insufficient data for meta-analysis.
Lateral Position Variability	4	67	Based on limited evidence, the combination of drugs increases lateral position variability by approximately 0.5 standard deviations, on average, relative to alcohol in experimental studies. However, both the strength and direction of the effect vary substantially based on unknown moderating factors. More research is needed to fully characterize how the combination of drugs, and alcohol alone, differ in terms of their effects on lateral position variability.
Lane Excursions	2	98	No compelling evidence that the combination of drugs reliably changes rates of lane excursions relative to alcohol in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Time Out of Lane	1	18	Based on limited evidence, the combination of drugs generally increases time out of lane by approximately 0.4 to 0.6 standard deviations, on average, relative to alcohol in experimental studies.
Speed	3	111	No compelling evidence that the combination of drugs reliably changes speed relative to alcohol in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Speed Variability	1	12	No compelling evidence that the combination of drugs reliably changes speed variability relative to alcohol in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Speed Exceedances	1	80	No compelling evidence that the combination of drugs reliably changes rates of speed exceedances relative to alcohol in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Time Speeding	0	0	Insufficient data for meta-analysis.

This meta-analysis indicates that the combination of cannabis and alcohol is more detrimental to driving performance relative to alcohol in isolation. Again, the literature is small, and the meta-analyses suffer from imprecision.

Finally, the effects of the combination of cannabis and alcohol on driving performance and behaviour relative to cannabis are considered. Results are summarized in Table 73, below.

Table 73. Summary of the effects of the combination of cannabis and alcohol on driving performance and behaviour, relative to cannabis.

Measure	k	N	Results
Crashes	1	80	No compelling evidence that the combination of drugs reliably changes crash rates relative to cannabis in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Hazard RT	4	129	No compelling evidence that the combination of drugs reliably changes hazard RT relative to cannabis in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Headway	0	0	Insufficient data for meta-analysis.
Headway Variability	0	0	Insufficient data for meta-analysis.
Lateral Position Variability	4	68	Based on limited evidence, the combination of drugs generally increases lateral position variability by approximately 0.3 standard deviations, on average, relative to cannabis in experimental studies. However, both the strength and direction of the effect vary substantially based on unknown moderating factors. More research is needed to fully characterize how the combination of drugs, and cannabis alone, differ in terms of their effects on lateral position variability.
Lane Excursions	2	98	No compelling evidence that the combination of drugs reliably changes rates of lane excursions relative to cannabis in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Time Out of Lane	1	18	Based on limited evidence, the combination of drugs generally increases time out of lane by approximately 0.3 to 0.5 standard deviations, on average, relative to cannabis in experimental studies.
Speed	3	112	No compelling evidence that the combination of drugs reliably changes speed relative to cannabis in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Speed Variability	1	12	No compelling evidence that the combination of drugs reliably changes speed variability relative to cannabis in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Speed Exceedances	1	80	No compelling evidence that the combination of drugs reliably changes rates of speed exceedances relative to cannabis in experimental studies. Lack of compelling evidence is primarily due to lack of data, which results in imprecision. More research is needed.
Time Speeding	0	0	Insufficient data for meta-analysis.

Consistent with comparisons of the combination of drugs on driving performance relative to alcohol in isolation, the combination of drugs has a more detrimental effect on driving performance than cannabis in isolation.

Theoretical Implications

The influence of cannabis on lateral control and speed have several theoretical implications. First, Fuller (2005) conceptualizes speed as the mechanism by which drivers regulate effort (and when required, risk) while driving. The observed reductions in speed associated with cannabis indicate that drivers experience increased difficulty and/or risk while completing the driving task while under the influence of cannabis, compared to while sober (i.e., baseline). Interestingly, cannabis was also found to increase lateral position variability and lane exceedances. This suggests that drivers' attempts to compensate for increased driving difficulty may not be entirely successful, and detriments in driving performance (i.e., increased lateral position variability) may still occur.

The consideration of drivers' compensatory attempts while driving under the influence of cannabis within the context of Fuller's (2005) theory generates important hypotheses related to real-world driver behaviour. Compensatory behaviours can occur not only at the tactical level (i.e., changes in driving speed), but also at the strategic level, which "defines the general planning stage of a trip, including the determination of trip goals, route, and modal choice, plus an evaluation of the costs and risks involved" (Michon, 1985, p. 481). Within an experimental driving study, participants are essentially limited to compensating at a tactical level, such as through adjustments to speed and following distance. However, in the real world, drivers may also compensate at a strategic level. The rationale for this hypothesis rests on an important assumption – namely, that drivers make a conscious choice to compensate for increased driving task difficulty (versus reducing their speed as a consequence of a more automatic, perceptually-driven process; Ward & Dye, 1999), and that their conscious choice is not simply a demand characteristic arising from observation by researchers. Indeed, studies have found that some

individuals who drive while under the influence of cannabis do report making conscious efforts to compensate for their intoxicated state by decreasing their driving speed, as well as by increasing their following distance and engaging in generally more “careful” or more “cautious” driving (Watson et al., 2019; Brooks-Russell et al., 2019; MacDonald et al., 2008). As predicted by Fuller’s (2005) model, research has also found that some individuals have reported compensating at a strategic level while under the influence of cannabis, such as by delaying the start of the drive or limiting the amount of cannabis consumed prior driving (Watson et al., 2019; Swift et al., 2010). Strategic-level compensatory mechanisms have important implications for the crash risk associated with cannabis (Rogeberg & Elvik, 2016). However, self-reports of compensatory strategies are fundamentally limited, and more objective data from more representative samples would aid in more fully characterizing how and when different subpopulations of cannabis users (i.e., infrequent users, regular users, medical users, dependent users) compensate at both the tactical and strategic level.

Although it is not clear whether reductions in speed observed in experimental driving studies reflect a conscious decision on the part of the driver, or a more automatic, perceptually-driven process, or some combination of both (Ward & Dye, 1999), both are consistent with Fuller’s (2005) model. However, the former would be consistent with cases where drivers respond to increases in perceived risk (rather than general increases in task demands) while driving under the influence of cannabis. Differentiating the relative contributions of automatic and conscious processes to speed changes are an important avenue for future research (see *Future Research*, below).

Next, the differential effects of cannabis and alcohol can also be interpreted in terms of Fuller’s (2005) theory. Fuller’s (2005) theory posits that drivers use speed to regulate effort

while driving; when the driving task becomes difficult, drivers slow down, and when the driving task becomes easier, drivers speed up. In the case of cannabis, it is clear that the lateral position of the vehicle becomes more difficult to maintain, and drivers attempt to compensate for increased task difficulty by slowing down. However, drivers under the influence of alcohol also, on average, experience lateral control difficulties (i.e., more lateral position variability, increased lane excursions, more time out of lane), as well as more crashes, slowed hazard RT, and longitudinal control difficulties (i.e., increased headway variability, increased speed variability). Despite this, drivers under the influence of alcohol do not slow down – instead, the results indicate that on average, they *increase* their driving speed in the included studies. It is not entirely clear why this occurs, but consideration of Fuller's (2005) model allows for some hypotheses to be generated. Perhaps drivers temporarily adopt a higher threshold for effort. Alternatively, participants under the influence of alcohol may not have realized that the driving task had become more difficult due to some sort of perceptual failure. Again, the perceptual and/or cognitive mechanisms underlying changes in driving speed while under the influence of cannabis and/or alcohol should be investigated in the future.

Finally, based on limited evidence, the combination of cannabis and alcohol has negative effects on driving ability compared not only to sober (i.e., baseline) driving, but also to either drug in isolation. Based on Fuller's (2005) model, driving performance decrements observed with the combination of cannabis and alcohol, which are greater in magnitude than either in isolation, should be accompanied speed reductions that are also of a greater magnitude than either in isolation. However, the results of this meta-analysis also indicate that alcohol does not lead to decreases in speed despite clearly impaired lane keeping, which makes predictions about the influence of the combination of the two drugs on driving speed difficult.

Some researchers have suggested that when cannabis and alcohol are combined, they effectively cancel each other out: specifically, it has been hypothesized that the propensity toward speeding while under the influence of alcohol and the propensity toward slowing down while under the influence of alcohol are additive, resulting in a null effect on speed (Ronen et al., 2010; Hartman et al., 2016). Although the combination of cannabis and alcohol was not associated with a change in speed relative to baseline, the results of this meta-analysis do not provide evidence that the two drugs counteract each other. A lack of evidence for an effect is not evidence for a null effect. The lack of an effect reported here is due to the small number of included studies, which lead to imprecision. In contrast, if a very small effect size and narrow confidence interval were observed, there may be reason to believe that the drugs counteract each other. Given that this was not one of the results of this meta-analysis, the hypothesis that cannabis and alcohol counteract each other on speed is not substantiated here. Future research is needed to verify how the combination of cannabis and alcohol affect driving speed relative to baseline and either drug in isolation.

Practical Implications

The results of this meta-analysis have important implications for real-world drivers. Does driving under the influence of cannabis constitute impaired driving? The results of the meta-analyses clearly indicate that driving under the influence of cannabis is impaired driving, and there is no evidence that cannabis improves driving ability, as some drivers would like to believe (Watson et al., 2019; Brooks-Russell et al., 2019; Swift et al., 2010; Terry & Wright, 2005). Although experimental driving studies indicate that cannabis is associated with slower driving speeds, impaired lane keeping persists. Additionally, based on limited evidence, the combination of cannabis and alcohol is more detrimental to lateral control than either alcohol or cannabis in

isolation. However, the literature is quite limited. Few studies incorporating the experimental driving measures reviewed here assess the influence of the combination of cannabis and alcohol to either in drug isolation. Again, more research is sorely needed in this area.

Evidence for additive effects of cannabis and alcohol warrants careful consideration of the need for and selection of appropriate countermeasures. In Canada, it is a criminal offence to drive within two hours of having a prohibited level of either alcohol or THC in the blood (Government of Canada, 2018). The prohibited limit for alcohol is 80 mg per decilitre of blood, and the prohibited limit for THC is 2 ng per millilitre of blood for a summary offence and 5 ng per millilitre of blood for a hybrid offence (Government of Canada, 2018). However, the combination of drugs is subject to special provisions: it is also an offence to drive within two hours of having both 50 mg of alcohol per decilitre (i.e., 0.05% BAC) and 2.5 ng of THC per millilitre of blood concurrently (Government of Canada, 2018). The implicit assumption behind the adjustment to the blood alcohol limit during the simultaneous presence of THC is that the effects of cannabis and alcohol are additive. Indeed, the results of the present meta-analysis are not at odds with this assumption. Additionally, driving under the combined influence of both cannabis and alcohol is not an uncommon behaviour. Research conducted in Canada, Switzerland, New Zealand, France, Australia, Italy and the United States indicates that the concurrent presence of alcohol is common among suspected impaired drivers who test positive for cannabis (Senna et al., 2010; Couper et al., 2014; Wood & Salomonsen-Sautel, 2016), as well as among cannabis-positive drivers involved in injury crashes (Mura et al., 2003; Longo et al., 2000; Favretto et al., 2018) and in fatal crashes (Poulsen et al., 2012; Laumon et al., 2005; Romano et al., 2017; Davey et al., 2020; Drummer et al., 2003; Beasley et al., 2011). However,

the implementation of THC limits may not be the most appropriate approach to managing cannabis-impaired driving, with or without concurrent alcohol impairment, for several reasons.

First, THC limits do not appear to have a strong empirical foundation. One paper often cited as a source for THC limits is an influential analytic study conducted by Grotenhermen and colleagues (2005). According to Grotenhermen and colleagues (2005), per se limits from alcohol were primarily based on epidemiological studies focused on quantifying crash risk. However, in the absence of sufficient epidemiological data, Grotenhermen and colleagues (2005) also considered experimental data. In particular, the two meta-analyses by Berghaus and colleagues (1998a, 1998b), which are described in the *Introduction* of this dissertation, formed the basis of Grotenhermen and colleagues' (2005) analysis. As discussed, Berghaus and colleagues' (1998a, 1998b) research syntheses are limited in a number of ways: they included studies focused on driving-related skills, which have an ambiguous relationship with safety (Shinar, 2017, p. 659); THC concentrations associated with observed effects were often imputed rather than measured directly; and, the syntheses used a vote-counting method, which is not a valid approach to meta-analysis (Borenstein et al., 2009, pp. 251-255, 325). Additionally, the results of the current meta-analysis suggest that the assumptions underlying Berghaus and colleagues' (1998, 1998b) syntheses are invalid. To illustrate, Grotenhermen and colleagues' (2005) reiterate the following limitation identified by Berghaus and colleagues (1998a, 1998b) in relation to their own two syntheses:

The methodology of comparative meta-analyses assumes implicitly that if a set of THC and alcohol concentrations produces the same impairment ratio in experimental studies, it also produces the same actual accident risk under real traffic conditions. This assumption

will not be valid if drivers under traffic conditions compensate differently for the impairment caused by THC and alcohol. (Grotenhermen et al., 2005, p. 30)

As indicated in the current meta-analysis, drivers under the influence of cannabis in experimental driving studies decrease their driving speed, whereas drivers under the influence of alcohol increase their driving speed. Thus, drivers under the influence of cannabis compensate for increased task demands or risk while driving, while drivers under the influence of alcohol do not. Consequently, the assumptions underlying Berghaus and colleagues' (1998a, 1998b) comparative meta-analyses do not hold, and suggestions for THC limits based on those meta-analyses, such as those offered by Grotenhermen and colleagues (2005, 2007), are not valid for this reason, among others discussed above.

Second, as discussed in the introduction, cannabis and alcohol differ in their respective pharmacokinetic and pharmacodynamic profiles. Because cannabis and alcohol are dissimilar substances, it is overly simplistic to attempt to approach the detection of and countermeasures to cannabis-impaired driving in the same manner as alcohol-impaired driving (e.g., by implementing legislated limits). Research indicates that blood THC concentration is not a good marker of whether someone is fit or unfit to drive which complicates attempts to select and justify any particular THC limit. For example, Logan and colleagues (2016) investigated whether THC concentrations could be used to predict "impairment" measured as Drug Recognition Expert (DRE) evaluation performance. Overall, Logan et al. (2016) reported that errors on most DRE indicators were not associated with THC concentrations, and performance on most indicators did not differ between individuals with THC concentrations above and below 5 ng/mL of THC. Additionally, DRE indicators failed to discriminate between individuals with THC concentrations above and below 5 ng/mL. Based on these findings, Logan and colleagues stated

simply: “the data do not support science-based per se limits for THC” (p. 28). Logan and colleagues’ (2016) interpretation of their results was later criticized by Capler and colleagues (2017), who questioned the use of DRE evaluation performance as an indicator for impairment. However, similar null findings were observed in a recent Canadian study that sampled excess routine blood work collected by physicians from drivers injured in police-investigated collisions. This study found that there was no increase in crash responsibility at blood THC levels less than 2 ng/mL (the summary offence limit), between 2 ng/mL and 5 ng/mL, or over 5 ng/mL (the hybrid offence limit) of whole blood (Brubacher et al., 2019).

In its analysis of Bill C-46 in September 2017, the Canadian Bar Association (CBA) stated: “The CBA Section recommends that the federal government base any measurement of blood drug concentration on proven scientific evidence that links the concentration to impairment” (CBA, 2017, p. 5). Currently, the scientific evidence is weak. Researchers disagree on the extent to which THC concentrations are a valid indicator of driver impairment, in part due to disagreements about what exactly constitutes impairment. Indeed, constitutional challenges to Canada’s impaired driving legislation targeting cannabis-positive drivers are anticipated (e.g., The Canadian Press, 2019a).

Finally, it is not known whether per se limits for THC reduce the prevalence of cannabis-impaired drivers or prevents injuries or fatalities (Anderson & Rees, 2015; Logan et al., 2016). However, it is generally accepted that alcohol per se limits were only partially responsible for the historical decrease in alcohol-involved fatal crashes since their implementation; increased visibility of enforcement, as well as revolutionary changes in societal norms and opinions about alcohol-impaired following the work of grassroots organizations such as MADD, also played an important role (Evans, 2004). The belief that cannabis impairs driving ability is associated with

decreased intent to drive under its influence (Davis et al., 2016; Swift et al., 2010; Jones et al., 2007). To decrease rates of cannabis-impaired driving and cannabis-involved crashes, societal opinions about driving under the influence of cannabis – specifically, that it is safe, or potentially even makes one a better driver – need to change. This meta-analysis clearly indicates that cannabis impairs driving. Furthermore, engaging in compensatory strategies are likely insufficient to mitigate against cannabis impairment.

Given these issues, it is the opinion of this author that a more pragmatic approach than the implementation of THC limits may be a universal adjustment to existing BAC limits. Based on the subgroup analyses (i.e., of crashes, hazard RT, lateral position variability, lane excursions and speed variability), cannabis appears to affect driving performance to a similar level as low levels of alcohol. If the detrimental effects of cannabis on driving (i.e., a doubling in crash risk) are conceptualized as a benchmark against which to judge the necessity for legislative interventions in response to other forms of impaired driving, and the detrimental effects of cannabis on driving performance appear to be similar in magnitude to levels of alcohol below the current Canadian BAC limit of 80 mg of alcohol per decilitre of blood (i.e., 0.08% BAC), then it becomes apparent that the appropriateness of the current BAC limit needs to be revisited once more (for a previous argument to lower the Canadian BAC limit, see Chamberlain & Solomon, 2002). Specifically, it follows that there is a case for lowering the blood alcohol limit from 80 mg of alcohol per decilitre of blood, which is associated with a quadrupling of crash risk (Compton & Berning, 2015), to 50 mg of alcohol per decilitre of blood (i.e., 0.05% BAC), which is associated with an approximately doubled crash risk (Compton & Berning, 2015). Indeed, 50 mg of alcohol per decilitre of blood was the proposed limit previously suggested by Chamberlain and Solomon (2002) nearly two decades ago. There is evidence that lowering blood alcohol

limits from 0.08% BAC to 0.05% BAC decreases rates of injuries and fatalities not only among drivers with blood alcohol levels targeted by the reduced limit, but at all BAC levels (Fell & Scherer, 2017; Fell & Voas, 2014; Mann et al., 2001). Thus, a universal BAC limit of 0.05% could be used to not only to charge the same suspected cannabis-and-alcohol impaired drivers to whom the current regulations apply, but it could also potentially deter other cases of alcohol-impaired driving.

Limitations

This meta-analysis is not without limitations. Notably, the meta-analysis suffers from data loss, which is particularly problematic given that the extant literature is already small. As discussed, the meta-analysis suffers from imprecision. However, much of the eligible literature also fails to report the complete set of data needed to compute effect sizes. First, in order to compute standardized mean difference effect sizes for within-subjects studies, which comprise the majority of the literature, the correlation between pairs of scores is required. However, in most cases, these are not reported and are irretrievable. This issue is not unique to the experimental driving literature – it is a common issue for meta-analysis in general. Because most pre-post correlations were unknown, and recovered pre-post correlations were discordant from one another, sensitivity analyses were conducted using pre-post correlation values of zero, 0.5 and 0.9. This wide range of values is theorized to capture the range of actual plausible correlations, but it cannot be known for certain. Additionally, approximately one third of the studies eligible for inclusion were not included due to incomplete reporting of statistical data, including means and standard deviations. It is unknown whether the exclusion of those studies biases the results of the current meta-analysis in any particular direction.

Small study effects were observed for the effects of alcohol on lateral position variability and lane excursions relative to baseline. Both of these measures are related to the construct of lateral control. It is unknown whether the small study effects reflect publication bias or a legitimate relationship between the magnitude of an effect and its standard error. In the absence of a compelling case for either, the values reported here are unadjusted. However, they should be interpreted with this in mind.

Finally, there are limitations to the generalizability of the results reported in this meta-analysis due to the demographics of the included participants. As discussed previously, most of the included studies focused on the effects of cannabis on driving reflect young adults, and it is unclear whether the results are generalizable to older and younger drivers. Additionally, many of the included studies excluded heavier cannabis and/or alcohol users, who, as previously discussed, may be more likely to drive while intoxicated. Thus, the results of this meta-analysis may only be generalizable to a subset of drivers and/or substance users. Future studies investigating the effects of cannabis on driving performance and behaviour should focus on young and inexperienced drivers, older drivers, naïve cannabis users, heavy and chronic cannabis users, and medical cannabis-using drivers. This, and other future research directions, are discussed next.

Future Research

In completing this meta-analysis, several future research considerations were identified. First, a number of next steps are indicated based on meta-analytic findings. Additionally, there are some obvious gaps in the literature. Importantly, a number of study quality issues were identified that should be addressed in future work. In the following two sections, specific research directions, and quality considerations for future studies, are described.

Future research directions. First, there is a clear paucity of research in the areas covered within the scope of the current analysis. More data is needed related to the effects of cannabis, alcohol and their combination on experimental driving measures other than those related to lane keeping.

Second, consideration of findings within the context of Fuller's (2005) Task-Capability Interface model lead to the generation of a number of hypotheses and avenues for future research. As previously discussed, drivers under the influence of cannabis slow their driving speed in experimental driving studies. It is unclear whether this reflects a conscious decision on the part of the driver, a more perceptually-driven process, or some combination of both. Research should be conducted to determine if drivers' perceptions of speed change while under the influence of cannabis, and whether such changes lead to the adoption of slower driving speeds. The potential role of the same mechanisms should also be investigated with respect to the increase in speed observed while driving under the influence of alcohol. More data is needed to verify whether the relationship between alcohol dose and speed increases is linear across BAC levels as low as 0.01% and as high as 0.12%. Additionally, the prediction intervals associated with Bin 3, which ranges from 0.07 to 0.09% BAC, indicate that in some cases, drivers may actually *decrease* their speed while under the influence of alcohol at a level of BAC. It is unclear whether the negative values within the prediction intervals are statistical artifacts, or whether it is probable that drivers reliably decrease their speed in certain circumstances (e.g., by compensating for increased driving task difficulty) while under the influence of alcohol. More research is needed to determine whether drivers who are under the influence of alcohol attempt to compensate for their impaired state, and if so, whether BAC level moderates the relationship.

Third, research should be conducted to better understand why alcohol slows response time to tangible, on-road hazards. Although slowed response time has an obvious relationship with crashing, researchers should also focus on how alcohol affects rates of hits, misses, false alarms and correct rejections with respect to those hazards. This would allow researchers more context to interpret slowing in on-road hazards. For example, if hazard response time slows while under the influence of alcohol, is this because drivers experience diminished sensitivity to on-road hazards, or is it because drivers adopt a higher threshold for what constitutes an on-road hazard? These questions also apply to cannabis-impaired driving. Relatedly, the literatures focused on visual scanning during states of acute cannabis intoxication, within the context of driving, appear small. If cannabis does lead to more cautious driving, then a possible research direction is to investigate whether visual scanning behaviours change such that a greater amount of time is spent deliberately searching for hazards.

Fourth, cannabis preparations with a greater variety of cannabinoid compositions should be studied within the context of driving. Cannabidiol (CBD), a cannabinoid that naturally occurs in varying concentrations in the cannabis plant (Russo & Guy, 2006; Huestis, 2007), has become a popular item on the market (e.g., The Canadian Press, 2019b). Both CBD-containing oils and dry cannabis flowers are available to purchase for recreational use in Canada. Although CBD-containing cannabis is now readily available, most of the studies considered for inclusion reported the THC content, but not the CBD content, of the administered cannabis, and only one study in this meta-analysis investigated whether cannabis containing CBD affects driving performance and behaviour differently or similarly than cannabis containing negligible amounts of CBD (i.e., Arkell et al., 2019). Although several reviews suggest that CBD may have the benefit of lessening some of the more negative effects of THC (Russo & Guy, 2006; Fischer et

al., 2017), Arkell and colleagues (2019) reported that cannabis containing balanced concentrations of THC and CBD was “no less impairing” than cannabis containing THC and only negligible concentrations of CBD. However, this conclusion appears to have been made based on an indirect comparison. Although there was a significant difference between high-THC, low-CBD cannabis and placebo, as well as no significant difference between high-THC, high-CBD cannabis and placebo, they did not appear to test whether the two drug-driving conditions differed from each other in terms of SDLP. However, they did directly compare high-THC, low-CBD cannabis and high-THC, high-CBD cannabis to each other with respect to subjective effects, and no differences were observed between the conditions.

Overall, more research is needed to understand if and how the interaction of CBD and THC affect driving performance and behaviour. For instance, if CBD attenuates the negative influence of THC on driving performance, then fewer driving performance decrements would be expected with cannabis containing both THC and CBD, compared to high-THC, low-CBD cannabis. In contrast, if the influence of CBD is limited to, or has a greater influence on, the subjective effects of THC (compared to the negative effects of THC on driving performance), then high-THC, high-CBD cannabis could also be theorized to affect driving more similarly to alcohol. That is, if high-THC, high-CBD cannabis has the same negative influence on driving performance as high-THC, low-CBD cannabis, but drivers are less aware that they are impaired due to a less intense-feeling high, they may not compensate for their impaired state to the same extent as drivers under the influence of high-THC, low-CBD cannabis. For now, there is insufficient evidence to support the hypothesis that high-THC, high-CBD cannabis is a safer alternative to high-THC, low-CBD cannabis with respect to driving.

A fifth future research direction concerns the influence of cannabis on driving performance and behaviour for young and inexperienced drivers, as well as older drivers. As indicated in Table 1, most of the studies where cannabis was administered involved participants who were, on average, in their twenties. In Canada, the prevalence of past-year cannabis use in 2017 was highest among young adults aged 20 to 24 (33%), followed by youth and young adults aged 15 to 19 (19%) and adults over the age of 25 (13%) (Government of Canada, n.d.-c). Few participants in the cannabis studies appeared to be teenagers or young adults, or older adults (i.e., age 65 and over). Young, inexperienced drivers are known for having a higher risk of crashing than older, more experienced drivers, and observational studies indicate that they are also more susceptible to the detrimental effects of alcohol while driving (Peck et al., 2008; Voas et al., 2012). Although there appears to be less data focused on young novice drivers' crash risk in association with driving under the influence of cannabis, it seems reasonable to posit that acute cannabis intoxication could increase young novice drivers' vulnerability to crashing. Future experimental driving studies should be conducted to understand how young novice drivers' performance is affected by acute cannabis intoxication. Additionally, although the *prevalence* of cannabis use is lower among adults over the age of 25, most past-year cannabis users in Canada are over the age of 25 (Government of Canada, n.d.-c). Part of this group includes drivers over the age of 65, who are also at an elevated risk of crashing per vehicle mile travelled (Ryan et al., 1998; Evans, 2004). In Canada, individuals aged 65 and older also represent the fastest-growing group of cannabis consumers, and 27% of new cannabis users in the second and third quarters of 2019 belonged to this age group (Statistics Canada, 2019). Future research should also focus on understanding how older drivers' driving performance and behaviour is affected by acute cannabis intoxication.

A sixth research direction concerns medical cannabis users. Over a third of past-year cannabis users in Canada in 2017 reported use of cannabis for medical purposes (Government of Canada, n.d.-c). Although medical users represent an important part of the cannabis-using population in Canada, the current meta-analysis was focused exclusively on non-clinical participants, and the degree to which the results of the meta-analysis generalize to medical cannabis users is unclear. In reviewing studies for eligibility within the current meta-analysis, the literature focused on the effects of cannabis on medical cannabis users' driving performance and behaviour appears to be limited. An important question to ask is whether the costs of driving under the influence of a drug, such as medical cannabis, are greater than the costs of driving in an untreated state – that is, while experiencing the symptoms that the drug has been prescribed to treat, such as pain (Shinar, 2017, p. 657). Future experimental driving studies should be conducted with participants within the medical-cannabis using population.

Finally, future research should be conducted for the purposes of providing empirical support for recommendations to drivers about when it is safe to drive after a period of acute cannabis intoxication. Fischer and colleagues (2017), who authored the review upon which Canada's lower-risk cannabis use guidelines are based (see CAMH, 2019), suggest – based on a “substantial” level of evidence – that “users abstain from driving for at least the acute period of impairment identified by current scientific evidence” (p. e7), which they deem to be at least six hours after consuming cannabis but possibly longer. However, most the studies cited in support of their recommendation do not come from the driving performance and behaviour literature; most report on the effects of cannabis on physiological measures, subjective measures and driving-related skills. Again, different approaches to measuring driver impairment need to be reconciled before researchers can offer a clear answer to the question of how long impairment

lasts after consuming cannabis. Additionally, the time that drivers need to wait until pre-intoxication driving performance is restored, and the time that drivers need to wait until blood THC levels have dropped to a permissible level, are not necessarily the same. While an individual could reasonably estimate in advance how much they could drink in order to ensure that their blood alcohol concentration is not over the legal limit by the time they plan to drive (e.g., Watson et al., 1981), the same is not true of cannabis (e.g., Huestis et al., 1992). Drivers who do not wish to commit a criminal offence in Canada must ensure that their blood THC concentration does not exceed established limits within two hours of driving; however, this is not something that drivers can actually do (Fischer et al., 2017). Ultimately, the Government of Canada (n.d.-d) states on its website about drug-impaired driving that “there is no guidance to drivers about how much cannabis can be consumed before it is unsafe to drive or how long a driver should wait to drive after consuming cannabis” (*How cannabis impairs drivers*, line 5). One possible research question to address is whether drivers, after consuming cannabis, experiencing acute cannabis intoxication and waiting for the effects to dissipate, can reliably detect the point at which they have returned to their normal, pre-intoxicated state. In other words, are drivers’ perceptions of their own post-cannabis sobriety accurate? Experimental driving studies should be conducted in the future to help provide recommendations to drivers about how long they should wait to drive after consuming cannabis, and/or whether subjective feelings of sobriety are reliable indicators of actual sobriety. Ideally, such research should incorporate infrequent users, regular users, medical users and dependent users. Medical users and dependent users, however, pose a unique challenge in that they may not have a clear normal, pre-intoxicated state to compare acute intoxication against, either due to heavy use or due to the presence of

symptoms during periods of sobriety. Additionally, cannabis oils and edibles, which may elicit stronger or longer-lasting effects (Huestis, 2007), should be incorporated within this work.

Quality considerations for future research. The current meta-analysis offers a snapshot of the extant experimental literature focused on the effects of cannabis on driving performance and behaviour. As discussed, there are many avenues and opportunities for future work. As studies are published in the future, the current meta-analysis will accordingly require updating. However, it is hoped that the study quality issues reported here, and the recommendations for addressing them, present an enduring contribution to the literature as it evolves. Overall, researchers are encouraged to focus on addressing quality issues related to participant recruitment, blinding, reporting and theory.

Recruitment. First, researchers interested in conducting studies focused on the effects of cannabis and/or alcohol on driving performance and behaviour in the future should consider novel approaches to recruitment that serve to avoid the potential for selection bias. As discussed in the results, most of the studies used recruitment methods that involved posters, advertisements and other self-selection methods. With the increasing prevalence of legalized medicinal and recreational cannabis, there are new opportunities to start addressing this issue by employing new methods to participant recruitment. A novel approach to participant recruitment could take the form of interviewing cannabis purchasers leaving dispensaries, with the goal of characterizing eligible individuals (i.e., active cannabis users who drive) who are and who are not interested in participating in experimental driving studies. This would allow researchers to gain more insight about whether their studies are likely to be at risk of selection bias or not.

Blinding. Second, researchers should be aware that de-blinding to drug conditions in experimental driving studies is a common problem that poses a threat to validity but is difficult

to avoid. Researchers should anticipate blinding issues with standard inactive cannabis controls. Casarett (2018) describes the issue in relation to medical cannabis research and suggests a number of approaches to preventing or controlling for deblinding, including the use of psychoactive controls rather than inactive placebo controls, the recruitment of non-users, manipulation checks and the use of high-CBD strains. Some of these suggestions are likely more feasible than others within the context of experimental driving studies. First, the selection of an appropriate psychoactive control is not easy (Casarett, 2018). It is unclear which psychoactive control would be best suited for experimental driving research. Second, the recruitment of non-users for experimental driving studies is likely to present ethical issues, and the knowledge benefits that stand to be gained by administering cannabis to non-users, who do not belong to the population of interest (i.e., cannabis users who drive), is questionable. However, manipulation checks designed to assess whether there is any relationship between participants' belief of or actual knowledge of whether or not they have received active cannabis or a control should be standard in future experimental driving studies focused on assessing the influence of cannabis on driving. Finally, the use of high CBD cannabis, rather than low CBD cannabis, may be useful in assessing the degree to which THC affects driving performance and behaviour. However, as previously discussed, Arkell and colleagues (2019) found that the subjective effects experienced by participants did not differ between conditions involving THC-only cannabis and the balanced CBD-THC cannabis. Although more research is needed, it does not appear to be the case that cannabis containing CBD will necessarily diminish the subjective effects associated with THC to the point that it cannot be distinguished from placebo.

Reporting. Third, researchers who conduct studies focused on the effects of cannabis and/or alcohol in the future are encouraged to strive for high quality in reporting study methods

and results. During the study quality and risk of bias assessment, it was often difficult to assess how many participants were recruited, whether any dropouts or withdrawals occurred after recruitment, and whether the measures reported in a paper reflected the complete set of measures collected as part of a single project. For guidance on good quality reporting, researchers are encouraged to consult the CONSORT 2010 statement and flow diagram (Schulz et al., 2010) as Brands et al. (2019) have done. Although the statement is focused on randomized controlled trials, the elements included in the checklist and flow diagram are also applicable to both between-subjects and within-subjects experimental driving studies. Additionally, researchers are urged to be transparent about cases where data from a single set of participants is divided into multiple papers or re-reported upon following additional or revised analyses. In several instances, overlap was not identified until statistical data describing participant demographics or experimental driving measures were compared, or when effect sizes were visualized in funnel plots. Often, authors needed to be contacted directly to verify whether multiple papers were independent of one another. Finally, authors are encouraged to report tables of means and standard deviations. Lack of statistical reporting led to the exclusion of over 30 eligible studies in this meta-analysis. When space is limited, authors are encouraged to submit these tables as online supplementary materials.

Theory. Finally, researchers need to incorporate theory in future work. In reviewing the literature in preparation of this meta-analysis, as well as in reviewing full-texts to evaluate their eligibility for inclusion, it became apparent that experimental driving studies are largely atheoretical. Consequently, driver impairment is conceptualized by researchers in many different ways, with a variety of measures and a variety of interpretations for what those measures mean in relation to safety. These disagreements hamper the ability for researchers to clearly answer the

question, “is driving while under the influence of cannabis unsafe?” In this meta-analysis, the selection of measures, and their interpretation with respect to answering the question of whether cannabis impairs driving, was guided by Fuller’s (2005) framework. Measures that do not have a clear relationship to safety, when considered within the context of that framework, were not included in this meta-analysis. For example, it has been suggested that automatic functions are affected by cannabis more than cognitive functions under the conscious control of the driver, such as passing other vehicles (Grotenhermen et al., 2005; Sewell et al., 2009). However, changes in automatic functions measured in a laboratory setting may not necessarily scale up to driving performance decrements when drivers have the ability to adjust their driving behaviour to compensate for those decrements, and impairment within the context of cognitive functions is ambiguous. What exactly does it mean, for example, for cannabis to have a negative effect on passing? Impairment is more than a statistically significant change in behaviour from sober driving to driving under the influence – changes must be considered within the context of real-world safety. If a driver elects to pass up more opportunities to overtake a vehicle while under the influence of cannabis, is it because the driver fails to see safe opportunities to pass due to cannabis-induced perceptual deficits, or is it because the driver has adjusted to a more cautious, conservative driving style to reduce task demands or risk? The two have opposite implications for safety. Experimental driving research needs to move toward the incorporation of theory to prevent this type of ambiguity from occurring. When a measure is chosen for inclusion in an experimental driving study, the selection of the measure should be theoretically defensible, and hypotheses generated about how experimental manipulations will affect the chosen measure should be falsifiable. Researchers should avoid making post-hoc interpretations of their results within the context of safety in the absence of formal, theoretically-driven hypotheses.

Conclusion

This meta-analysis focused on the effects of cannabis and alcohol on driving performance and behaviour, both alone and in combination with one another. To date, this is the first meta-analysis to focus on a comprehensive set of measures of driving performance and behaviour within the context of cannabis and alcohol. Studies focused on purely cognitive and driving-related skills, which do not have a clear relationship with safety, were excluded. The results indicate that cannabis impairs driving performance. Most notably, cannabis is detrimental to lateral control of the vehicle even though drivers under the influence of cannabis attempt to compensate for their impaired state by slowing their driving speed. In contrast, individuals under the influence of alcohol generally increase their driving speed, which indicates a lack of awareness for their impaired state. Finally, the combination of both drugs is generally more detrimental to driving performance than either in isolation. However, the literature reviewed is small and in need of more data, and important quality issues and future directions identified in the current meta-analysis can help guide further scientific inquiry.

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Appendix A: Search Strategy

Table A1. Search strategy for PsycINFO, Embase and MEDLINE.

1	driving under the influence/
2	drunken driving/
3	1 or 2
4	tetrahydrocannabinol/
5	cannabinoids/
6	cannabis/
7	hashish/
8	marijuana/
9	4 or 5 or 6 or 7 or 8
10	3 or 9
11	“driv*” .m_titl.
12	“simulat*” .m_titl.
13	11 or 12
14	10 and 13

Table A2. Search strategy for Academic Search Complete, CINAHL and SportDISCUS.

S1	SU Cannabis OR Hashish OR SU Marijuana
S2	SU alcoholic beverages
S3	TI driv* OR TI simulat*
S4	S1 OR S2
S5	S3 AND S4

Note: All searches limited to Academic Journals.

The search strategy for Scopus was as follows:

TITLE (alcohol OR dronabinol OR nabilone OR tetrahydrocannabinol OR thc OR cannabis OR hash* OR marijuana OR marihuana) AND TITLE (driv* OR simulat*) AND (LIMIT-TO (SUBJAREA, “MEDI”) OR LIMIT-TO (SUBJAREA, “SOCI”) OR LIMIT-TO (SUBJAREA, “PHAR”) OR LIMIT-TO (SUBJAREA, “PSYC”)) AND (LIMIT-TO (DOCTYPE, “ar”) OR LIMIT-TO (DOCTYPE, “re”) OR LIMIT-TO (DOCTYPE, “cp”)) AND (LIMIT-TO (EXACTKEYWORD, “Automobile Driving”) OR LIMIT-TO (EXACTKEYWORD, “Car Driving”))

The search strategy for TRID, which was limited to only articles and papers, was as follows:

(alcohol OR dronabinol OR nabilone OR tetrahydrocannabinol OR thc OR cannabis OR hash* OR marijuana OR marihuana) AND (driv* OR simulat*)

Appendix B: Eligible Studies Excluded for Insufficient Data

Table B1. Studies that met inclusion criteria but did not report enough data for effect size computation.

Study	Setting	Included N	M Age (SD)	Relevant IV's	Relevant DV's
Allen & Stein, 1996	Simulator	33 (Unclear F)	Not reported.	Alcohol, Placebo	Lat. Control: <i>SD of Lane Deviation</i>
Attwood et al., 1980	Closed Course	6 (0 F)	Range 22 - 25	Alcohol, Placebo Control	Lat. Control: <i>Lane Position Variance</i> Speed: <i>Mean Velocity</i> Headway: <i>Mean Headway</i> Long. Control: <i>Velocity Variance, Headway Variance</i>
Attwood et al., 1981	Closed-Course	8 (0 F)	Range 20 - 28	Cannabis, Alcohol, Combination ² , Placebo Control	Lat. Control: <i>SD Lane Position</i> Speed: <i>Mean Velocity</i> Long. Control: <i>SD Velocity, SD Headway</i> Headway: <i>Headway</i>
Barkley et al., 2006	Simulator	39 (Unclear F) ¹	Unclear; M = 29.2 (8.2) for original 46 participants	Alcohol, Placebo Control	RT: <i>Total Brake Reaction Time [Hazard]</i> Speed: <i>Average Speed</i> Long. Control: <i>Variability of Speed</i> Crashes: <i>Collisions</i>
Burian et al., 2002	Simulator	13 (0 F)	M = 31, Range 23 – 43	Alcohol, Placebo	Speed: <i>Driving Speed</i>
Chen & Chen, 2017	Simulator	16 (0 F)	Range 18 – 24	Alcohol, Placebo	Lat. Control: <i>SDLP</i> Speed: <i>Mean Speed</i>
de Waard & Brookhuis, 1991	On-Road	20 (0 F)	Range 25 – 40	Alcohol, Untreated Control	Lat. Control: <i>SD of Lateral Position</i> Headway: <i>Time Headway</i>

Study	Setting	Included N	M Age (SD)	Relevant IV's	Relevant DV's
Fairclough & Graham, 1999	Simulator	32 (0 F) ²	M = 30.63, Range 20 – 46 for <i>n</i> = 16 <i>Control</i> group participants; M = 30.68, Range 20 – 50 for <i>n</i> = 16 <i>Alcohol</i> group participants	Alcohol, Placebo Control	Headway: <i>Time Headway</i> Lat. Control: <i>Lane Crossings</i> Long. Control: <i>Speed Variability</i> Crashes: <i>Accidents</i>
Hartley et al., 2019	Simulator	30 (0 F)	M = 21.50 (3.26), Range 20 – 34.	Cannabis, Placebo	Lat. Control: <i>SDLP</i>
Hartman et al., 2016	Simulator	18 (5 F)	M = 26.3 (4.2), Range 21 - 37	Cannabis, Alcohol, Combination, Placebo Control	Speed: <i>Mean Speed Relative to the Speed Limit, Percent Speed High</i> Long. Control: <i>SD Speed</i> Headway: <i>Mean Following Distance</i>
Laurell & Tornros, 1991	Simulator	24 (Unclear F)	Range 20 – 32.	Alcohol, Untreated Control	Speed: <i>Average Speed</i> Crashes: <i>Crashes</i>
Lenne et al., 2010	Simulator	33 (Unclear F)	Unclear. Original 47 participants included <i>n</i> = 22, Range 18 – 21; and, <i>n</i> = 25 Range 25 – 40.	Cannabis, Alcohol, Combination, Placebo Control	Lat. Control: <i>SD Lateral Position</i> Speed: <i>Mean Speed</i> Long. Control: <i>SD Speed, SD Headway</i> Headway: <i>Mean Headway</i>
Leung & Starmer, 2005	Simulator	32 (14 F)	M = 20 (0.9), Range 18 – 21 for <i>n</i> = 16 younger drivers; M = 28 (2.7), Range = 25 – 35 for <i>n</i> = 16 “mature” drivers	Alcohol, Placebo	Speed: <i>Mean Speed</i>

Study	Setting	Included N	M Age (SD)	Relevant IV's	Relevant DV's
Li et al., 2016	Simulator	52 (18 F)	M = 38.2, Range 21 – 61	Alcohol, Untreated Control	RT: <i>FB Module Braking RT</i> [Hazard] Lat. Control: <i>SDLP</i> Long. Control: <i>SD of Speed</i>
Liu & Fu, 2007	Simulator	8 (2 F)	Range 20 – 24 for $n = 4$, Range 25 – 30 for $n = 4$.	Alcohol, Untreated Control	Lat. Control: <i>Variance in Lateral Lane Position</i> Speed: <i>Mean Speed</i> Long. Control: <i>Speed Variance</i> Crashes: <i>Number of Accidents</i>
Liu & Ho, 2010 ⁴	Simulator	8 (2 F)	M = 24.125 (1.88), Range 22 – 27	Alcohol, Placebo	Long. Control: <i>Variance of Longitudinal Speed</i>
Martin, 1971	Simulator	12 (0 F)	Median = 25, Range 22 – 28.	Alcohol, Placebo	Lat. Control: <i>Time Off Target</i>
Micallef et al., 2018	Simulator, On-Road	15 (0 F), Simulator; 11 (0 F), On-Road	Unclear. For original $N = 20$, Range 25 – 35.	Cannabis, Placebo	Lat. Control: <i>SDLP, Inappropriate Line Crossings</i>
Mortimer & Sturgis, 1979	On-Road	40 (17 F)	Median = 30, Range 19 – 56	Alcohol, Placebo	Lat. Control: <i>Lateral Path Error Variance</i> Speed: <i>Speed</i> Long. Control: <i>Headway Variance, Speed Maintenance</i> Headway: <i>Mean Headway</i>
Mortimer & Howat, 1986	Closed Course	14 (7 F)	Range 21 – 32	Alcohol, Placebo	Long. Control: <i>Absolute Mean Error In Speed, Speed Maintenance</i>

Study	Setting	Included N	M Age (SD)	Relevant IV's	Relevant DV's
Moskowitz et al., 1976	Simulator	23 (0 F)	Unclear. For original $N = 24$, $M = 24$, Range 21 - 32	Cannabis, Placebo	<p>Speed: <i>Average Speed During the Event (MPH), Speed at the Beginning of the Event (MPH), Speed at the End of the Event (MPH)</i></p> <p>Speed Var: <i>Within Subject SD of Average Speed During the Event (MPH), Within Subject SD of Speed at the Beginning of the Event (MPH), Within Subject SD of Speed at the End of the Event (MPH)</i></p>
Moskowitz et al., 2000	Simulator	168 (84 F)	M = 34 years, 11 months for $n = 84$ males; M = 33 years, 2 months for $n = 84$ females; participants divided into “youthful drivers” (age 19-20; M = 19 years, 8 months), “young adult drivers” (age 21-24; M = 22 years, 5 months), “adult drivers” (age 25-50; M = 32 years, 8 months), “older drivers” (age 51-69; M = 61 years, 7 months), each with 21 males and 21 females per group	Alcohol, Placebo	<p>Lat. Control: <i>Lane Deviation Variability</i></p> <p>Speed: <i>Times Over Speed Limit</i></p> <p>Long. Control: <i>Speed Variability</i></p> <p>Crashes: <i>Collisions</i></p>

Study	Setting	Included N	M Age (SD)	Relevant IV's	Relevant DV's
Quillian et al., 1999	Simulator	28 (0 F)	For $n = 14$ middle-aged group, $M = 36.2$ (5.8), Range 30 – 50. For $n = 14$ older group, $M = 69.4$ (5.2), Range 60 – 77.	Alcohol, Non-Alcohol (unclear if placebo or untreated control)	Lat. Control: <i>Off Road Speed</i> Speed: <i>High Speed, SD Speed</i> Crashes: <i>Crashes, Bump Collisions</i>
Rafaelsen et al., 1973a	Simulator	8 (0 F)	Range 21 - 29	Cannabis, Alcohol, Placebo	Speed: <i>Mean Speed</i>
Rafaelsen et al., 1973b ³	Simulator	8 (0 F)	Range 21 – 29	Cannabis, Alcohol, Placebo	Speed: <i>Mean Speed</i> Long. Control: <i>Variation of Speed</i>
Rakauskas et al., 2005	Simulator	48 (0 F)	$M = 22.3$, Range 21 – 29.	Alcohol, Placebo	RT: <i>Response Time to Pullout Events [Hazard]</i> Speed: <i>Speed at Curve Apex</i> Headway: <i>Median Time Headway</i> Lat. Control: <i>Lane Position Variability</i> Long. Control: <i>Time Headway Variability</i> Collisions: <i>Number of Collisions</i>
Ranney & Gawron, 1984 (Study 1)	On-Road	6 (0 F)	Range 21 – 55.	Alcohol, Placebo	Lat. Control: <i>SD of Lateral Position, Lane Deviation Frequency, Time Off Road</i> Speed: <i>Mean Velocity</i> Long. Control: <i>SD of Velocity</i> Collisions: <i>Accidents</i>
Ranney & Gawron, 1984 (Study 2)	Simulator	12 (0 F)	Range 21 – 55.	Alcohol, Placebo	Speed: <i>Speed, Speed Exceedances</i> Collisions: <i>Obstacles Struck</i>

Study	Setting	Included N	M Age (SD)	Relevant IV's	Relevant DV's
Roehrs et al., 1994	Simulator	12 (0 F)	Range 21 – 35	Alcohol, Placebo	Crashes: <i>Crashes</i>
Smiley et al., 1985	Simulator	35 (0 F)	Unclear. Inclusion criterion included age range 21 – 45.	Cannabis, Alcohol, Combination, Placebo Control	Lat. Control: <i>Lane Position Variability</i> Long. Control: <i>Speed Variability, Headway Variability</i> Crashes: <i>Number of Crashes</i>
Smiley et al., 1987	On-Road	52 (0 F)	Range 21 – 30.	Alcohol, Cannabis, Combination, Placebo	Speed: <i>Speed</i> Headway: <i>Headway</i> Long. Control: <i>SD of Velocity, Headway Variability</i>
Spaanjaars et al., 2011	Simulator	74 (74 F)	M = 21.85 (1.54), Range 19 – 25	Alcohol, Placebo	Lat. Control: <i>SD of Lateral Position</i> Speed: <i>Average Speed</i>
Stein, 1985	Simulator	12 (0 F)	For original $N = 13$, Range 21 – 65.	Cannabis, Alcohol, Combination, Placebo	Speed: <i>Mean Speed</i> Lat. Control: <i>Lane Position Variability</i> Long. Control: <i>Speed Variance</i> Crashes: <i>Crashes</i>
Sutton, 1983	Closed Course	9 (0 F)	M = 25.1	Cannabis, Alcohol, Combination, Placebo	Lat. Control: <i>Weaving Over Yellow Center Line, Leaving the Driving Course</i>
Vakulin et al., 2009	Simulator	20 (5 F)	M = 50.6 (10.1)	Alcohol, Placebo	RT: <i>Braking RT [Hazard]</i> Crashes: <i>Crash Frequency</i>
Wu et al., 2011	Simulator	13 (9 F)	Unclear. For original $N = 15$ group, Range 20 – 25.	Alcohol, Untreated Control	Speed: <i>High Velocity Time</i>

1. This study initially enrolled 56 (19 F) adults with ADHD and 46 (19 F) community controls. Only the final sample of 39 “community control” participants were eligible for inclusion in the meta-analysis.
2. Two sleep deprivation groups excluded. Only participants from non-sleep-deprived groups (i.e., *Control*, *Alcohol*) were eligible for inclusion in the meta-analysis.
3. Suspected duplicate of Rafaelsen et al., 1973a.
4. Suspected duplicate of Liu & Fu, 2007.

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Appendix C: Forest Plots (Primary Meta-Analyses)

Cannabis v. Baseline: Crashes

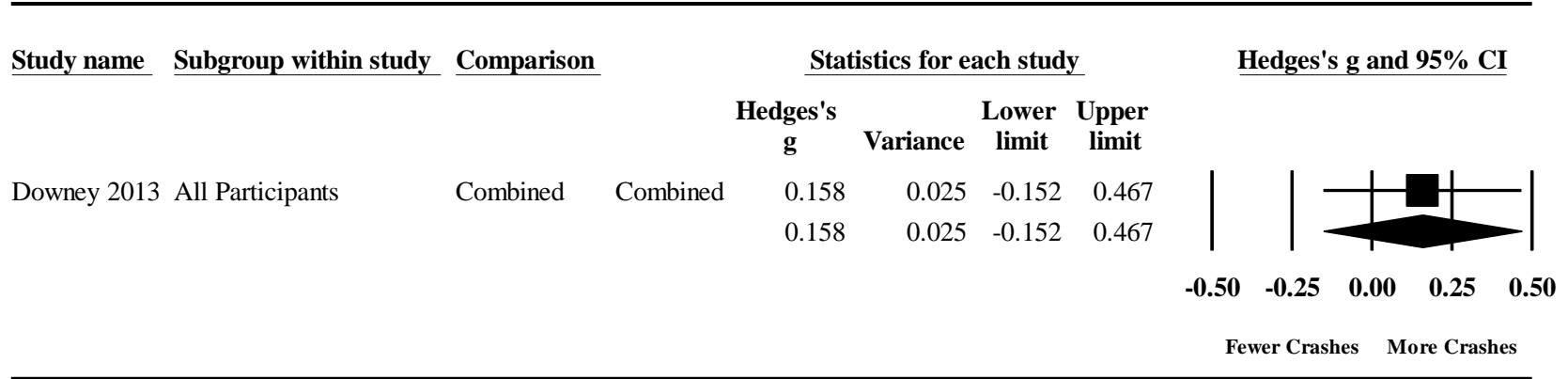


Figure C1. Forest plot illustrating *Cannabis v. Baseline: Crashes* (missing pre-post correlations set to $r = \text{zero}$).

Cannabis v. Baseline: Crashes

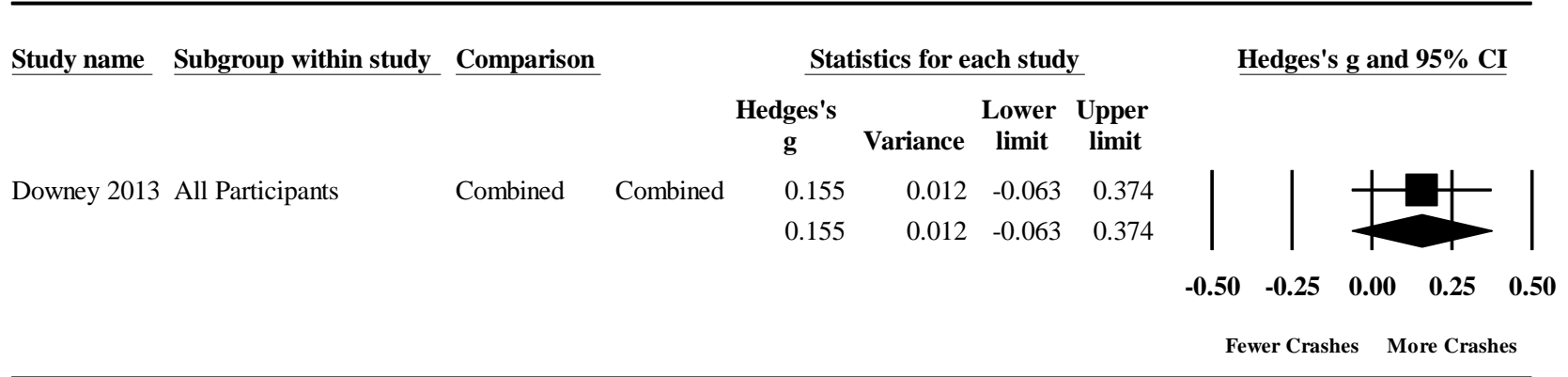


Figure C2. Forest plot illustrating *Cannabis v. Baseline: Crashes* (missing pre-post correlations set to $r = 0.5$).

Cannabis v. Baseline: Crashes

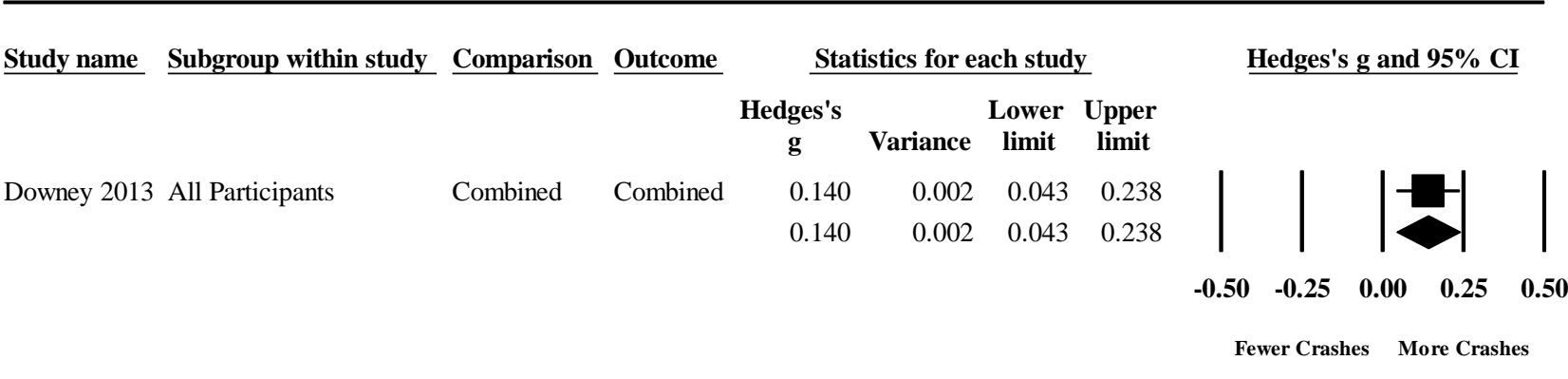


Figure C3. Forest plot illustrating Cannabis v. Baseline: Crashes (missing pre-post correlations set to $r = 0.9$).

Cannabis v. Baseline: Hazard RT

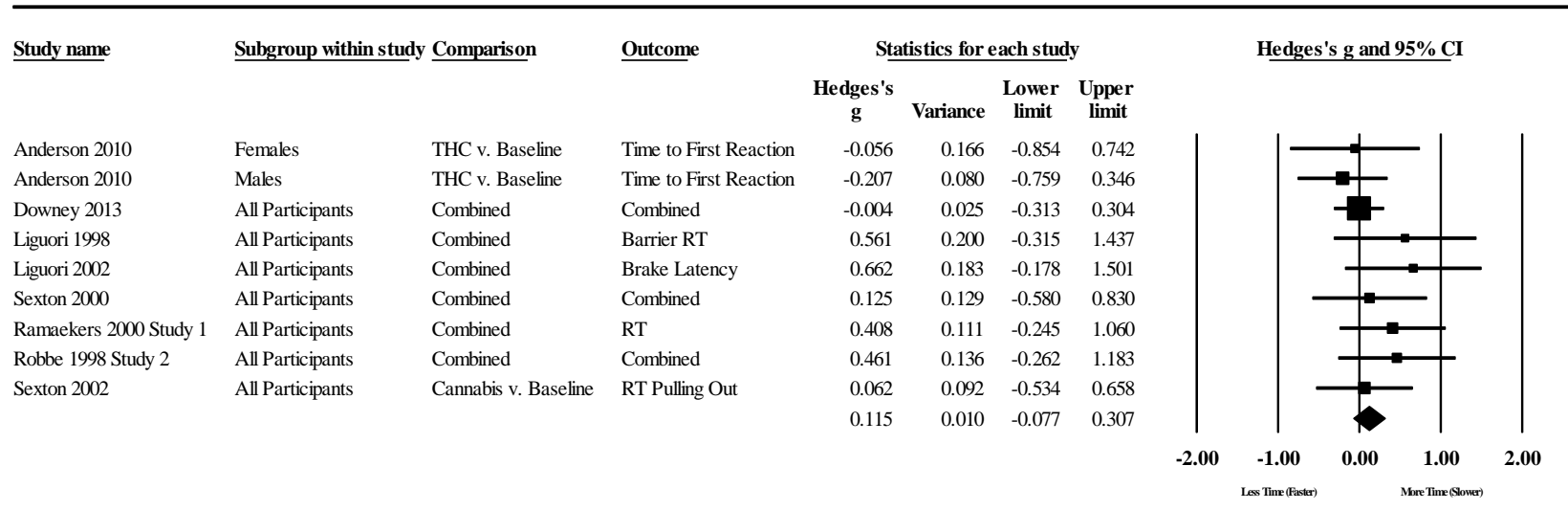


Figure C4. Forest plot illustrating Cannabis v. Baseline: Hazard RT (missing pre-post correlations set to $r = 0$).

Cannabis v. Baseline: Hazard RT

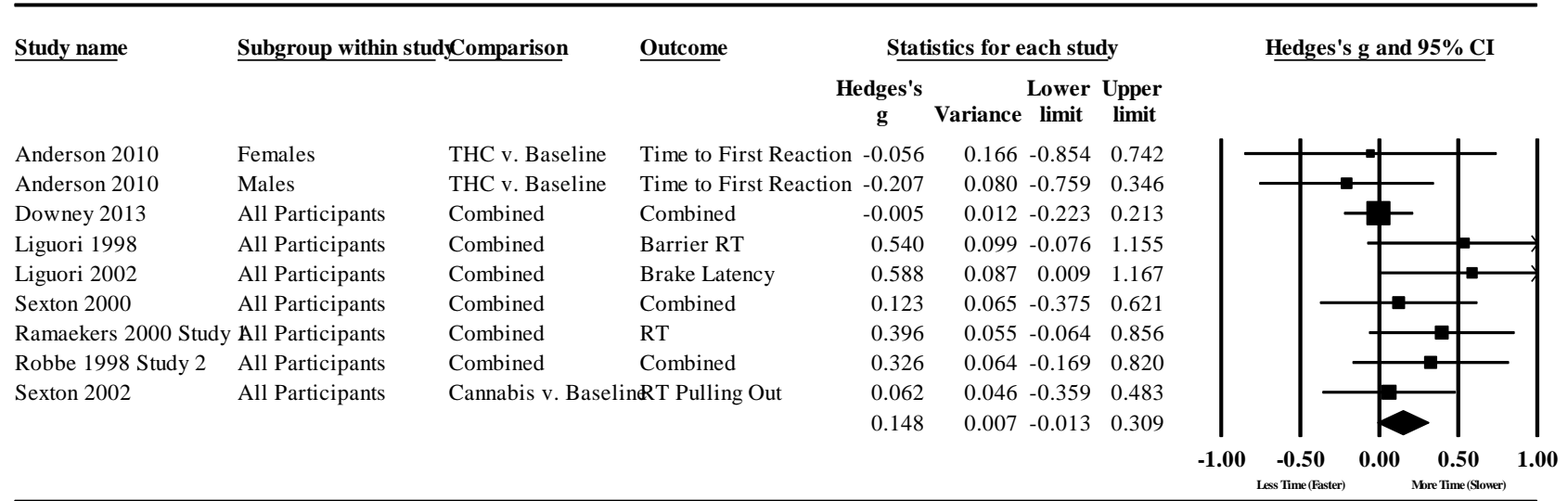


Figure C5. Forest plot illustrating *Cannabis v. Baseline: Hazard RT* (missing pre-post correlations set to $r = 0.5$).

Cannabis v. Baseline: Hazard RT

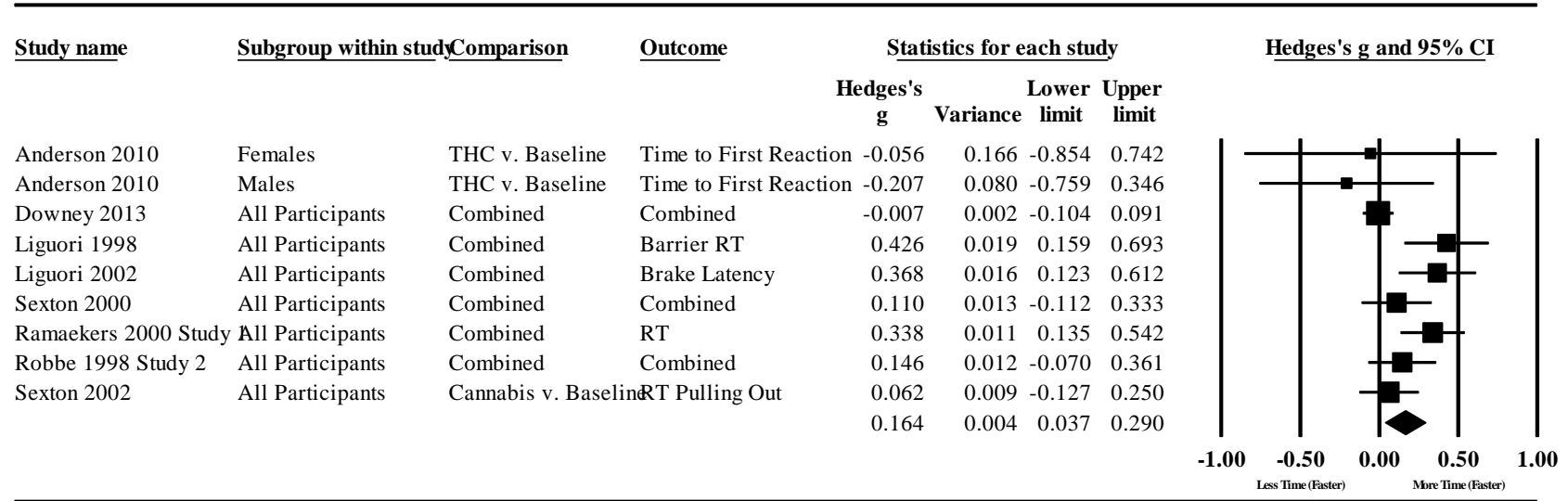


Figure C6. Forest plot illustrating Cannabis v. Baseline: Hazard RT (missing pre-post correlations set to $r = 0.9$).

Cannabis v. Baseline: Headway

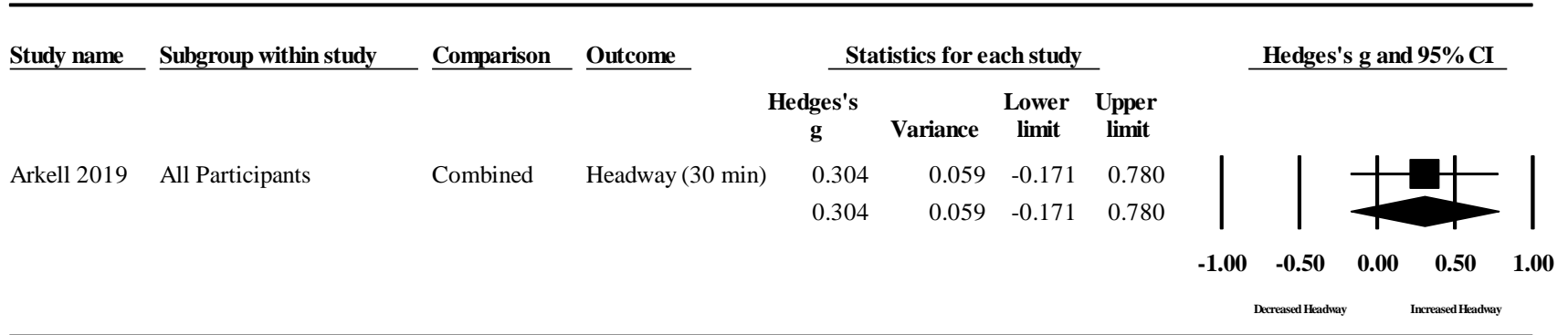


Figure C7. Forest plot illustrating Cannabis v. Baseline: Headway.

Cannabis v. Baseline: Headway Variability

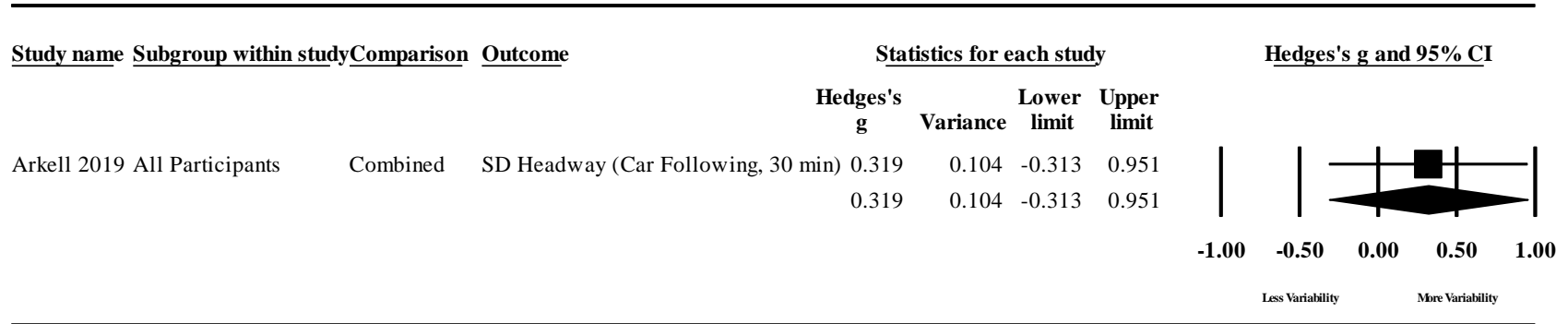


Figure C8. Forest plot illustrating Cannabis v. Baseline: Headway Variability.

Cannabis v. Baseline: Lateral Position Variability

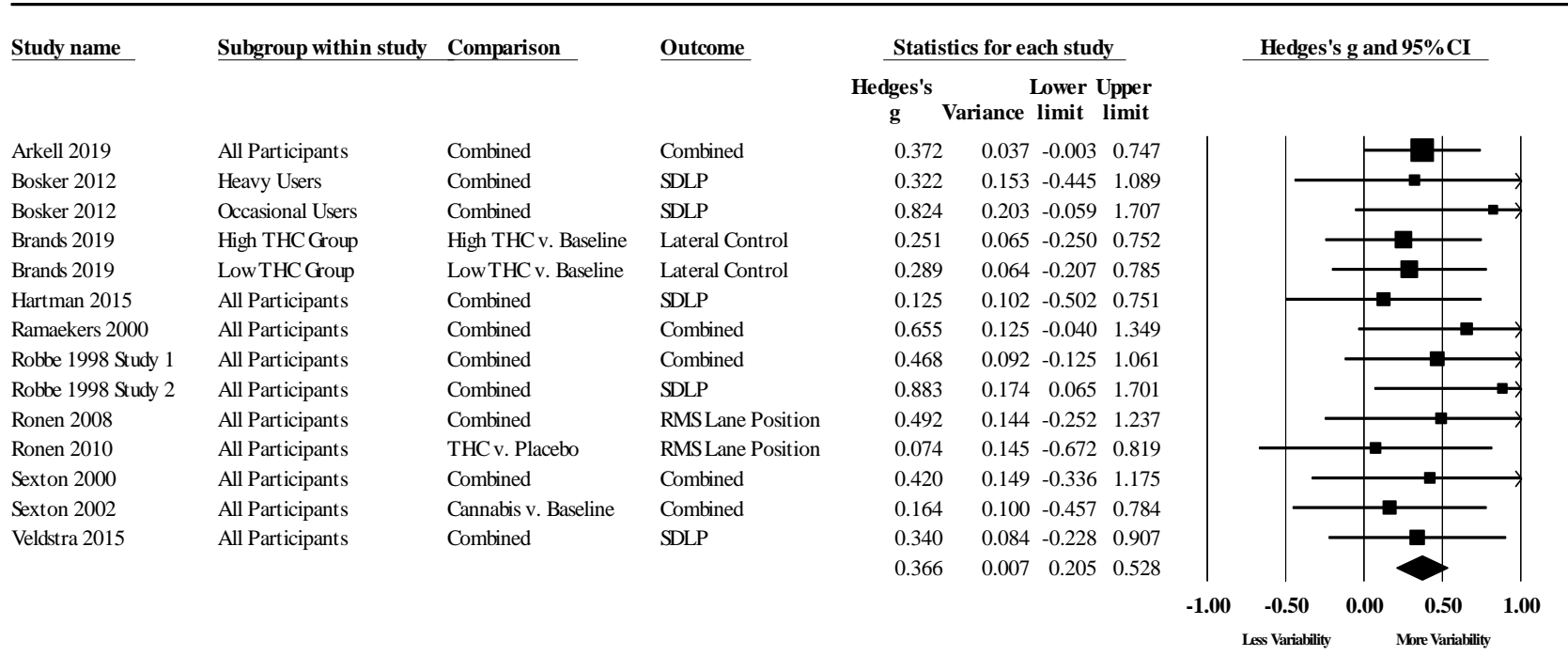


Figure C9. Forest plot illustrating Cannabis v. Baseline: Lateral Position Variability (missing pre-post correlations set to $r = \text{zero}$).

Cannabis v. Baseline: Lateral Position Variability

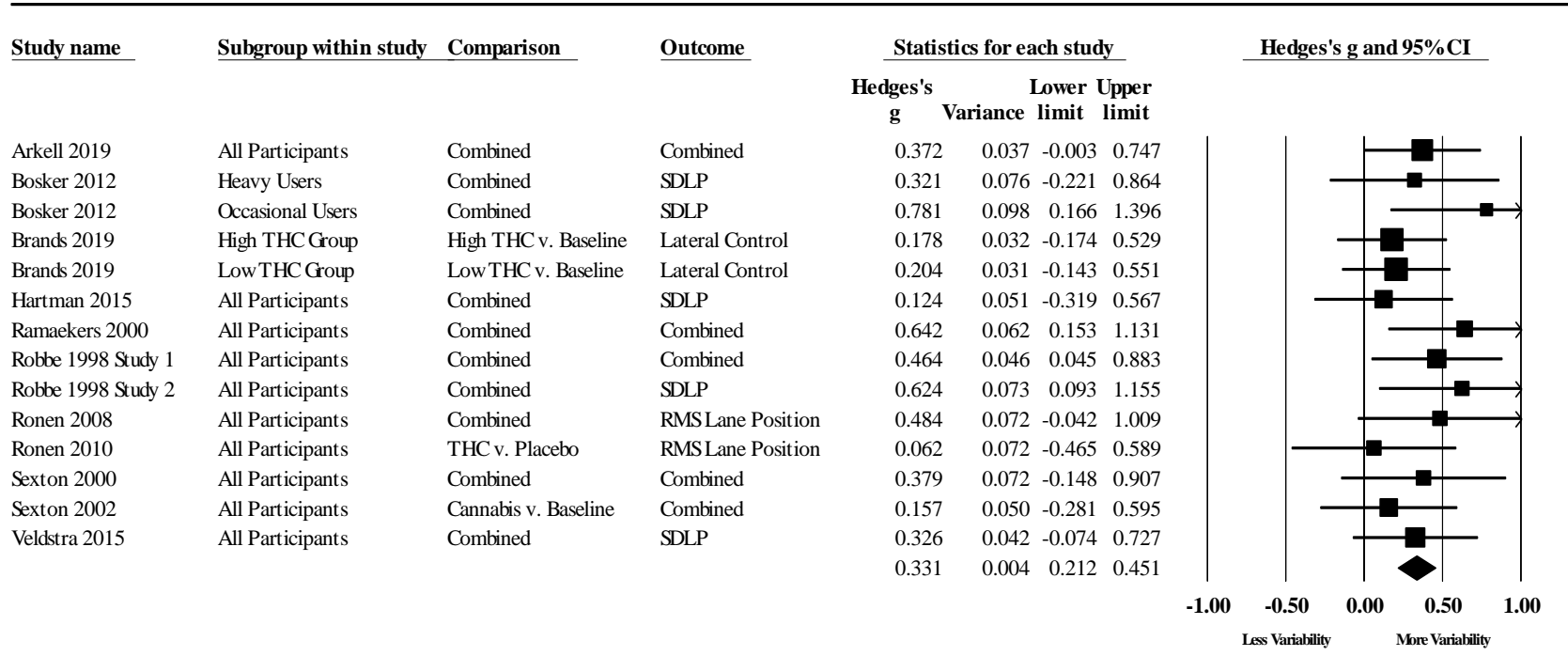


Figure C10. Forest plot illustrating Cannabis v. Baseline: Lateral Position Variability (missing pre-post correlations set to $r = 0.5$).

Cannabis v. Baseline: Lateral Position Variability

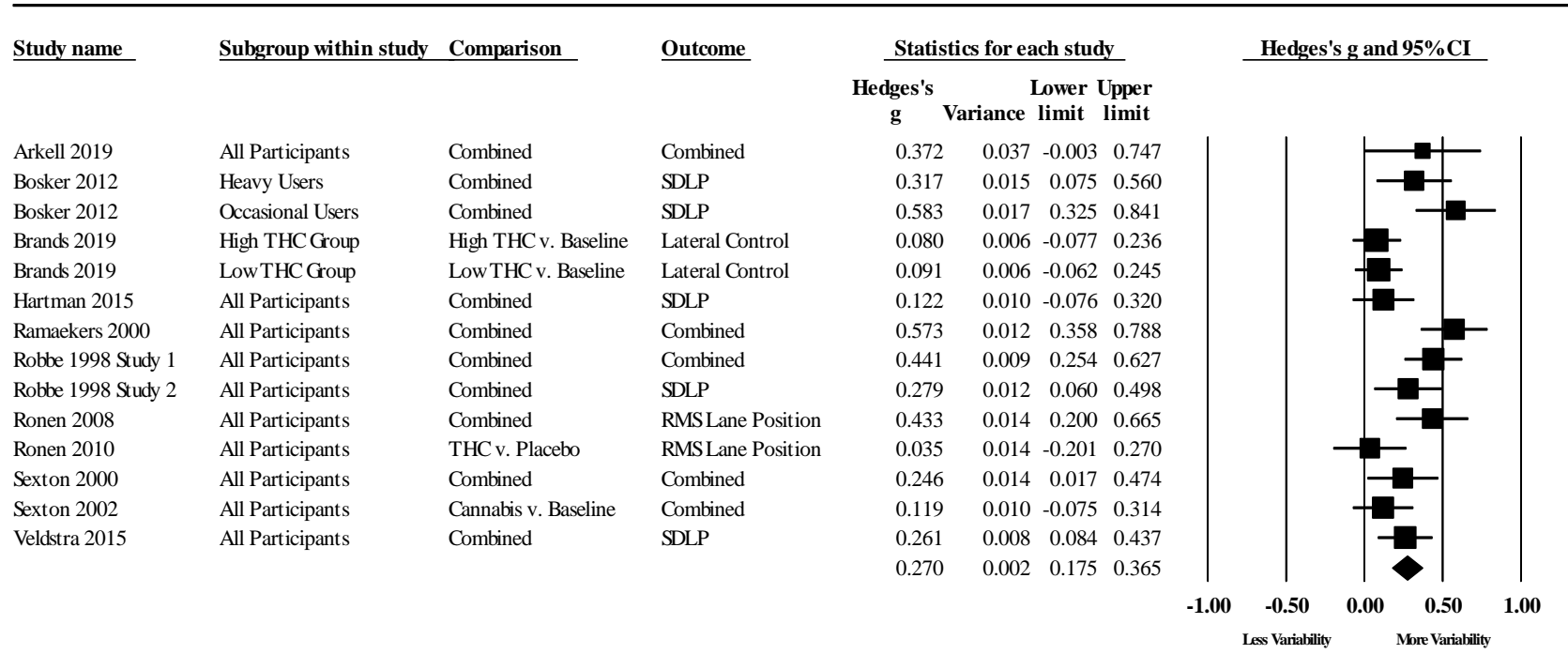


Figure C11. Forest plot illustrating *Cannabis v. Baseline: Lateral Position Variability* (missing pre-post correlations set to $r = 0.9$).

Cannabis v. Baseline: Lane Excursions

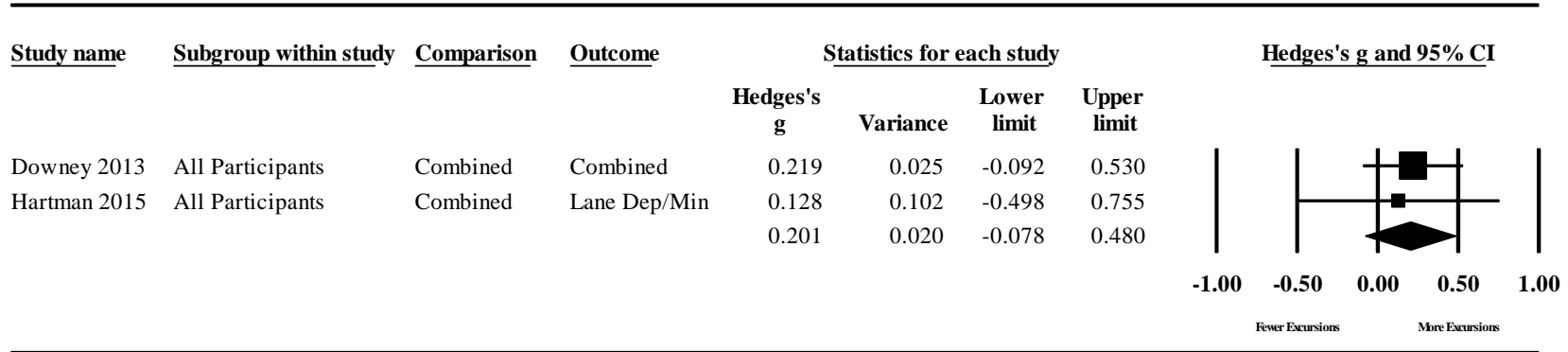


Figure C12. Forest plot illustrating Cannabis v. Baseline: Lane Excursions (missing pre-post correlations set to $r = \text{zero}$).

Cannabis v. Baseline: Lane Excursions

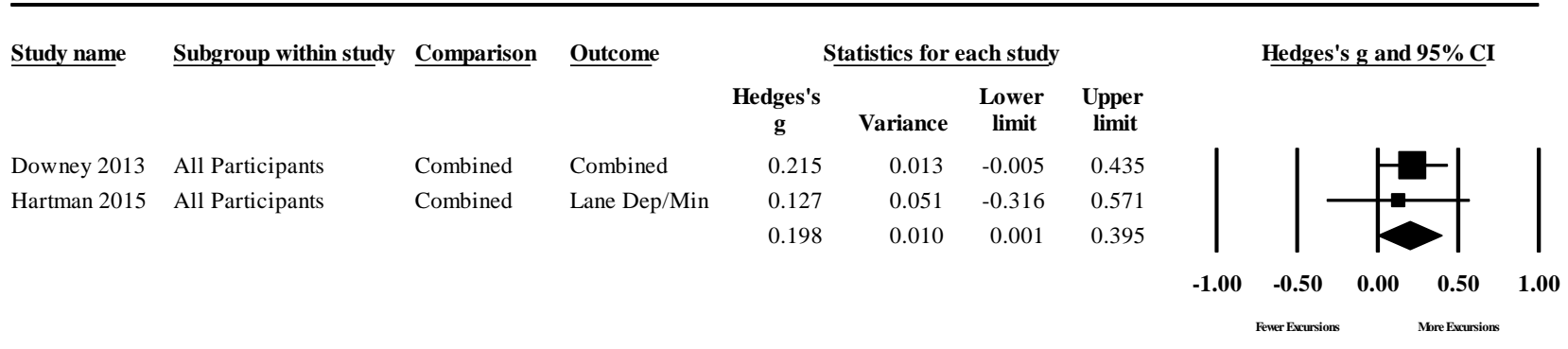


Figure C13. Forest plot illustrating Cannabis v. Baseline: Lane Excursions (missing pre-post correlations set to $r = 0.5$).

Cannabis v. Baseline: Lane Excursions

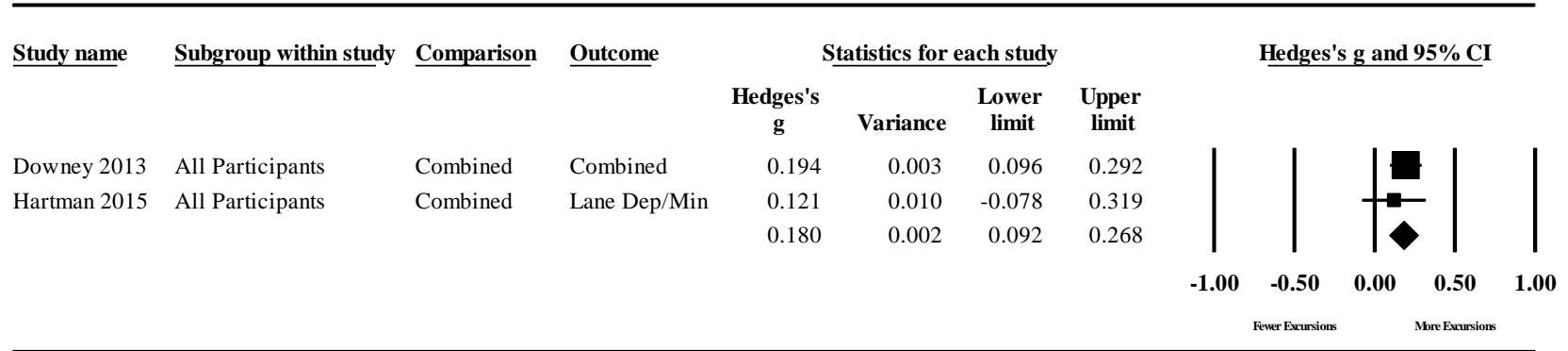


Figure C14. Forest plot illustrating Cannabis v. Baseline: Lane Excursions (missing pre-post correlations set to $r = 0.9$).

Cannabis v. Baseline: Time Out of Lane

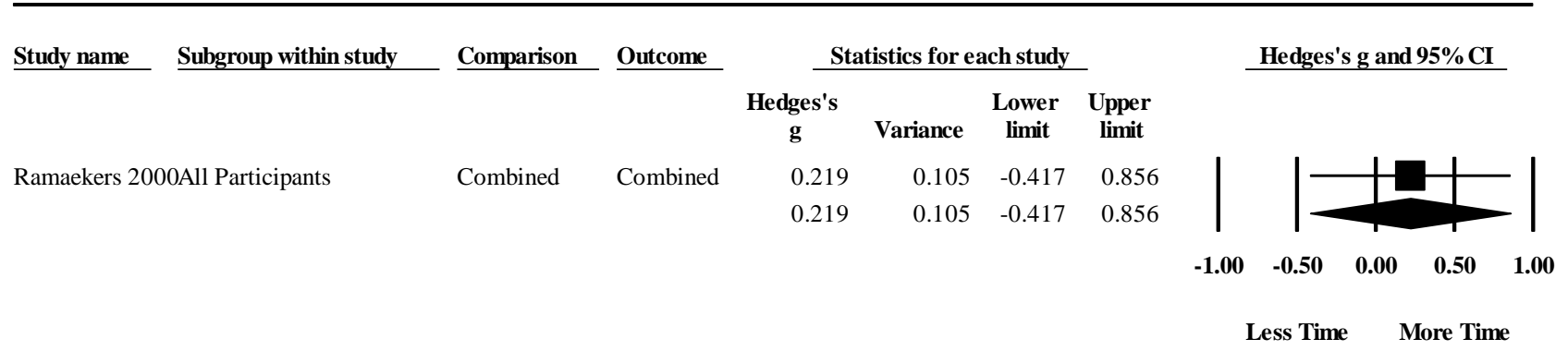


Figure C15. Forest plot illustrating Cannabis v. Baseline: Time Out of Lane (missing pre-post correlations set to $r = \text{zero}$).

Cannabis v. Baseline: Time Out of Lane

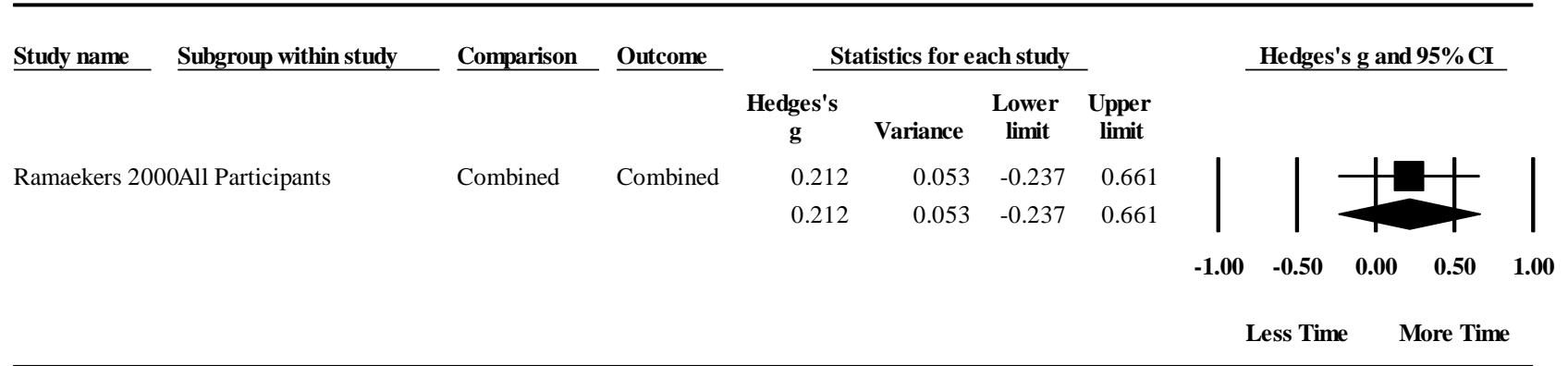


Figure C16. Forest plot illustrating Cannabis v. Baseline: Time Out of Lane (missing pre-post correlations set to $r = 0.5$).

Cannabis v. Baseline: Time Out of Lane

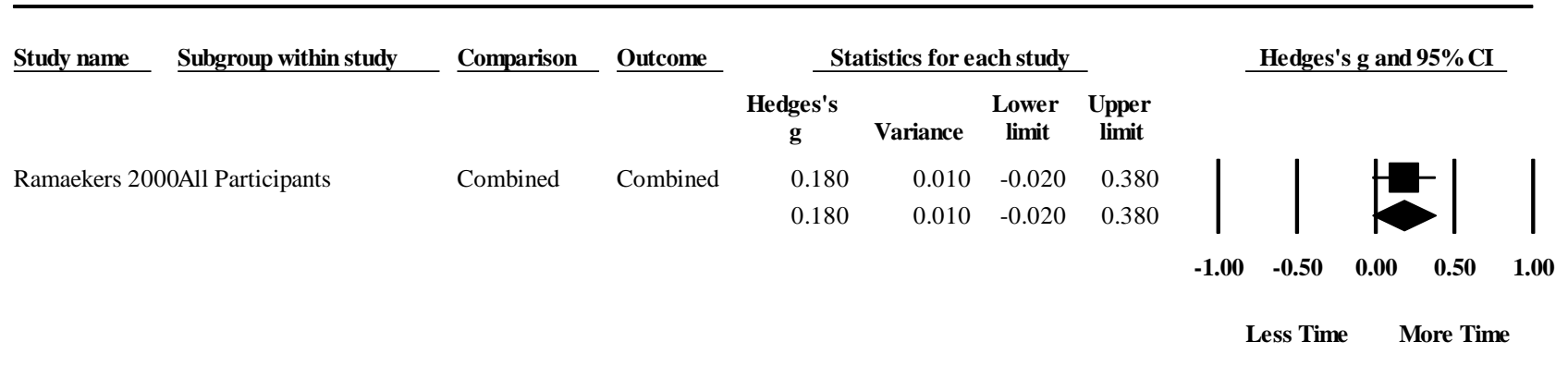


Figure C17. Forest plot illustrating Cannabis v. Baseline: Time Out of Lane (missing pre-post correlations set to $r = 0.9$).

Cannabis v. Baseline: Speed

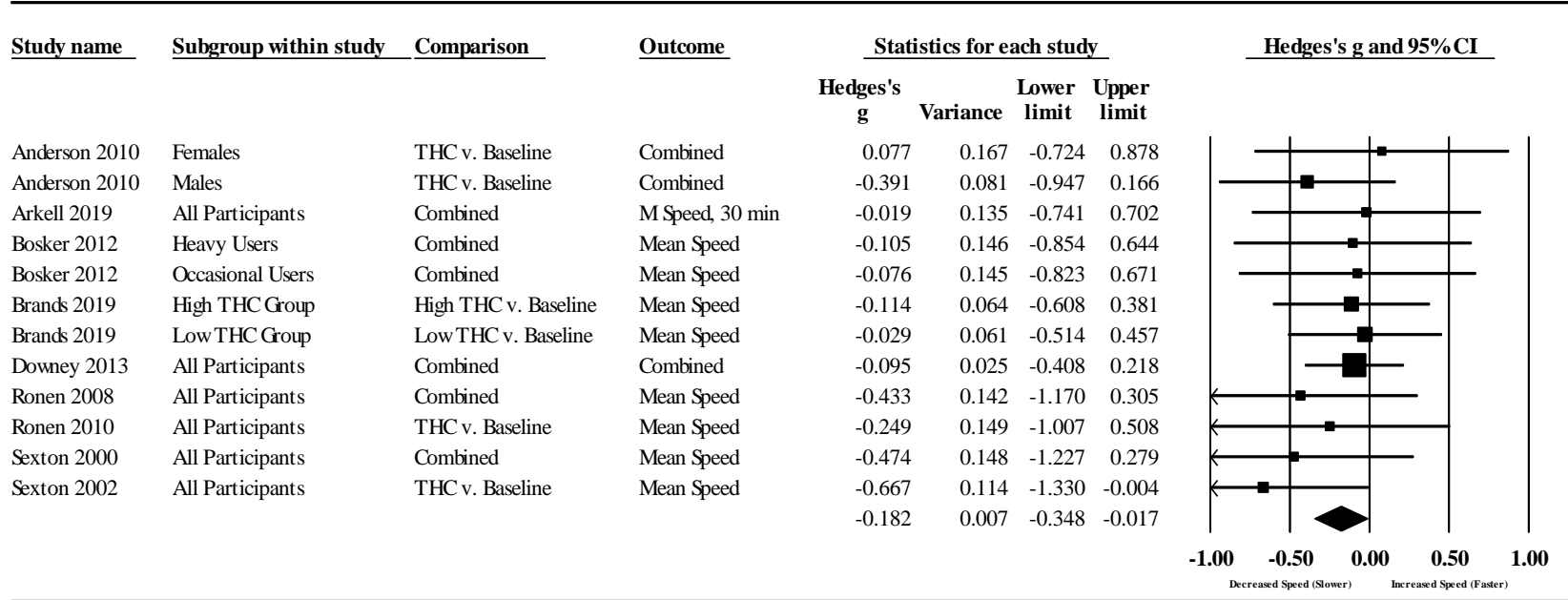


Figure C18. Forest plot illustrating Cannabis v. Baseline: Speed (missing pre-post correlations set to $r = \text{zero}$).

Cannabis v. Baseline: Speed

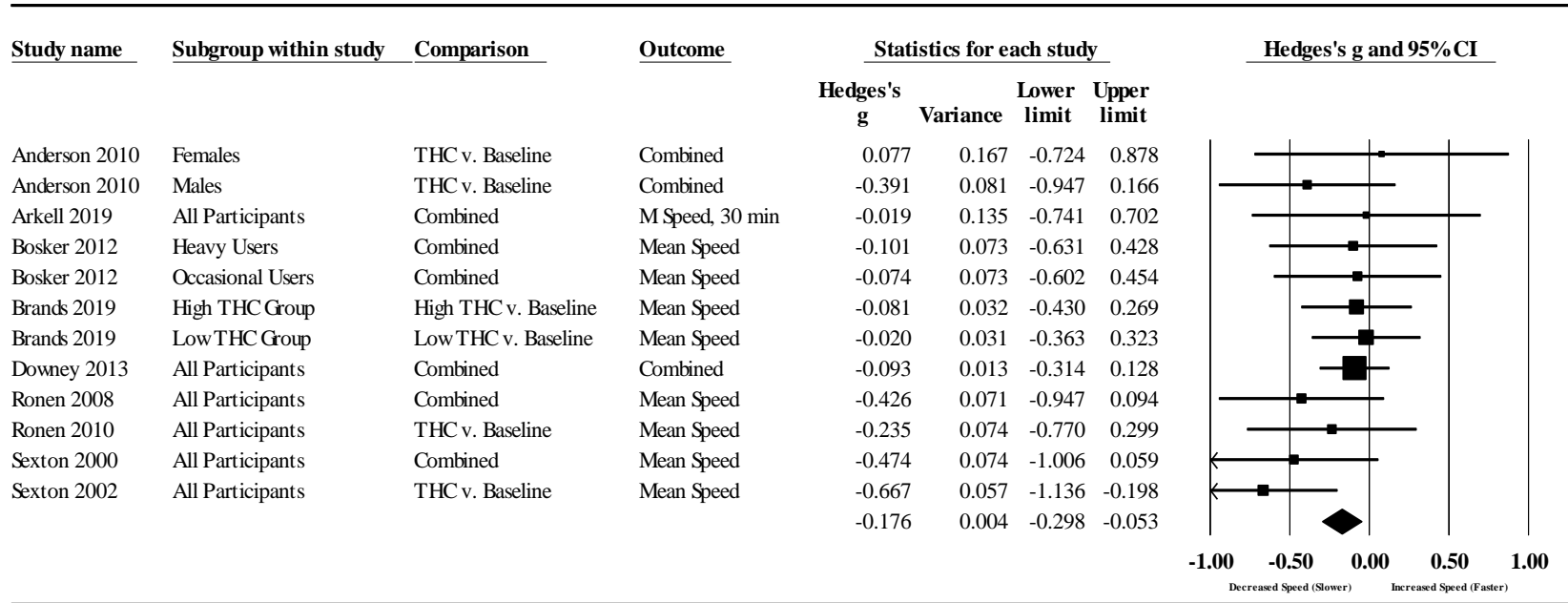


Figure C19. Forest plot illustrating Cannabis v. Baseline: Speed (missing pre-post correlations set to $r = 0.5$).

Cannabis v. Baseline: Speed

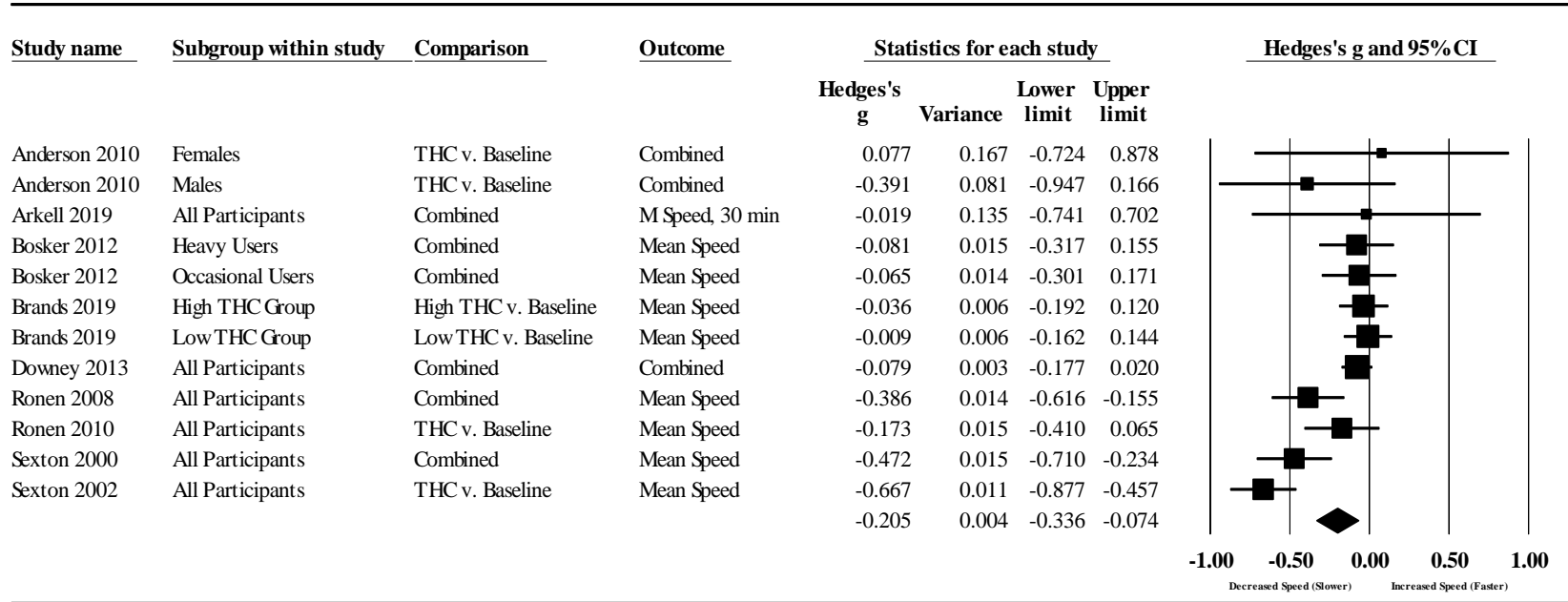


Figure C20. Forest plot illustrating Cannabis v. Baseline: Speed (missing pre-post correlations set to $r = 0.9$).

Cannabis v. Baseline: Speed Variability

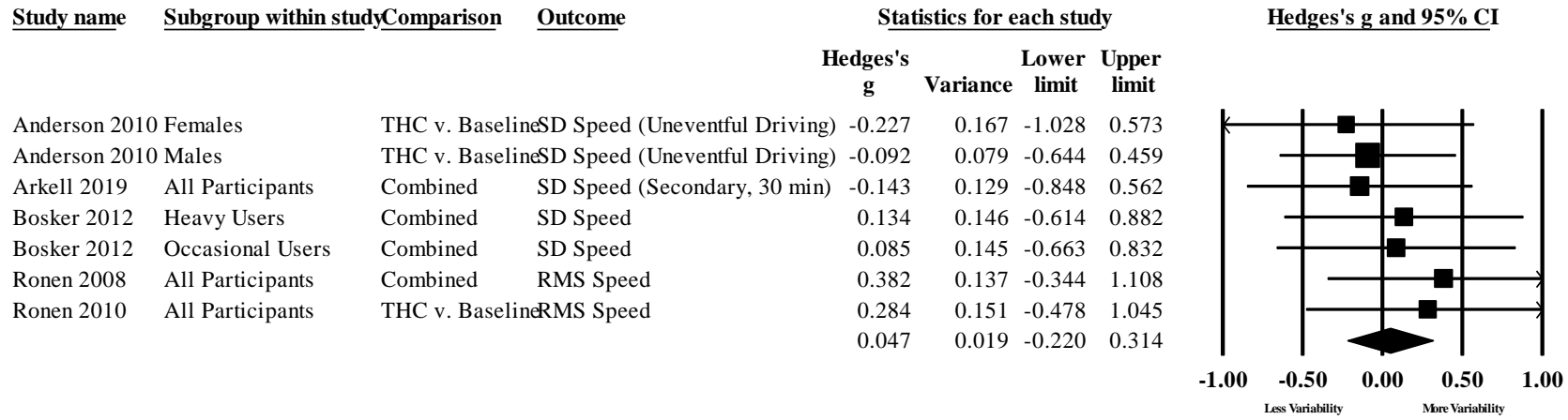


Figure C21. Forest plot illustrating Cannabis v. Baseline: Speed Variability (missing pre-post correlations set to $r = 0$).

Cannabis v. Baseline: Speed Variability

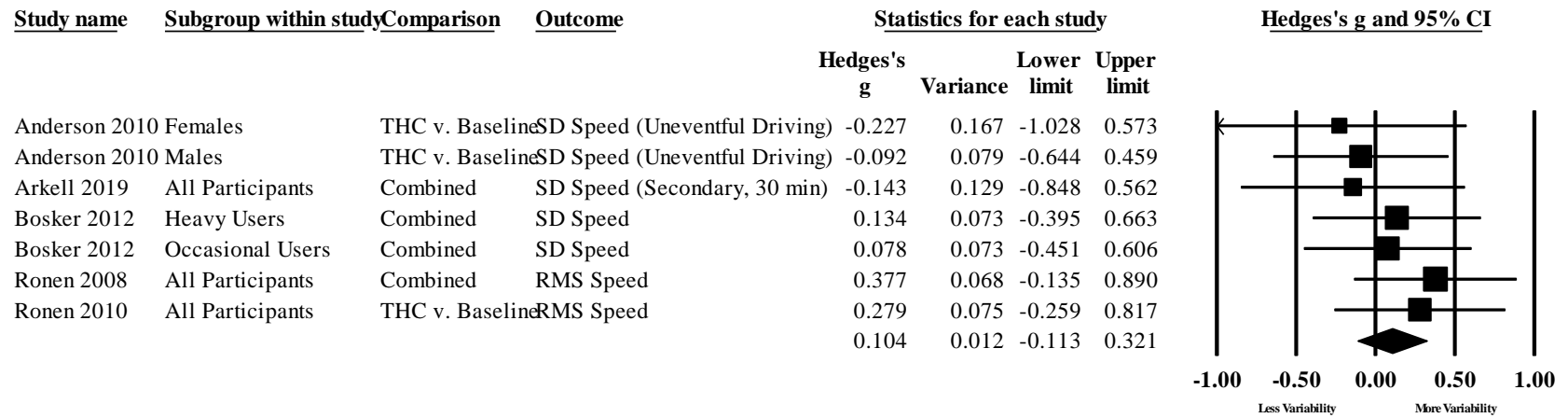


Figure C22. Forest plot illustrating *Cannabis v. Baseline: Speed Variability* (missing pre-post correlations set to $r = 0.5$).

Cannabis v. Baseline: Speed Variability

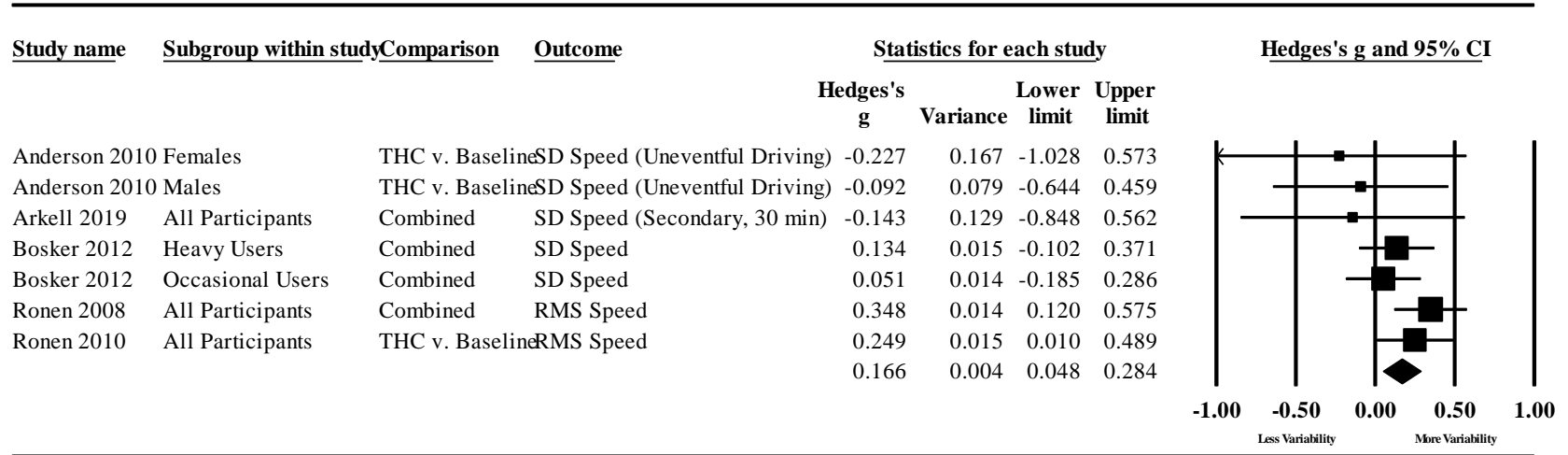


Figure C23. Forest plot illustrating Cannabis v. Baseline: Speed Variability (missing pre-post correlations set to $r = 0.9$).

Cannabis v. Baseline: Speed Exceedances

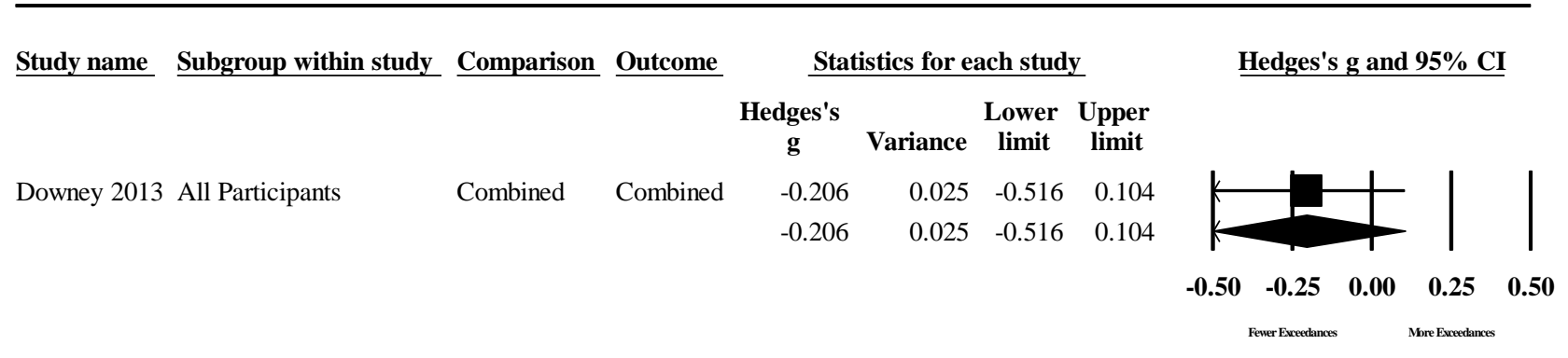


Figure C24. Forest plot illustrating Cannabis v. Baseline: Speed Exceedances (missing pre-post correlations set to $r = 0$).

Cannabis v. Baseline: Speed Exceedances

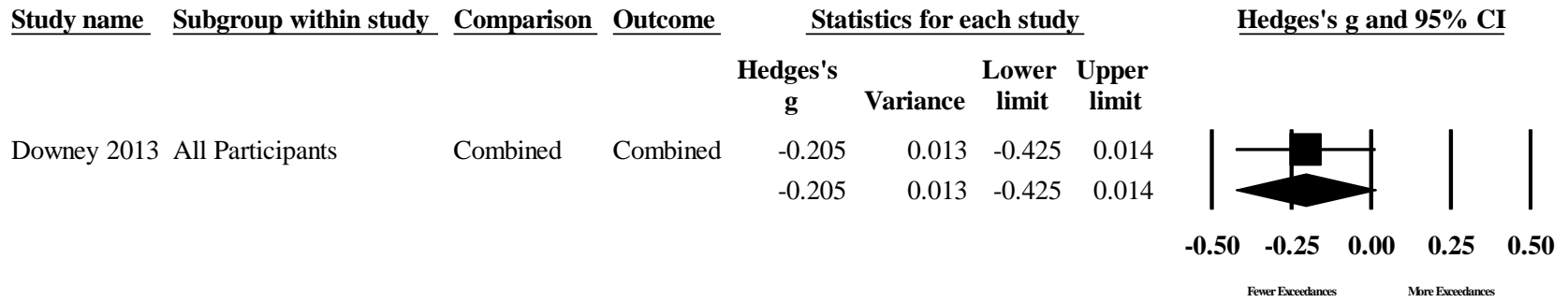


Figure C25. Forest plot illustrating *Cannabis v. Baseline: Speed Exceedances* (missing pre-post correlations set to $r = 0.5$).

Cannabis v. Baseline: Speed Exceedances

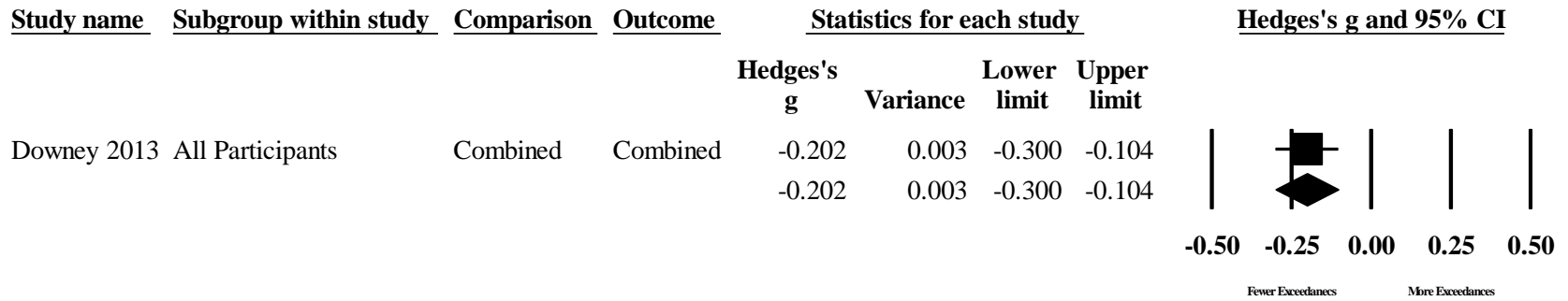


Figure C26. Forest plot illustrating *Cannabis v. Baseline: Speed Exceedances* (missing pre-post correlations set to $r = 0.9$).

Alcohol v. Baseline: Crashes

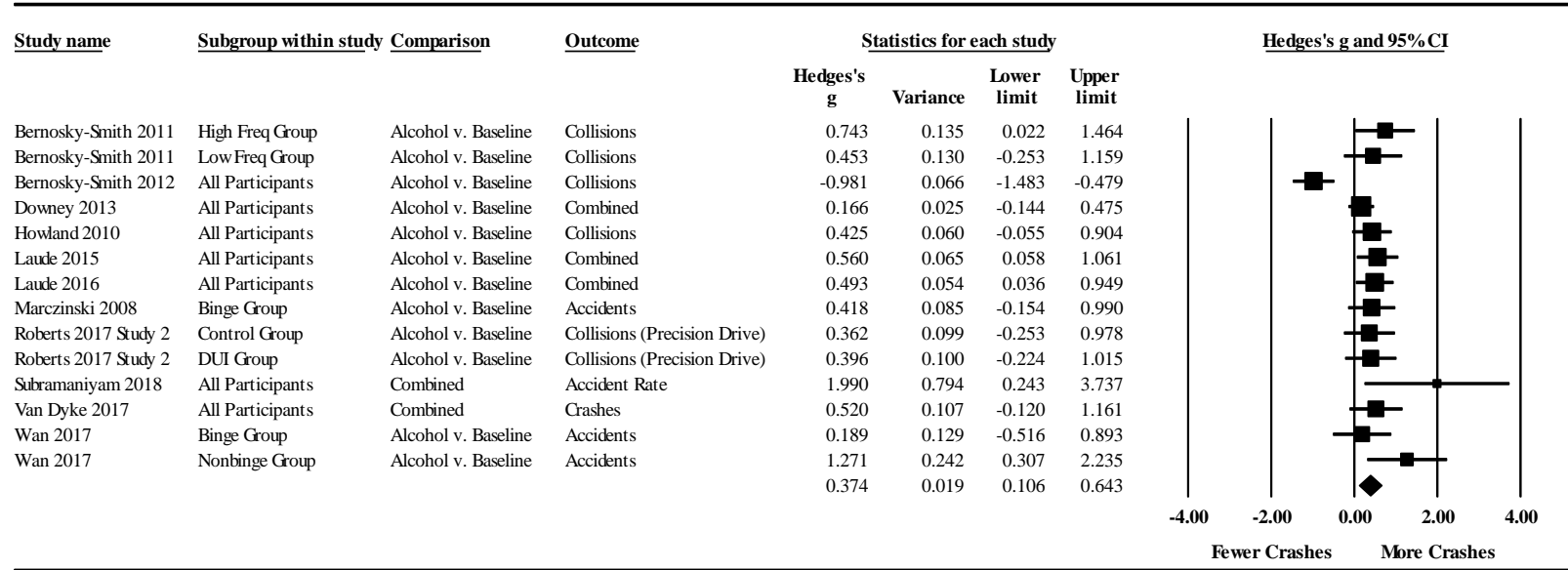


Figure C27. Forest plot illustrating Alcohol v. Baseline: Crashes (missing pre-post correlations set to $r = 0$).

Alcohol v. Baseline: Crashes

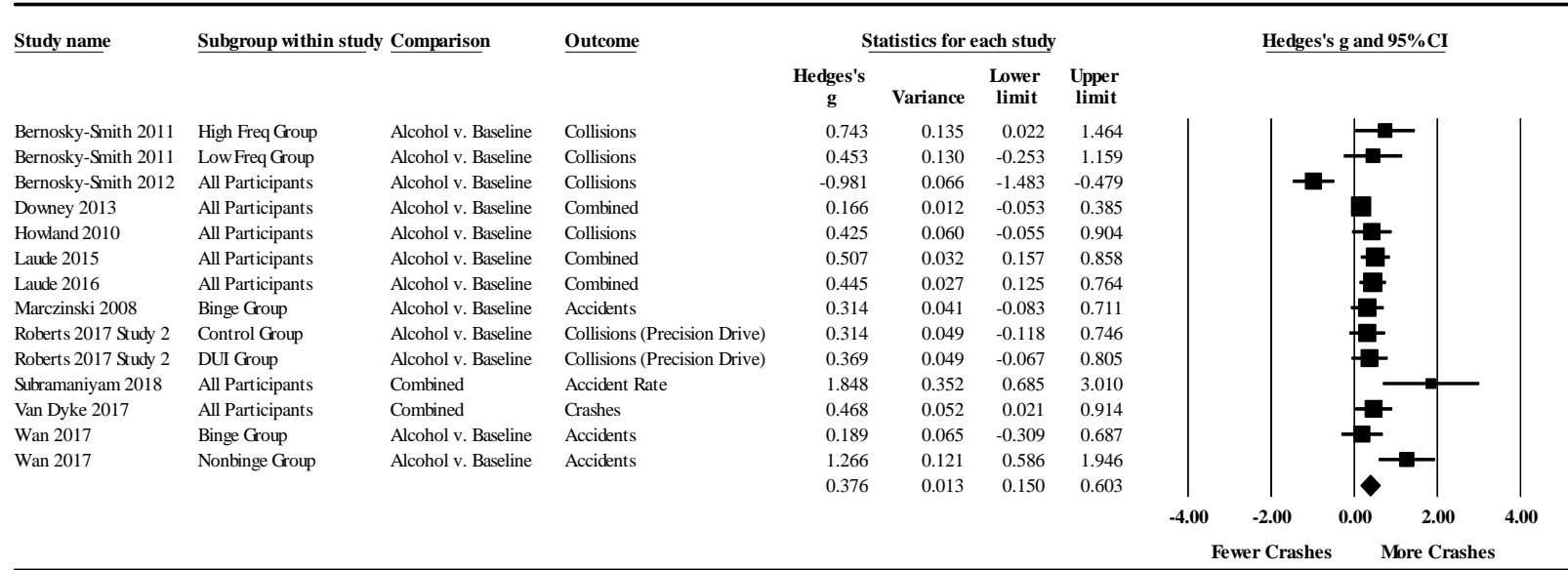


Figure C28. Forest plot illustrating Alcohol v. Baseline: Crashes (missing pre-post correlations set to $r = 0.5$).

Alcohol v. Baseline: Crashes

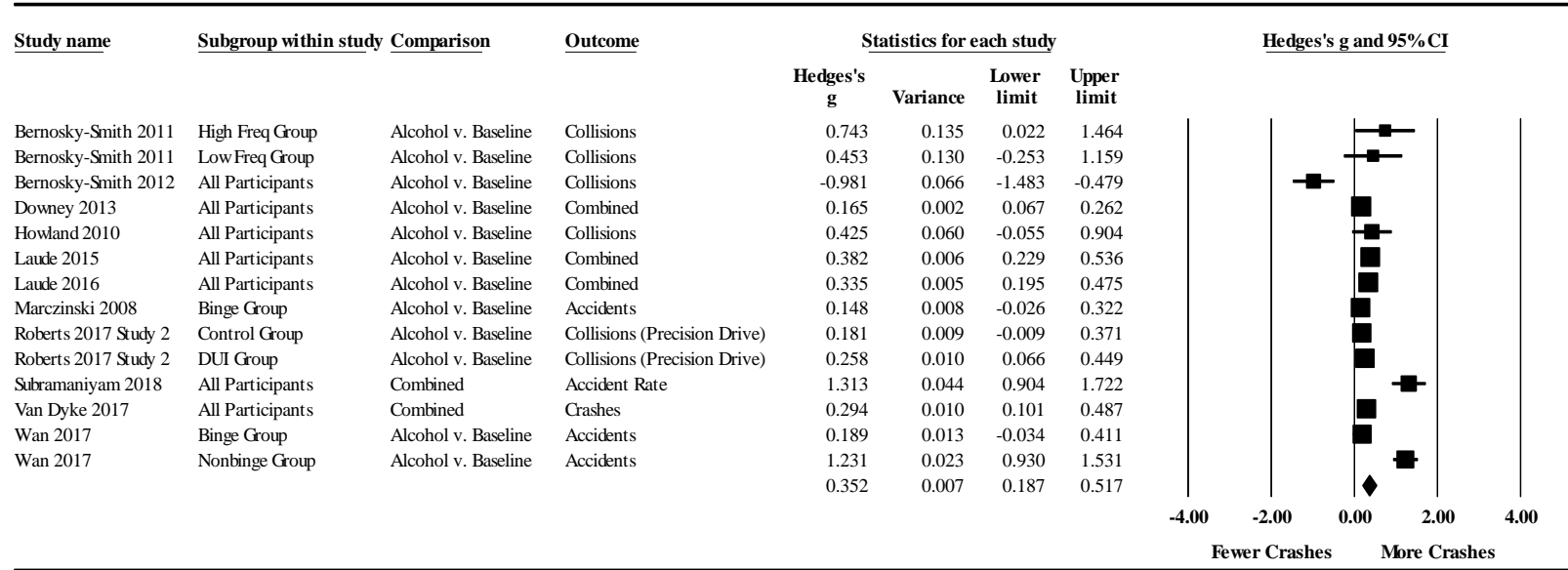


Figure C29. Forest plot illustrating Alcohol v. Baseline: Crashes (missing pre-post correlations set to $r = 0.9$).

Alcohol v. Baseline: Crashes

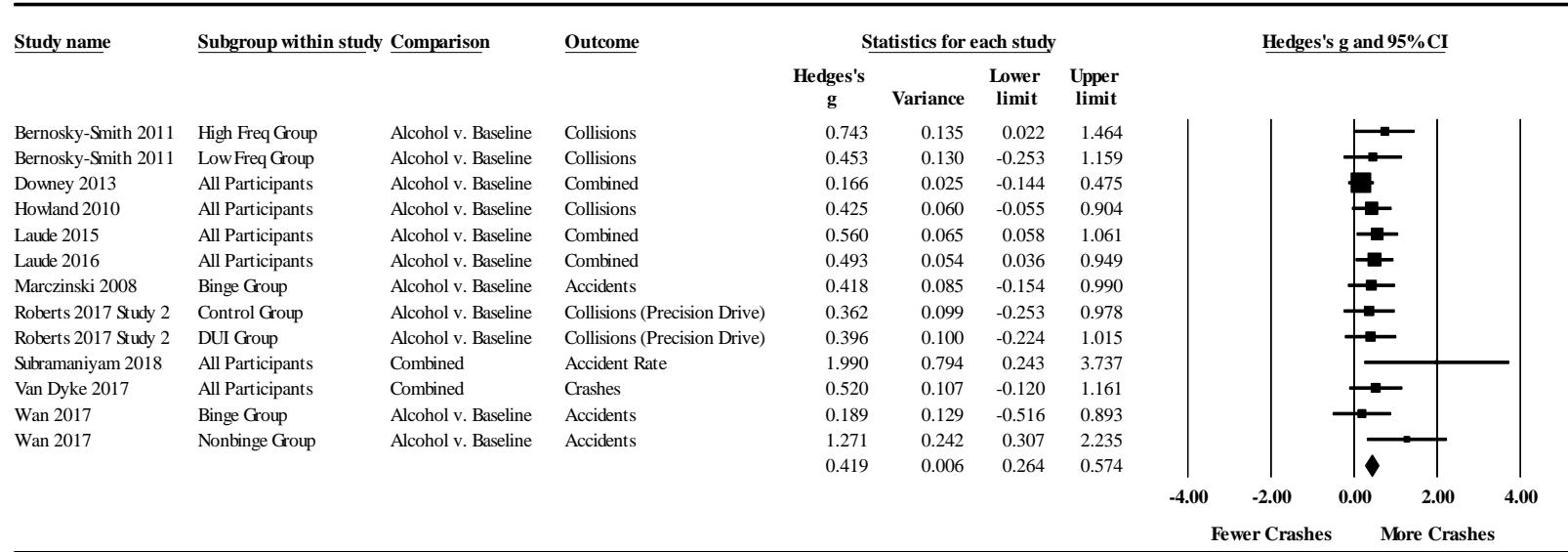


Figure C30. Forest plot illustrating Alcohol v. Baseline: Crashes (missing pre-post correlations set to $r =$ zero). Excludes Bernosky-Smith et al. (2012).

Alcohol v. Baseline: Crashes

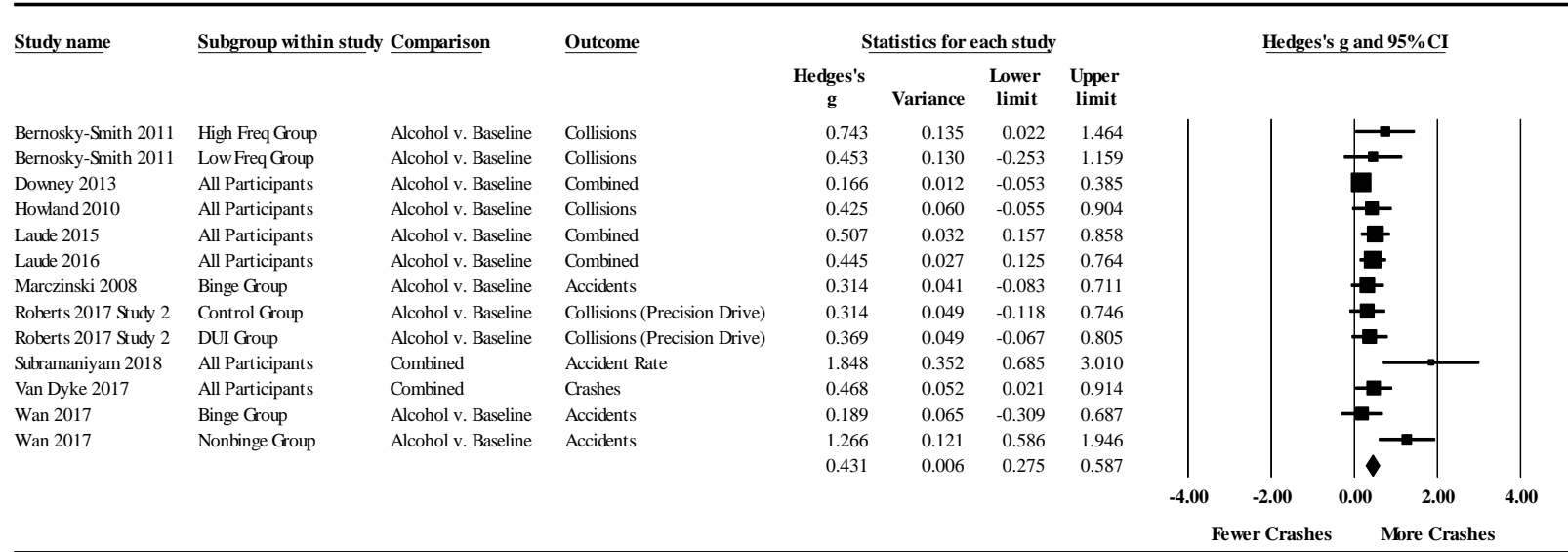


Figure C31. Forest plot illustrating Alcohol v. Baseline: Crashes (missing pre-post correlations set to $r = 0.5$). Excludes Bernosky-Smith et al. (2012).

Alcohol v. Baseline: Crashes

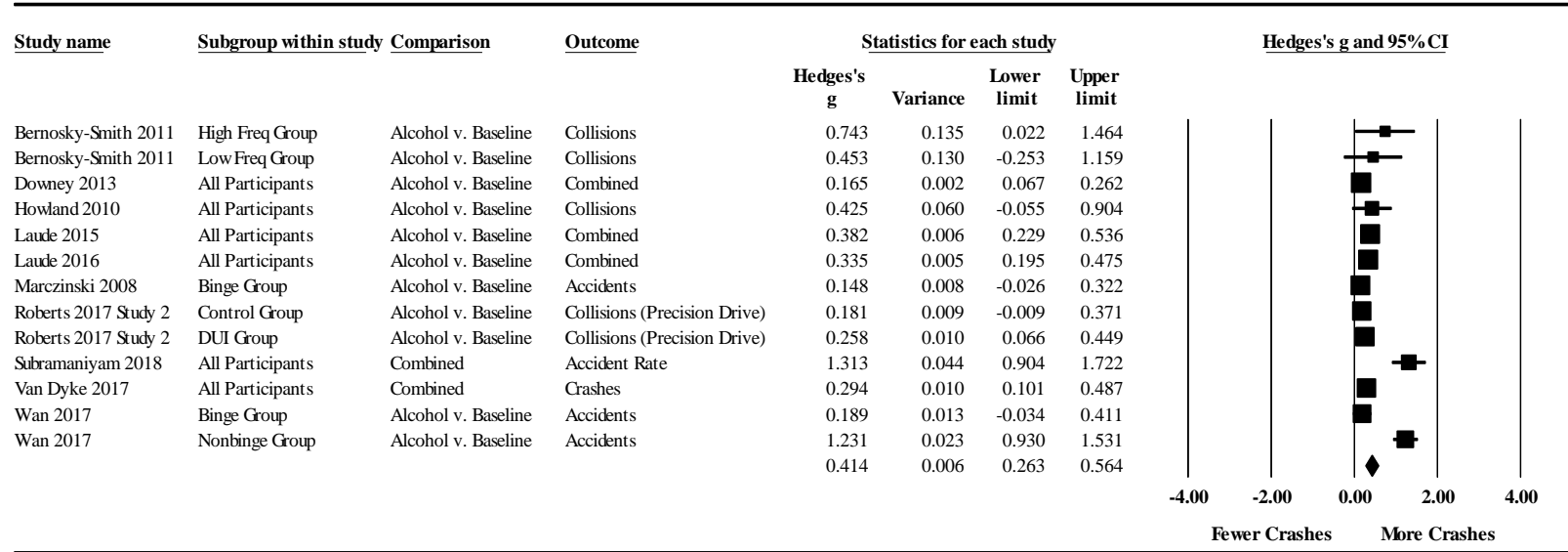


Figure C32. Forest plot illustrating *Alcohol v. Baseline: Crashes* (missing pre-post correlations set to $r = 0.9$). Excludes Bernosky-Smith et al. (2012).

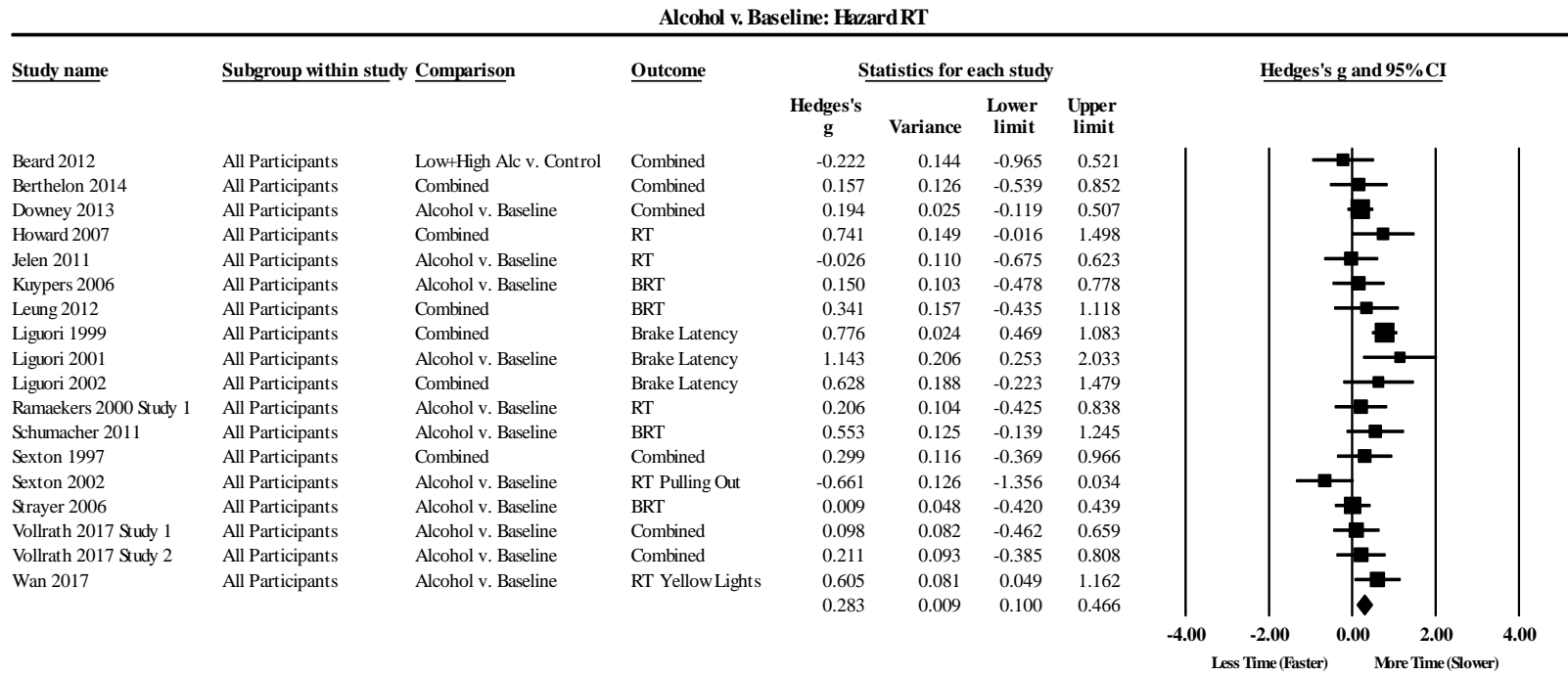


Figure C33. Forest plot illustrating *Alcohol v. Baseline: Hazard RT* (missing pre-post correlations set to $r = \text{zero}$).

Note that Schumacher et al. (2017) was eligible for inclusion in this meta-analysis but data was extracted and included from the 2011 paper.

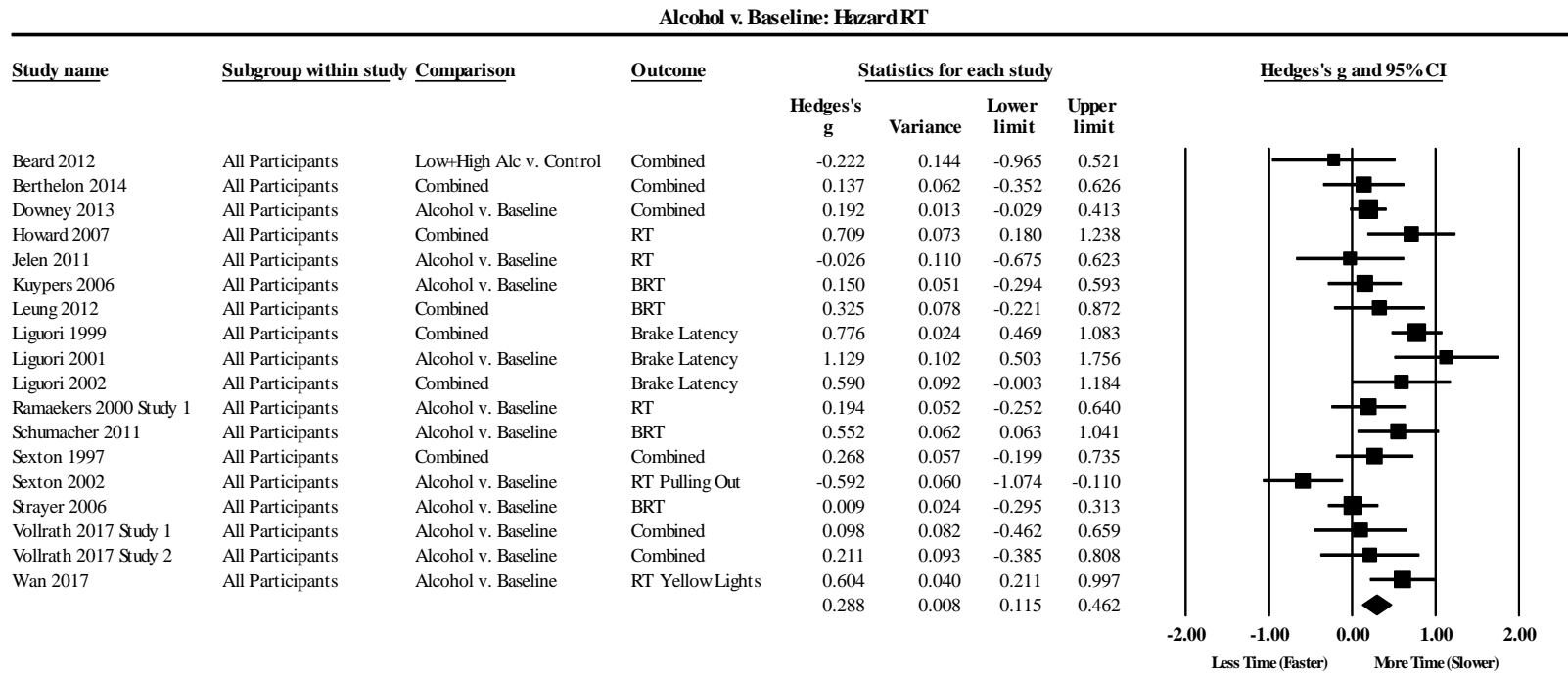


Figure C34. Forest plot illustrating *Alcohol v. Baseline: Hazard RT* (missing pre-post correlations set to $r = 0.5$).

Note that Schumacher et al. (2017) was eligible for inclusion in this meta-analysis but data was extracted and included from the 2011 paper.

Alcohol v. Baseline: Hazard RT

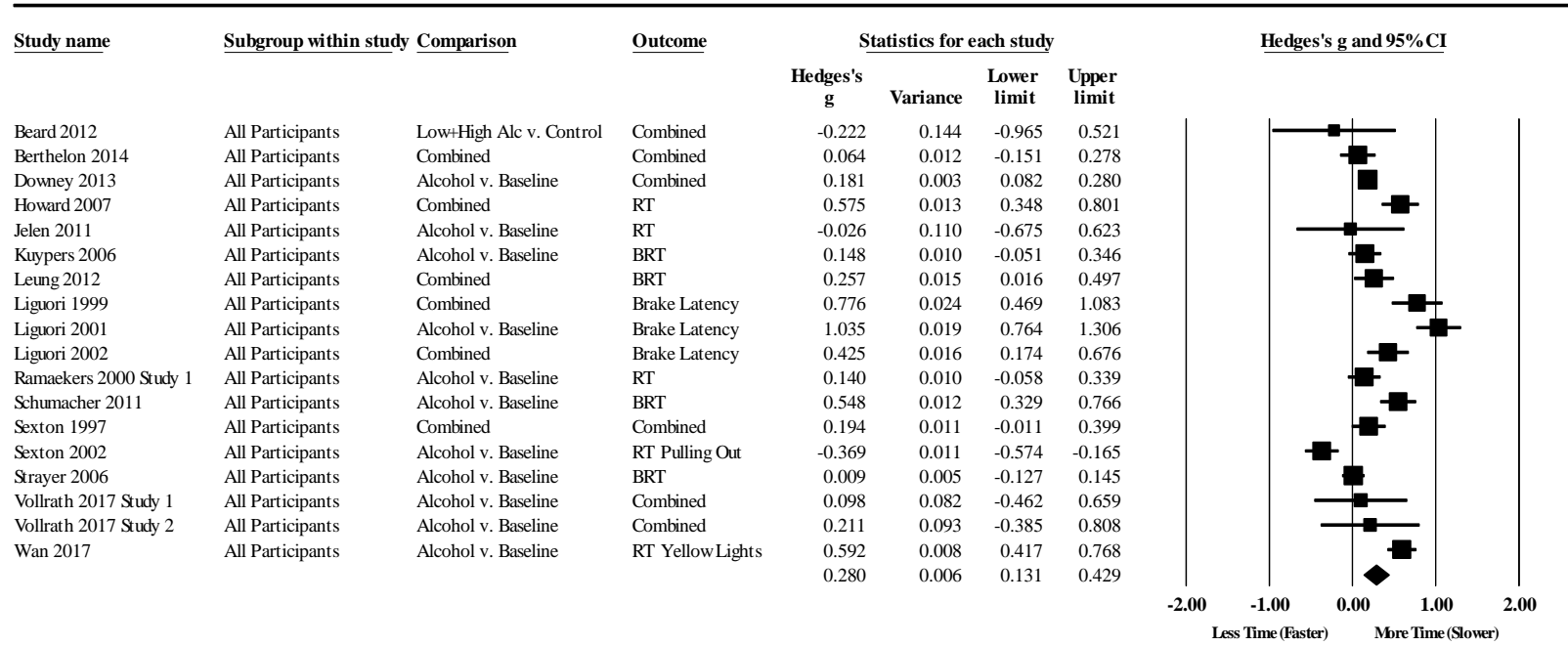


Figure C35. Forest plot illustrating Alcohol v. Baseline: Hazard RT (missing pre-post correlations set to $r = 0.9$).

Note that Schumacher et al. (2017) was eligible for inclusion in this meta-analysis but data was extracted and included from the 2011 paper.

Alcohol v. Baseline: Headway

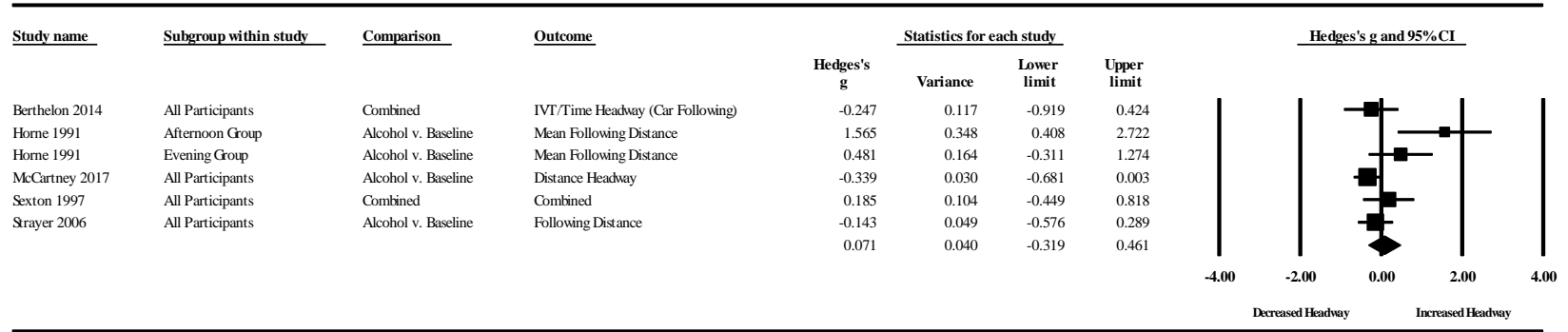


Figure C36. Forest plot illustrating Alcohol v. Baseline: Headway (missing pre-post correlations set to $r = 0$).

Alcohol v. Baseline: Headway

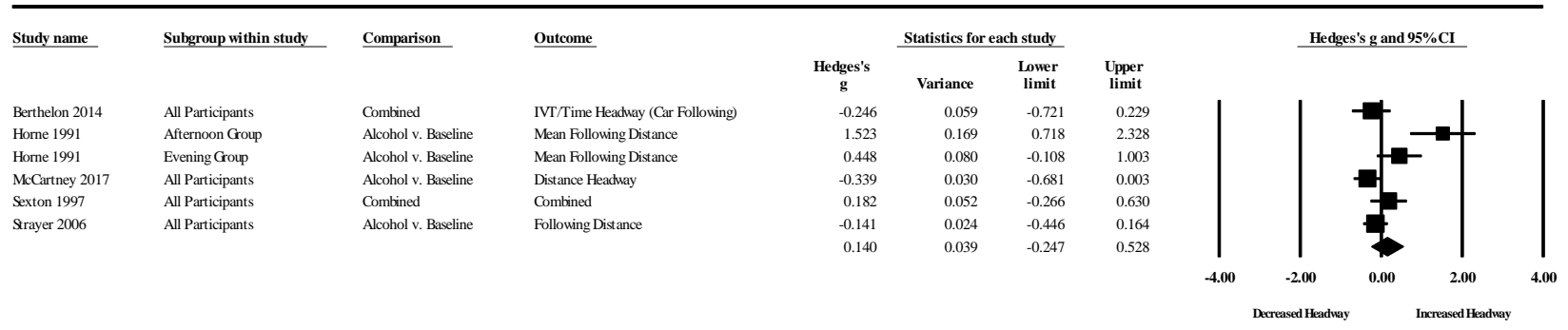


Figure C37. Forest plot illustrating Alcohol v. Baseline: Headway (missing pre-post correlations set to $r = 0.5$).

Alcohol v. Baseline: Headway

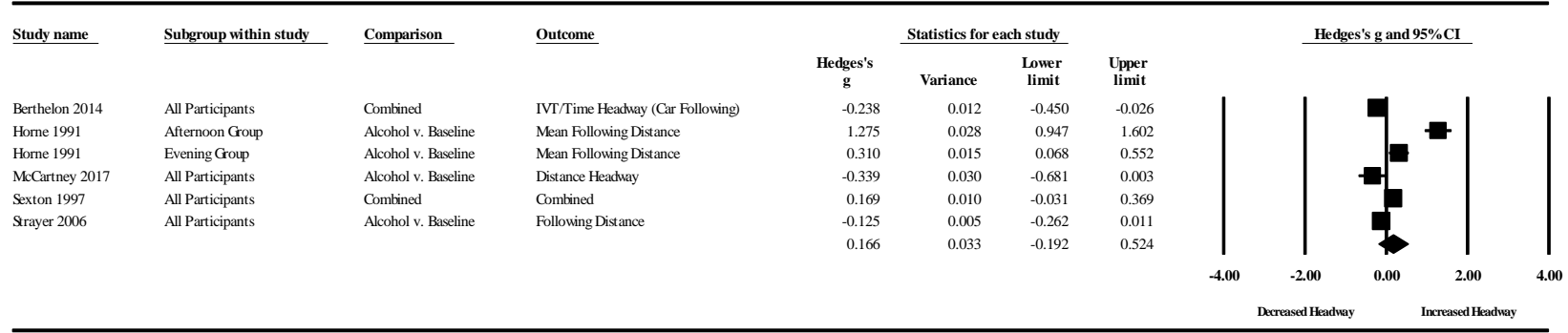


Figure C38. Forest plot illustrating Alcohol v. Baseline: Headway (missing pre-post correlations set to $r = 0.9$).

Alcohol v. Baseline: Headway Variability

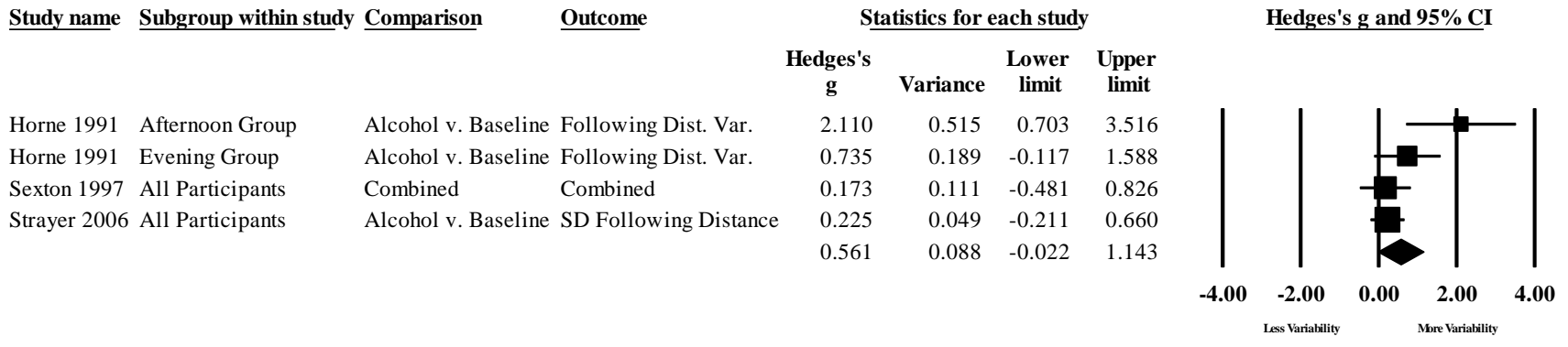


Figure C39. Forest plot illustrating Alcohol v. Baseline: Headway Variability (missing pre-post correlations set to $r = \text{zero}$).

Alcohol v. Baseline: Headway Variability

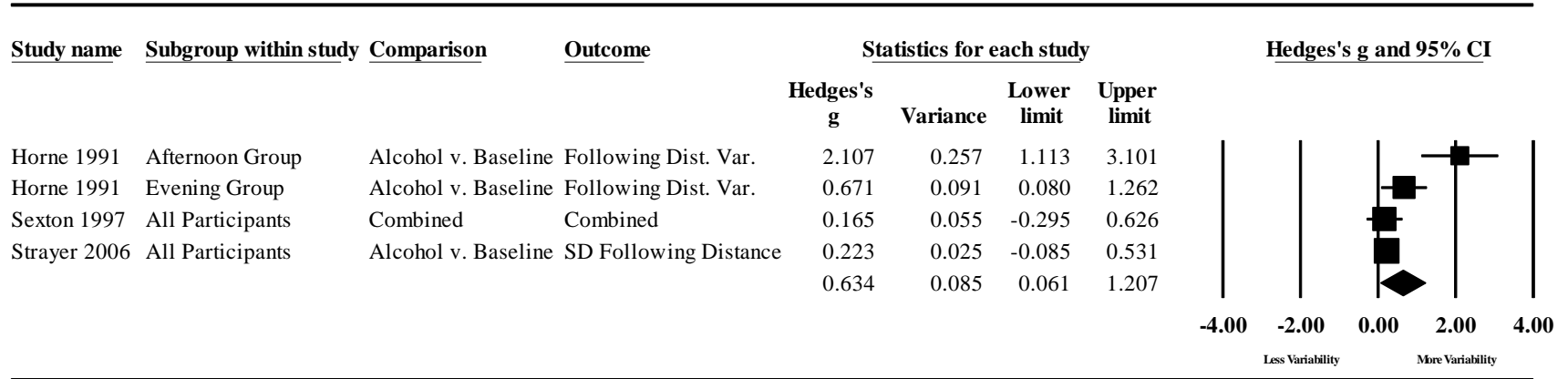


Figure C40. Forest plot illustrating Alcohol v. Baseline: Headway Variability (missing pre-post correlations set to $r = 0.5$).

Alcohol v. Baseline: Headway Variability

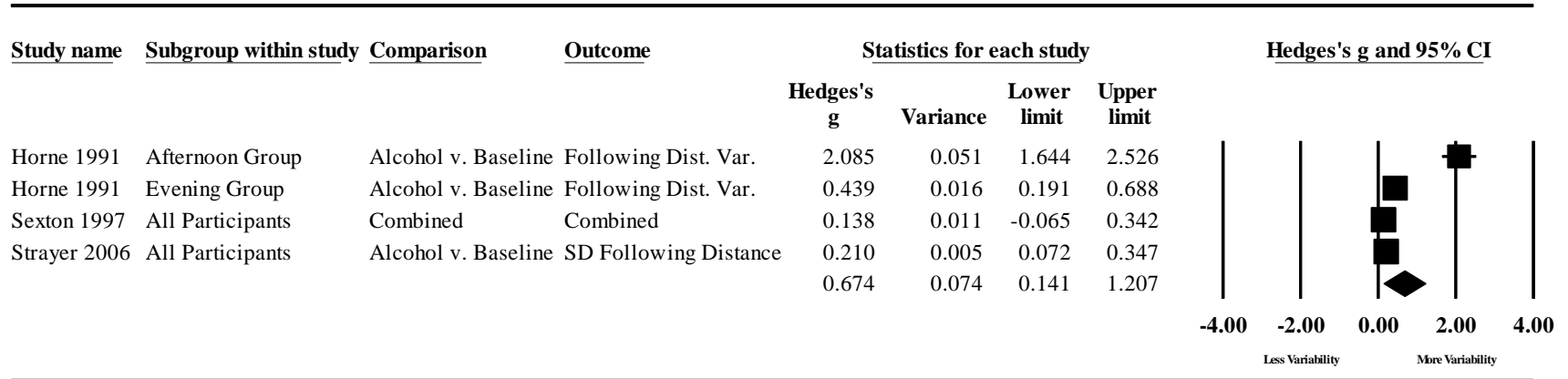


Figure C41. Forest plot illustrating Alcohol v. Baseline: Headway Variability (missing pre-post correlations set to $r = 0.9$).

Alcohol v. Baseline: Lateral Position Variability

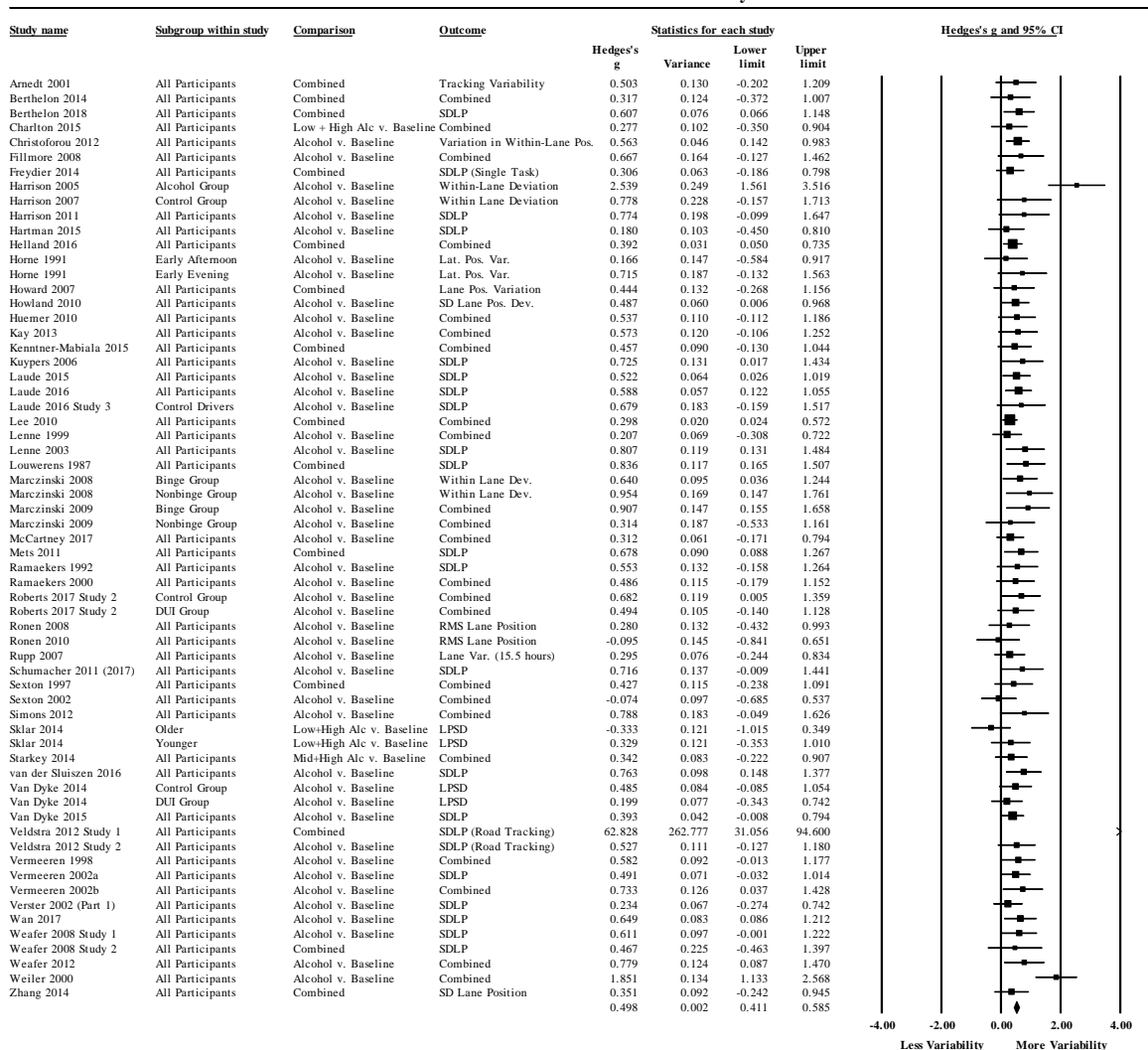


Figure C42. Forest plot illustrating *Alcohol v. Baseline: Lateral Position Variability* (missing pre-post correlations set to $r = \text{zero}$). Includes Study 1 from Veldstra et al. (2012).

Note that Schumacher et al. (2017) was eligible for inclusion in this meta-analysis but data was extracted and included from the 2011 paper.

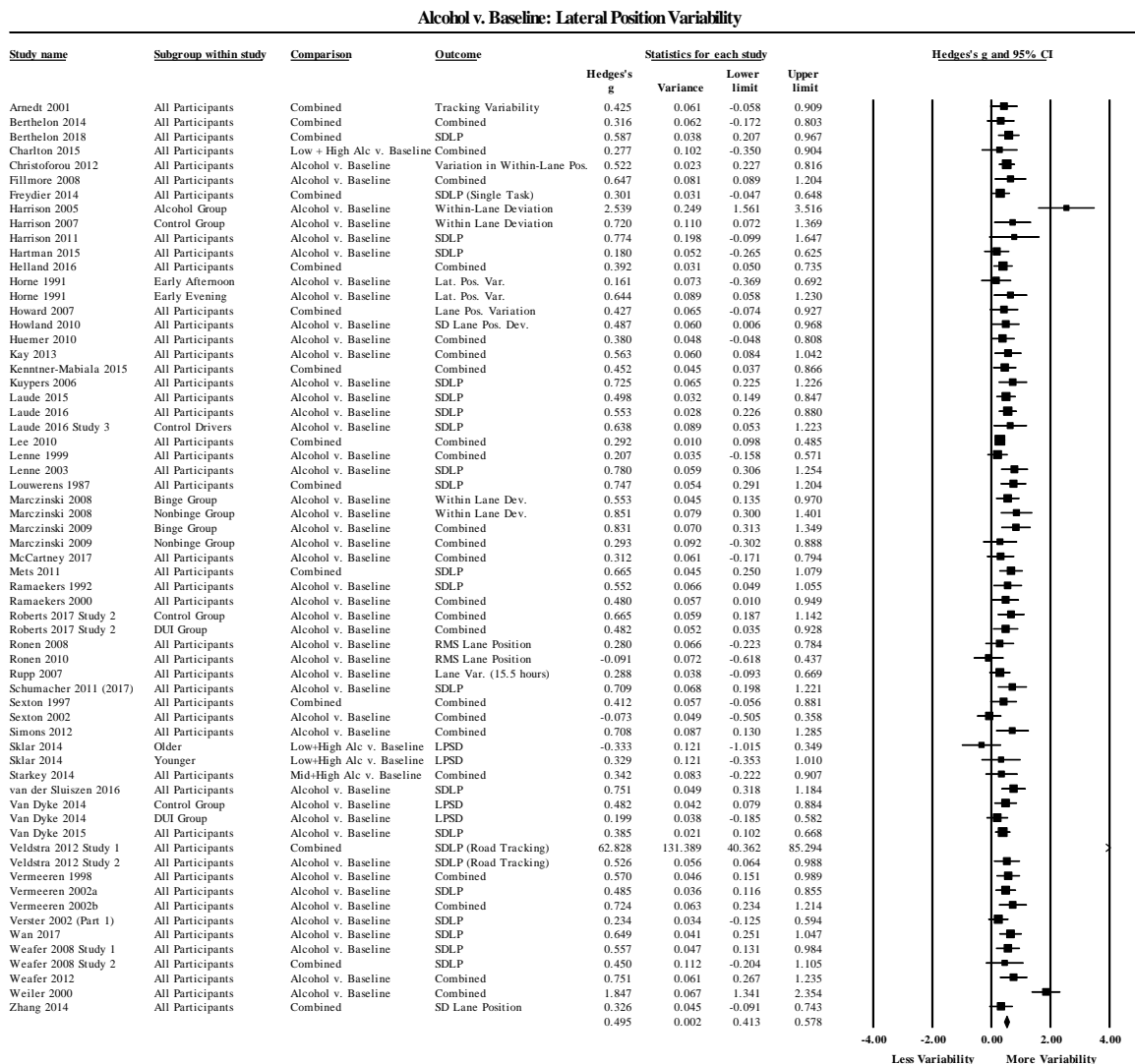


Figure C43. Forest plot illustrating *Alcohol v. Baseline: Lateral Position Variability* (missing pre-post correlations set to $r = 0.5$). Includes Study 1 from Veldstra et al. (2012).

Note that Schumacher et al. (2017) was eligible for inclusion in this meta-analysis but data was extracted and included from the 2011 paper.

Alcohol v. Baseline: Lateral Position Variability

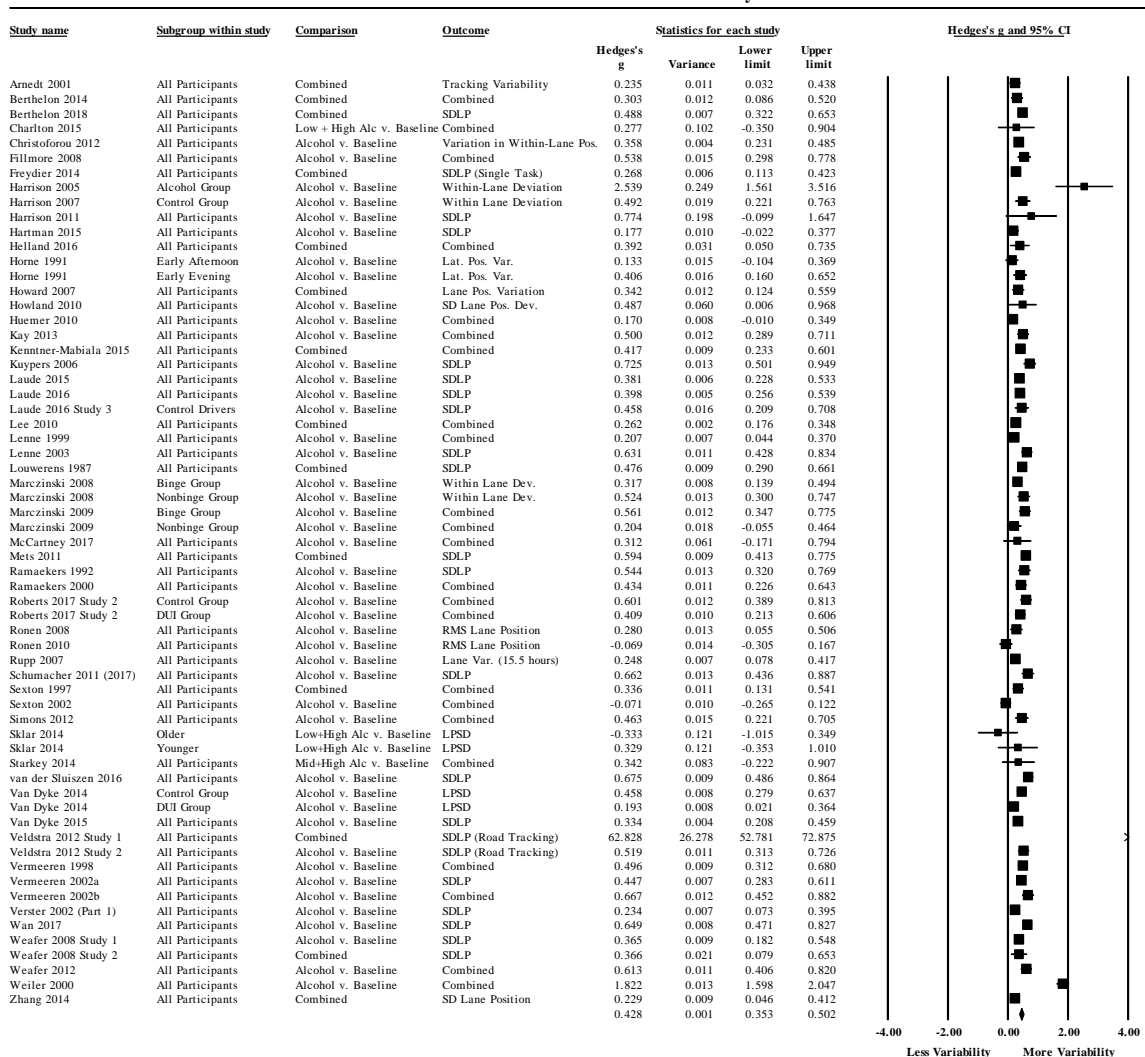


Figure C44. Forest plot illustrating *Alcohol v. Baseline: Lateral Position Variability* (missing pre-post correlations set to $r = 0.9$). Includes Study 1 from Veldstra et al. (2012).

Note that Schumacher et al. (2017) was eligible for inclusion in this meta-analysis but data was extracted and included from the 2011 paper.

Alcohol v. Baseline: Lateral Position Variability

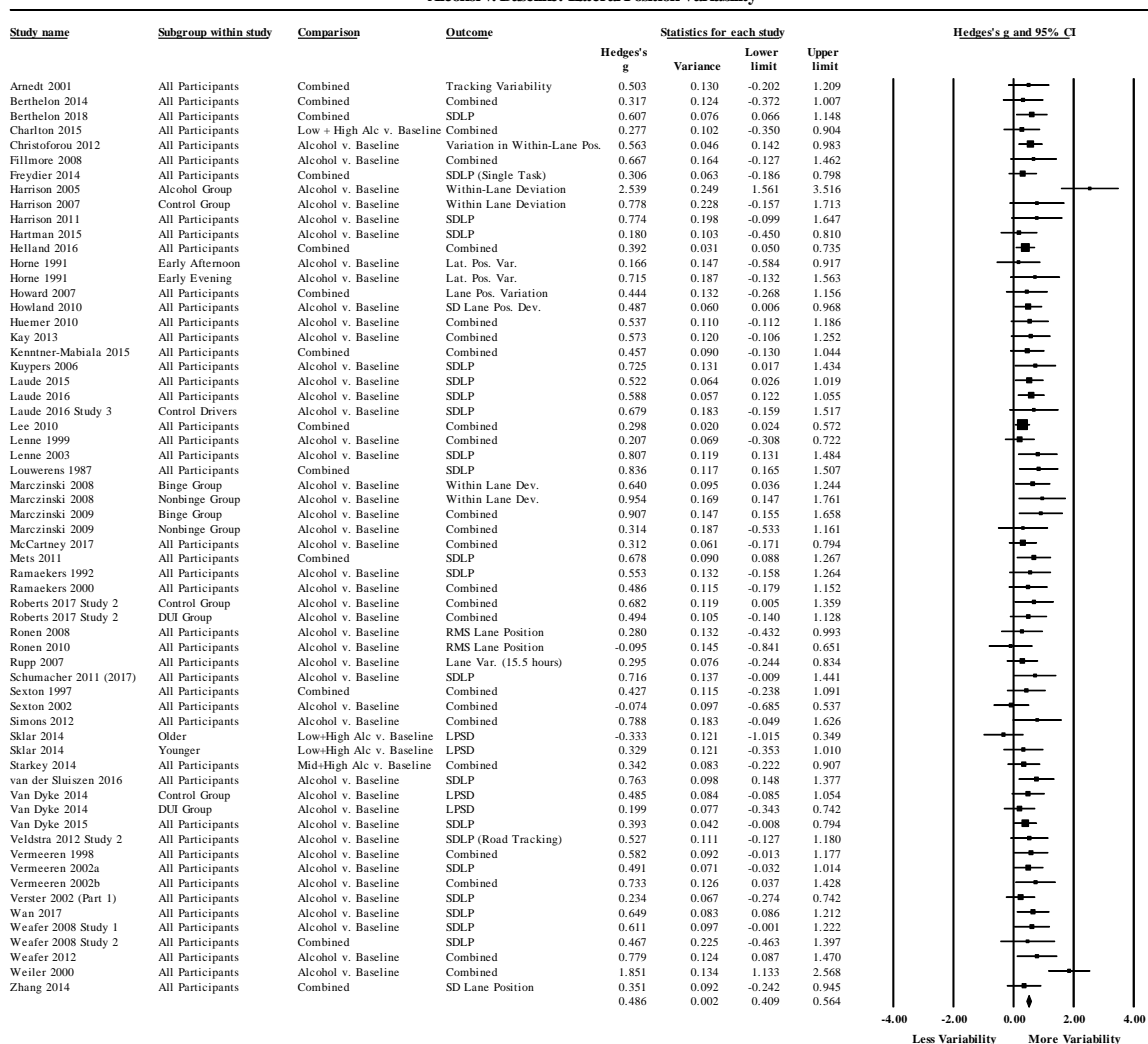


Figure C45. Forest plot illustrating *Alcohol v. Baseline: Lateral Position Variability* (missing pre-post correlations set to $r = \text{zero}$). Excludes Study 1 from Veldstra et al. (2012).

Note that Schumacher et al. (2017) was eligible for inclusion in this meta-analysis but data was extracted and included from the 2011 paper.

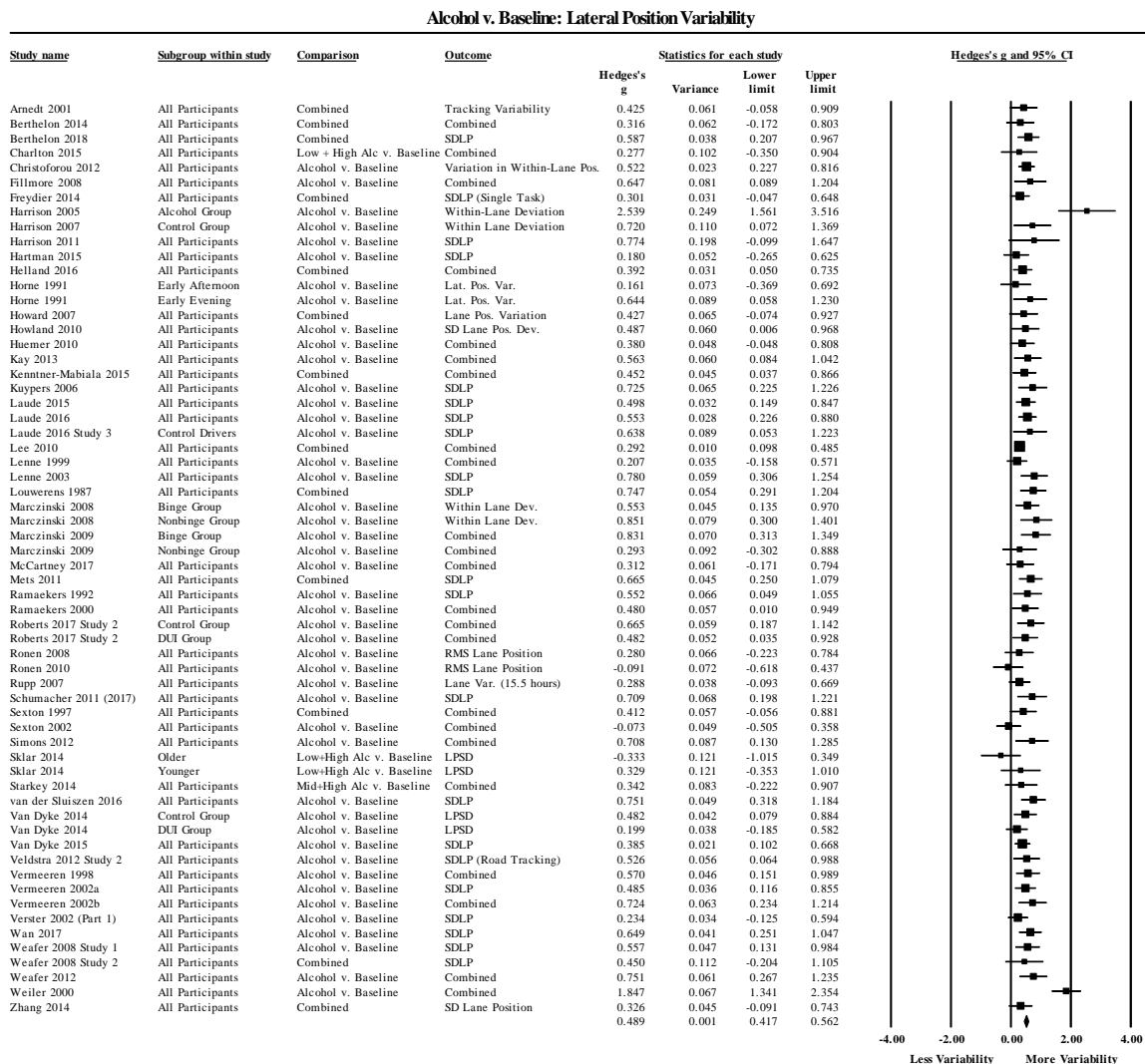


Figure C46. Forest plot illustrating *Alcohol v. Baseline: Lateral Position Variability* (missing pre-post correlations set to $r = 0.5$). Excludes Study 1 from Veldstra et al. (2012).

Note that Schumacher et al. (2017) was eligible for inclusion in this meta-analysis but data was extracted and included from the 2011 paper.

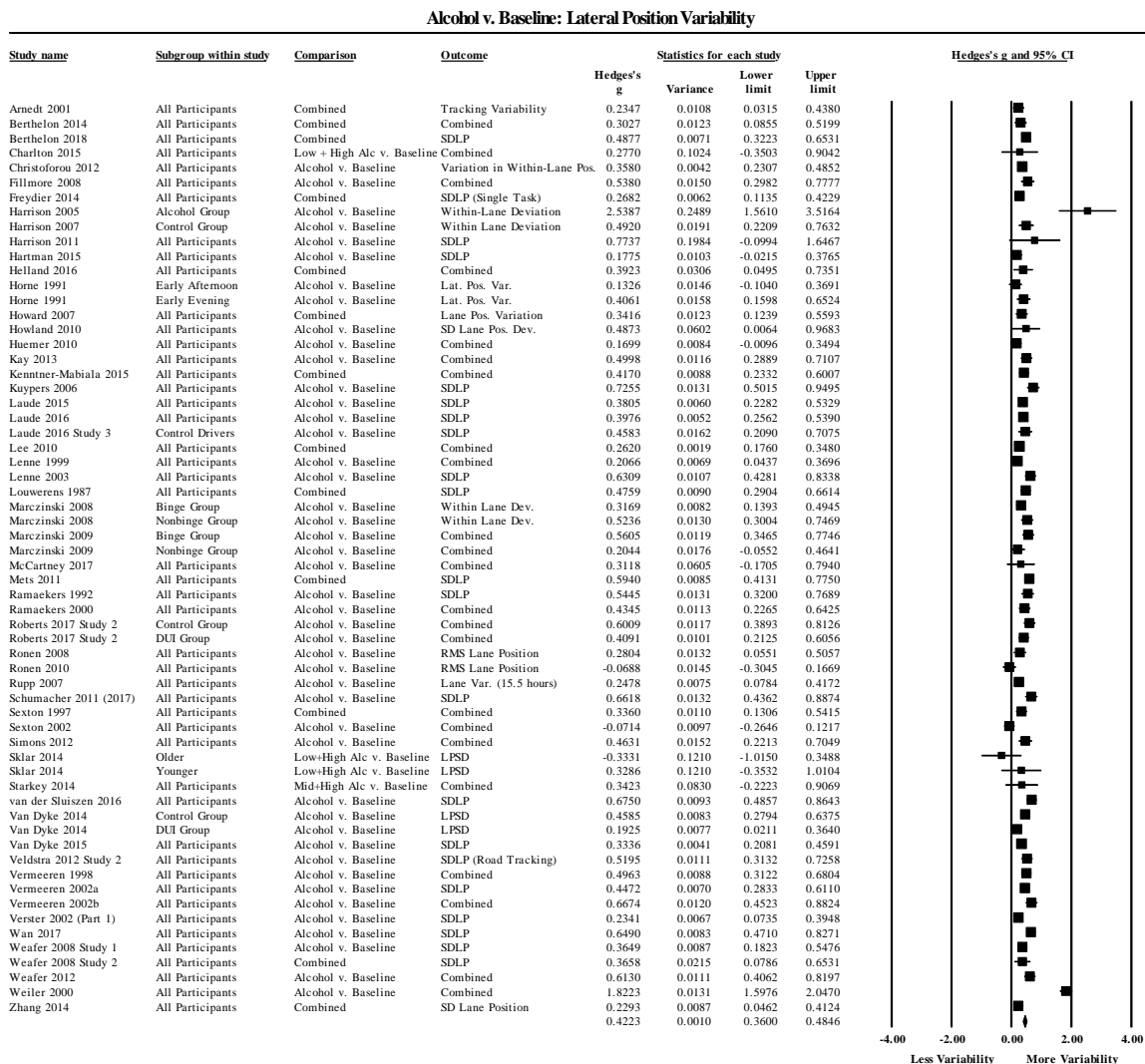


Figure C47. Forest plot illustrating *Alcohol v. Baseline: Lateral Position Variability* (missing pre-post correlations set to $r = 0.9$). Excludes Study 1 from Veldstra et al. (2012).

Note that Schumacher et al. (2017) was eligible for inclusion in this meta-analysis but data was extracted and included from the 2011 paper.

Alcohol v. Baseline: Lane Excursions

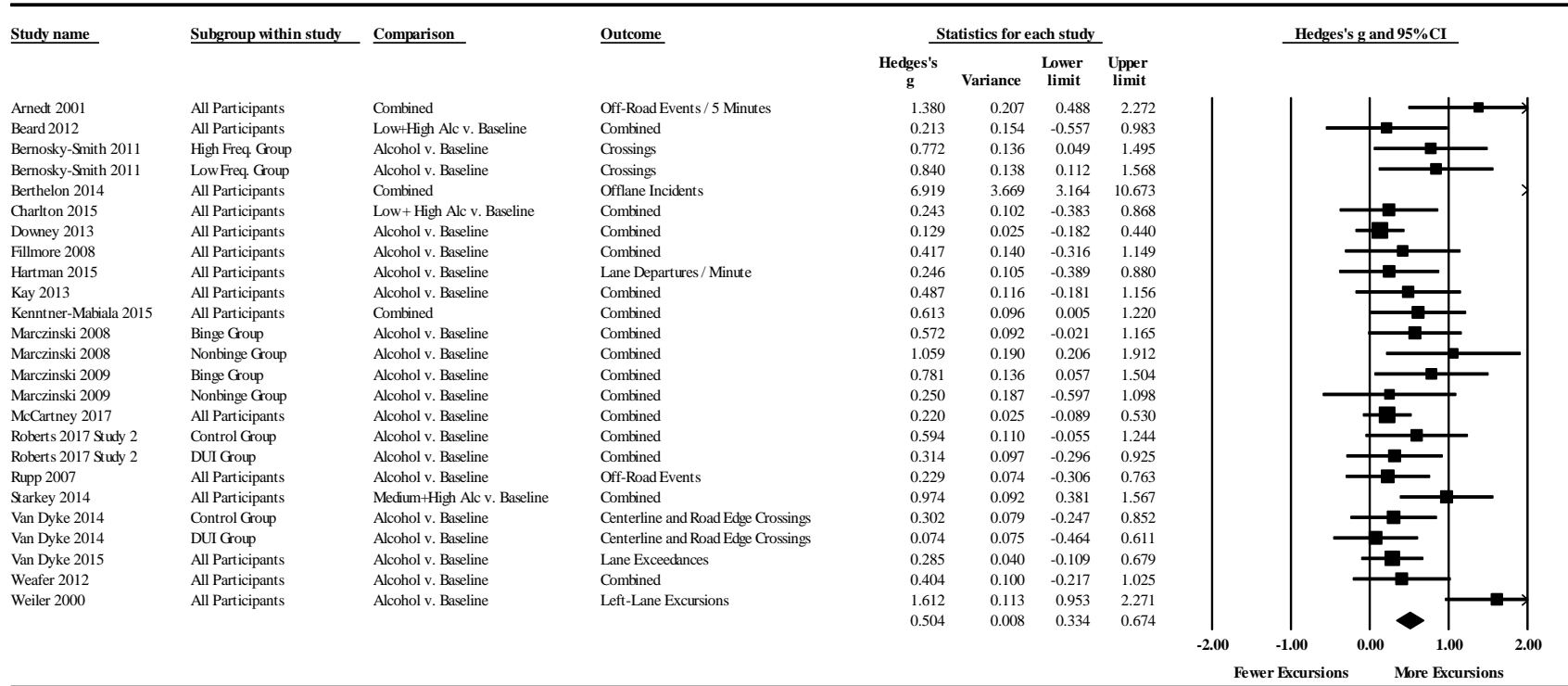


Figure C48. Forest plot illustrating Alcohol v. Baseline: Lane Excursions (missing pre-post correlations set to $r = zero$). Includes Berthelon & Gineyt (2014) and Weiler et al. (2000).

Alcohol v. Baseline: Lane Excursions

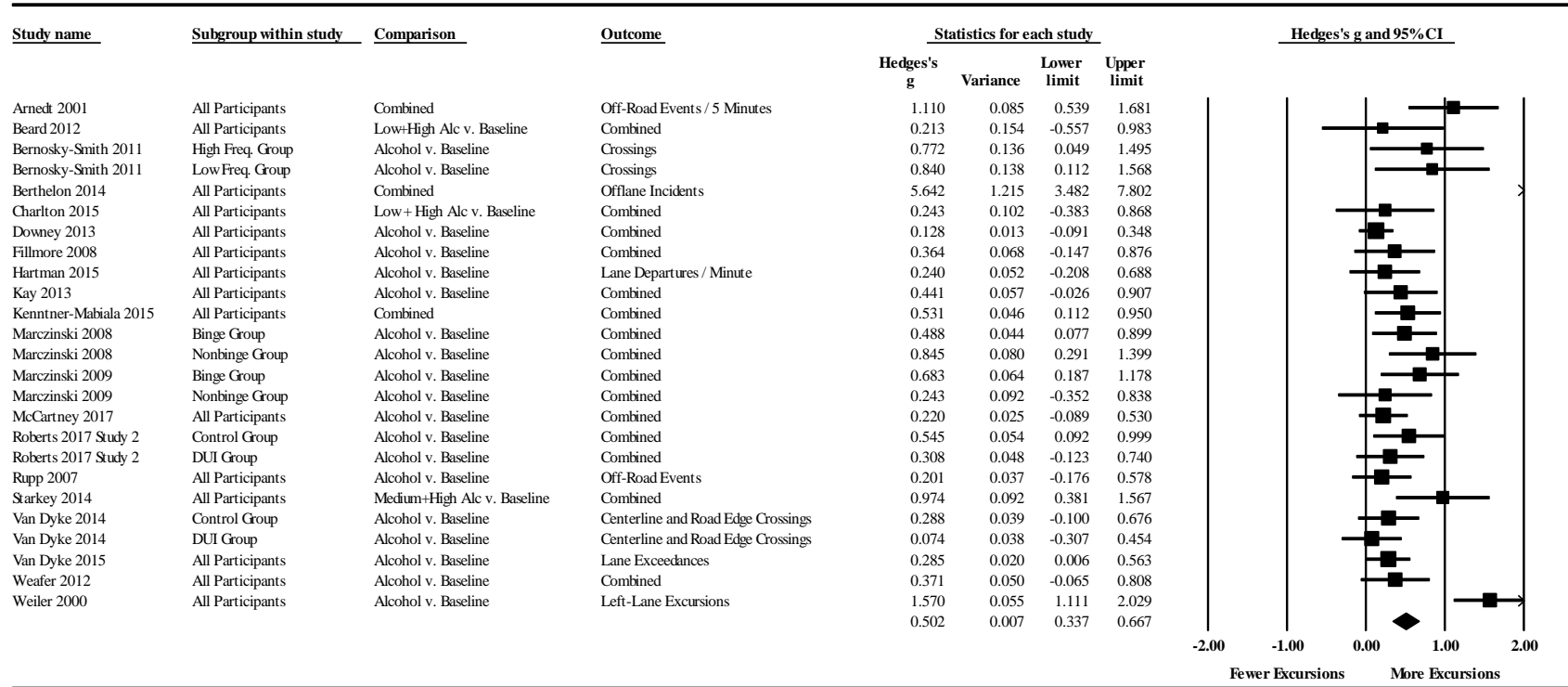


Figure C49. Forest plot illustrating Alcohol v. Baseline: Lane Excursions (missing pre-post correlations set to $r = 0.5$). Includes Berthelon & Giney (2014) and Weiler et al. (2000).

Alcohol v. Baseline: Lane Excursions

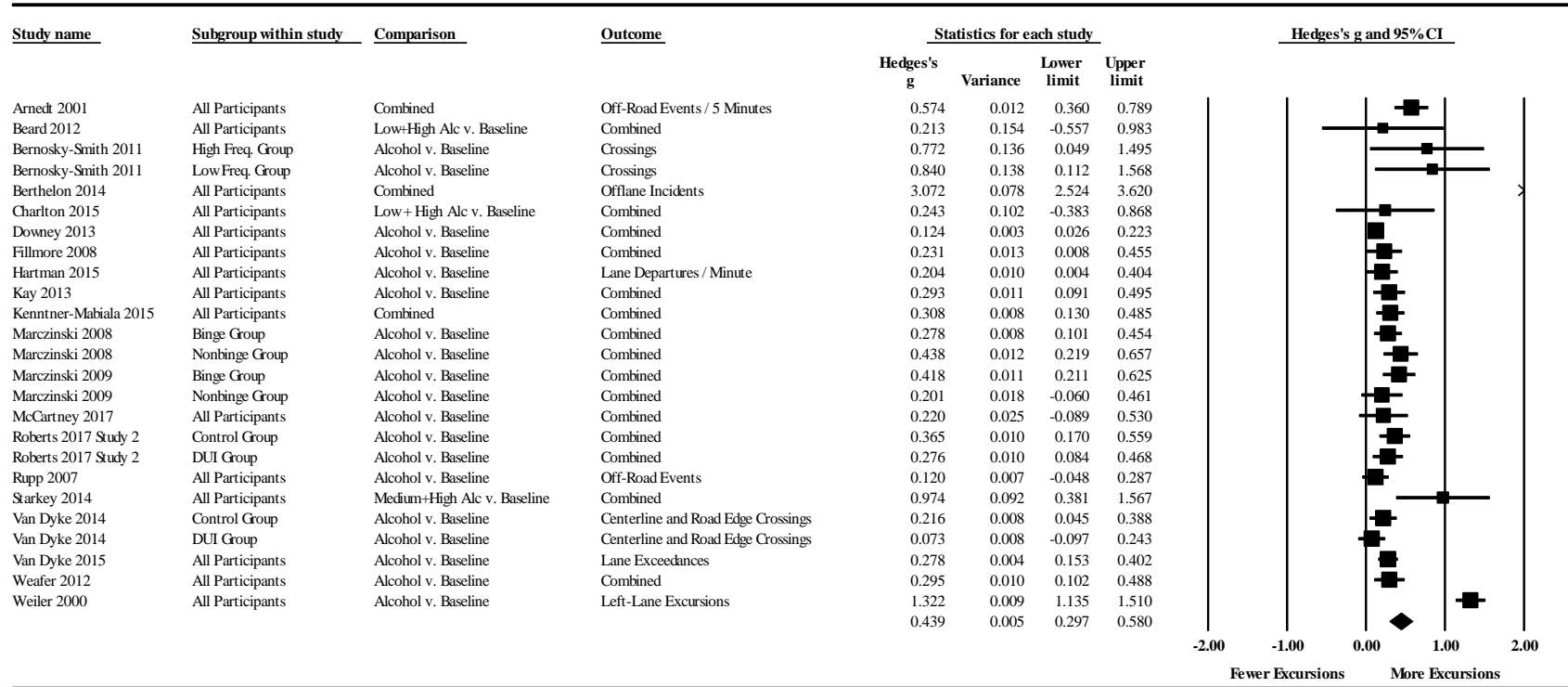


Figure C50. Forest plot illustrating Alcohol v. Baseline: Lane Excursions (missing pre-post correlations set to $r = 0.9$). Includes Berthelon & Gineyt (2014) and Weiler et al. (2000).

Alcohol v. Baseline: Lane Excursions

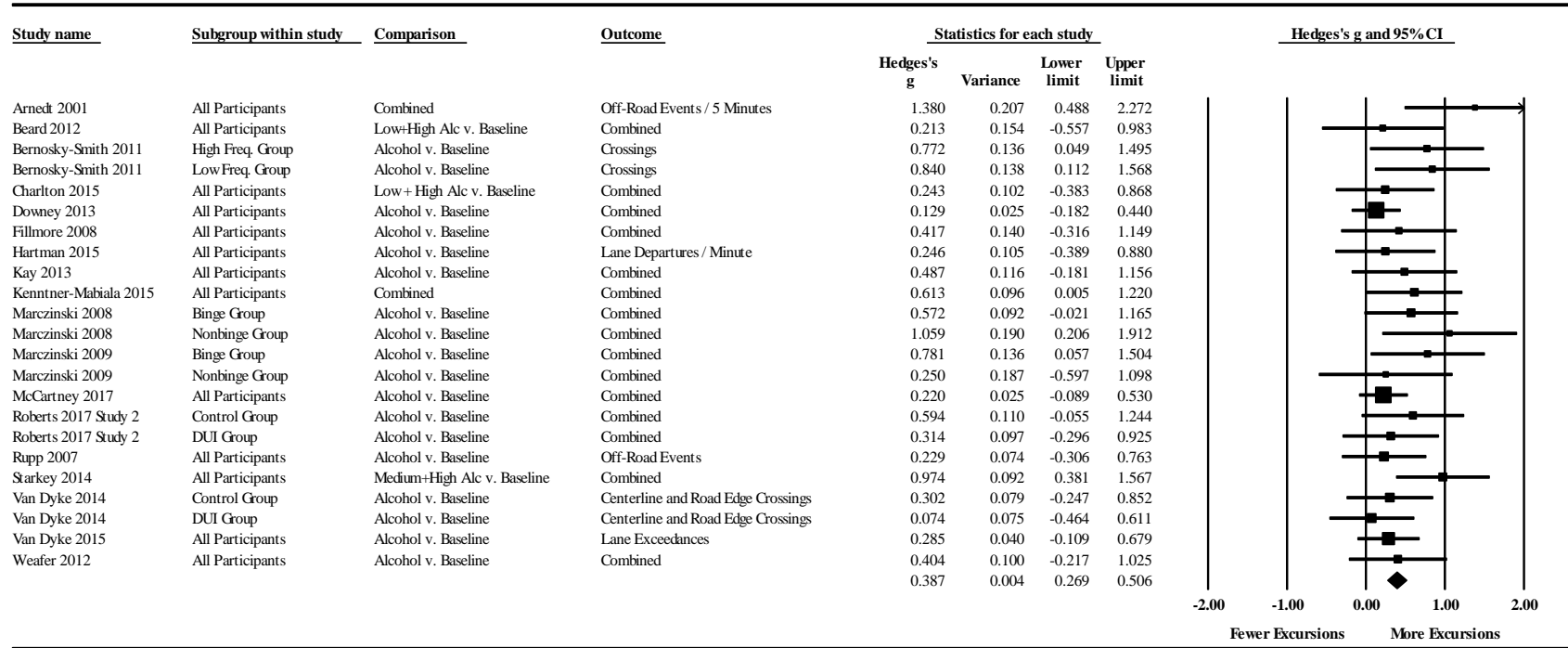


Figure C51. Forest plot illustrating Alcohol v. Baseline: Lane Excursions (missing pre-post correlations set to $r = 0$). Excludes Berthelon & Gineyt (2014) and Weiler et al. (2000).

Alcohol v. Baseline: Lane Excursions

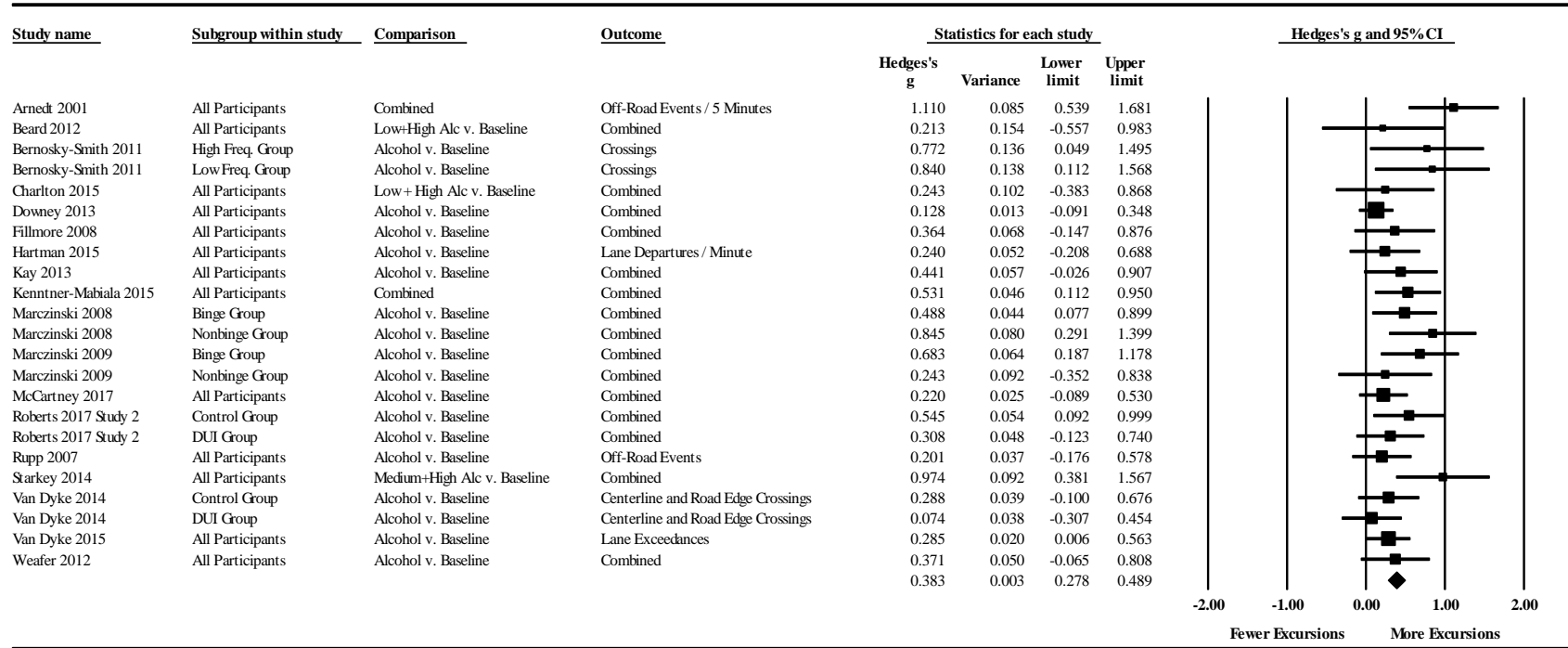


Figure C52. Forest plot illustrating Alcohol v. Baseline: Lane Excursions (missing pre-post correlations set to $r = 0.5$). Excludes Berthelon & Gineyt (2014) and Weiler et al. (2000).

Alcohol v. Baseline: Lane Excursions

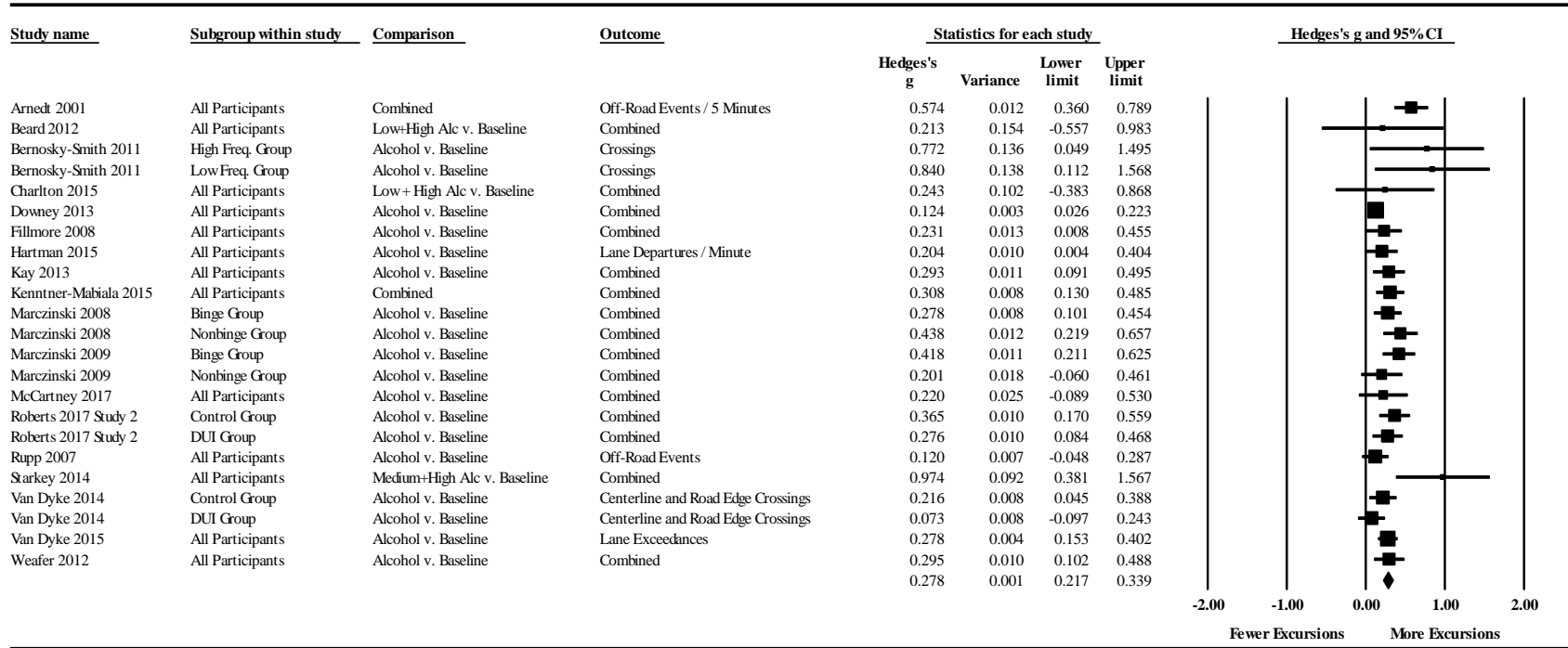


Figure C53. Forest plot illustrating Alcohol v. Baseline: Lane Excursions (missing pre-post correlations set to $r = 0.9$). Excludes Berthelon & Gineyt (2014) and Weiler et al. (2000).

Alcohol v. Baseline: Time Out of Lane

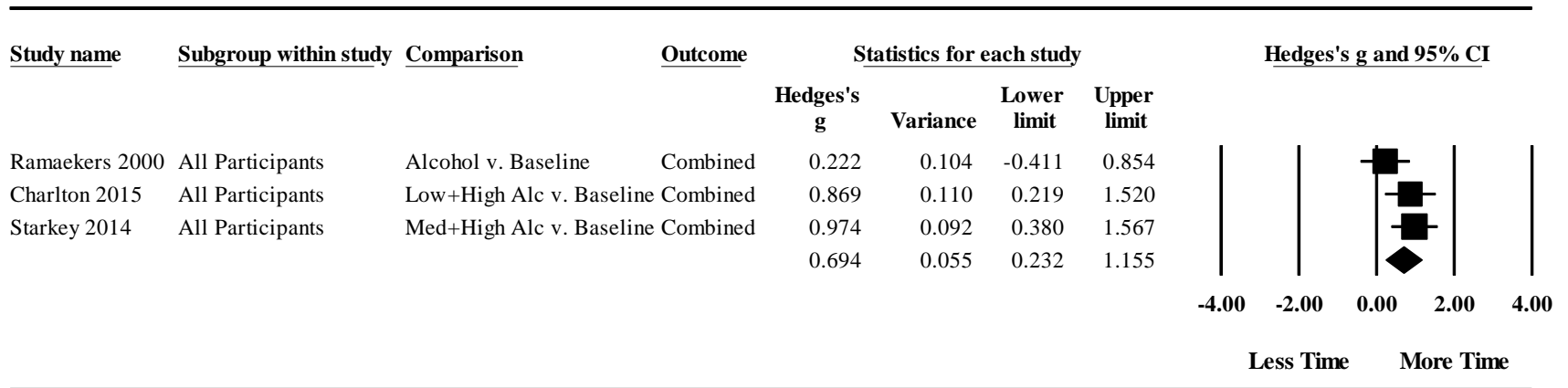


Figure C54. Forest plot illustrating Alcohol v. Baseline: Time Out of Lane (missing pre-post correlations set to $r = 0$).

Alcohol v. Baseline: Time Out of Lane

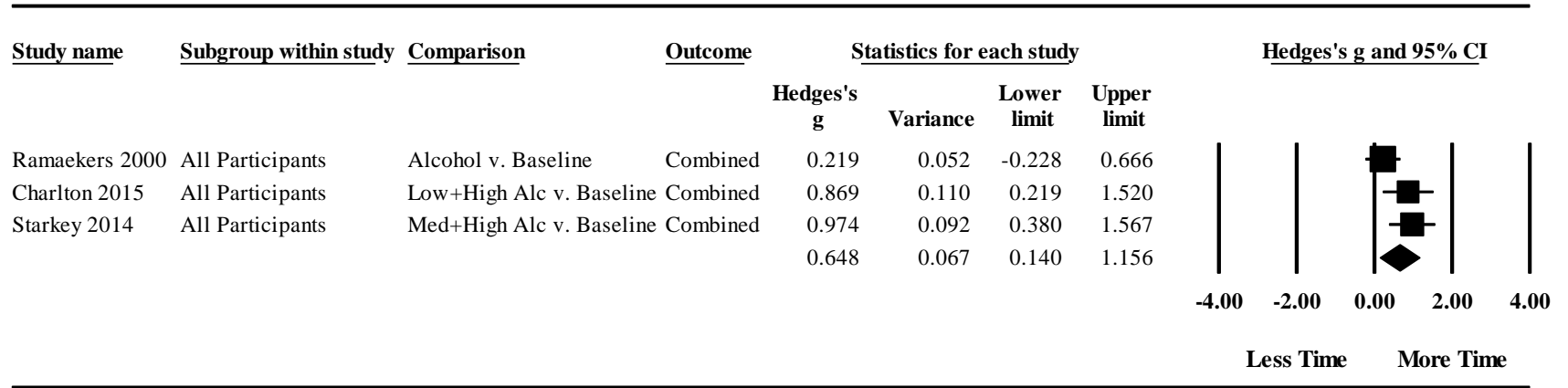


Figure C55. Forest plot illustrating Alcohol v. Baseline: Time Out of Lane (missing pre-post correlations set to $r = 0.5$).

Alcohol v. Baseline: Time Out of Lane

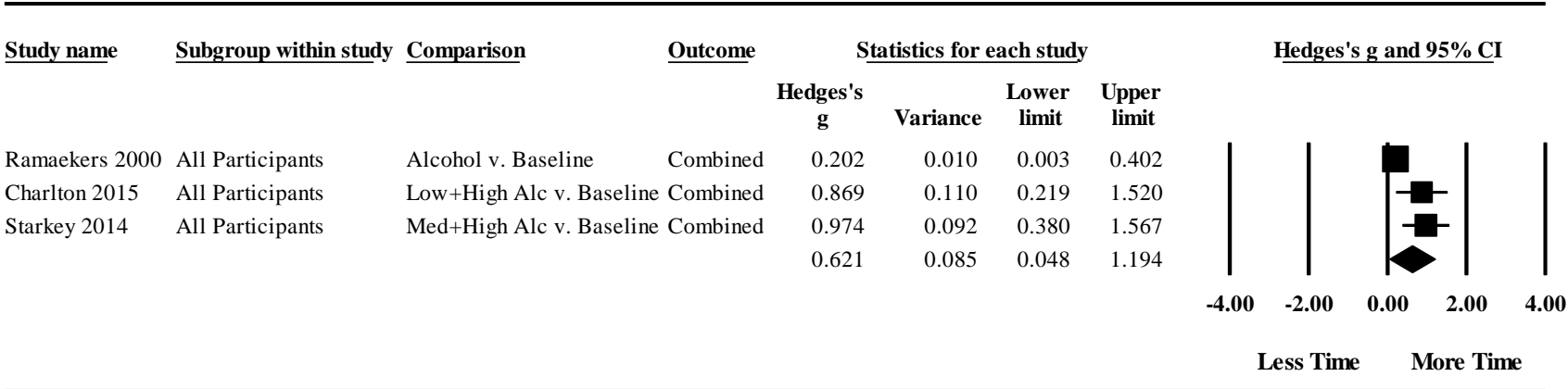


Figure C56. Forest plot illustrating Alcohol v. Baseline: Time Out of Lane (missing pre-post correlations set to $r = 0.9$).

Alcohol v. Baseline: Speed

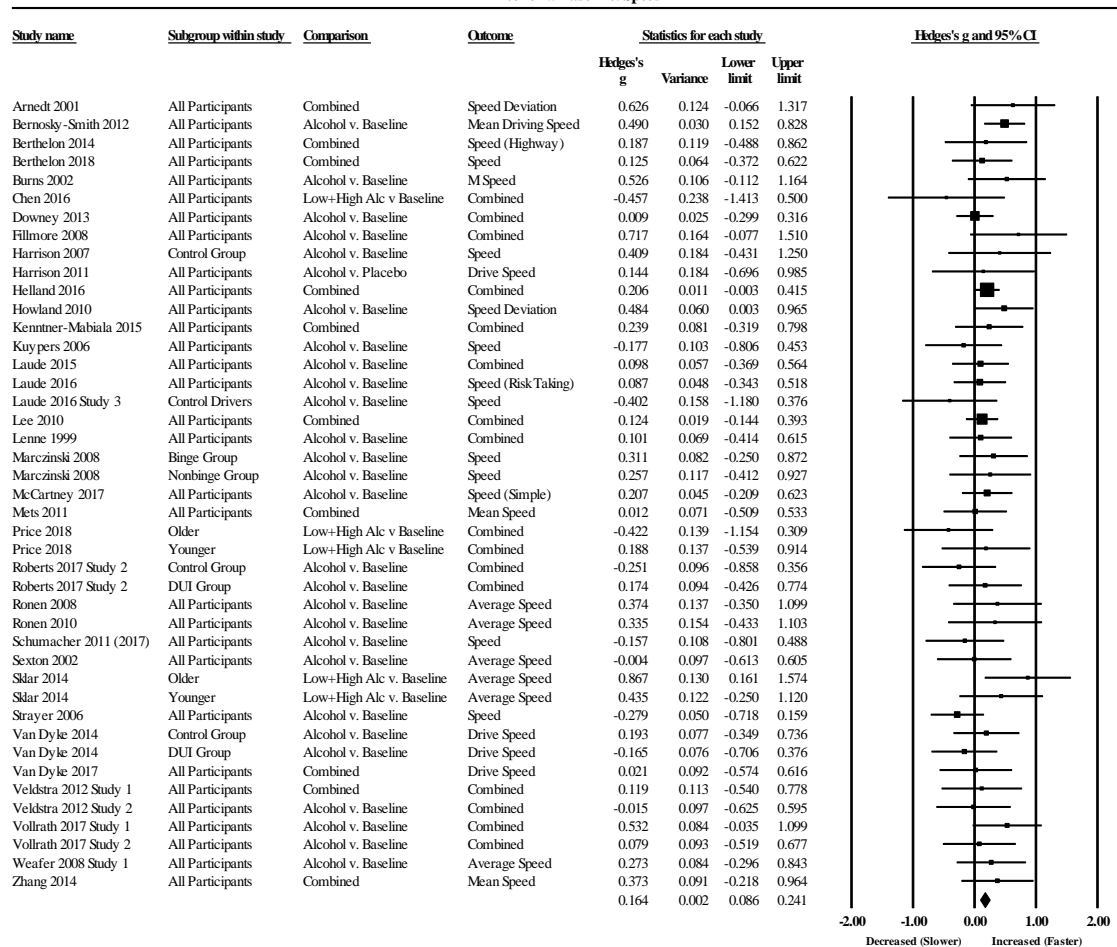


Figure C57. Forest plot illustrating Alcohol v. Baseline: Speed (missing pre-post correlations set to $r = 0$).

Note that Schumacher et al. (2017) was eligible for inclusion in this meta-analysis but data was extracted and included from the 2011 paper.

Alcohol v. Baseline: Speed

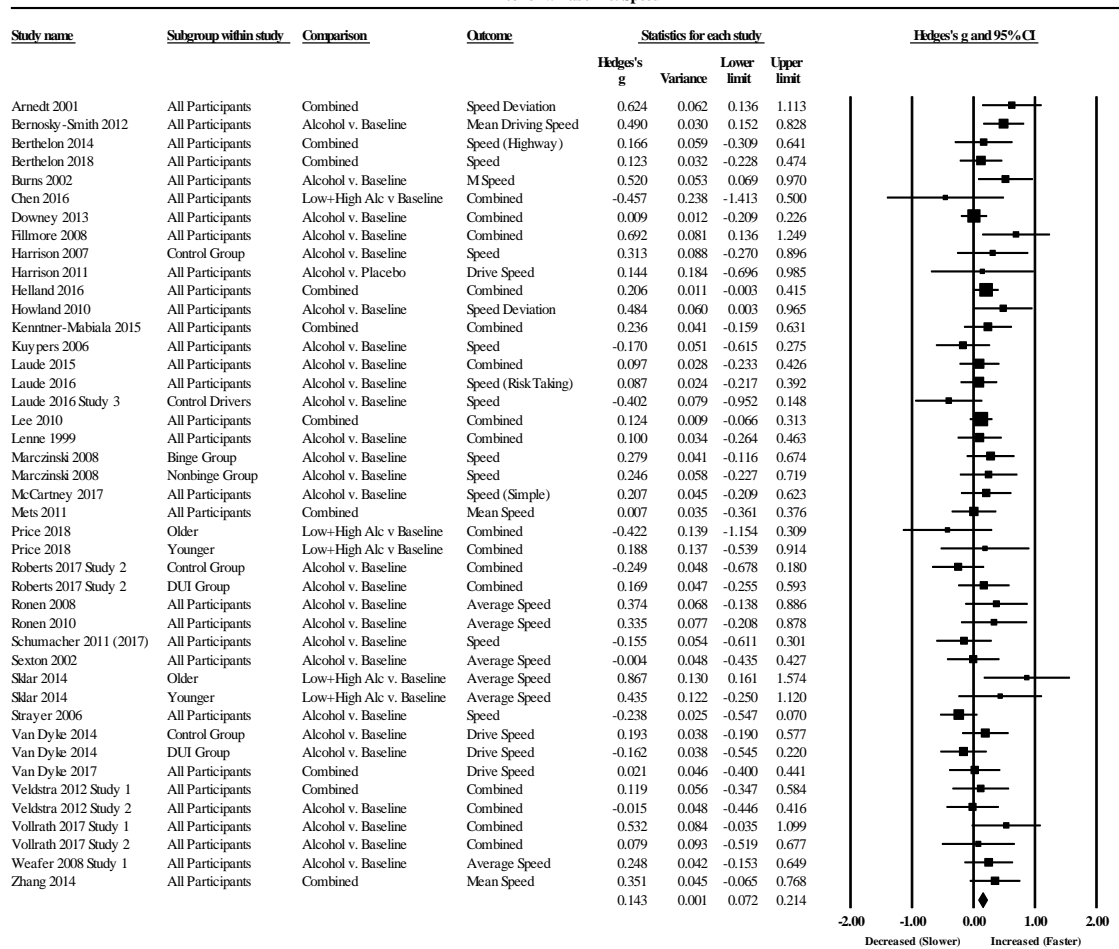


Figure C58. Forest plot illustrating Alcohol v. Baseline: Speed (missing pre-post correlations set to $r = 0.5$).

Note that Schumacher et al. (2017) was eligible for inclusion in this meta-analysis but data was extracted and included from the 2011 paper.

Alcohol v. Baseline: Speed

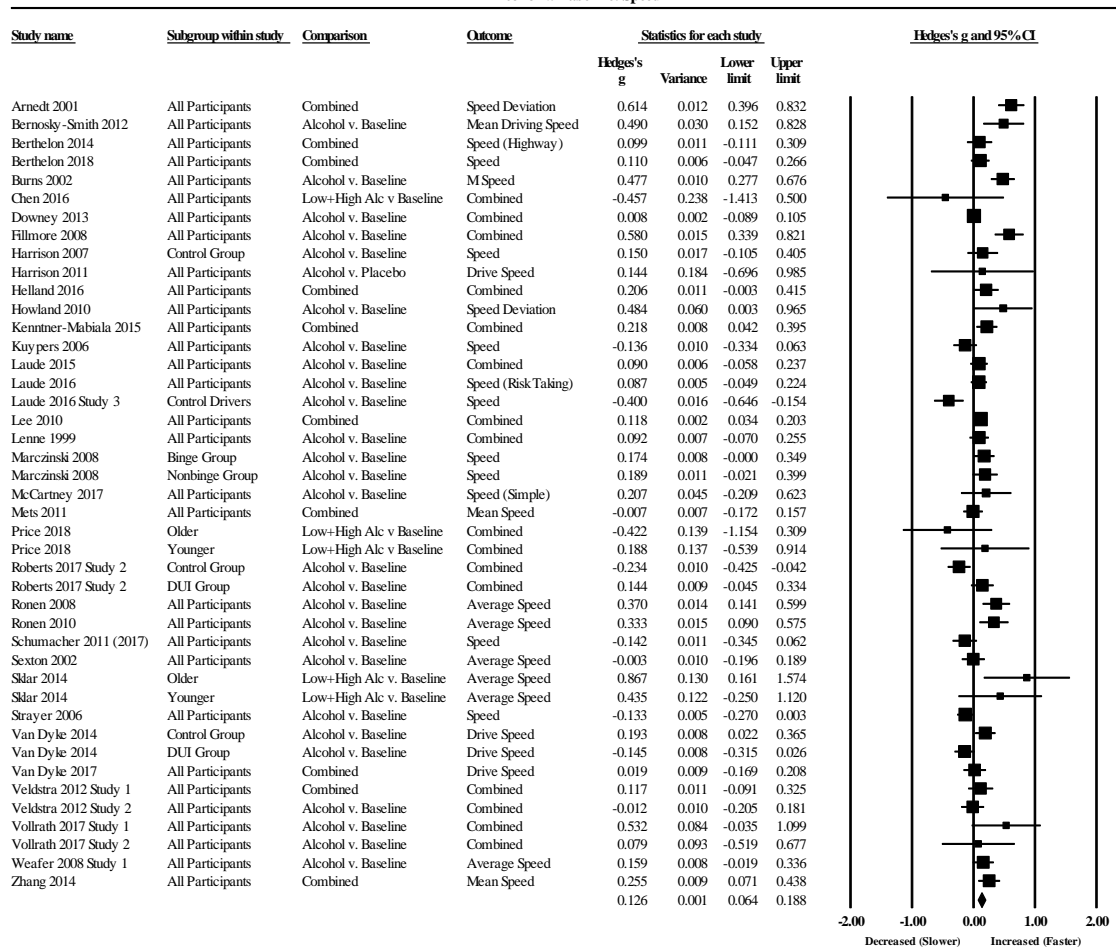


Figure C59. Forest plot illustrating Alcohol v. Baseline: Speed (missing pre-post correlations set to $r = 0.9$).

Note that Schumacher et al. (2017) was eligible for inclusion in this meta-analysis but data was extracted and included from the 2011 paper.

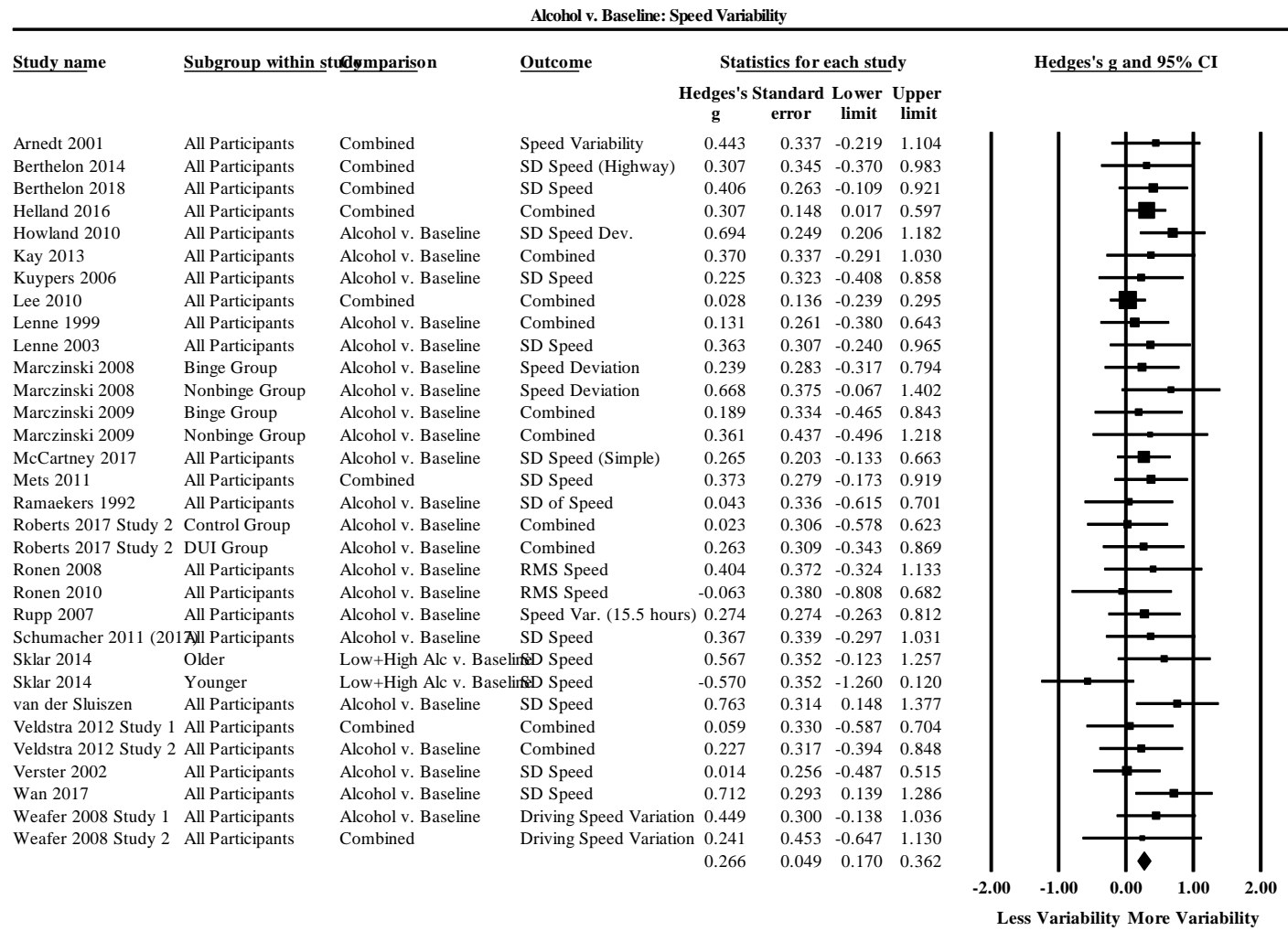


Figure C60. Forest plot illustrating Alcohol v. Baseline: Speed Variability (missing pre-post correlations set to $r = 0$).

Note that Schumacher et al. (2017) was eligible for inclusion in this meta-analysis but data was extracted and included from the 2011 paper.

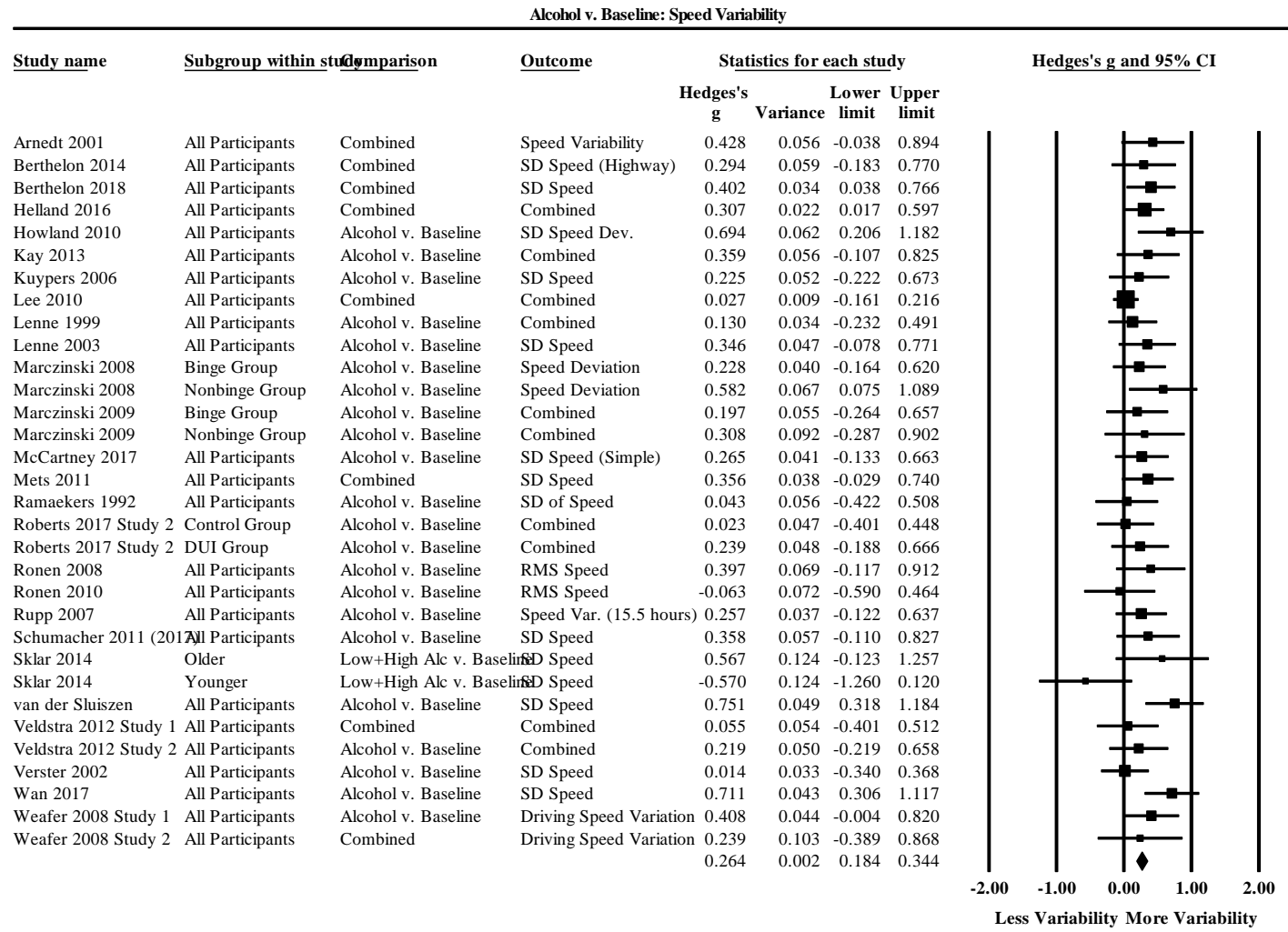


Figure C61. Forest plot illustrating Alcohol v. Baseline: Speed Variability (missing pre-post correlations set to $r = 0.5$).

Note that Schumacher et al. (2017) was eligible for inclusion in this meta-analysis but data was extracted and included from the 2011 paper.

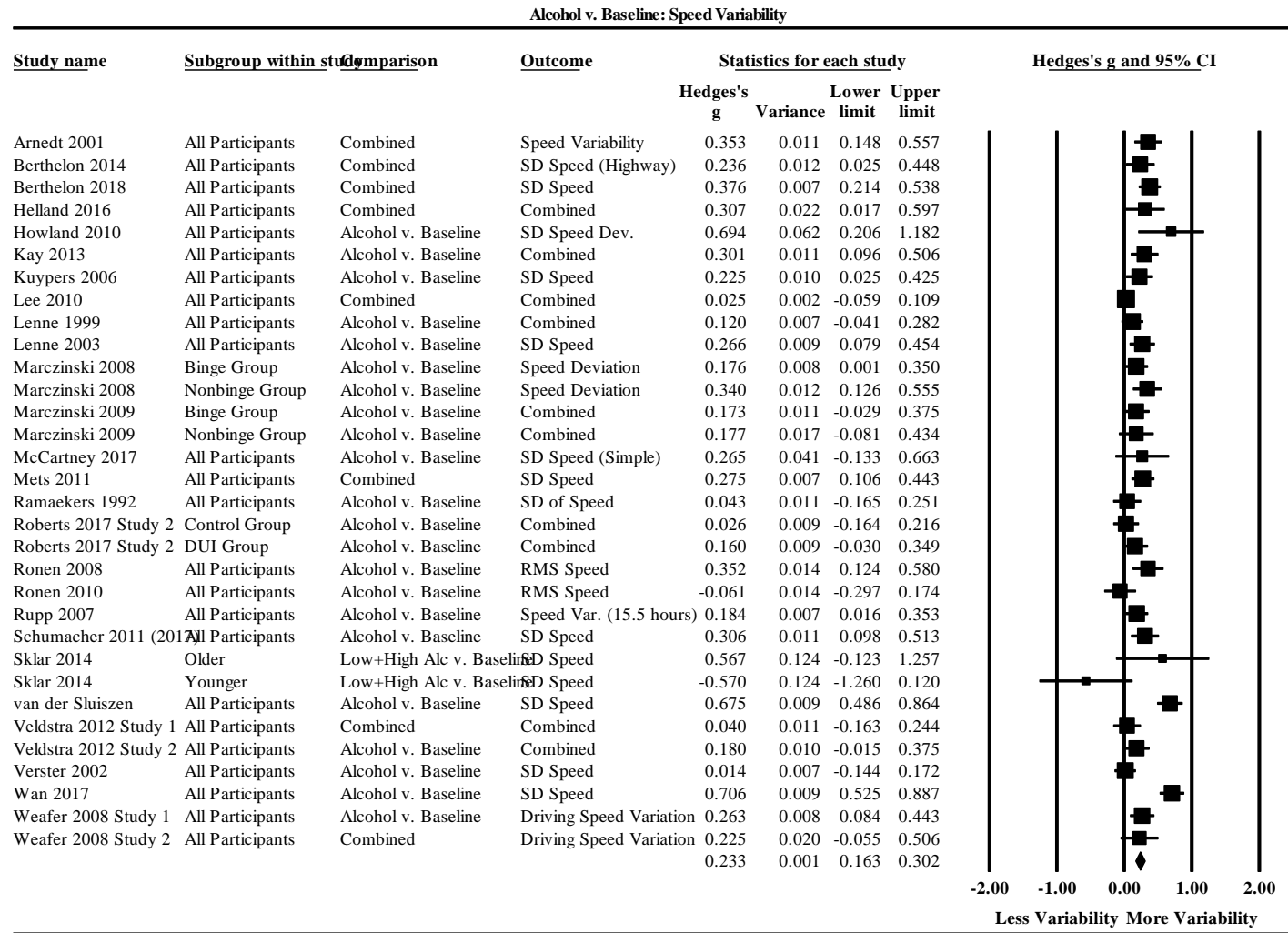


Figure C62. Forest plot illustrating *Alcohol v. Baseline: Speed Variability* (missing pre-post correlations set to $r = 0.9$).

Note that Schumacher et al. (2017) was eligible for inclusion in this meta-analysis but data was extracted and included from the 2011 paper.

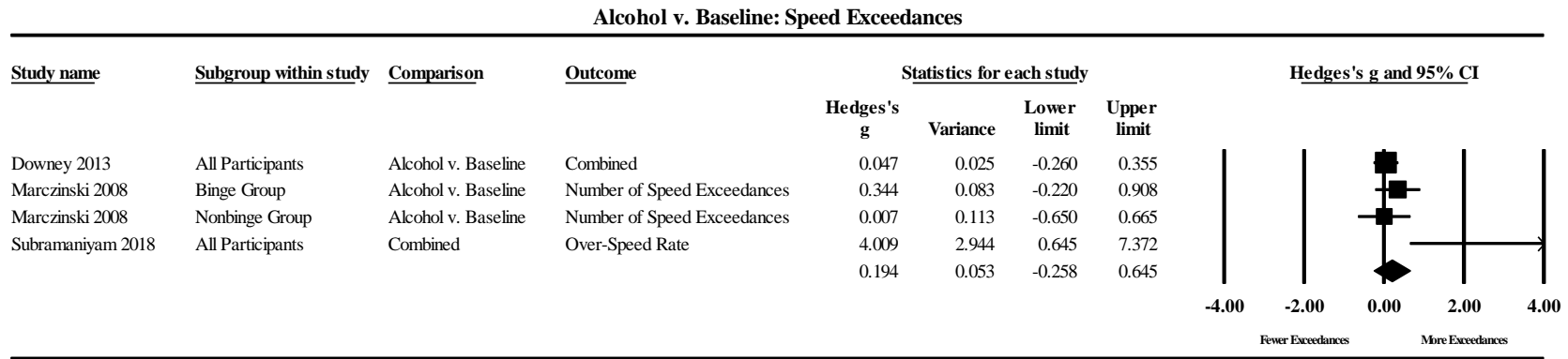


Figure C63. Forest plot illustrating *Alcohol v. Baseline: Speed Exceedances* (missing pre-post correlations set to $r = 0$).

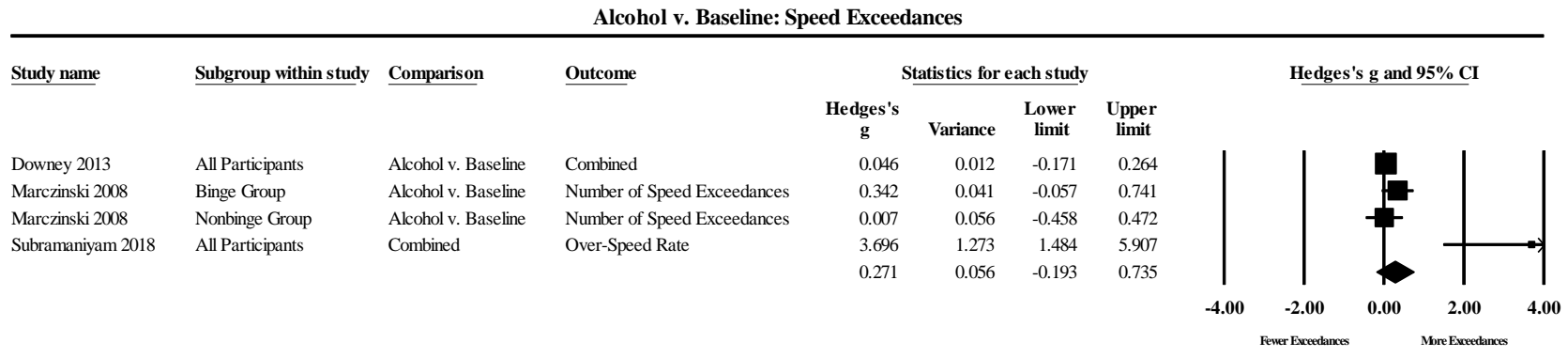


Figure C64. Forest plot illustrating *Alcohol v. Baseline: Speed Exceedances* (missing pre-post correlations set to $r = 0.5$).

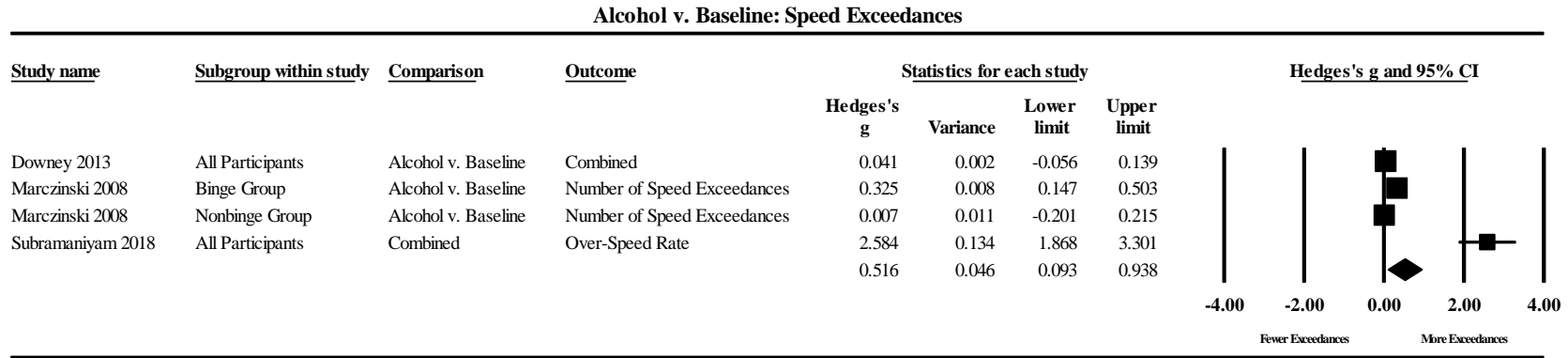


Figure C65. Forest plot illustrating *Alcohol v. Baseline: Speed Exceedances* (missing pre-post correlations set to $r = 0.9$).

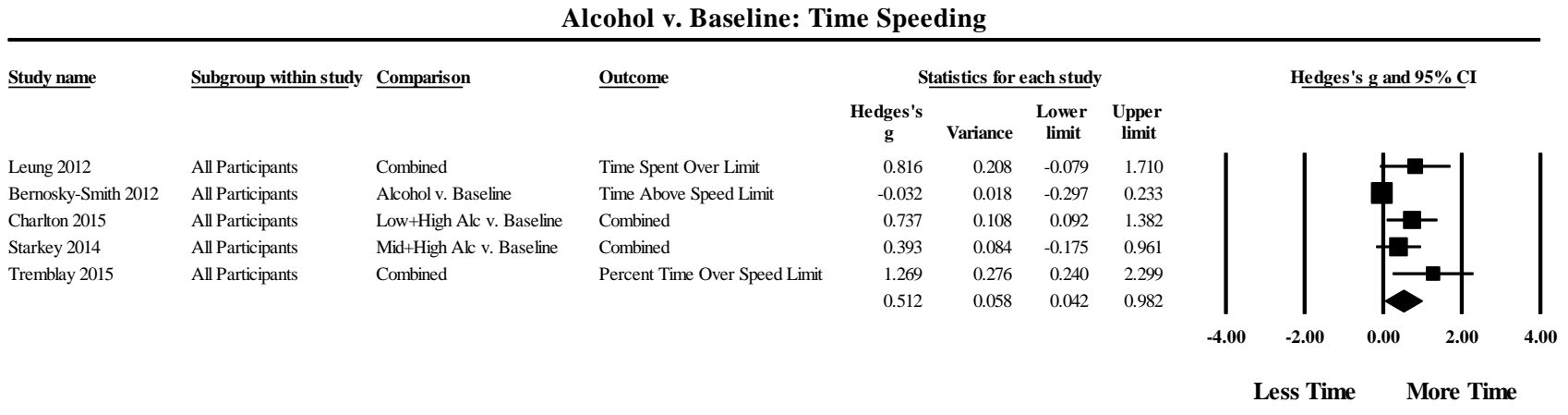


Figure C66. Forest plot illustrating *Alcohol v. Baseline: Time Speeding* (missing pre-post correlations set to $r = \text{zero}$).

Alcohol v. Baseline: Time Speeding

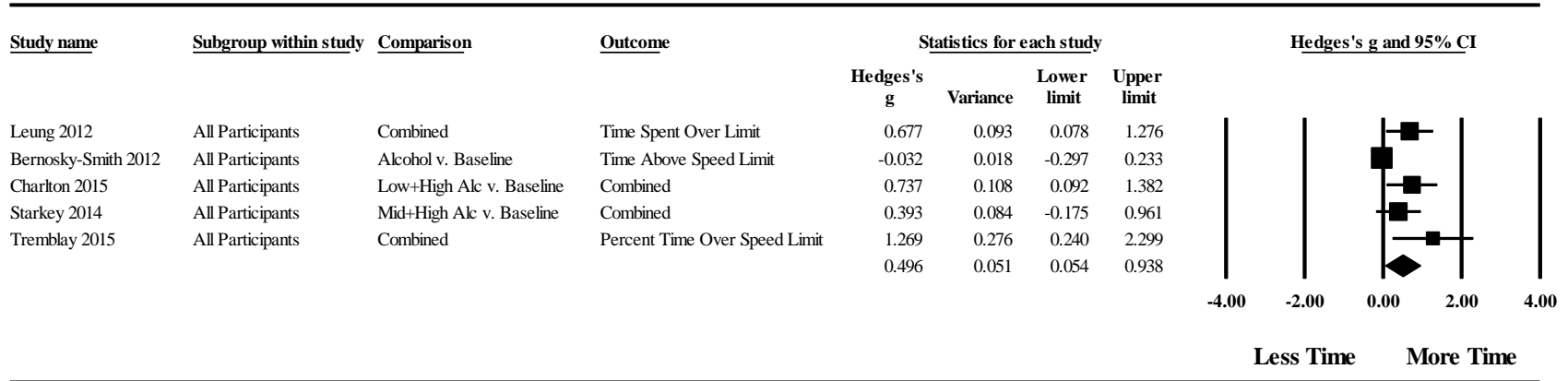


Figure C67. Forest plot illustrating *Alcohol v. Baseline: Time Speeding* (missing pre-post correlations set to $r = 0.5$).

Alcohol v. Baseline: Time Speeding

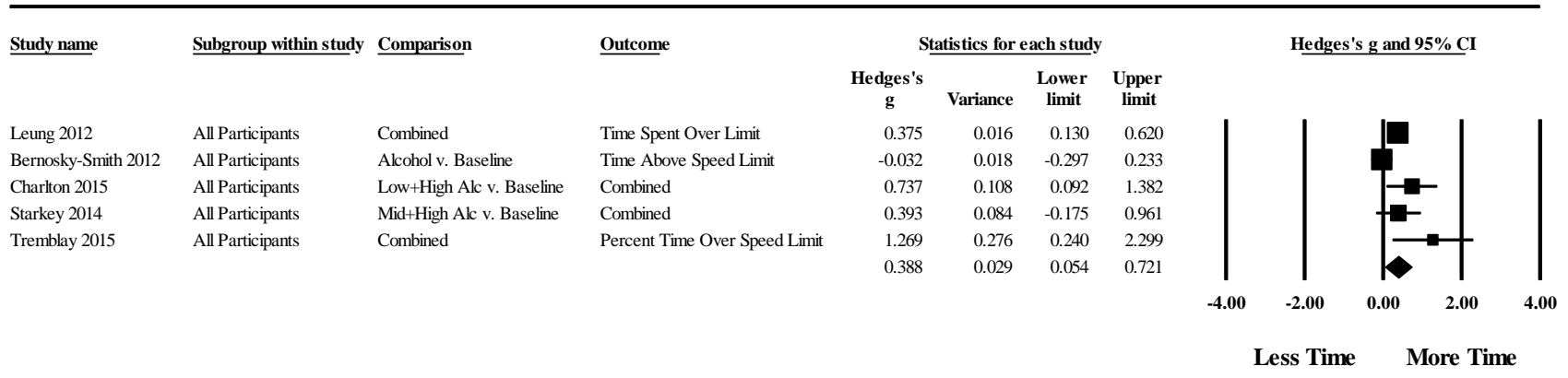


Figure C68. Forest plot illustrating *Alcohol v. Baseline: Time Speeding* (missing pre-post correlations set to $r = 0.9$).

Cannabis v. Alcohol: Crashes

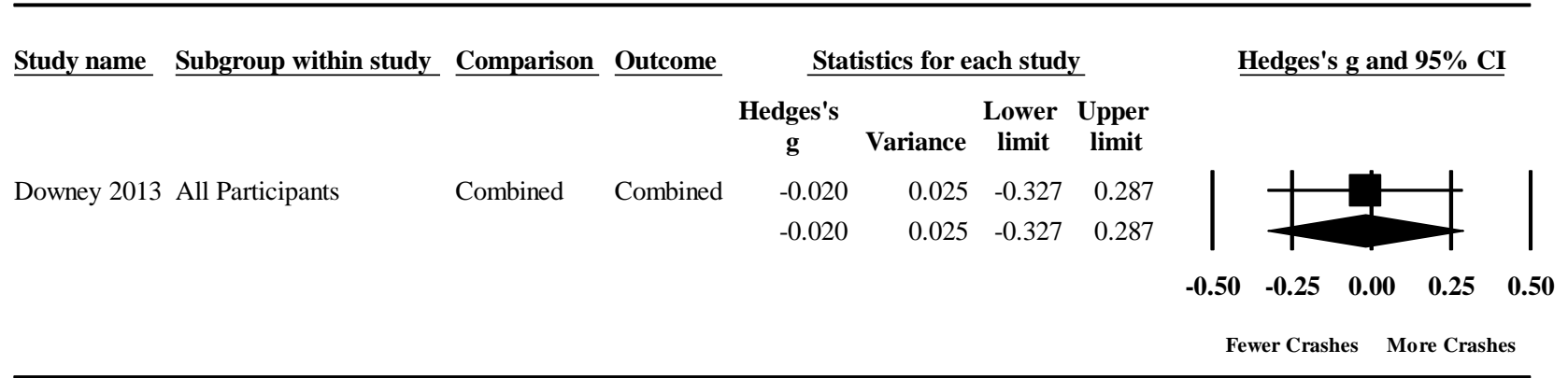


Figure C69. Forest plot illustrating *Cannabis v. Alcohol: Crashes* (missing pre-post correlations set to $r = 0$).

Cannabis v. Alcohol: Crashes

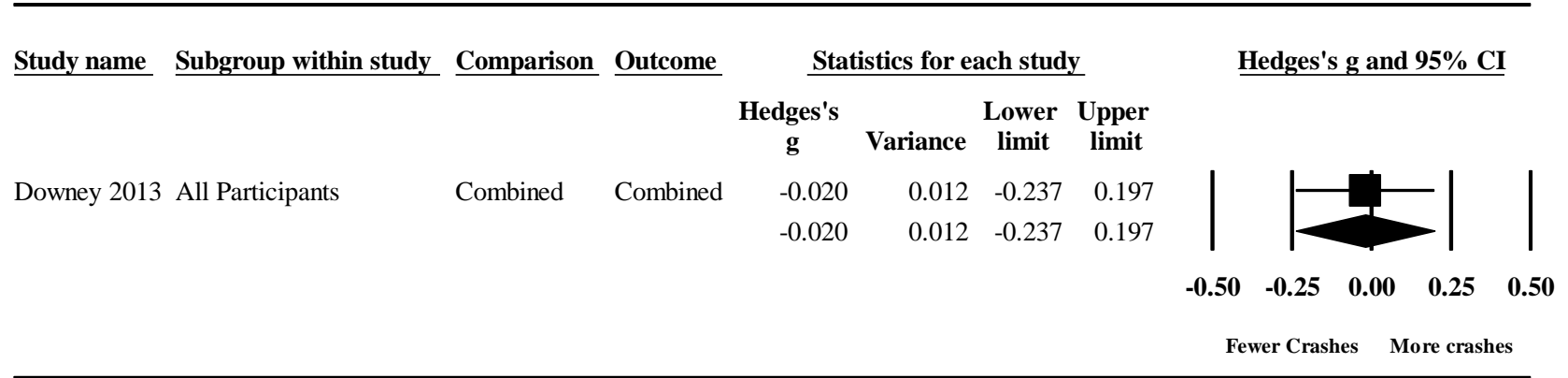


Figure C70. Forest plot illustrating *Cannabis v. Alcohol: Crashes* (missing pre-post correlations set to $r = 0.5$).

Cannabis v. Alcohol: Crashes

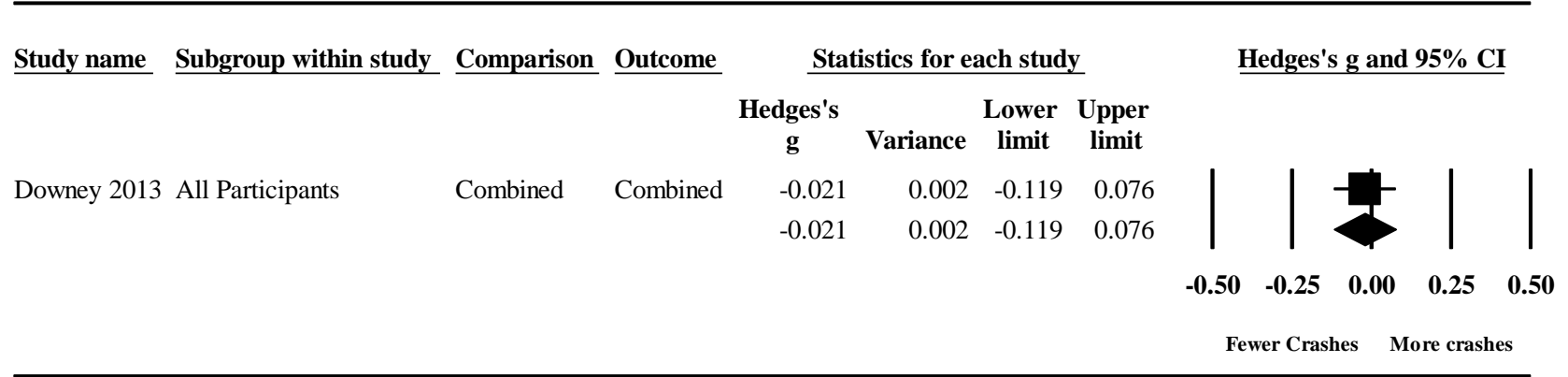


Figure C71. Forest plot illustrating Cannabis v. Alcohol: Crashes (missing pre-post correlations set to $r = 0.9$).

Cannabis v. Alcohol: Hazard RT

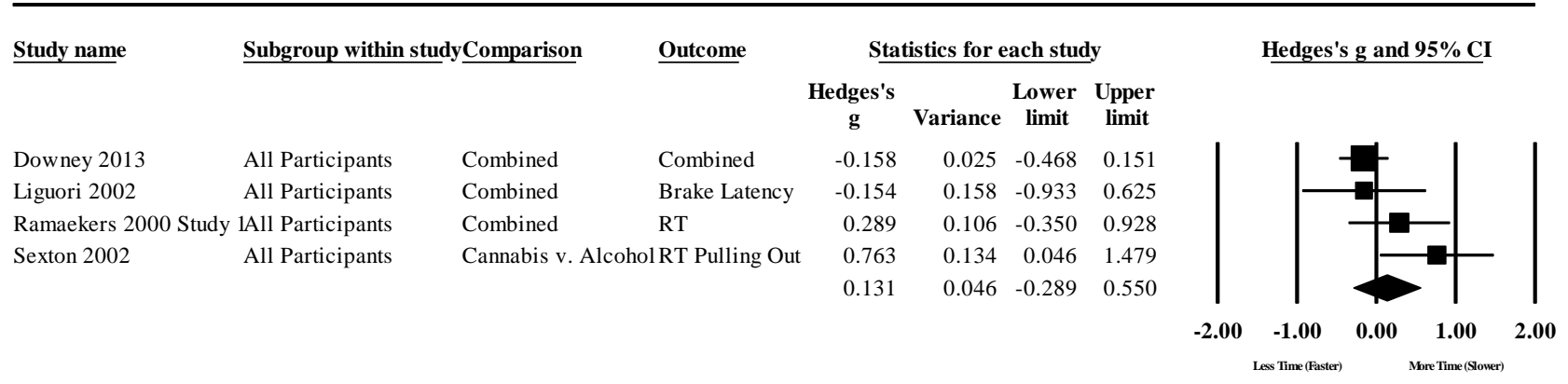


Figure C72. Forest plot illustrating Cannabis v. Alcohol: Hazard RT (missing pre-post correlations set to $r = \text{zero}$).

Cannabis v. Alcohol: Hazard RT

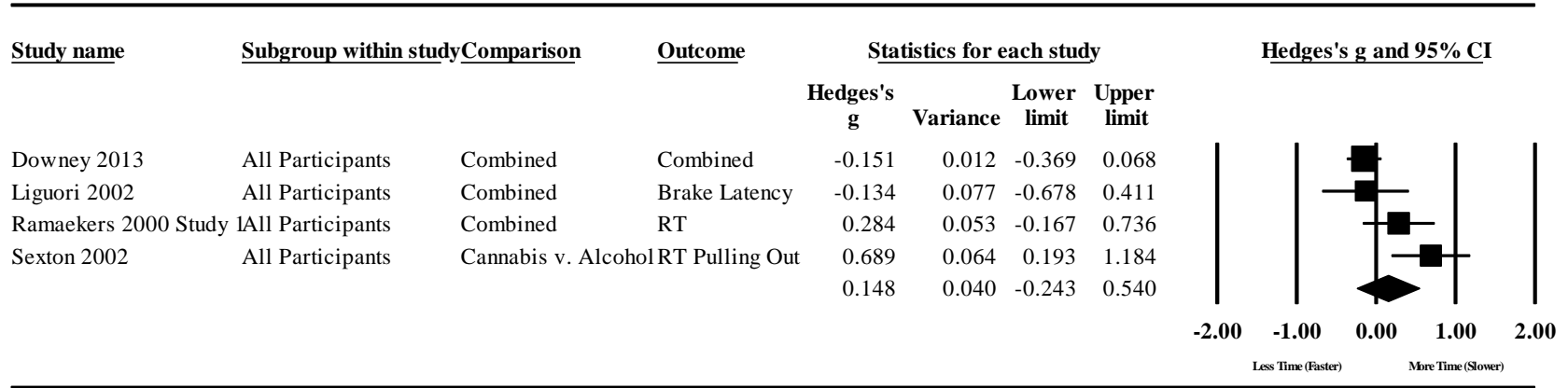


Figure C73. Forest plot illustrating Cannabis v. Alcohol: Hazard RT (missing pre-post correlations set to $r = 0.5$).

Cannabis v. Alcohol: Hazard RT

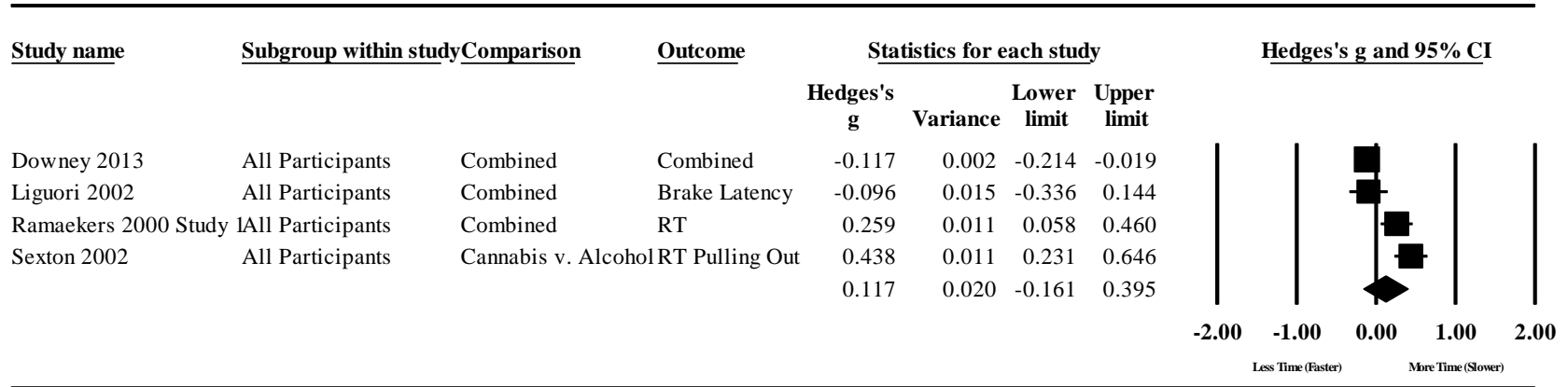


Figure C74. Forest plot illustrating Cannabis v. Alcohol: Hazard RT (missing pre-post correlations set to $r = 0.9$).

Cannabis v. Alcohol: Lateral Position Variability

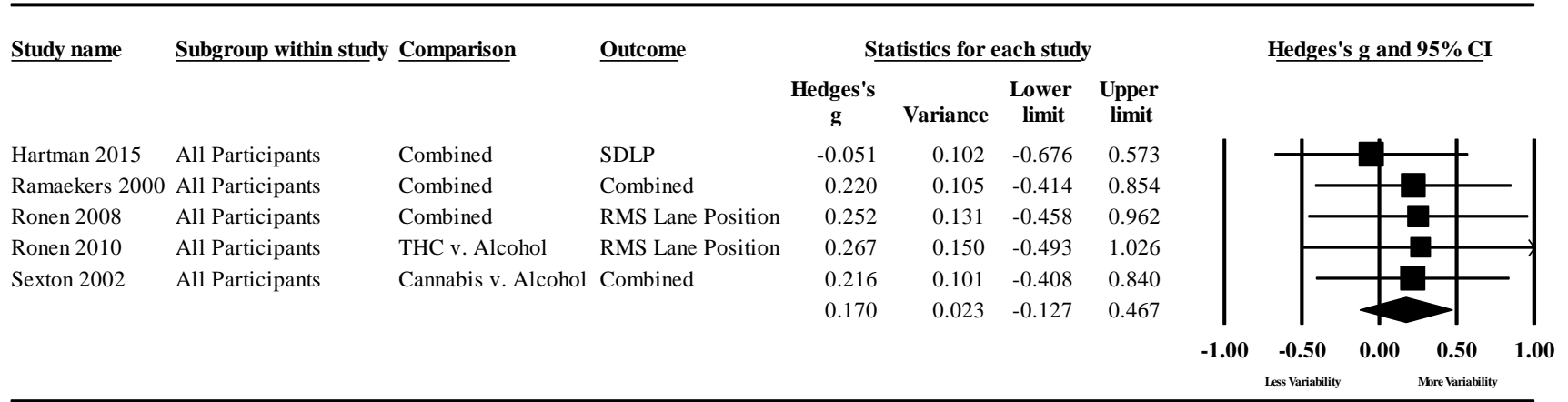


Figure C75. Forest plot illustrating Cannabis v. Alcohol: Lateral Position Variability (missing pre-post correlations set to $r = \text{zero}$).

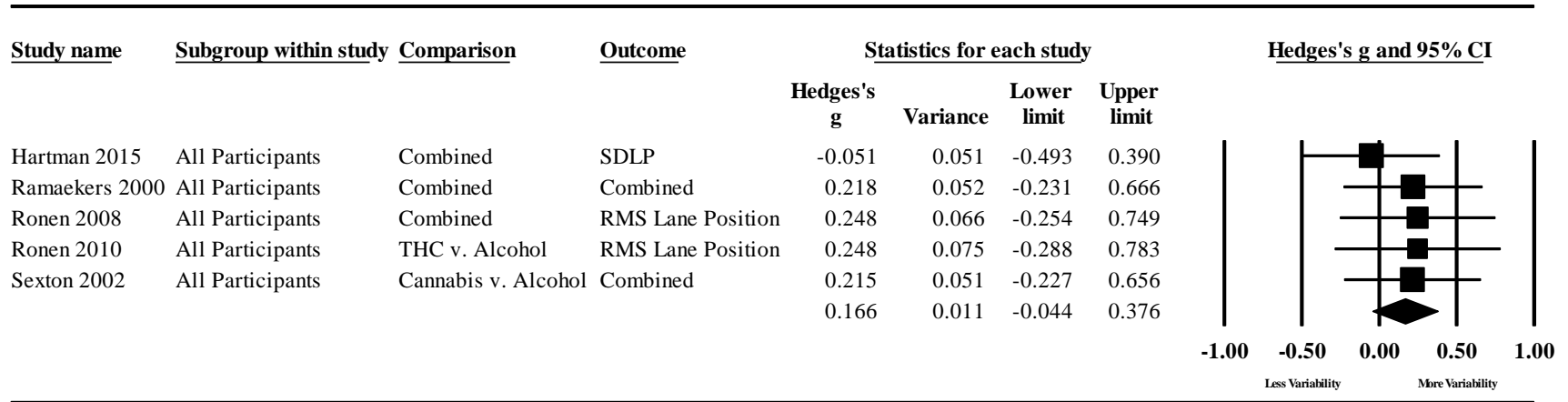


Figure C76. Forest plot illustrating Cannabis v. Alcohol: Lateral Position Variability (missing pre-post correlations set to $r = 0.5$).

Cannabis v. Alcohol: Lateral Position Variability

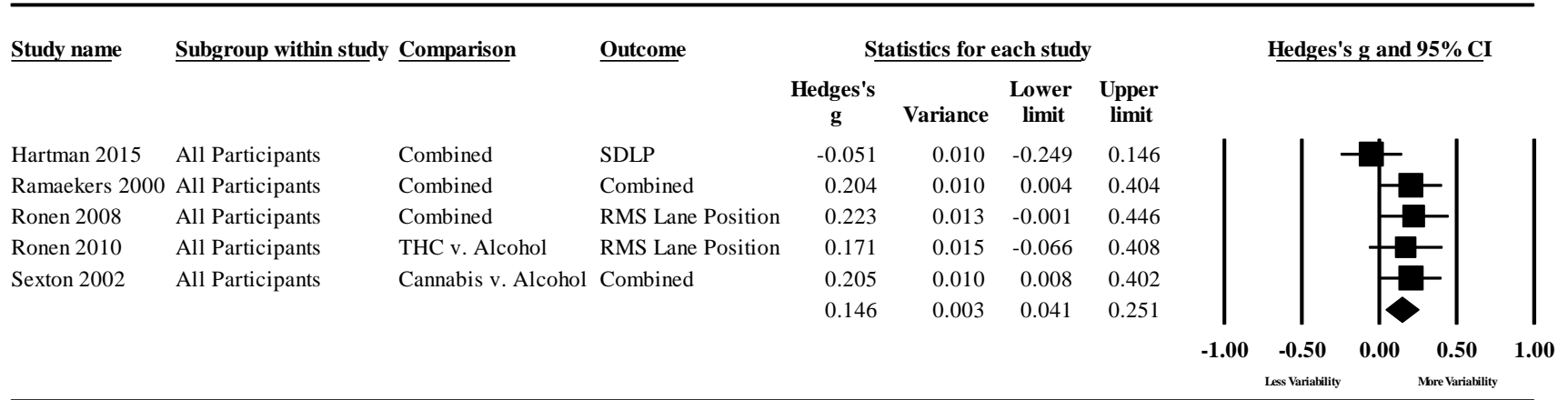


Figure C77. Forest plot illustrating Cannabis v. Alcohol: Lateral Position Variability (missing pre-post correlations set to $r = 0.9$).

Cannabis v. Alcohol: Lane Excursions

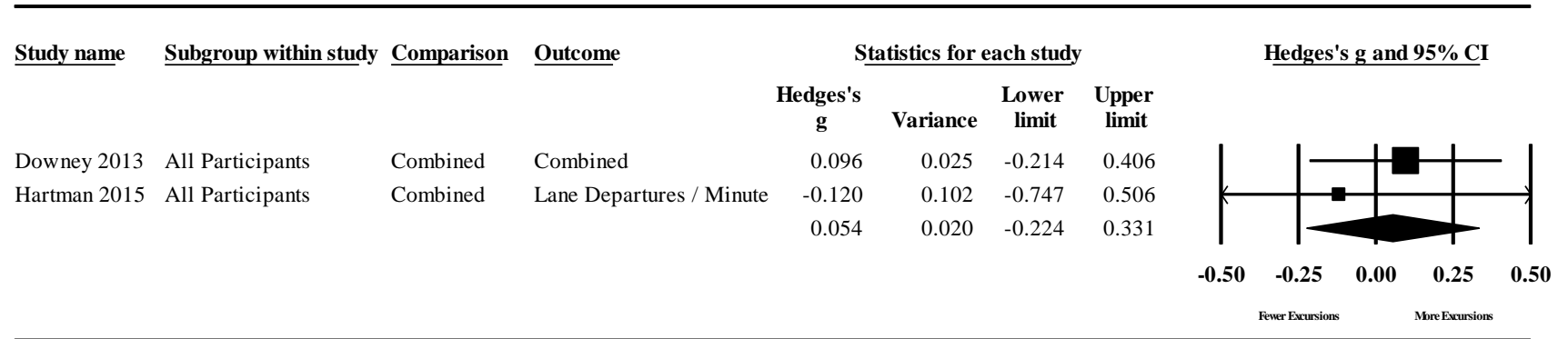


Figure C78. Forest plot illustrating Cannabis v. Alcohol: Lane Excursions (missing pre-post correlations set to $r = \text{zero}$).

Cannabis v. Alcohol: Lane Excursions

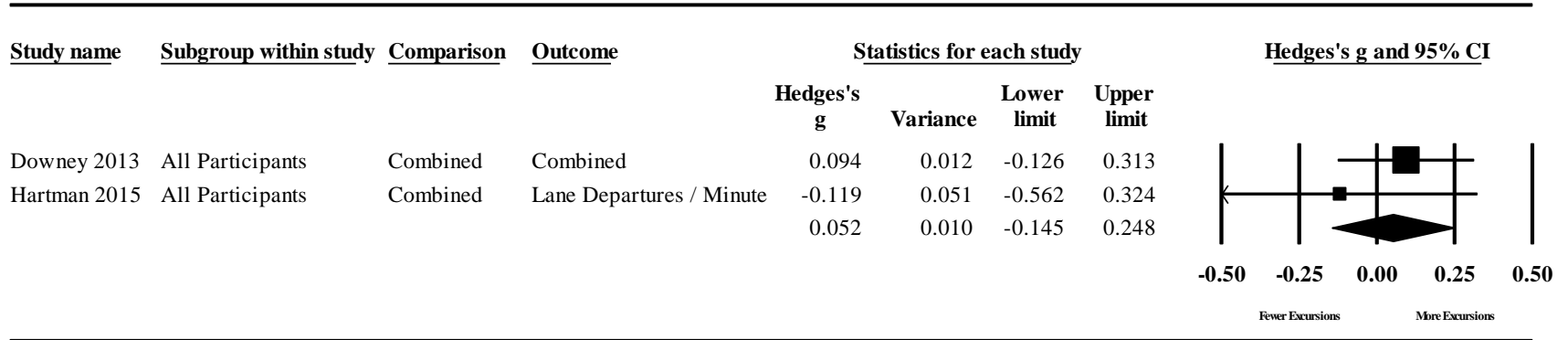


Figure C79. Forest plot illustrating Cannabis v. Alcohol: Lane Excursions (missing pre-post correlations set to $r = 0.5$).

Cannabis v. Alcohol: Lane Excursions

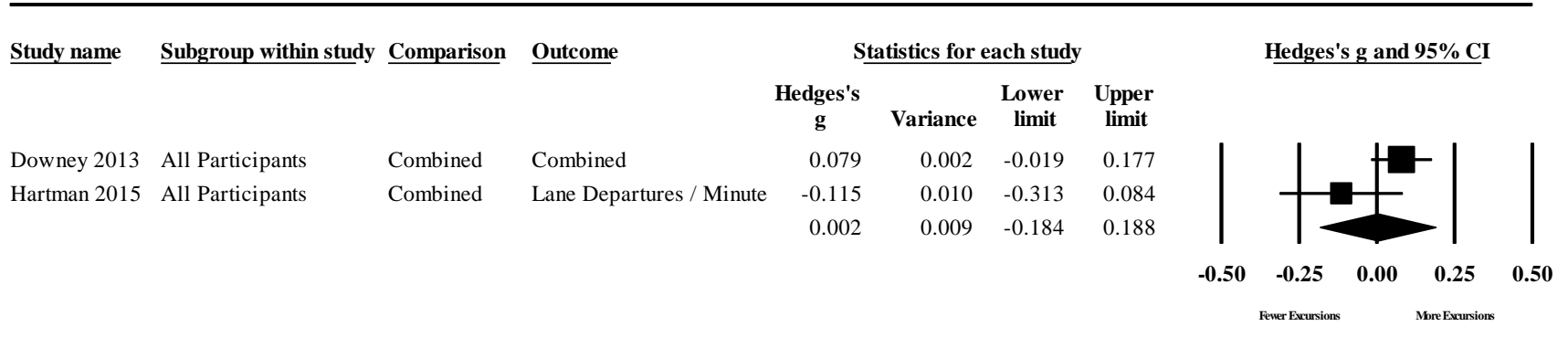


Figure C80. Forest plot illustrating Cannabis v. Alcohol: Lane Excursions (missing pre-post correlations set to $r = 0.9$).

Cannabis v. Alcohol: Time Out of Lane

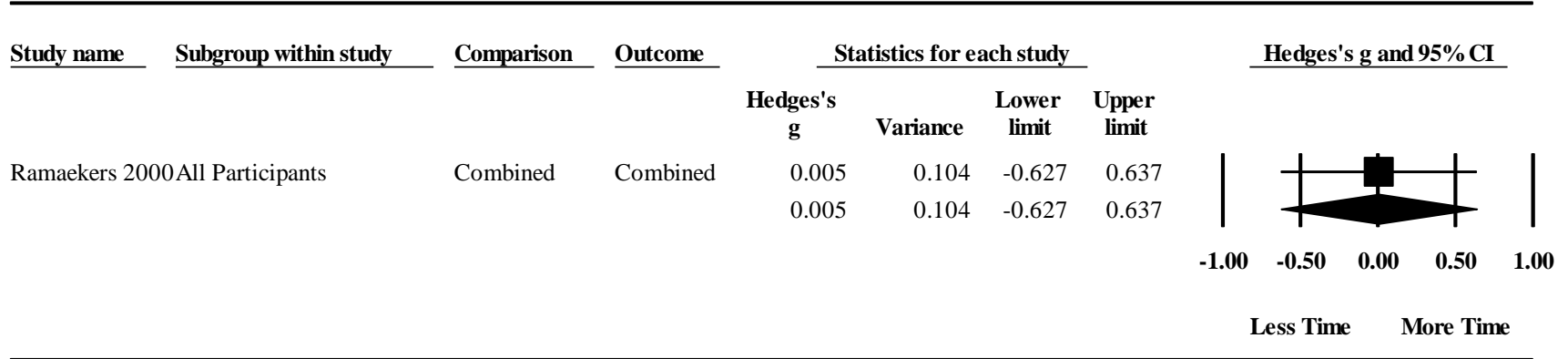


Figure C81. Forest plot illustrating Cannabis v. Alcohol: Time Out of Lane (missing pre-post correlations set to $r = \text{zero}$).

Cannabis v. Alcohol: Time Out of Lane

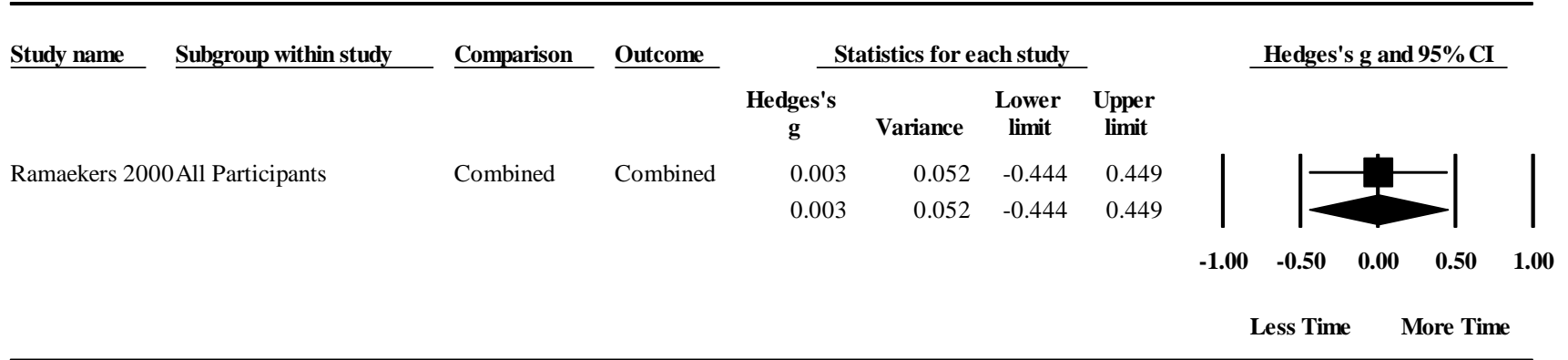


Figure C82. Forest plot illustrating Cannabis v. Alcohol: Time Out of Lane (missing pre-post correlations set to $r = 0.5$).

Cannabis v. Alcohol: Time Out of Lane

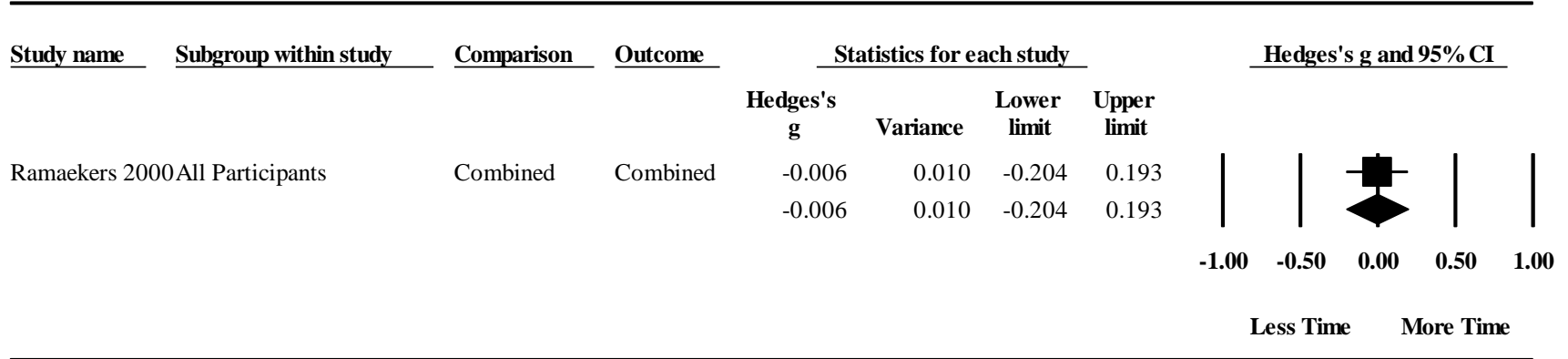


Figure C83. Forest plot illustrating *Cannabis v. Alcohol: Time Out of Lane* (missing pre-post correlations set to $r = 0.9$).

Cannabis v. Alcohol: Speed

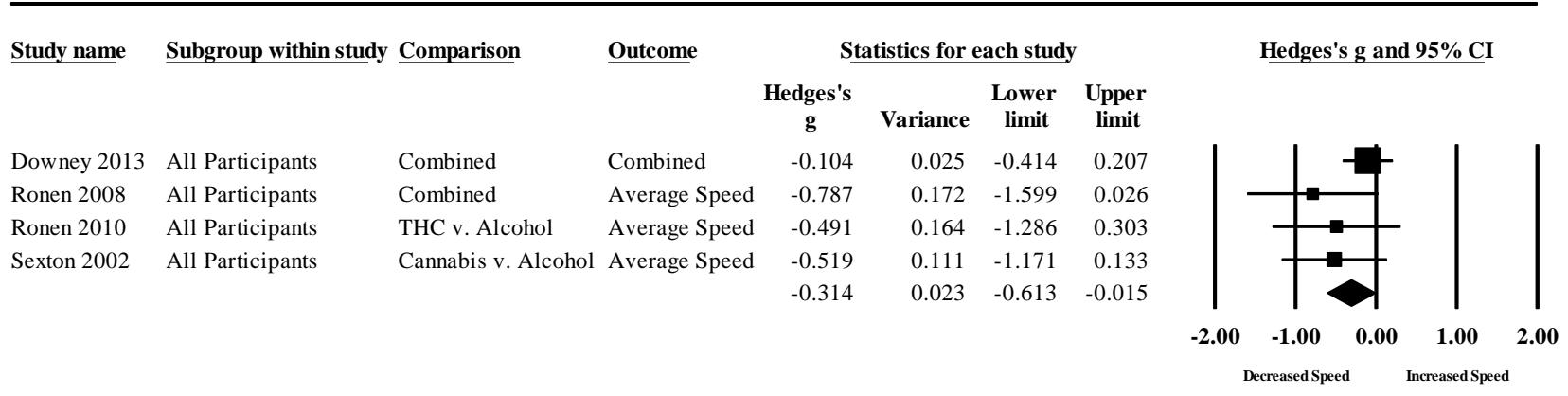


Figure C84. Forest plot illustrating *Cannabis v. Alcohol: Speed* (missing pre-post correlations set to $r = \text{zero}$).

Cannabis v. Alcohol: Speed

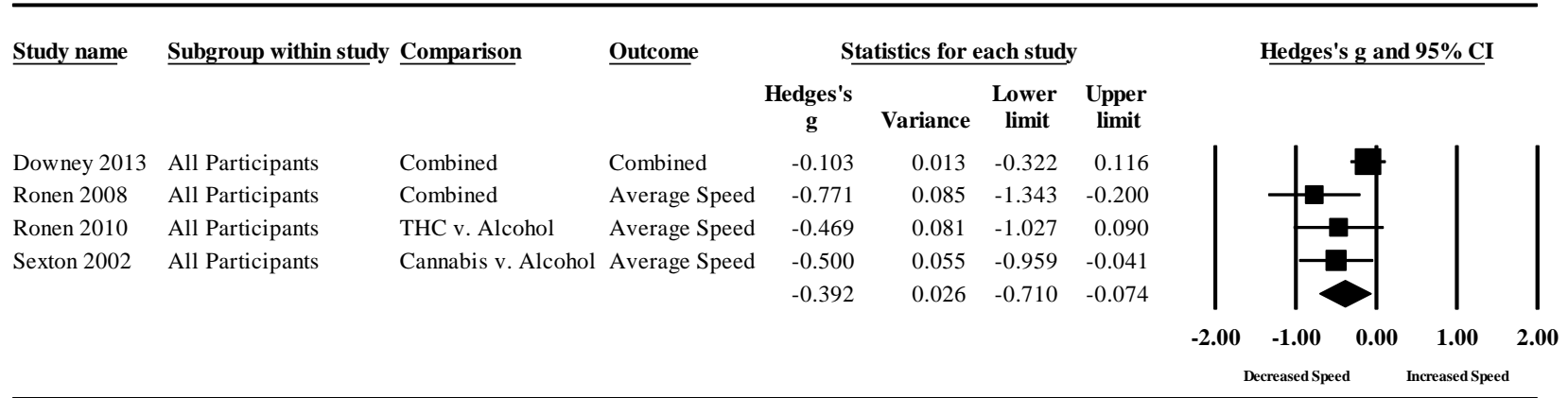


Figure C85. Forest plot illustrating Cannabis v. Alcohol: Speed (missing pre-post correlations set to $r = 0.5$).

Cannabis v. Alcohol: Speed

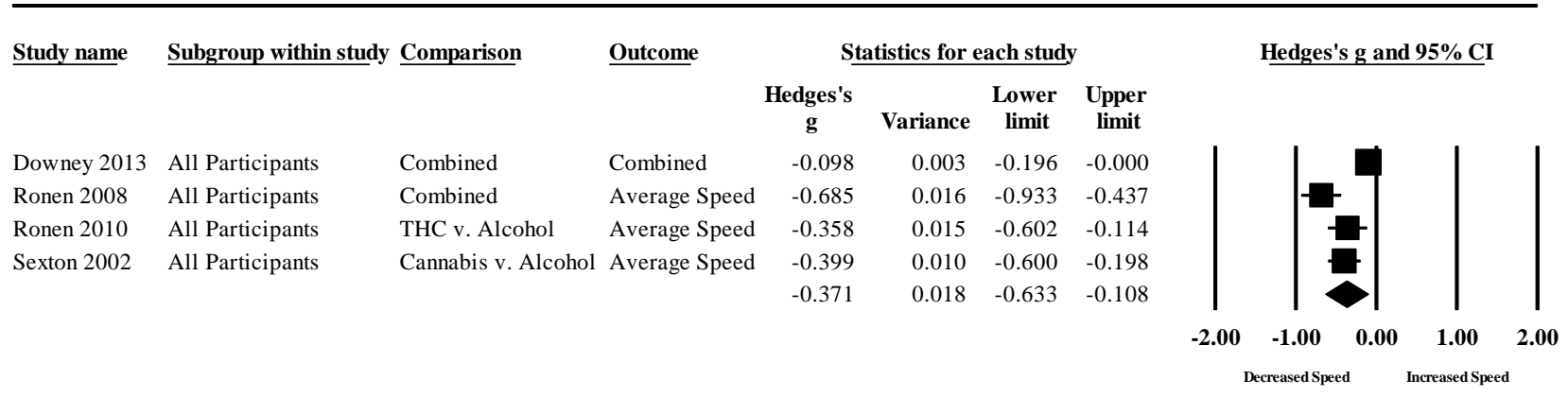


Figure C86. Forest plot illustrating Cannabis v. Alcohol: Speed (missing pre-post correlations set to $r = 0.9$).

Cannabis v. Alcohol: Speed Variability

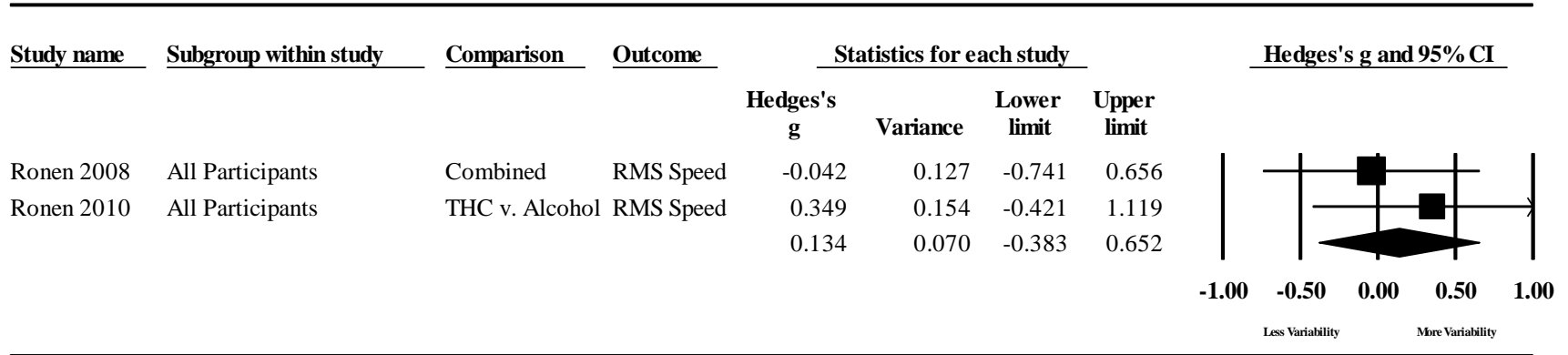


Figure C87. Forest plot illustrating *Cannabis v. Alcohol: Speed Variability* (missing pre-post correlations set to $r = \text{zero}$).

Cannabis v. Alcohol: Speed Variability

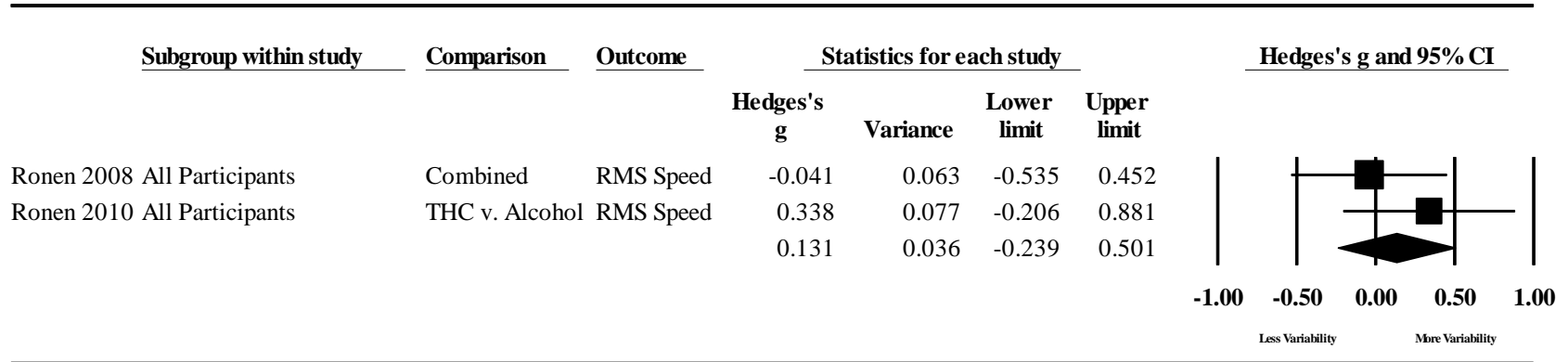


Figure C88. Forest plot illustrating *Cannabis v. Alcohol: Speed Variability* (missing pre-post correlations set to $r = 0.5$).

Cannabis v. Alcohol: Speed Variability

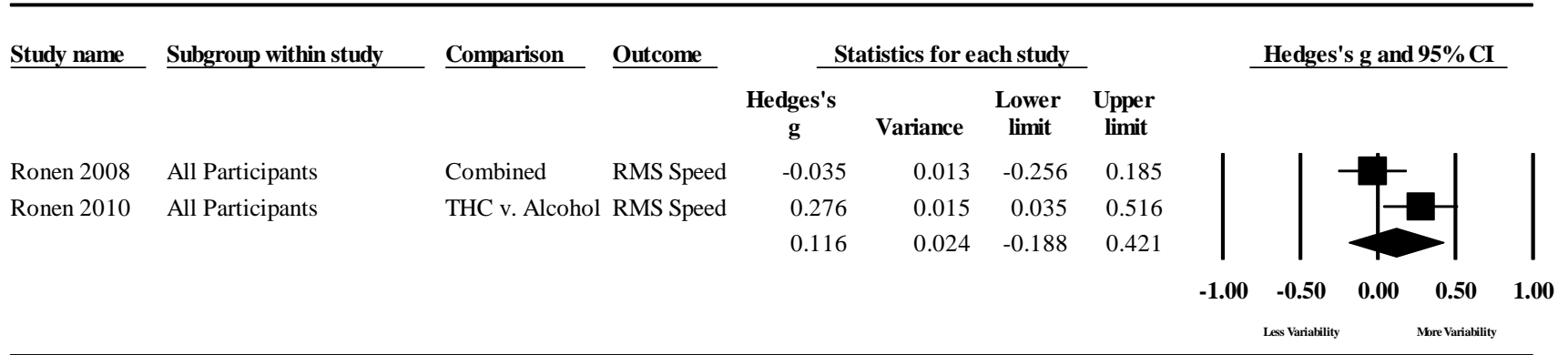


Figure C89. Forest plot illustrating *Cannabis v. Alcohol: Speed Variability* (missing pre-post correlations set to $r = 0.9$).

Cannais v. Alcohol: Speed Exceedances

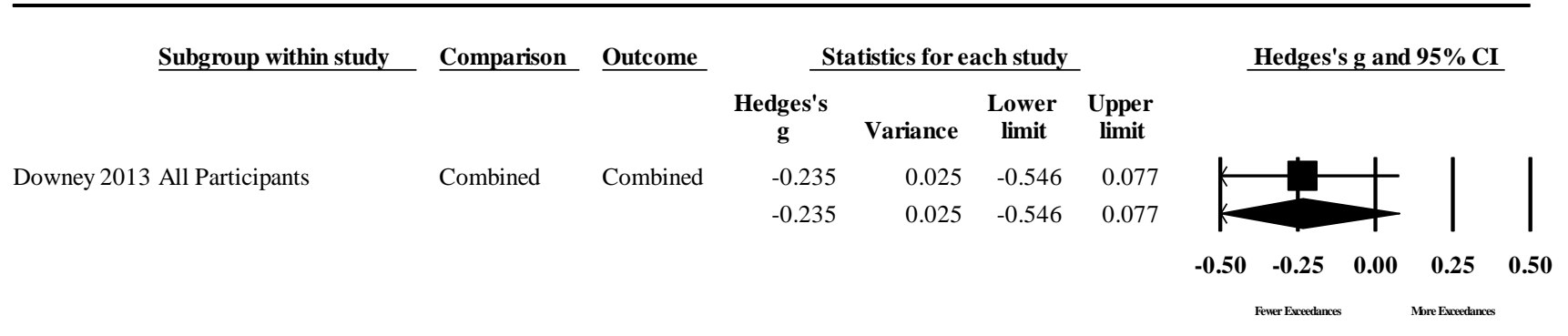


Figure C90. Forest plot illustrating *Cannabis v. Alcohol: Speed Exceedances* (missing pre-post correlations set to $r = \text{zero}$).

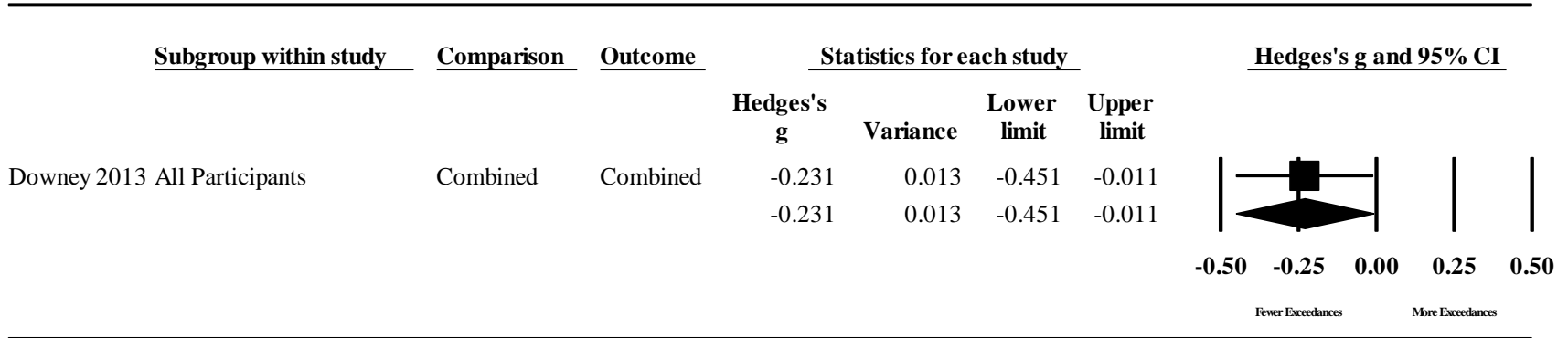


Figure C91. Forest plot illustrating *Cannabis v. Alcohol: Speed Exceedances* (missing pre-post correlations set to $r = 0.5$).

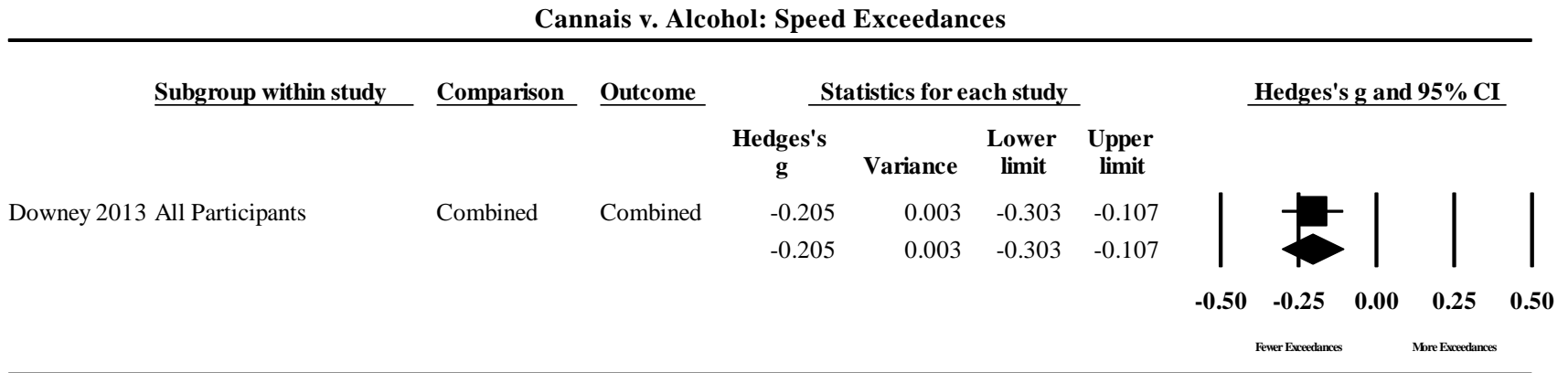


Figure C92. Forest plot illustrating *Cannabis v. Alcohol: Speed Exceedances* (missing pre-post correlations set to $r = 0.9$).

Combination v. Baseline: Crashes

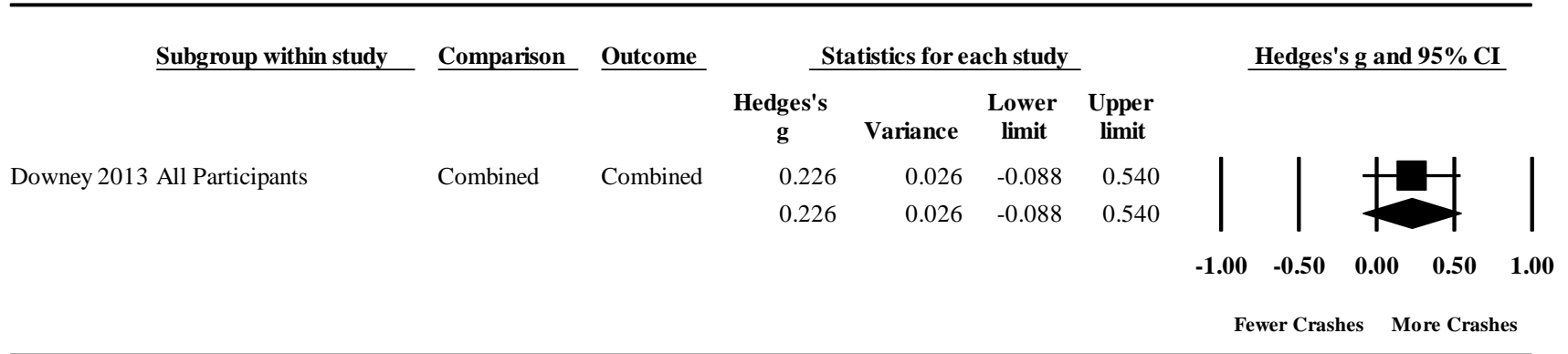


Figure C93. Forest plot illustrating *Combination v. Baseline: Crashes* (missing pre-post correlations set to $r = 0$).

Combination v. Baseline: Crashes

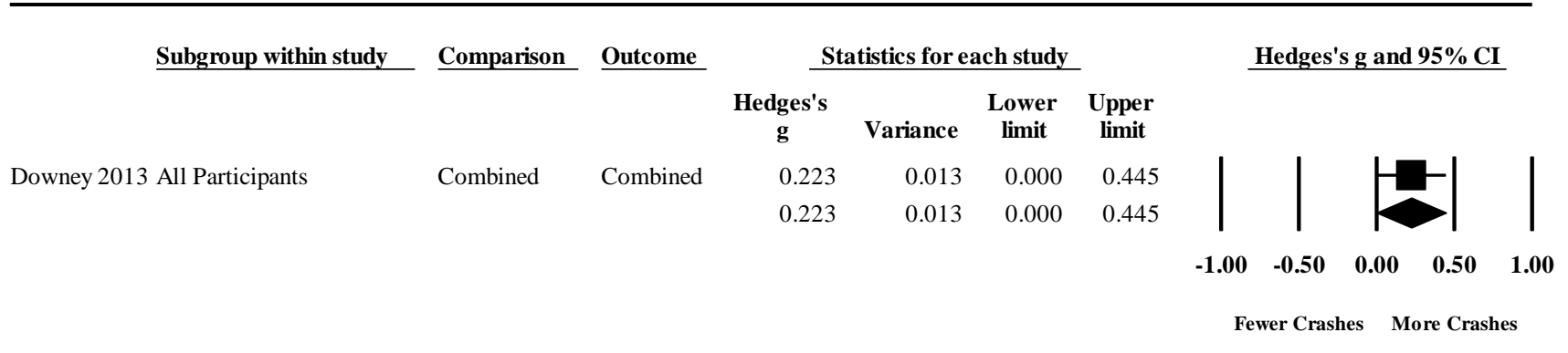


Figure C94. Forest plot illustrating *Combination v. Baseline: Crashes* (missing pre-post correlations set to $r = 0.5$).

Combination v. Baseline: Crashes

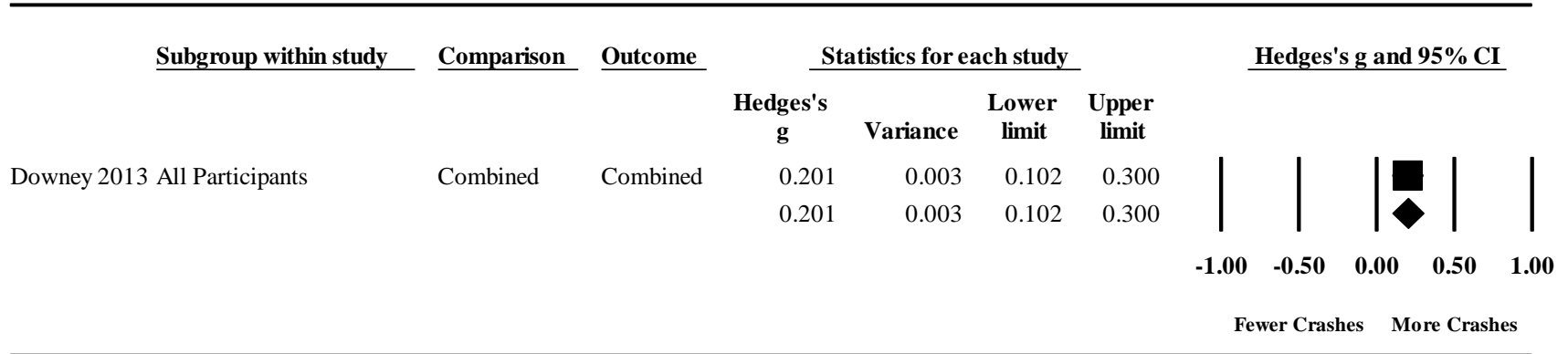


Figure C95. Forest plot illustrating *Combination v. Baseline: Crashes* (missing pre-post correlations set to $r = 0.9$).

Combination v. Baseline: Hazard RT

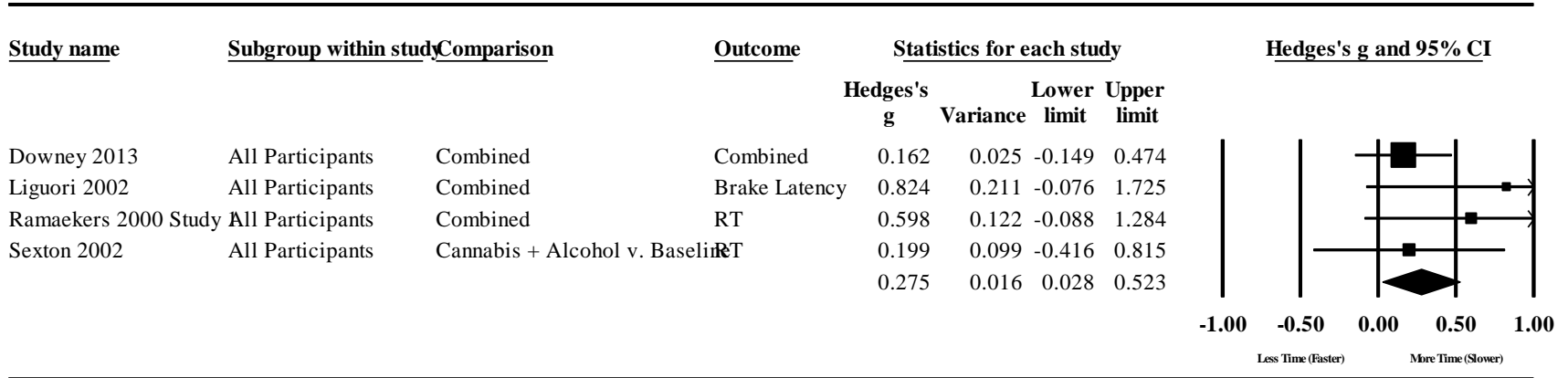


Figure C96. Forest plot illustrating *Combination v. Baseline: Hazard RT* (missing pre-post correlations set to $r = \text{zero}$).

Combination v. Baseline: Hazard RT

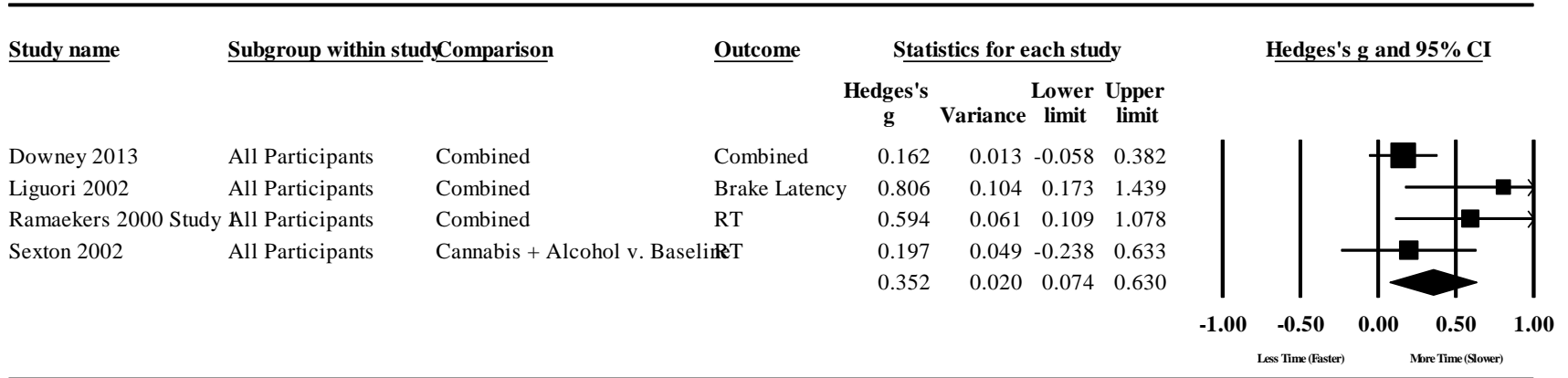


Figure C97. Forest plot illustrating *Combination v. Baseline: Hazard RT* (missing pre-post correlations set to $r = 0.5$).

Combination v. Baseline: Hazard RT

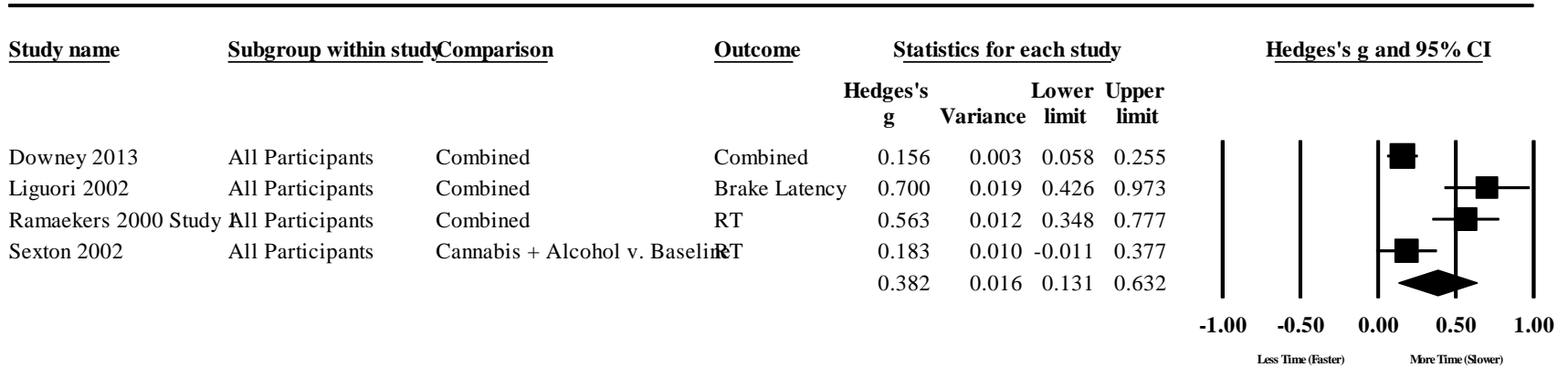


Figure C98. Forest plot illustrating *Combination v. Baseline: Hazard RT* (missing pre-post correlations set to $r = 0.9$).

Combination v. Baseline: Lateral Position Variability

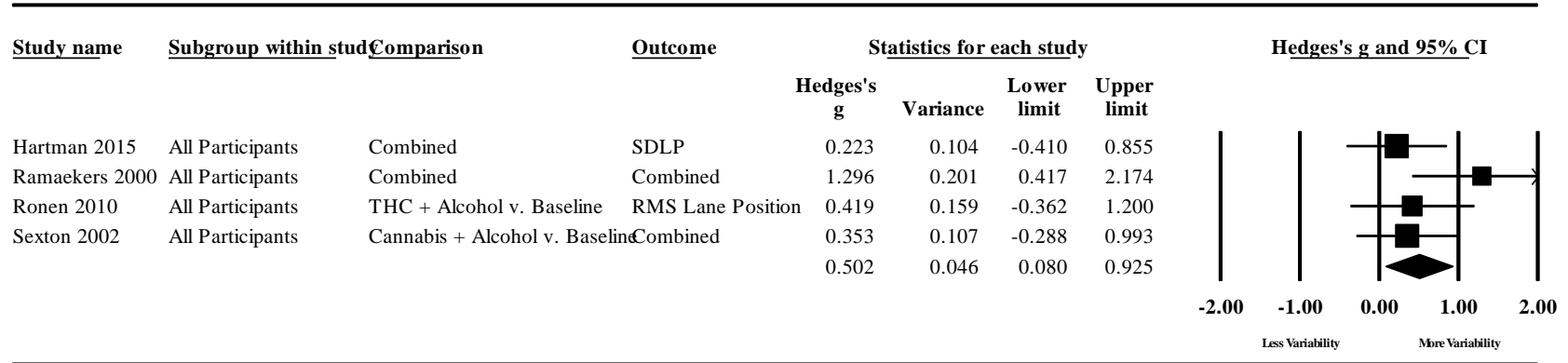


Figure C99. Forest plot illustrating *Combination v. Baseline: Lateral Position Variability* (missing pre-post correlations set to $r =$ zero).

Combination v. Baseline: Lateral Position Variability

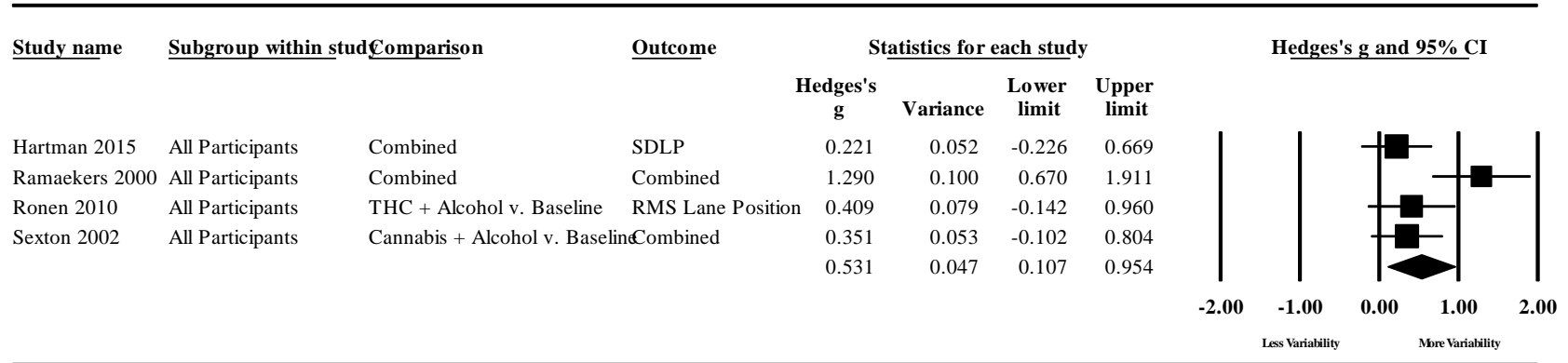


Figure C100. Forest plot illustrating *Combination v. Baseline: Lateral Position Variability* (missing pre-post correlations set to $r =$ 0.5).

Combination v. Baseline: Lateral Position Variability

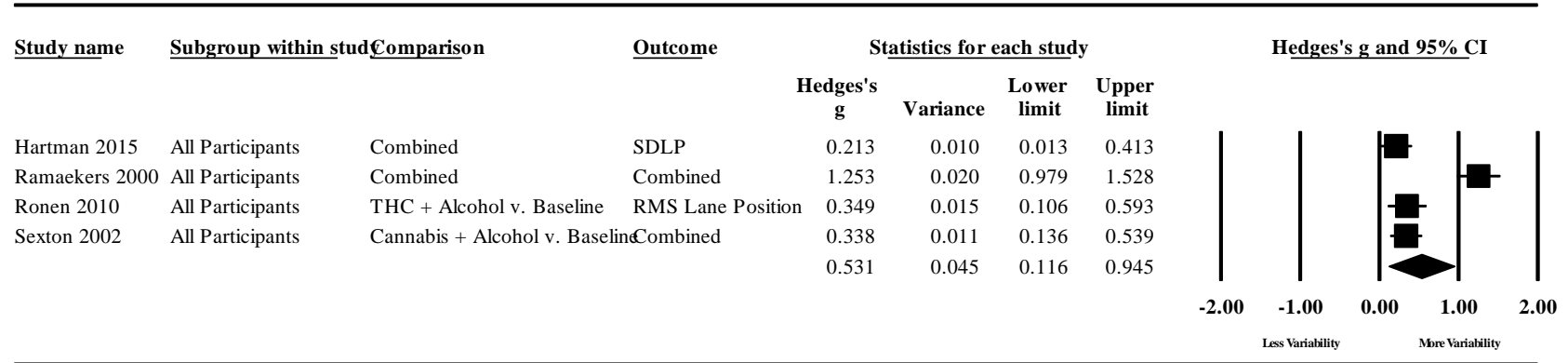


Figure C101. Forest plot illustrating *Combination v. Baseline: Lateral Position Variability* (missing pre-post correlations set to $r = 0.9$).

Combination v. Baseline: Lane Excursions

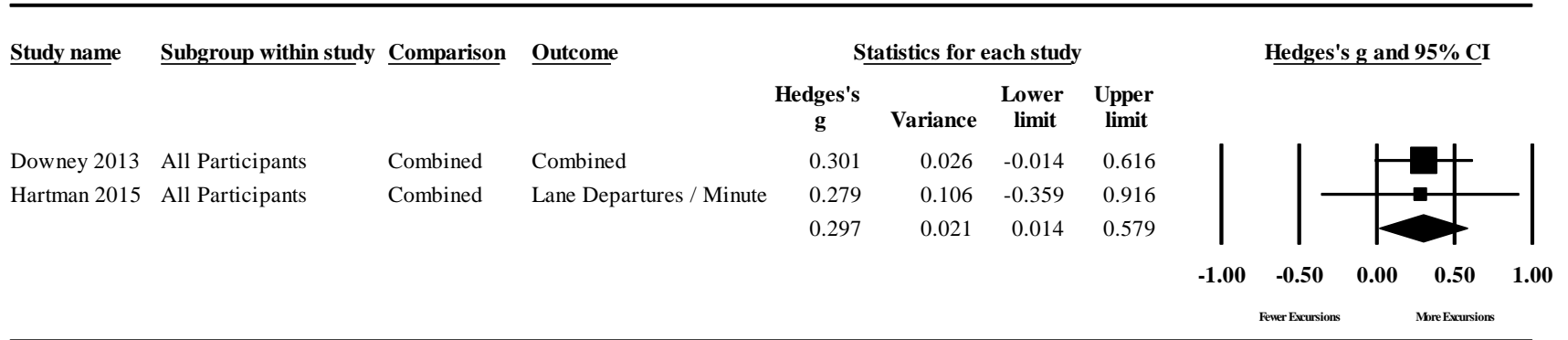


Figure C102. Forest plot illustrating *Combination v. Baseline: Lane Excursions* (missing pre-post correlations set to $r = \text{zero}$).

Combination v. Baseline: Lane Excursions

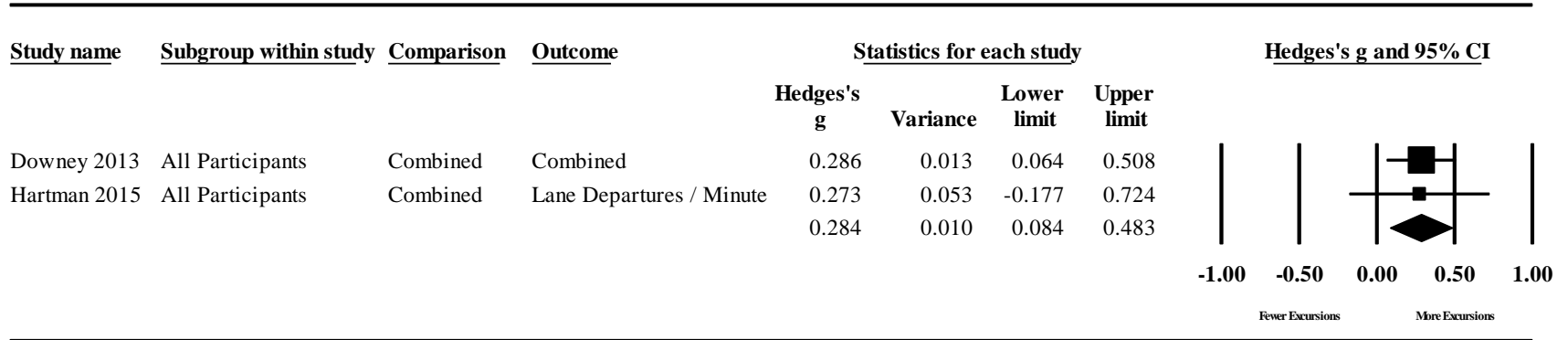


Figure C103. Forest plot illustrating *Combination v. Baseline: Lane Excursions* (missing pre-post correlations set to $r = 0.5$).

Combination v. Baseline: Lane Excursions

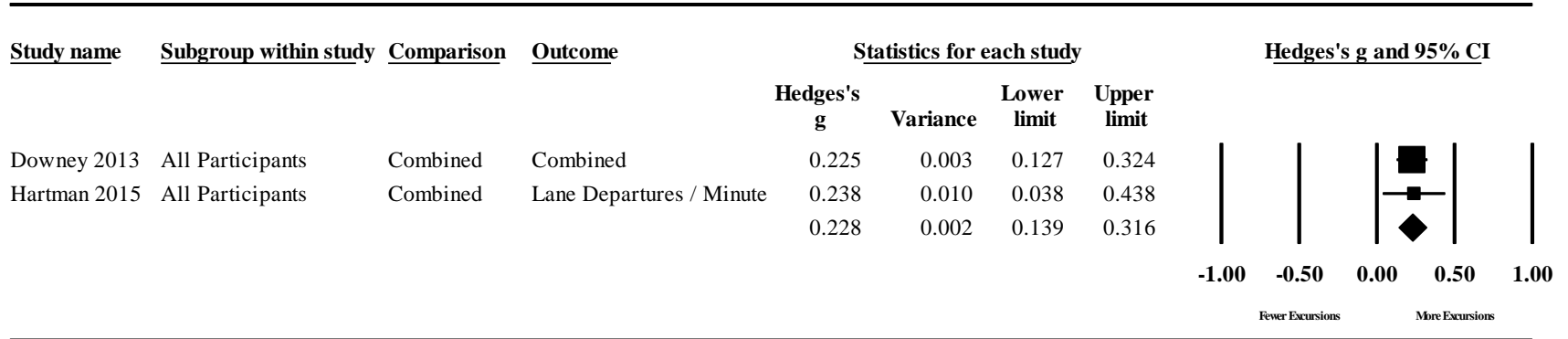


Figure C104. Forest plot illustrating *Combination v. Baseline: Lane Excursions* (missing pre-post correlations set to $r = 0.9$).

Combination v. Baseline: Speed

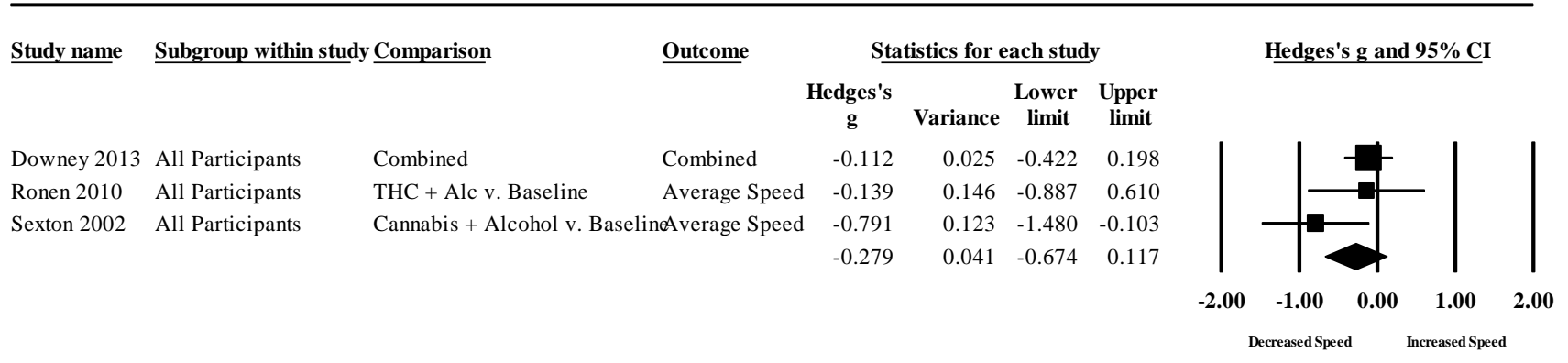


Figure C105. Forest plot illustrating *Combination v. Baseline: Speed* (missing pre-post correlations set to $r = 0$).

Combination v. Baseline: Speed

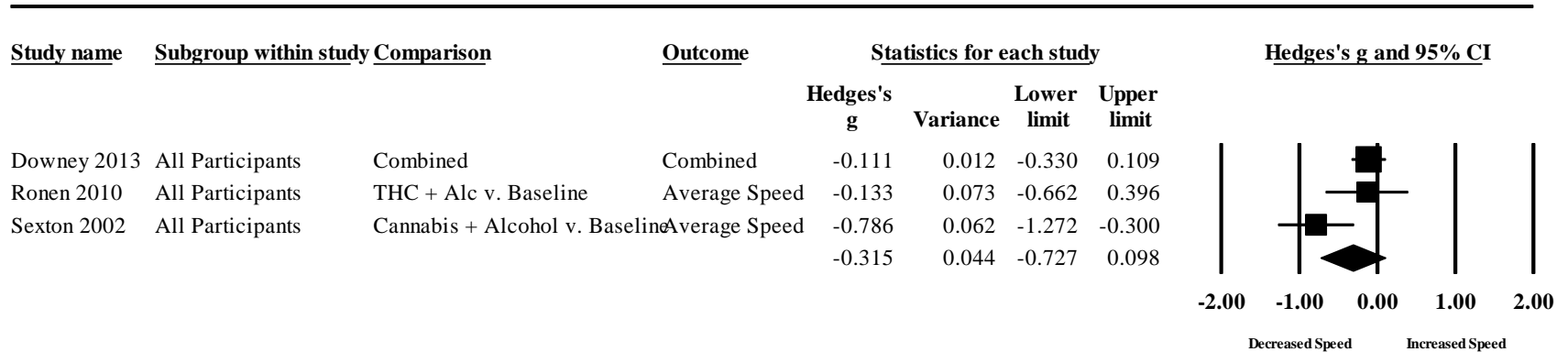


Figure C106. Forest plot illustrating *Combination v. Baseline: Speed* (missing pre-post correlations set to $r = 0.5$).

Combination v. Baseline: Speed

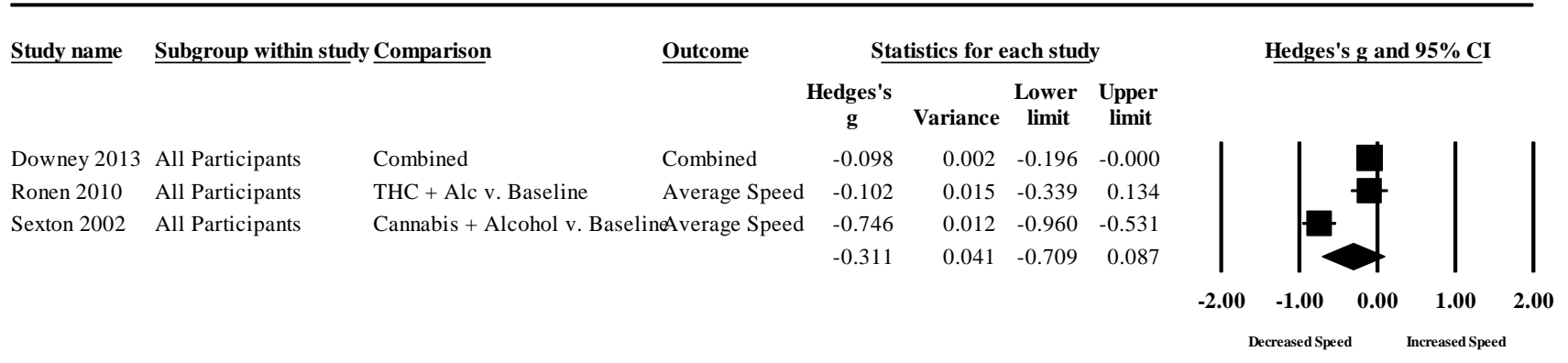


Figure C107. Forest plot illustrating *Combination v. Baseline: Speed* (missing pre-post correlations set to $r = 0.9$)

Combination v. Baseline: Speed Variability

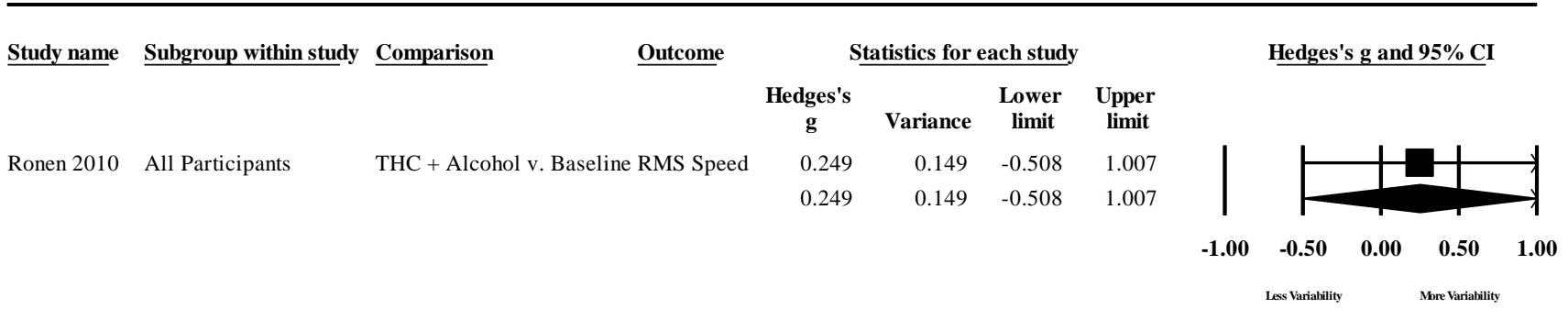


Figure C108. Forest plot illustrating *Combination v. Baseline: Speed Variability* (missing pre-post correlations set to $r = zero$).

Combination v. Baseline: Speed Variability

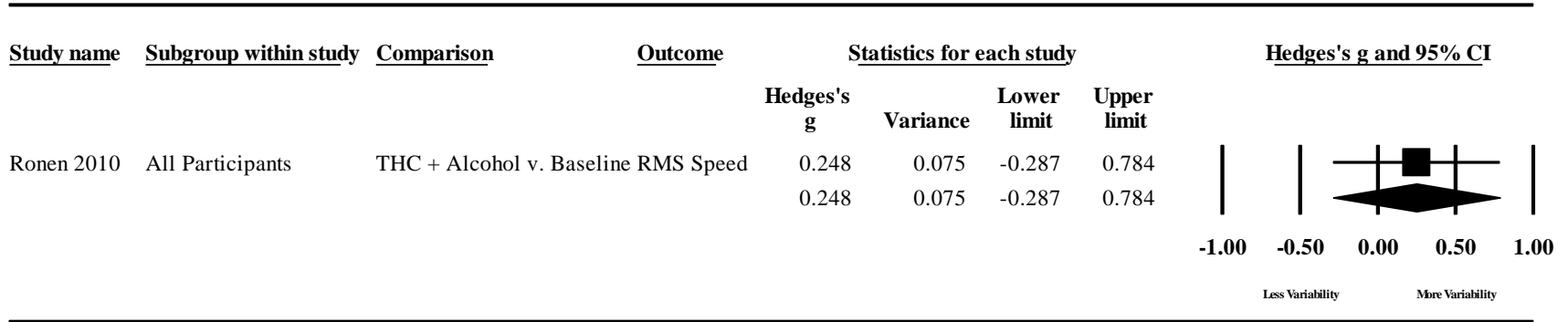


Figure C109. Forest plot illustrating *Combination v. Baseline: Speed Variability* (missing pre-post correlations set to $r = 0.5$).

Combination v. Baseline: Speed Variability

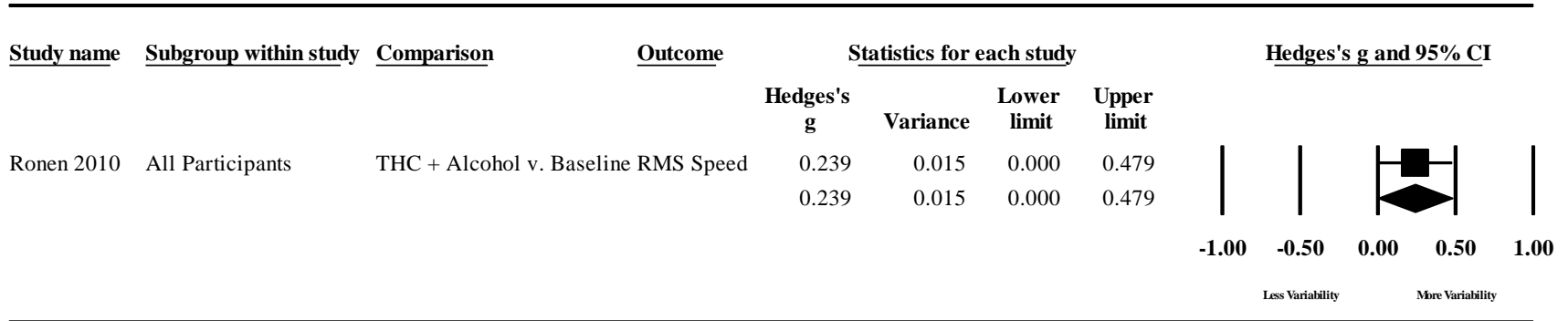


Figure C110. Forest plot illustrating *Combination v. Baseline: Speed Variability* (missing pre-post correlations set to $r = 0.9$).

Combination v. Baseline: Speed Exceedances

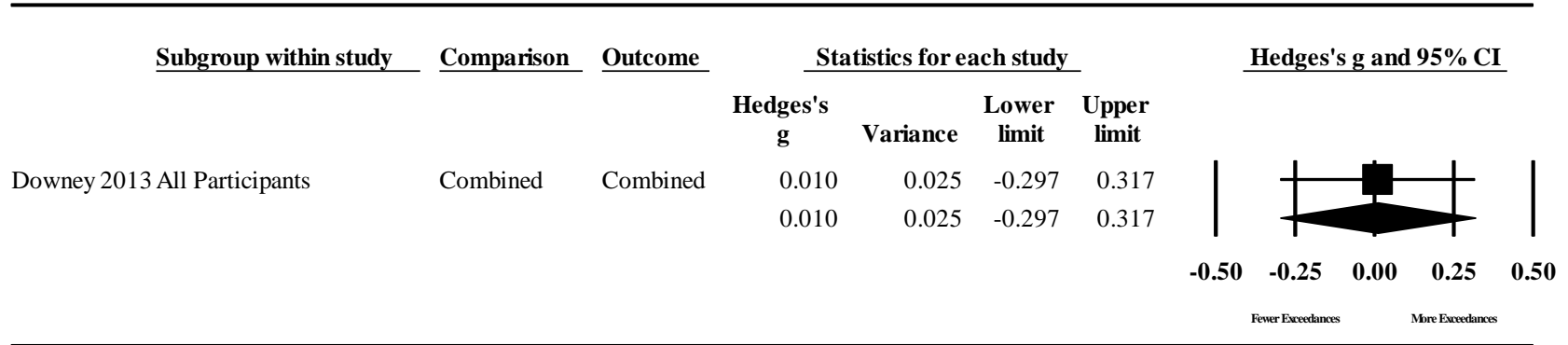


Figure C111. Forest plot illustrating *Combination v. Baseline: Speed Exceedances* (missing pre-post correlations set to $r = \text{zero}$).

Combination v. Baseline: Speed Exceedances

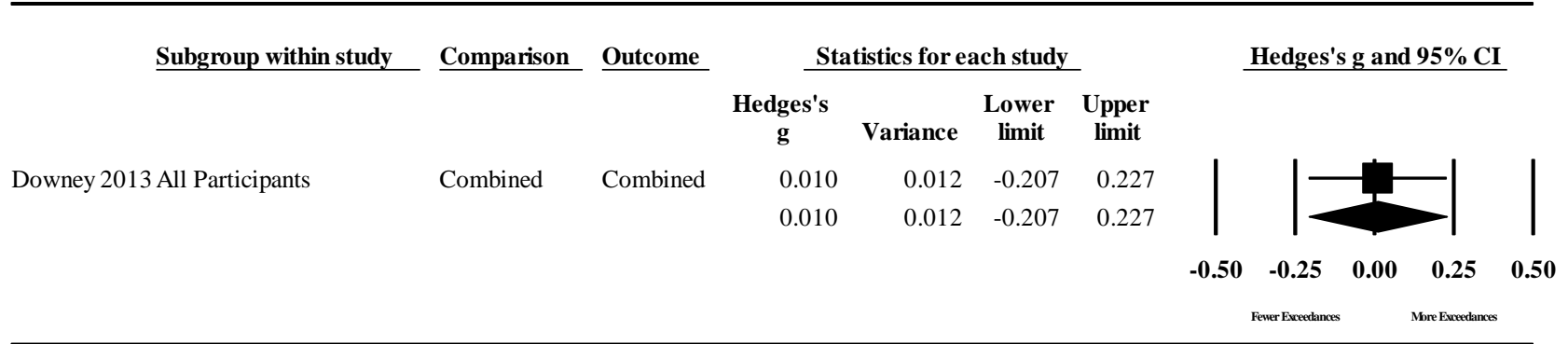


Figure C112. Forest plot illustrating *Combination v. Baseline: Speed Exceedances* (missing pre-post correlations set to $r = 0.5$).

Combination v. Baseline: Speed Exceedances

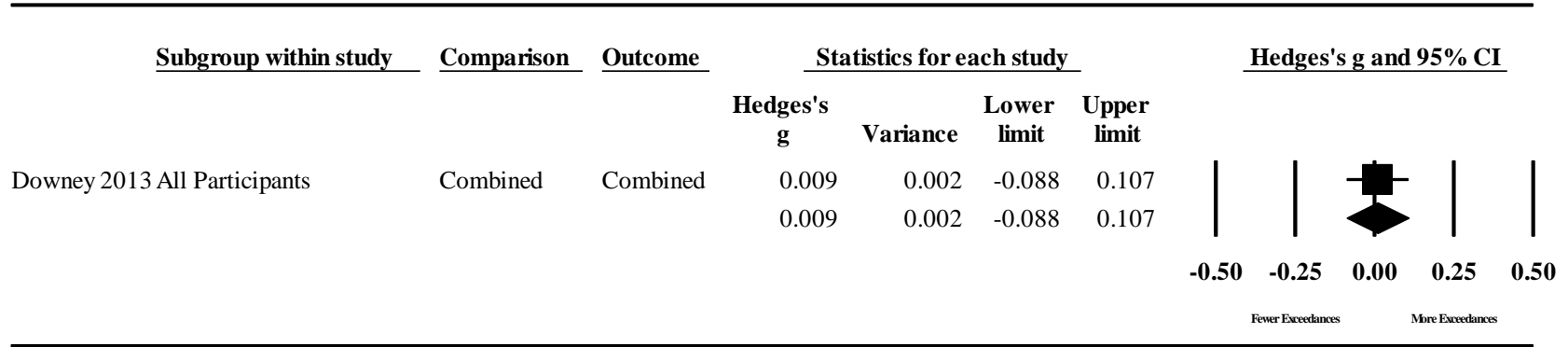


Figure C113. Forest plot illustrating *Combination v. Baseline: Speed Exceedances* (missing pre-post correlations set to $r = 0.9$).

Combination v. Baseline: Time Out of Lane

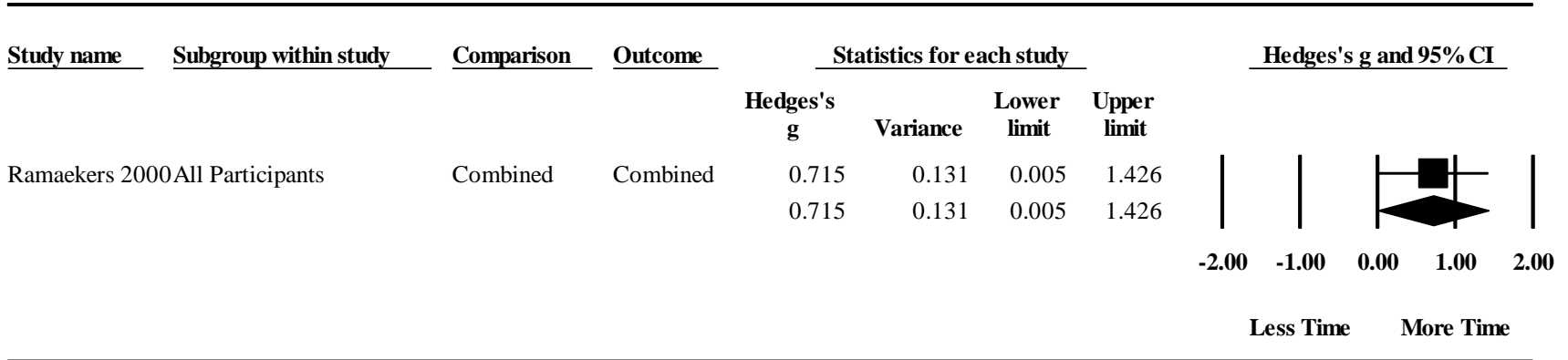


Figure C114. Forest plot illustrating *Combination v. Baseline: Time Out of Lane* (missing pre-post correlations set to $r = 0$).

Combination v. Baseline: Time Out of Lane

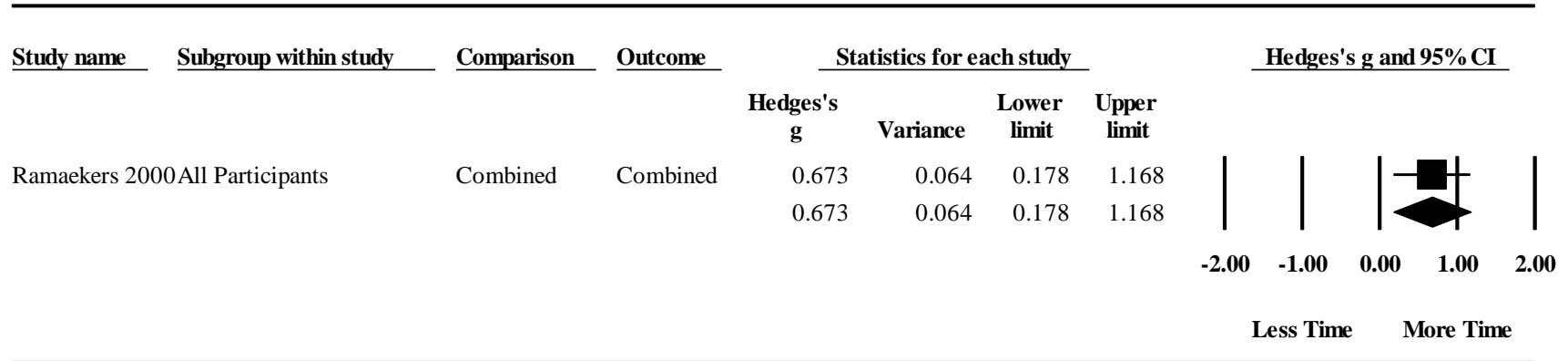


Figure C115. Forest plot illustrating *Combination v. Baseline: Time Out of Lane* (missing pre-post correlations set to $r = 0.5$).

Combination v. Baseline: Time Out of Lane

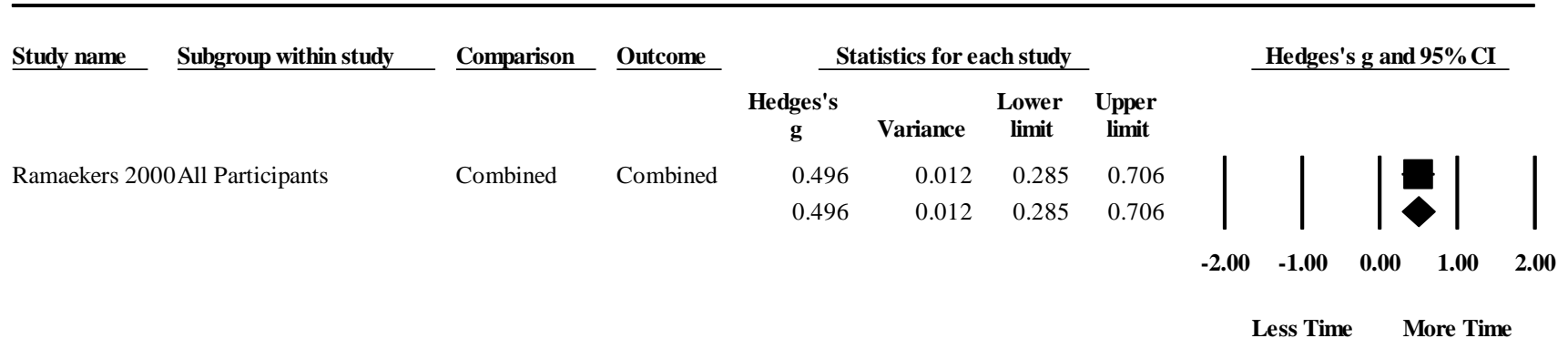


Figure C116. Forest plot illustrating *Combination v. Baseline: Time Out of Lane* (missing pre-post correlations set to $r = 0.9$).

Combination v. Alcohol: Crashes

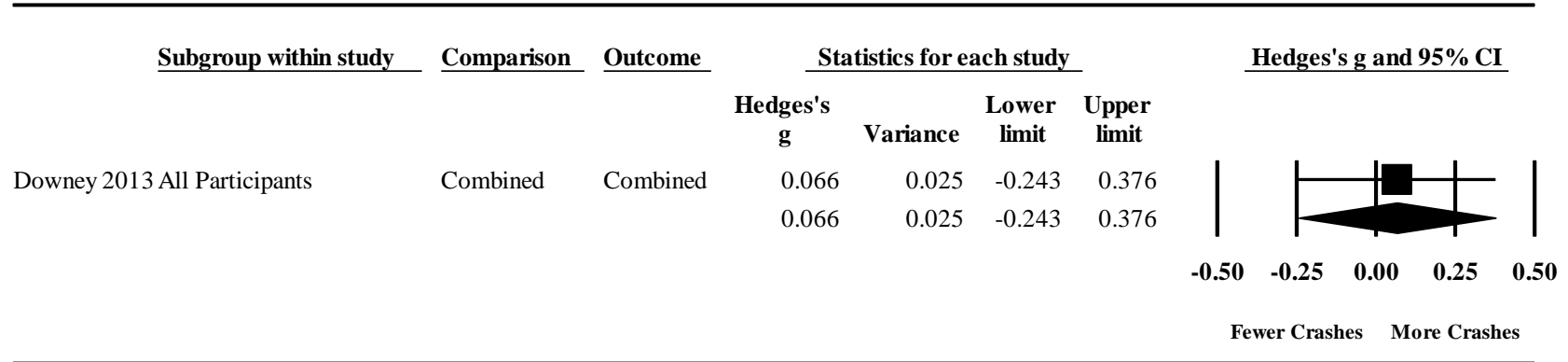


Figure C117. Forest plot illustrating *Combination v. Alcohol: Crashes* (missing pre-post correlations set to $r = \text{zero}$).

Combination v. Alcohol: Crashes

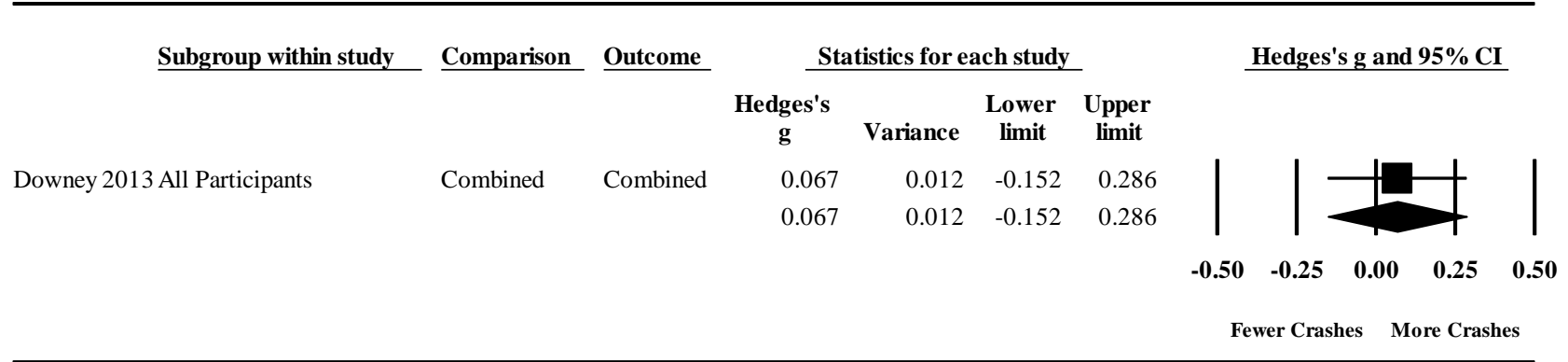


Figure C118. Forest plot illustrating *Combination v. Alcohol: Crashes* (missing pre-post correlations set to $r = 0.5$).

Combination v. Alcohol: Crashes

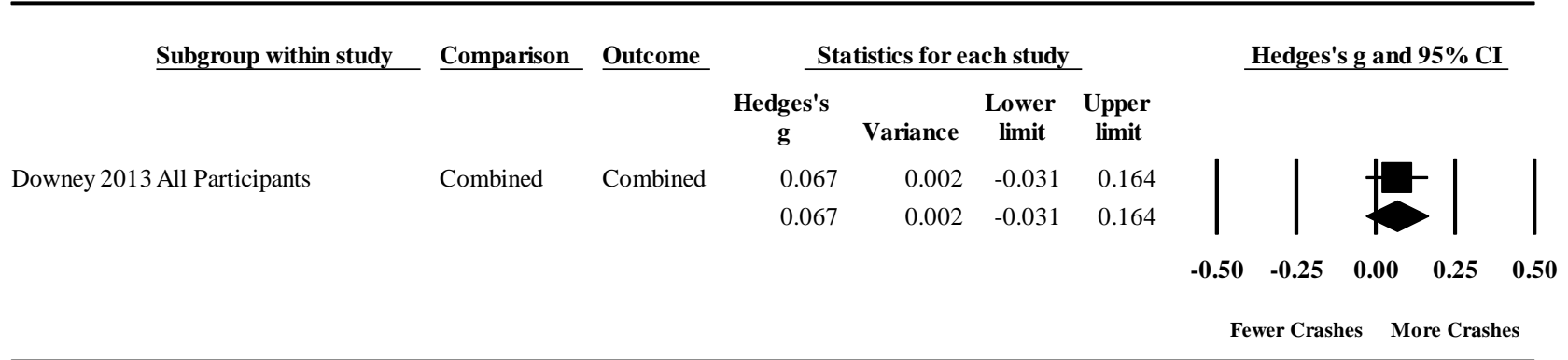


Figure C119. Forest plot illustrating *Combination v. Alcohol: Crashes* (missing pre-post correlations set to $r = 0.9$).

Combination v. Alcohol: Hazard RT

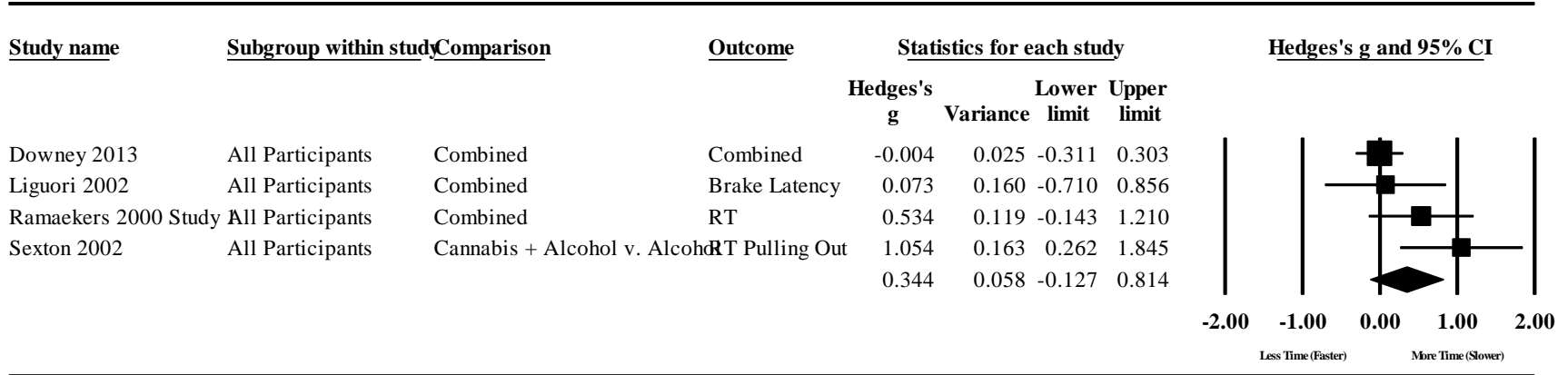


Figure C120. Forest plot illustrating *Combination v. Alcohol: Hazard RT* (missing pre-post correlations set to $r = zero$).

Combination v. Alcohol: Hazard RT

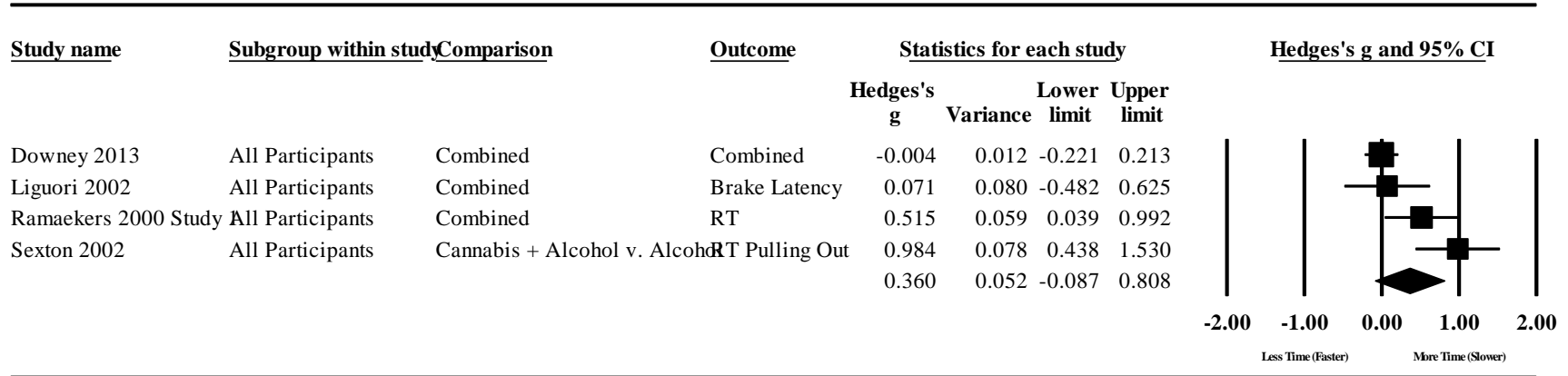


Figure C121. Forest plot illustrating *Combination v. Alcohol: Hazard RT* (missing pre-post correlations set to $r = 0.5$).

Combination v. Alcohol: Hazard RT

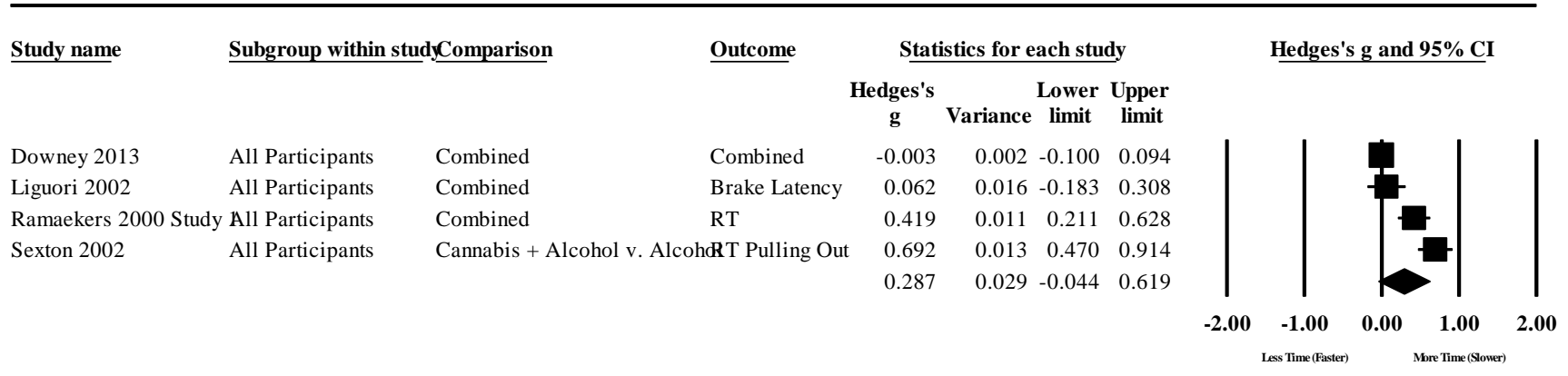


Figure C122. Forest plot illustrating *Combination v. Alcohol: Hazard RT* (missing pre-post correlations set to $r = 0.9$).

Combination v. Alcohol: Lateral Position Variability

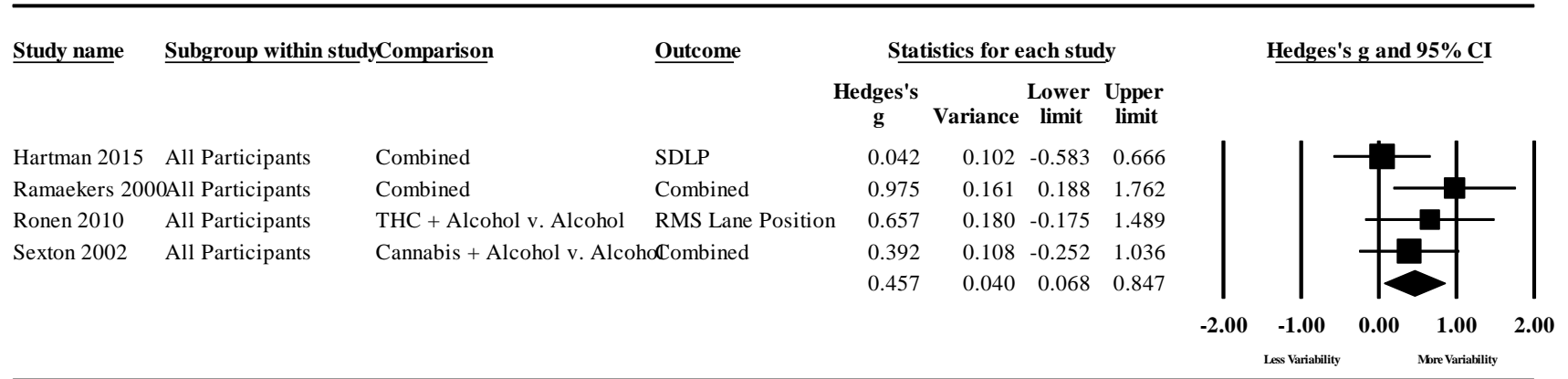


Figure C123. Forest plot illustrating *Combination v. Alcohol: Lateral Position Variability* (missing pre-post correlations set to $r =$ zero).

Combination v. Alcohol: Lateral Position Variability

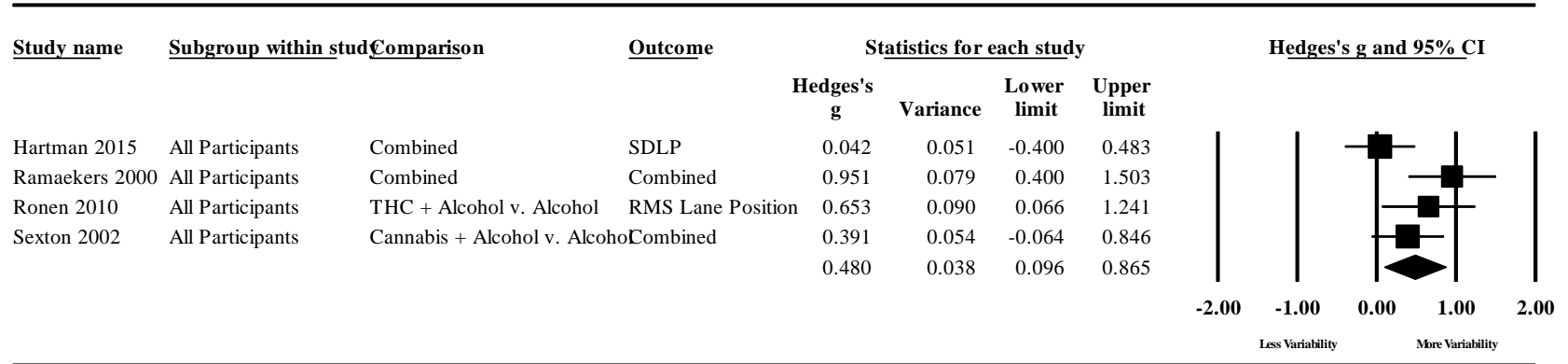


Figure C124. Forest plot illustrating *Combination v. Alcohol: Lateral Position Variability* (missing pre-post correlations set to $r = 0.5$).

Combination v. Alcohol: Lateral Position Variability

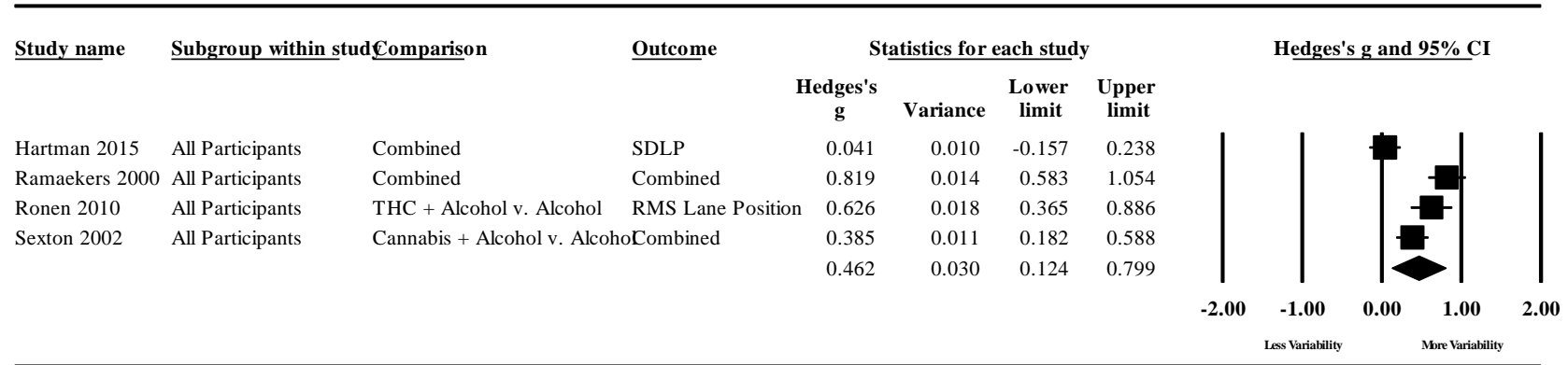


Figure C125. Forest plot illustrating *Combination v. Alcohol: Lateral Position Variability* (missing pre-post correlations set to $r = 0.9$).

Combination v. Alcohol: Lane Excursions

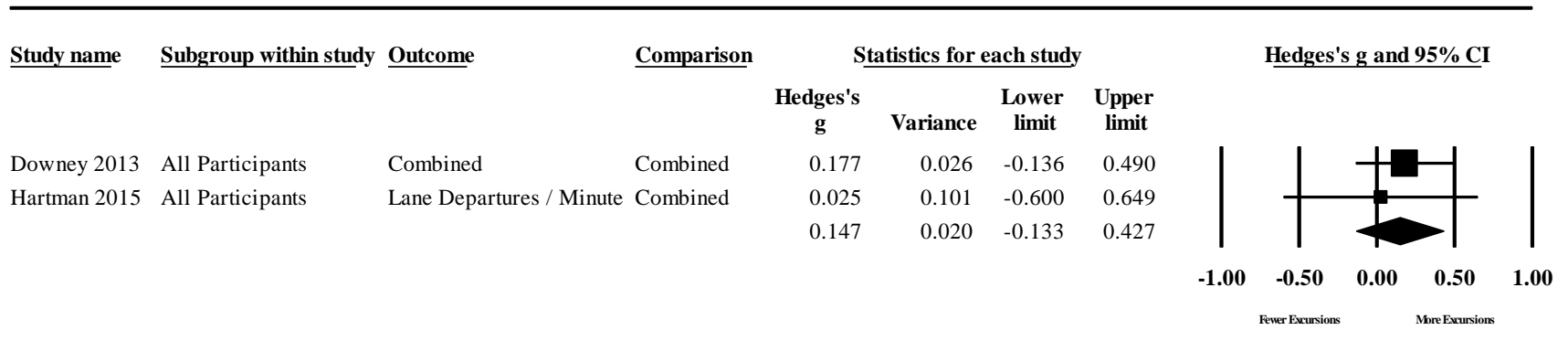


Figure C126. Forest plot illustrating *Combination v. Alcohol: Lane Excursions* (missing pre-post correlations set to $r = 0$).

Combination v. Alcohol: Lane Excursions

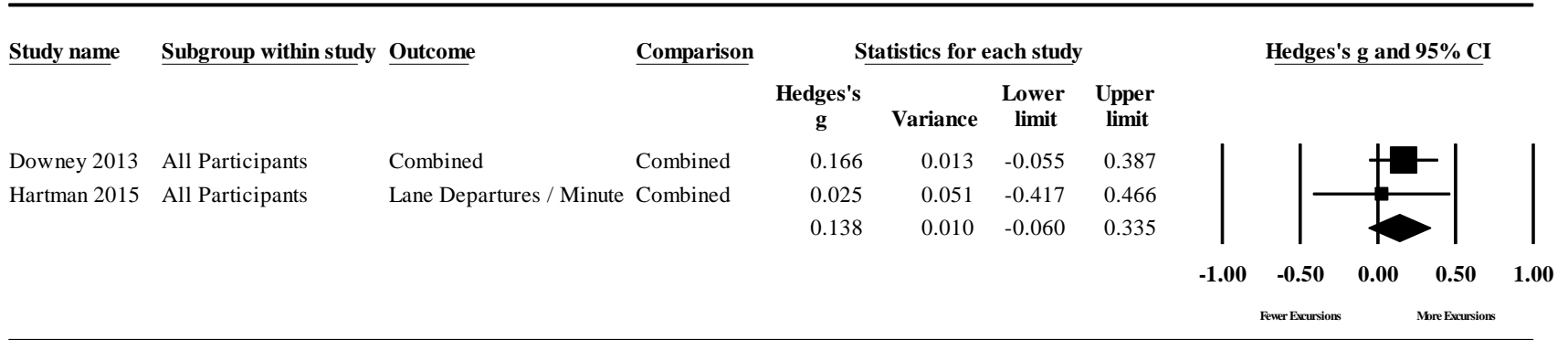


Figure C127. Forest plot illustrating *Combination v. Alcohol: Lane Excursions* (missing pre-post correlations set to $r = 0.5$).

Combination v. Alcohol: Lane Excursions

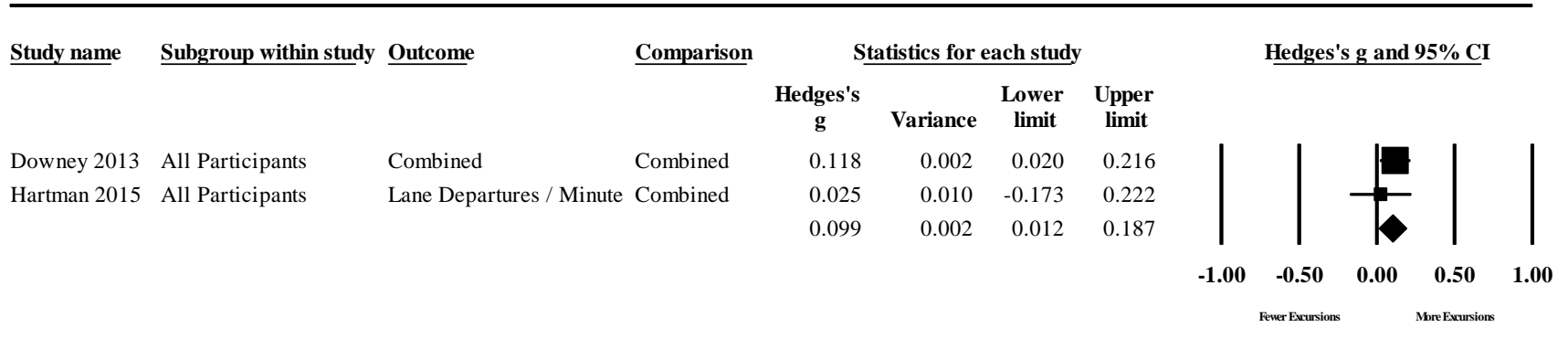


Figure C128. Forest plot illustrating *Combination v. Alcohol: Lane Excursions* (missing pre-post correlations set to $r = 0.9$).

Combination v. Alcohol: Time Out of Lane

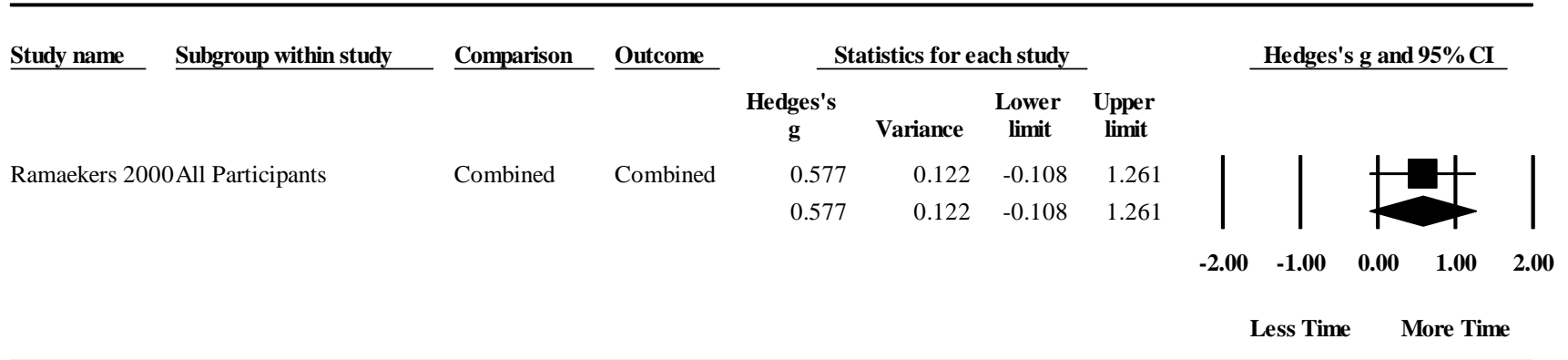


Figure C129. Forest plot illustrating *Combination v. Alcohol: Time Out of Lane* (missing pre-post correlations set to $r = \text{zero}$).

Combination v. Alcohol: Time Out of Lane

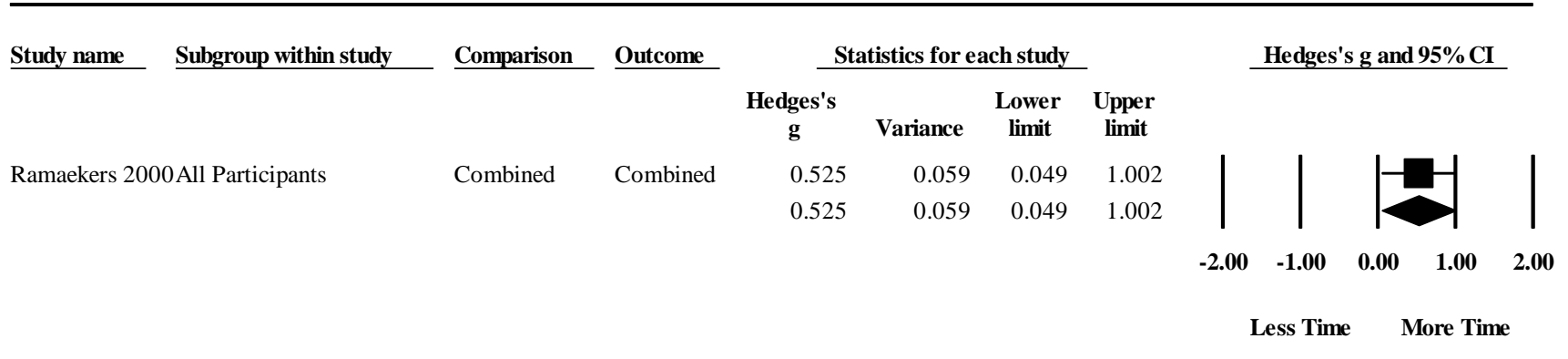


Figure C130. Forest plot illustrating *Combination v. Alcohol: Time Out of Lane* (missing pre-post correlations set to $r = 0.5$).

Combination v. Alcohol: Time Out of Lane

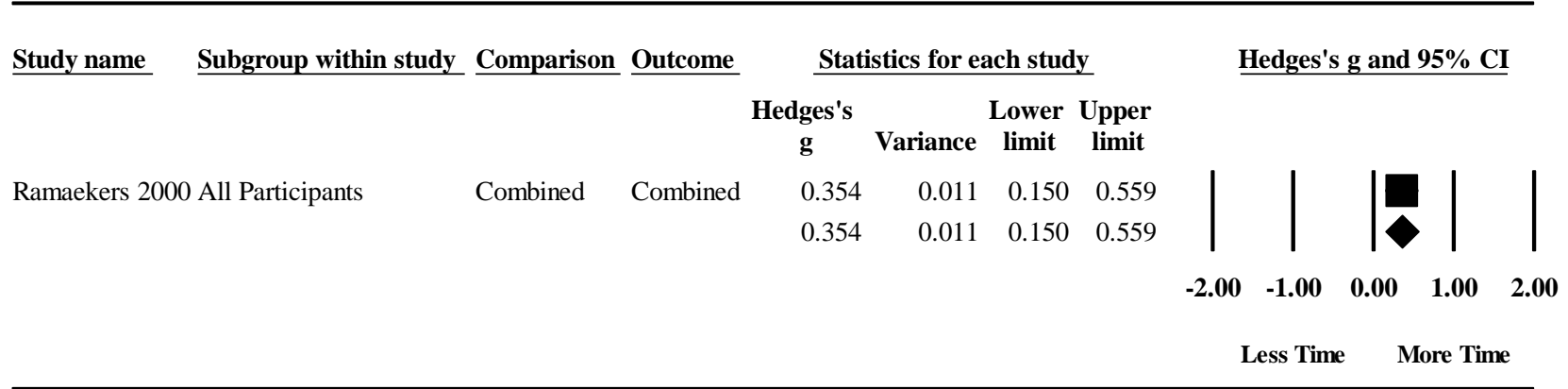


Figure C131. Forest plot illustrating *Combination v. Alcohol: Time Out of Lane* (missing pre-post correlations set to $r = 0.9$).

Combination v. Alcohol: Speed

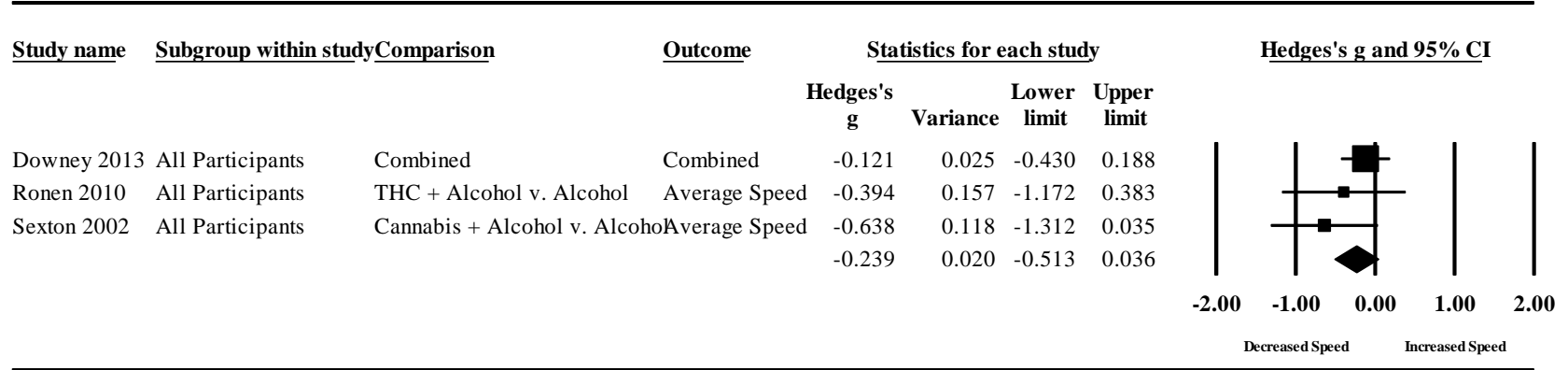


Figure C132. Forest plot illustrating *Combination v. Alcohol: Speed* (missing pre-post correlations set to $r = zero$).

Combination v. Alcohol: Speed

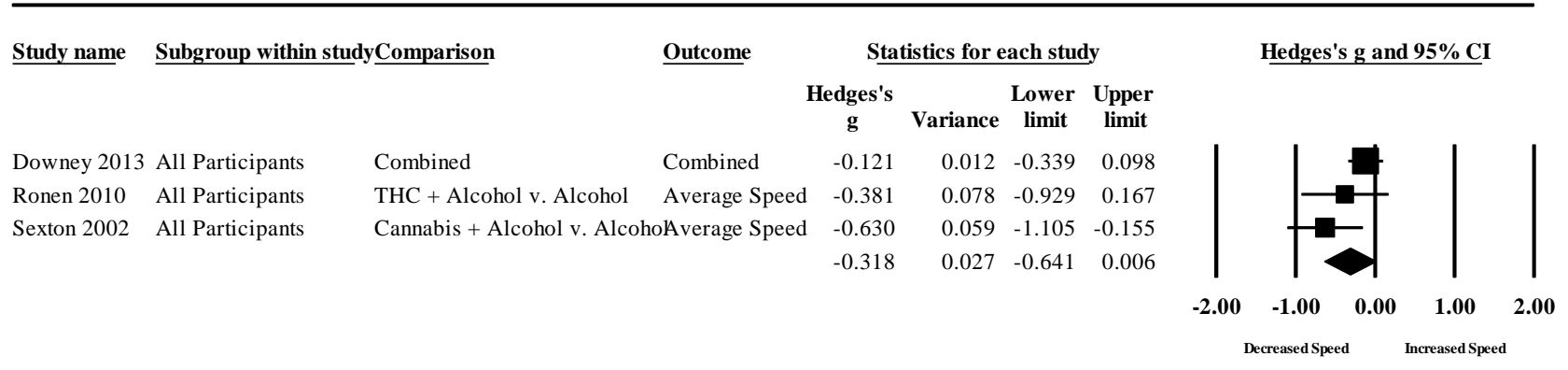


Figure C133. Forest plot illustrating *Combination v. Alcohol: Speed* (missing pre-post correlations set to $r = 0.5$).

Combination v. Alcohol: Speed

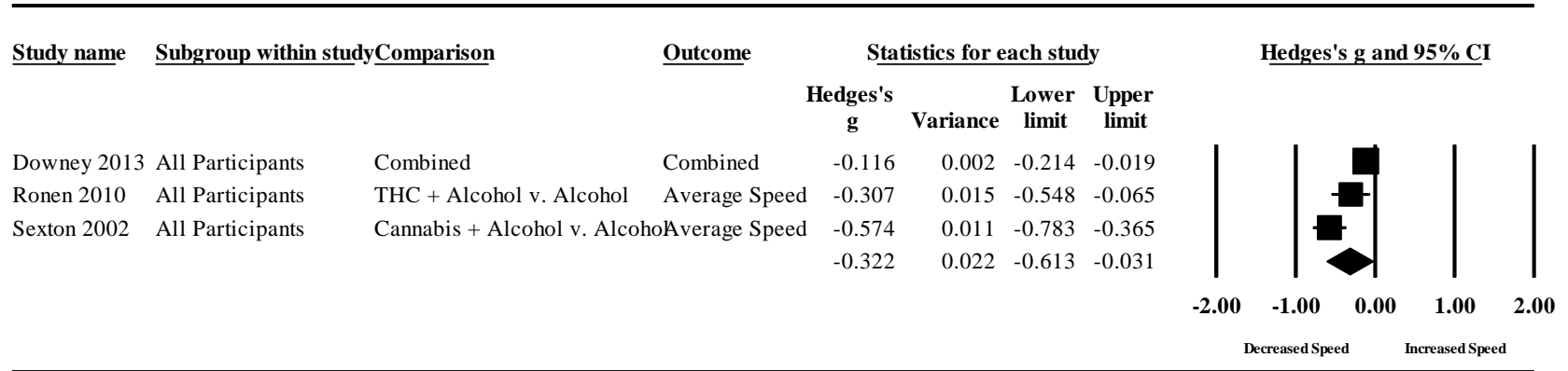


Figure C134. Forest plot illustrating *Combination v. Alcohol: Speed* (missing pre-post correlations set to $r = 0.9$).

Combination v. Alcohol: Speed Variability

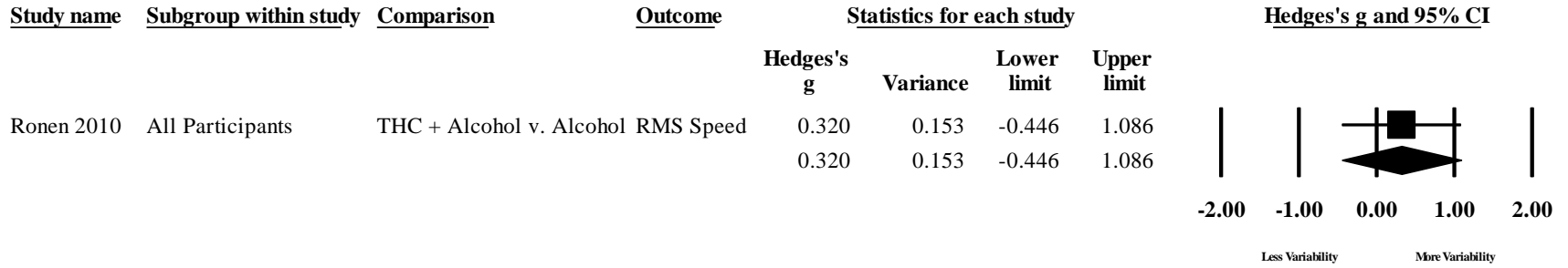


Figure C135. Forest plot illustrating *Combination v. Alcohol: Speed Variability* (missing pre-post correlations set to $r = 0$).

Combination v. Alcohol: Speed Variability

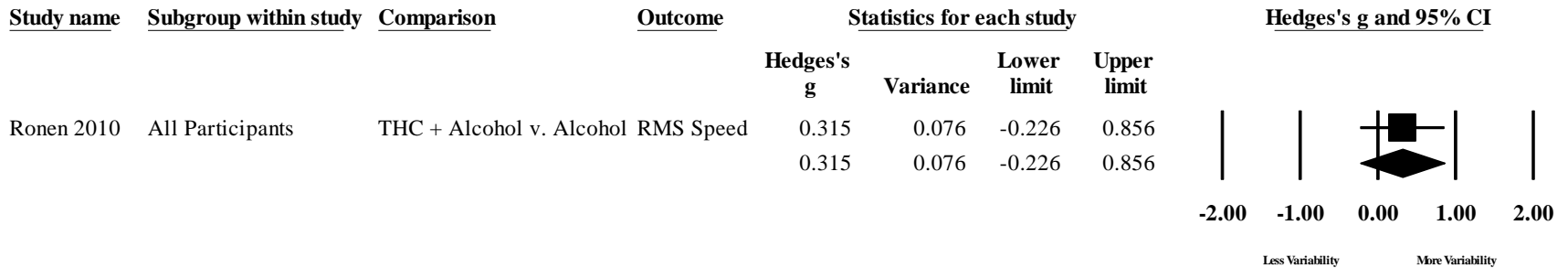


Figure C136. Forest plot illustrating *Combination v. Alcohol: Speed Variability* (missing pre-post correlations set to $r = 0.5$).

Combination v. Alcohol: Speed Variability

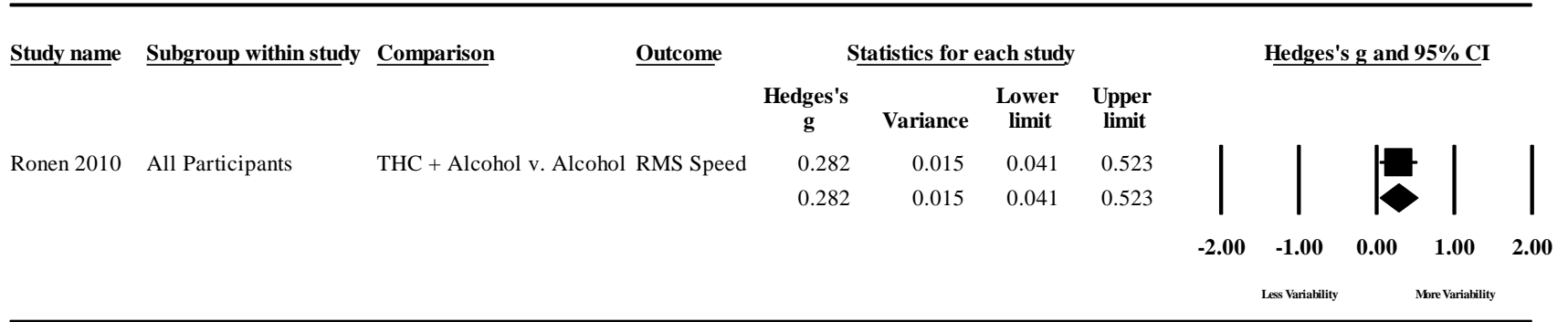


Figure C137. Forest plot illustrating *Combination v. Alcohol: Speed Variability* (missing pre-post correlations set to $r = 0.9$).

Combination v. Alcohol: Speed Exceedances

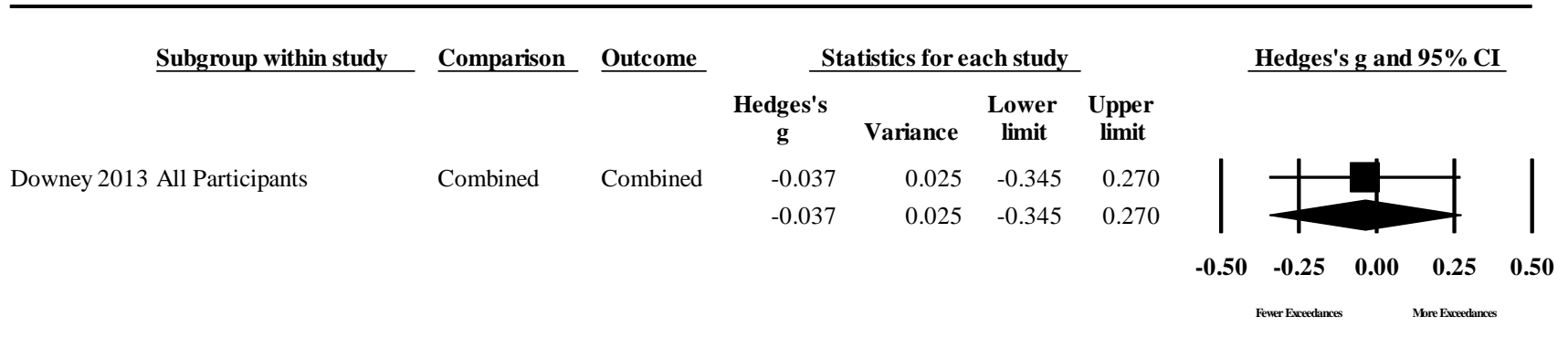


Figure C138. Forest plot illustrating *Combination v. Alcohol: Speed Exceedances* (missing pre-post correlations set to $r = zero$).

Combination v. Alcohol: Speed Exceedances

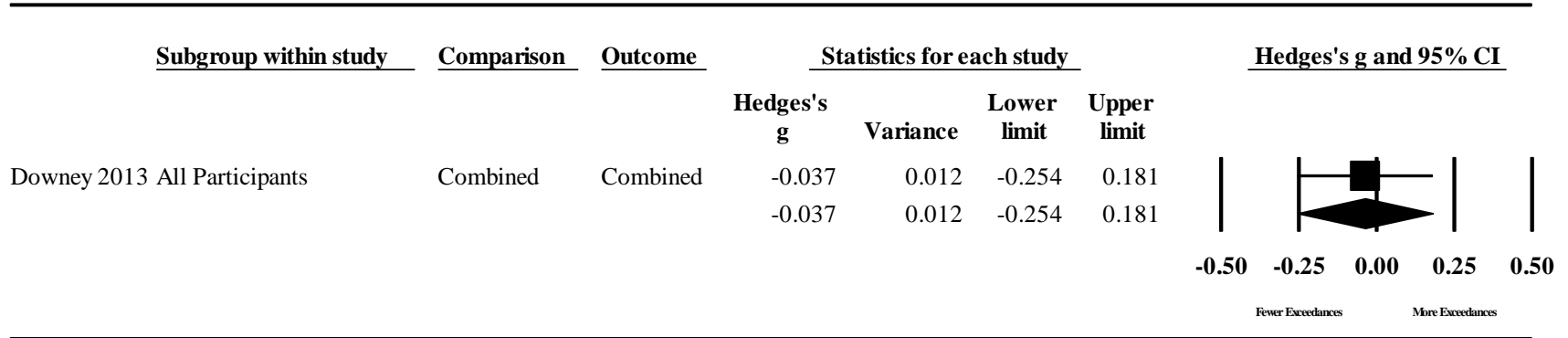


Figure C139. Forest plot illustrating *Combination v. Alcohol: Speed Exceedances* (missing pre-post correlations set to $r = 0.5$).

Combination v. Alcohol: Speed Exceedances

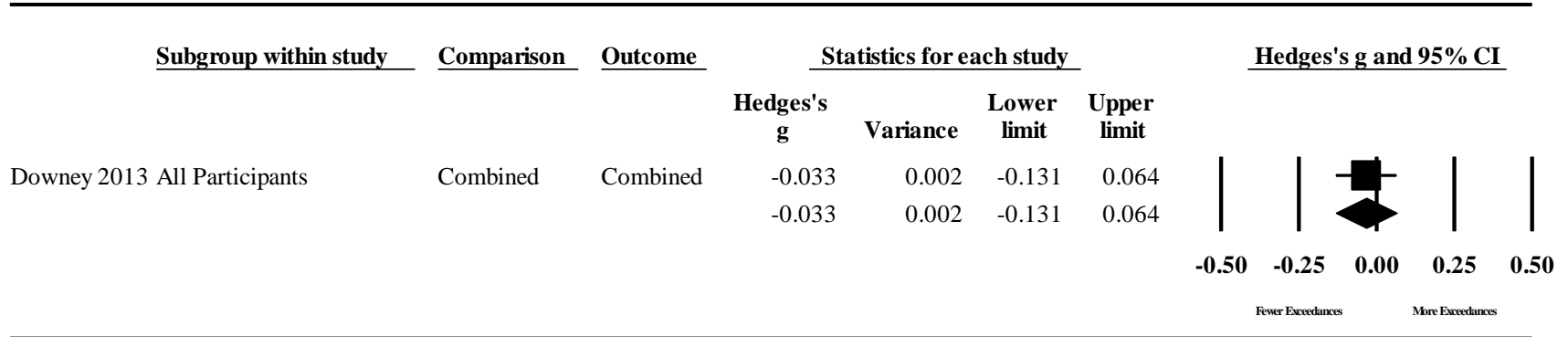


Figure C140. Forest plot illustrating *Combination v. Alcohol: Speed Exceedances* (missing pre-post correlations set to $r = 0.9$).

Combination v. Cannabis: Crashes

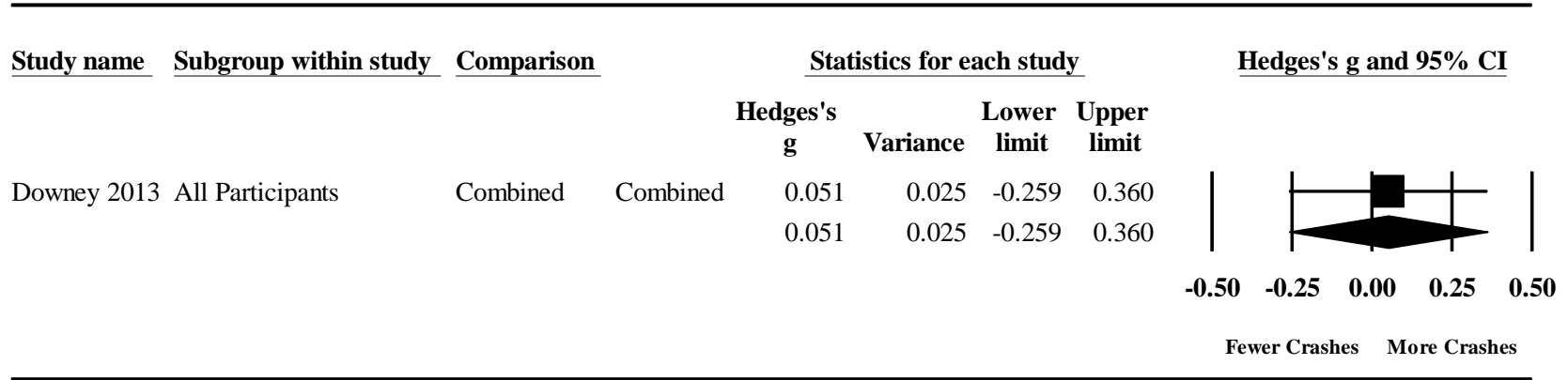


Figure C141. Forest plot illustrating *Combination v. Cannabis: Crashes* (missing pre-post correlations set to $r = \text{zero}$).

Combination v. Cannabis: Crashes

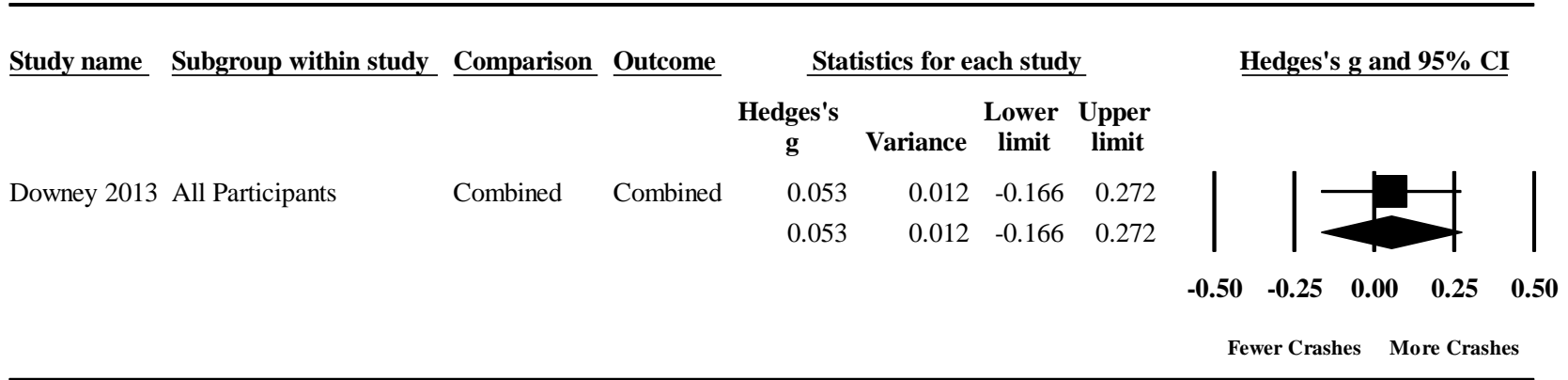


Figure C142. Forest plot illustrating *Combination v. Cannabis: Crashes* (missing pre-post correlations set to $r = 0.5$).

Combination v. Cannabis: Crashes

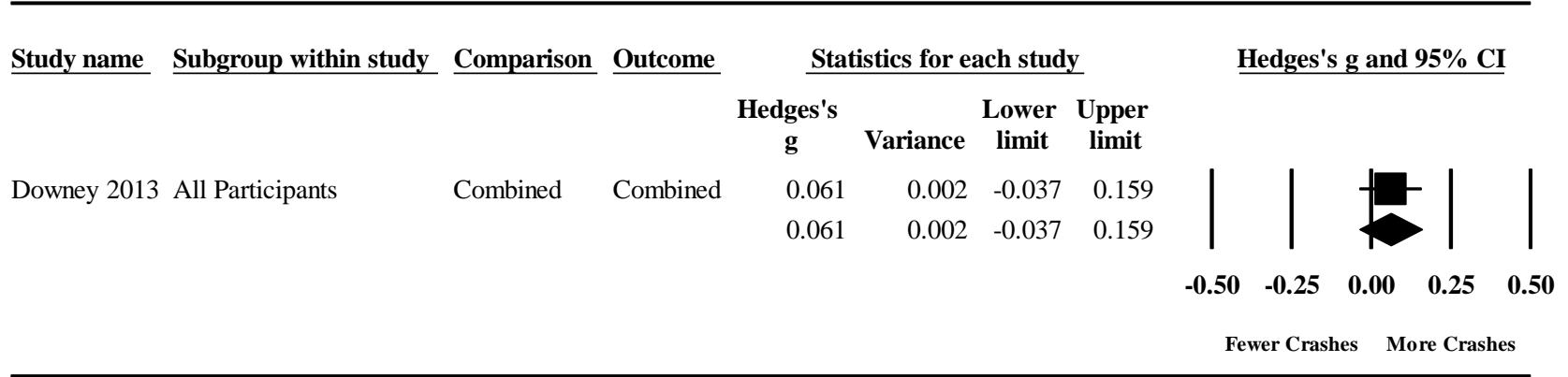


Figure C143. Forest plot illustrating *Combination v. Cannabis: Crashes* (missing pre-post correlations set to $r = 0.9$).

Combination v. Cannabis: Hazard RT

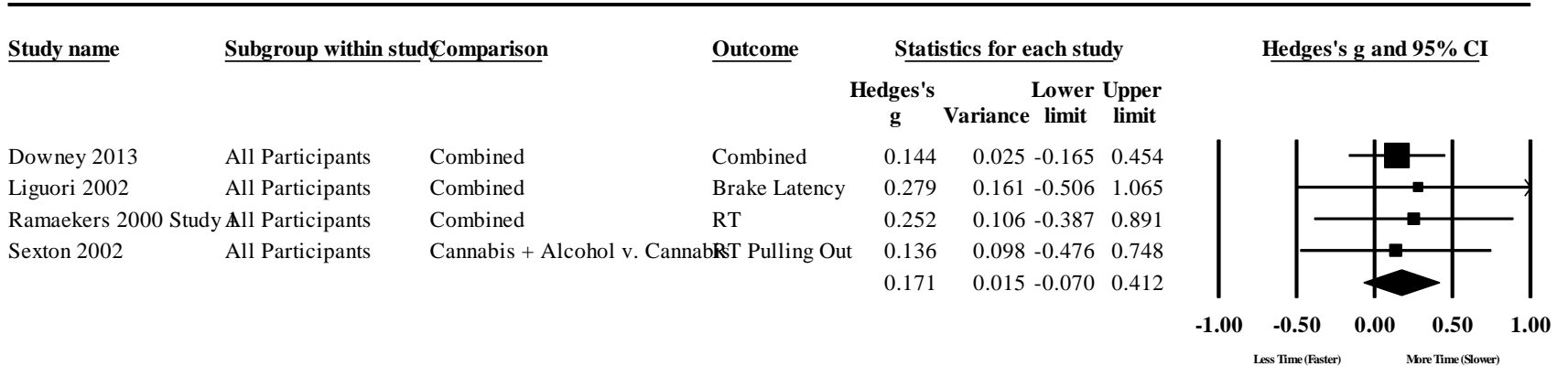


Figure C144. Forest plot illustrating *Combination v. Cannabis: Hazard RT* (missing pre-post correlations set to $r = \text{zero}$).

Combination v. Cannabis: Hazard RT

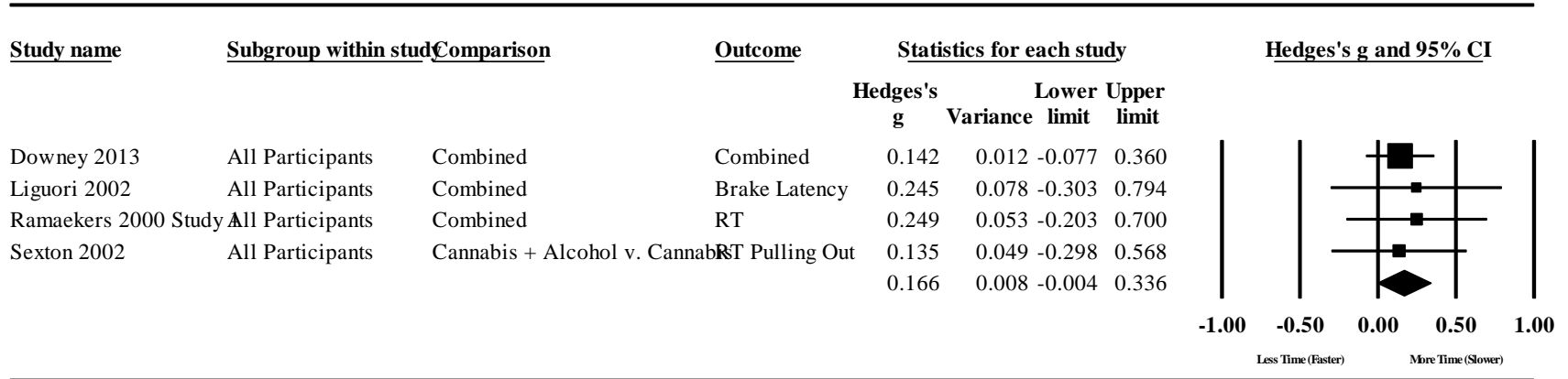


Figure C145. Forest plot illustrating *Combination v. Cannabis: Hazard RT* (missing pre-post correlations set to $r = 0.5$).

Combination v. Cannabis: Hazard RT

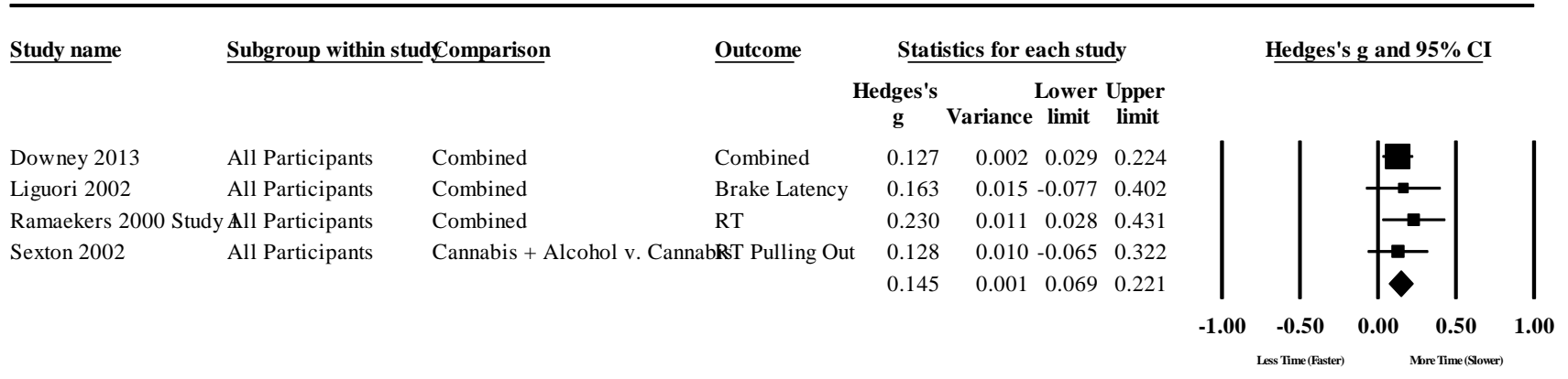


Figure C146. Forest plot illustrating *Combination v. Cannabis: Hazard RT* (missing pre-post correlations set to $r = 0.9$).

Combination v. Cannabis: Lateral Position Variability

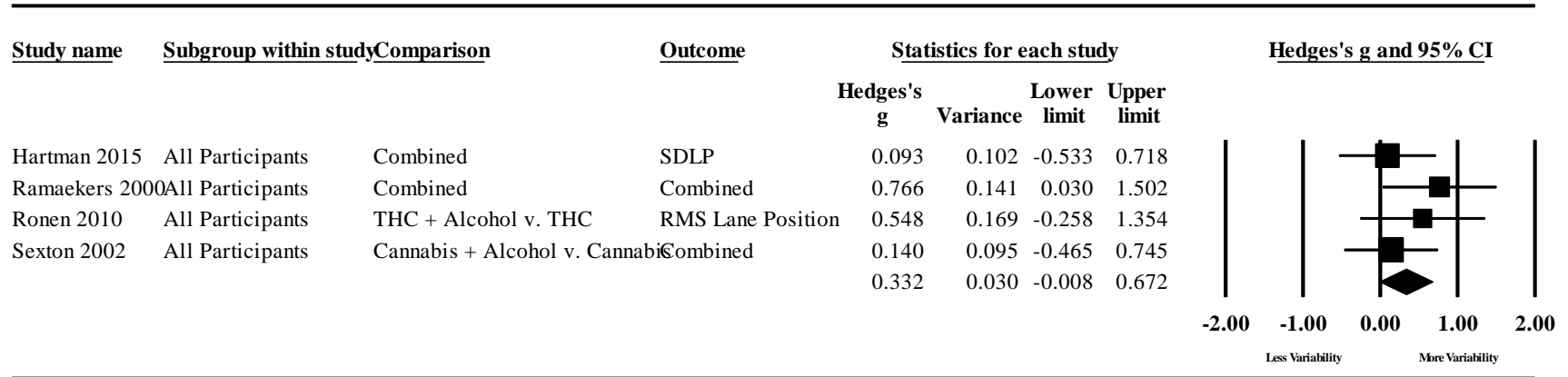


Figure C147. Forest plot illustrating *Combination v. Cannabis: Lateral Position Variability* (missing pre-post correlations set to $r =$ zero).

Combination v. Cannabis: Lateral Position Variability

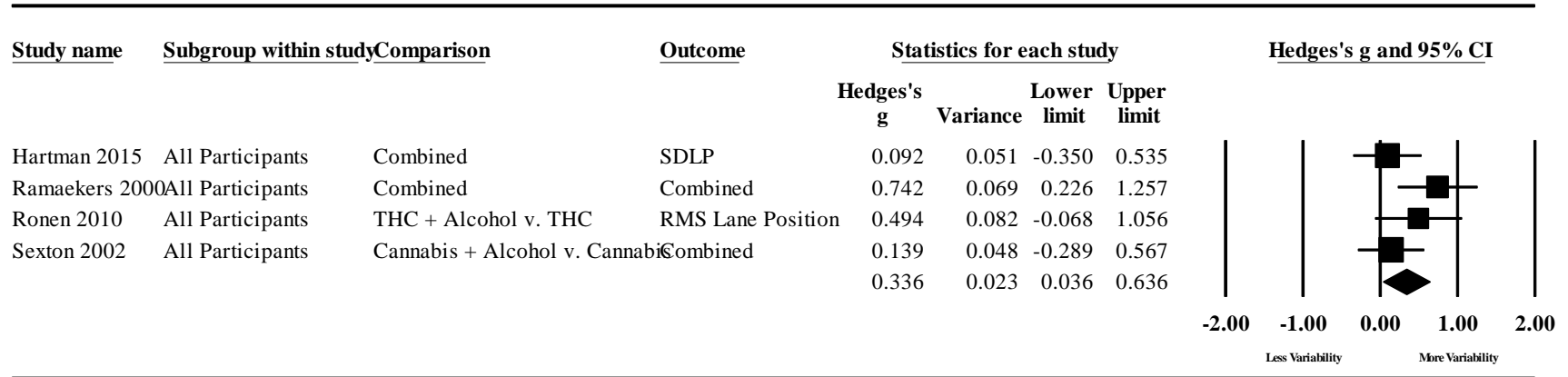


Figure C148. Forest plot illustrating *Combination v. Cannabis: Lateral Position Variability* (missing pre-post correlations set to $r = 0.5$).

Combination v. Cannabis: Lateral Position Variability

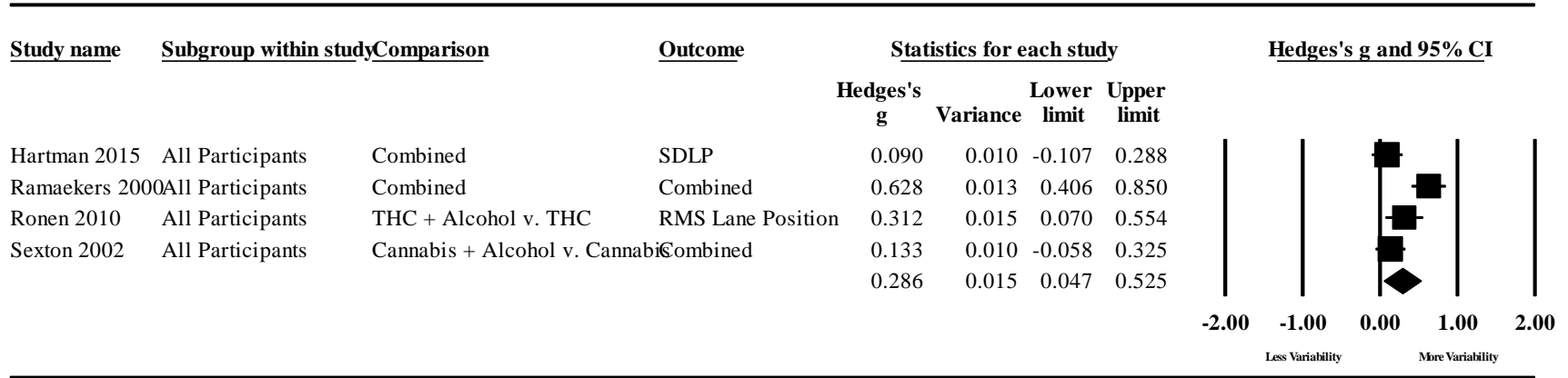


Figure C149. Forest plot illustrating *Combination v. Cannabis: Lateral Position Variability* (missing pre-post correlations set to $r = 0.9$).

Combination v. Cannabis: Lane Excursions

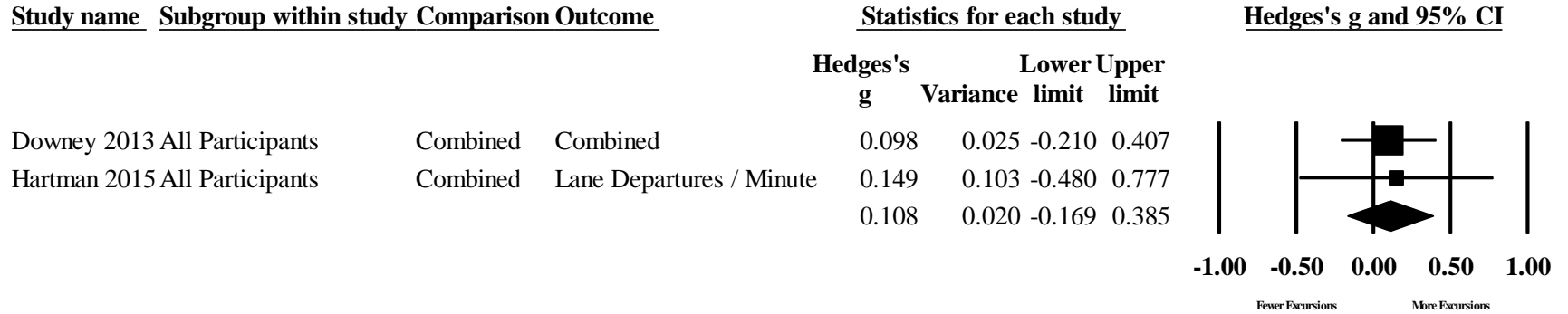


Figure C150. Forest plot illustrating *Combination v. Cannabis: Lane Excursions* (missing pre-post correlations set to $r = 0$).

Combination v. Cannabis: Lane Excursions

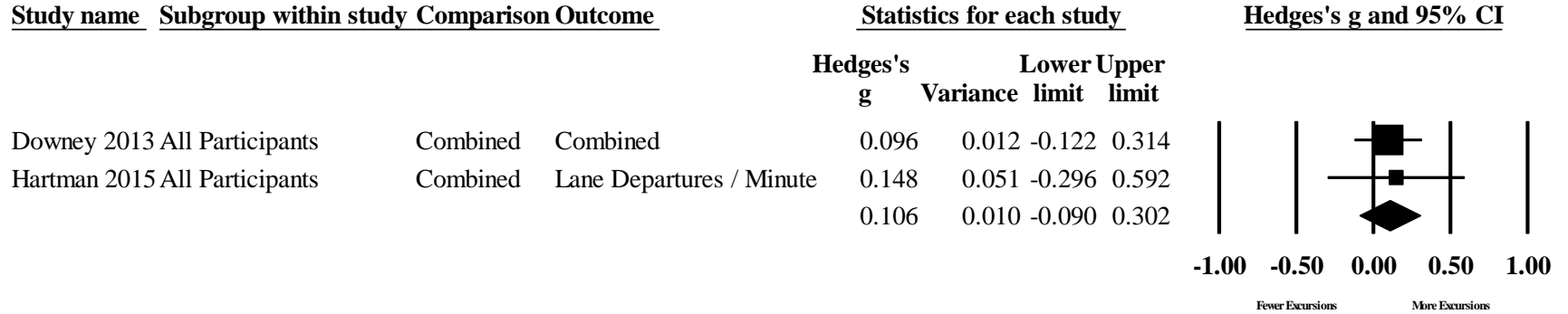


Figure C151. Forest plot illustrating *Combination v. Cannabis: Lane Excursions* (missing pre-post correlations set to $r = 0.5$).

Combination v. Cannabis: Lane Excursions

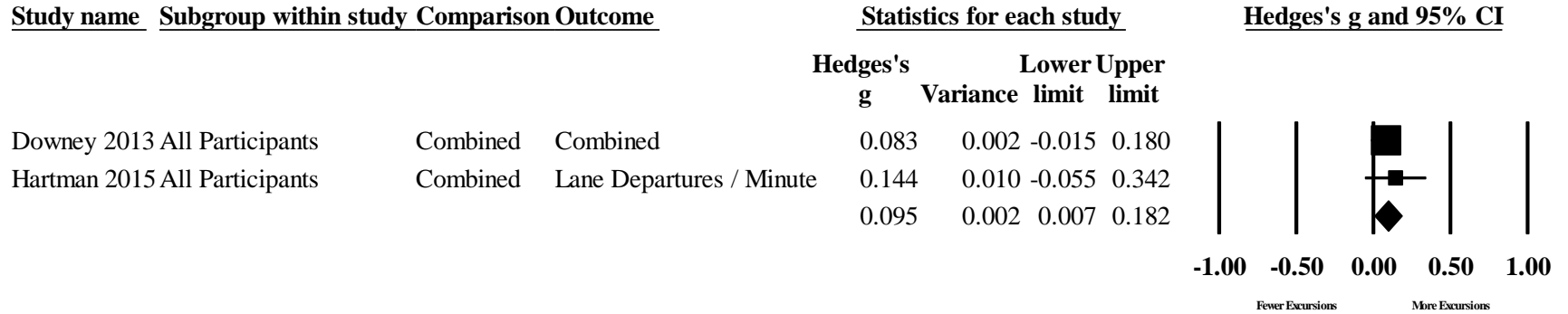


Figure C152. Forest plot illustrating *Combination v. Cannabis: Lane Excursions* (missing pre-post correlations set to $r = 0.9$).

Combination v. Cannabis: Time Out of Lane

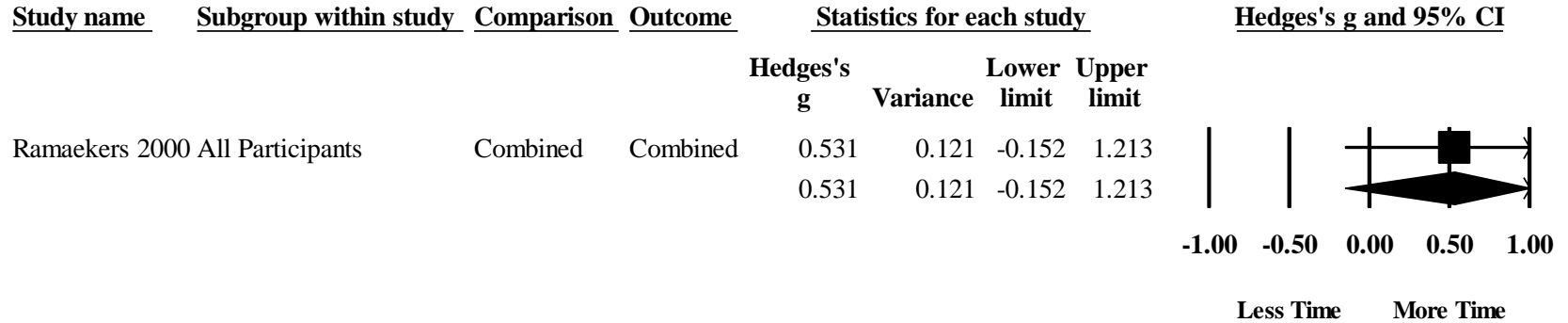


Figure C153. Forest plot illustrating *Combination v. Cannabis: Time Out of Lane* (missing pre-post correlations set to $r = \text{zero}$).

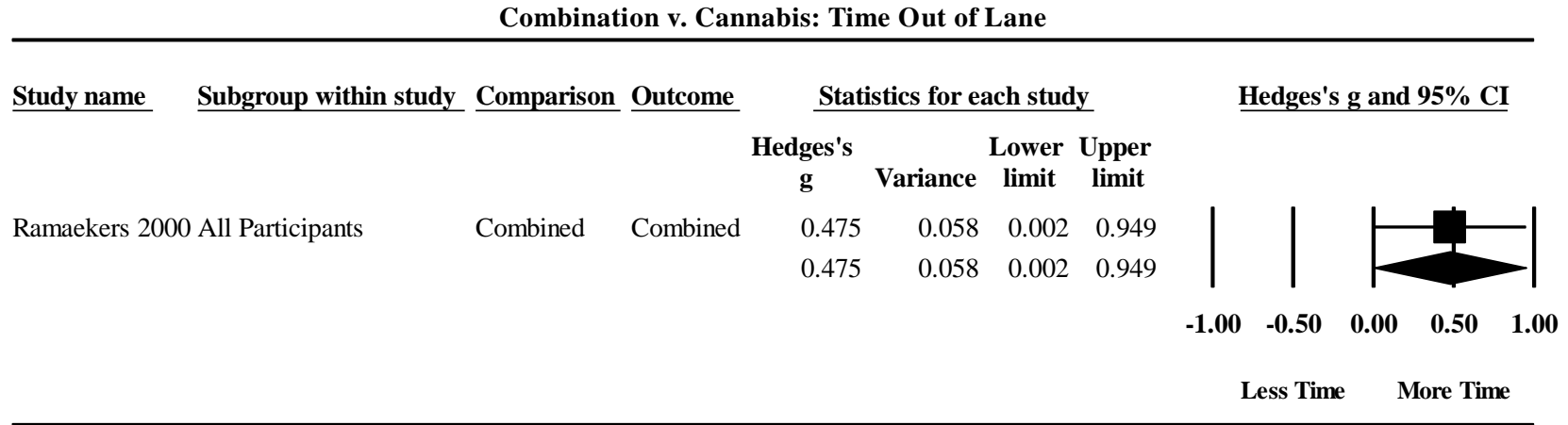


Figure C154. Forest plot illustrating *Combination v. Cannabis: Time Out of Lane* (missing pre-post correlations set to $r = 0.5$).

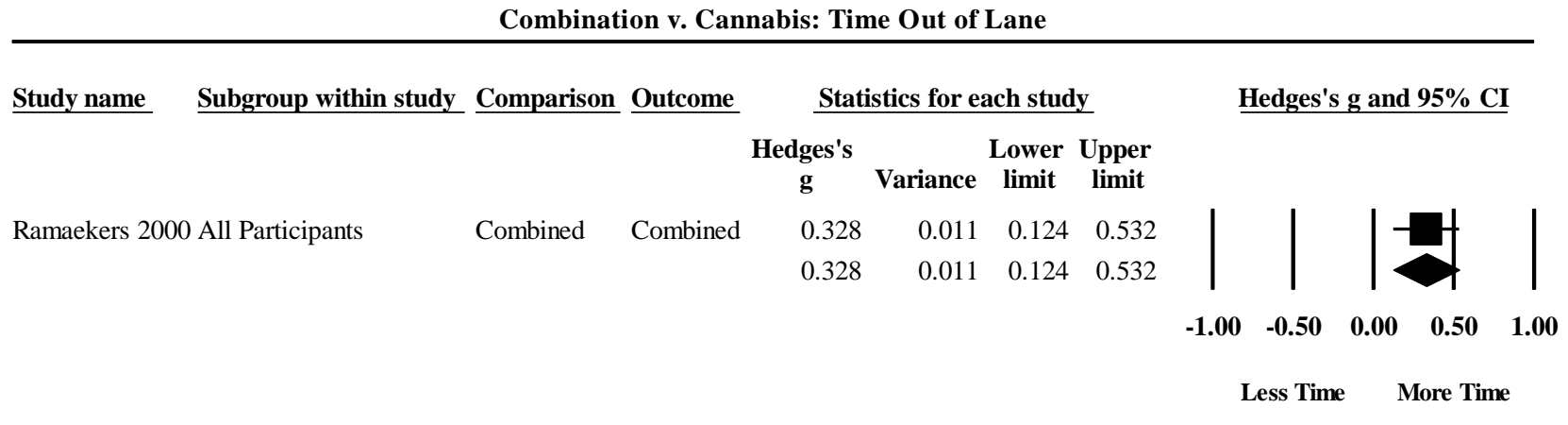


Figure C155. Forest plot illustrating *Combination v. Cannabis: Time Out of Lane* (missing pre-post correlations set to $r = 0.9$).

Combination v. Cannabis: Speed

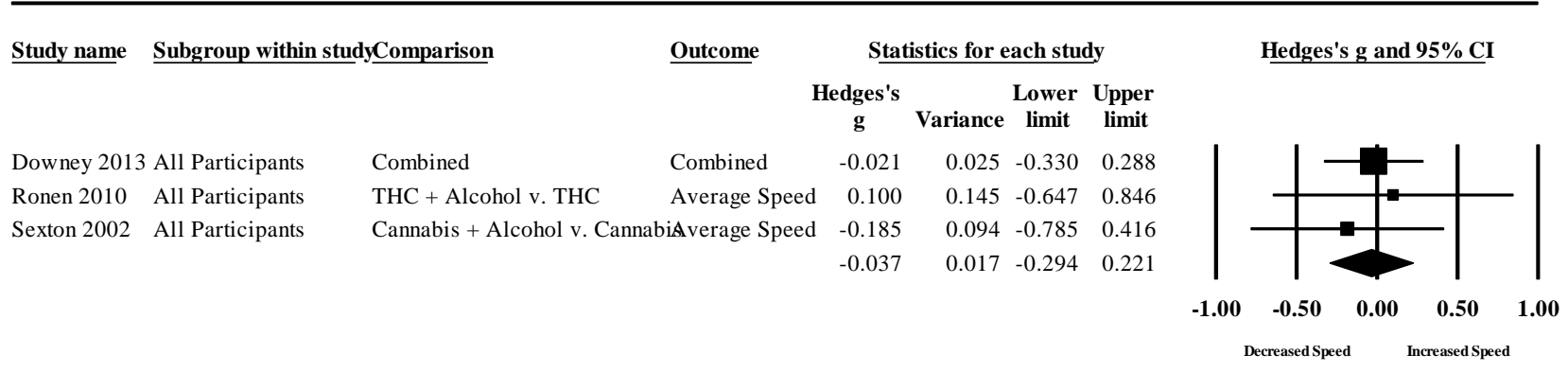


Figure C156. Forest plot illustrating *Combination v. Cannabis: Speed* (missing pre-post correlations set to $r = \text{zero}$).

Combination v. Cannabis: Speed

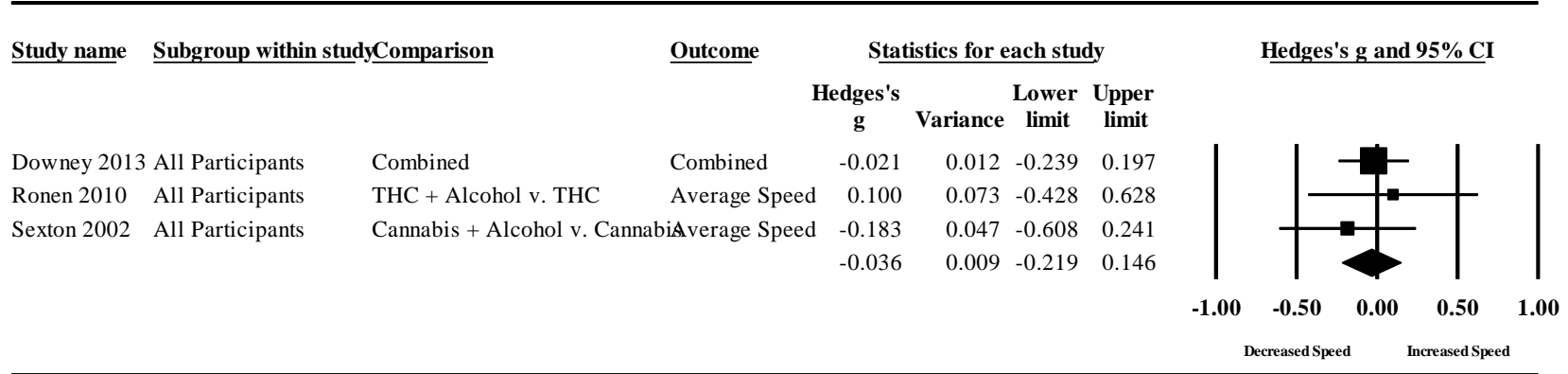


Figure C157. Forest plot illustrating *Combination v. Cannabis: Speed* (missing pre-post correlations set to $r = 0.5$).

Combination v. Cannabis: Speed

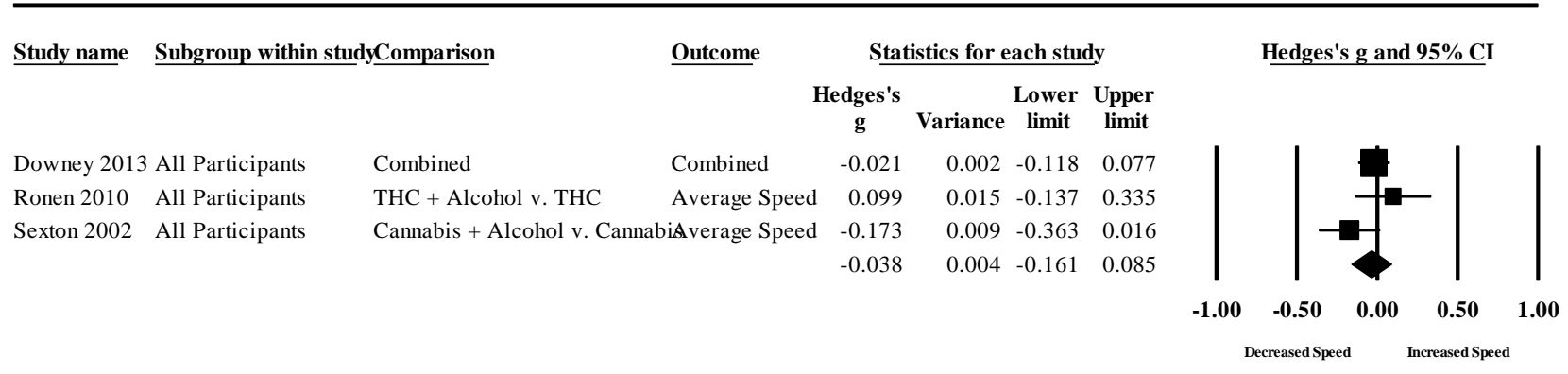


Figure C158. Forest plot illustrating *Combination v. Cannabis: Speed* (missing pre-post correlations set to $r = 0.9$).

Combination v. Cannabis: Speed Variability

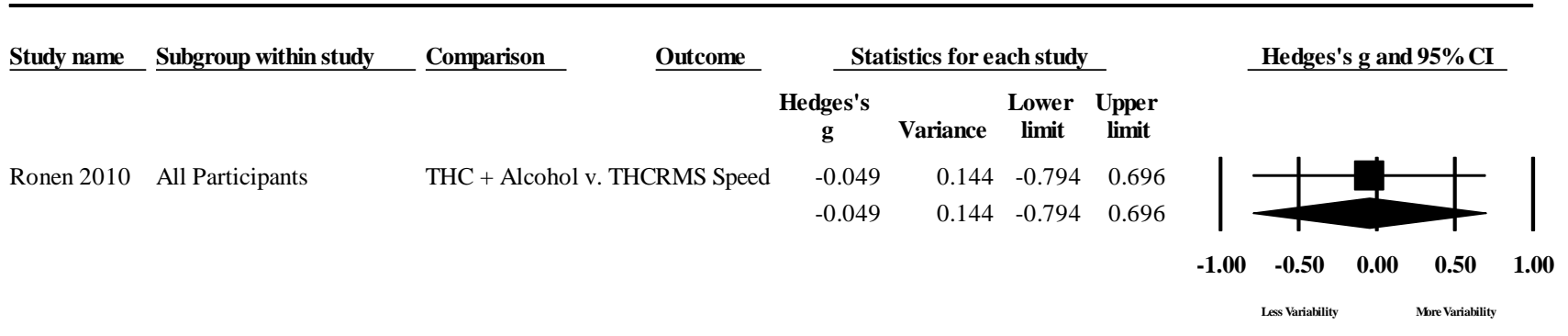


Figure C159. Forest plot illustrating *Combination v. Cannabis: Speed Variability* (missing pre-post correlations set to $r = 0$).

Combination v. Cannabis: Speed Variability

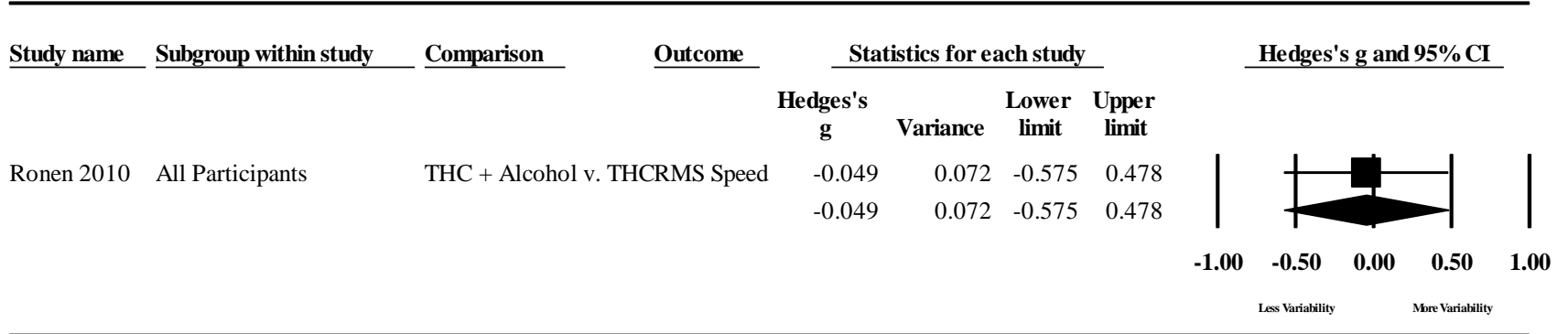


Figure C160. Forest plot illustrating *Combination v. Cannabis: Speed Variability* (missing pre-post correlations set to $r = 0.5$).

Combination v. Cannabis: Speed Variability

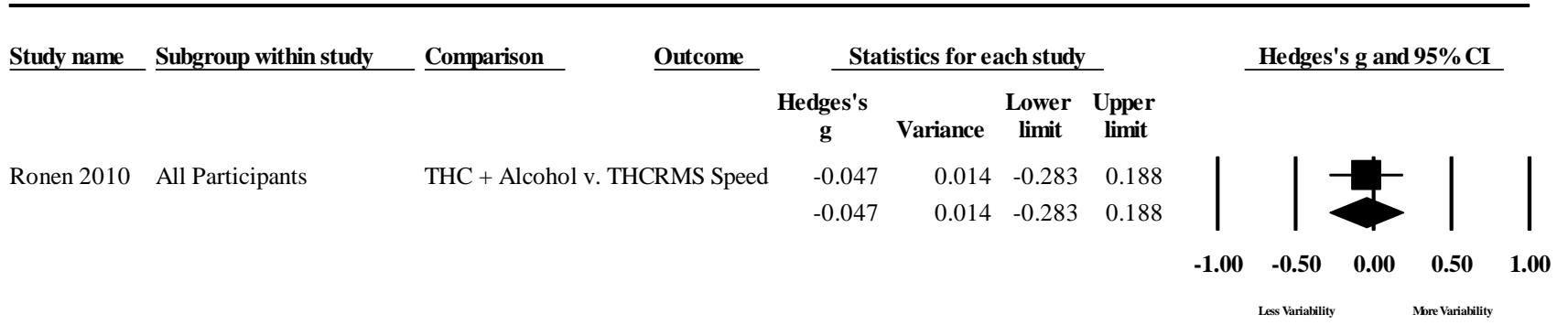


Figure C161. Forest plot illustrating *Combination v. Cannabis: Speed Variability* (missing pre-post correlations set to $r = 0.9$).

Combination v. Cannabis: Speed Exceedances

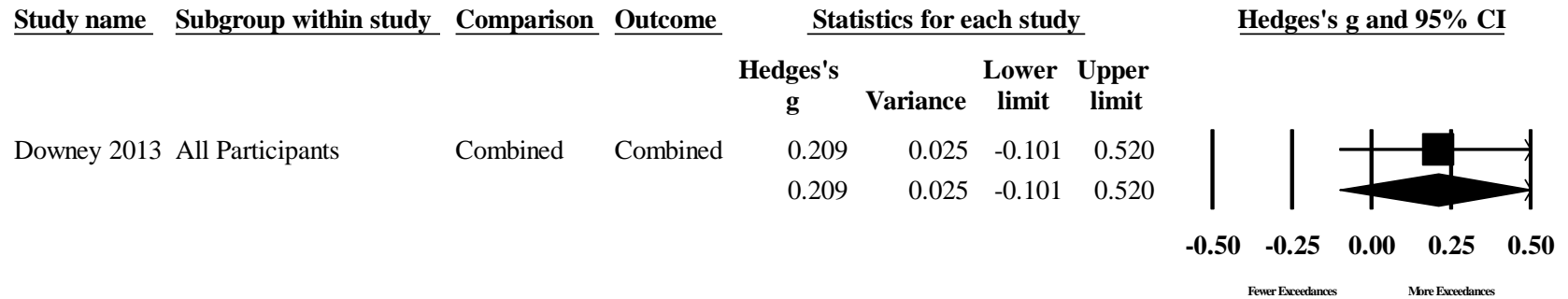


Figure C162. Forest plot illustrating *Combination v. Cannabis: Speed Exceedances* (missing pre-post correlations set to $r = 0$).

Combination v. Cannabis: Speed Exceedances

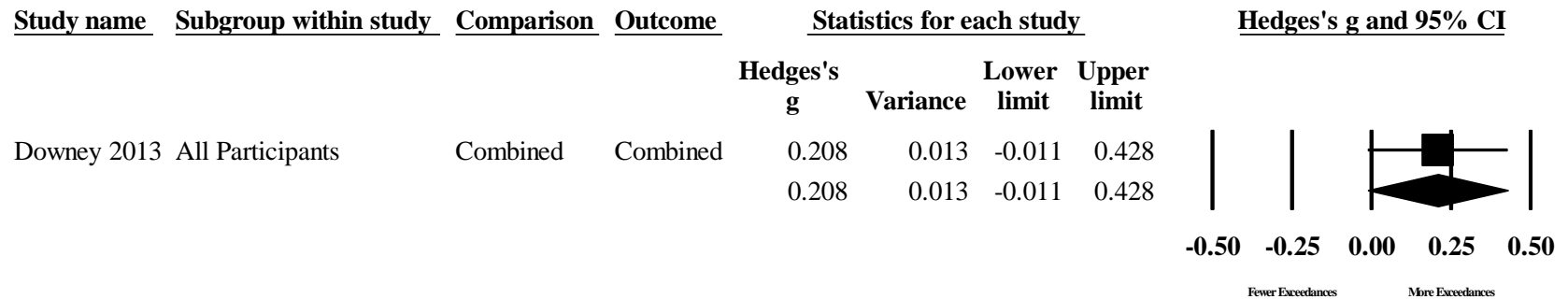


Figure C163. Forest plot illustrating *Combination v. Cannabis: Speed Exceedances* (missing pre-post correlations set to $r = 0.5$).

Combination v. Cannabis: Speed Exceedances

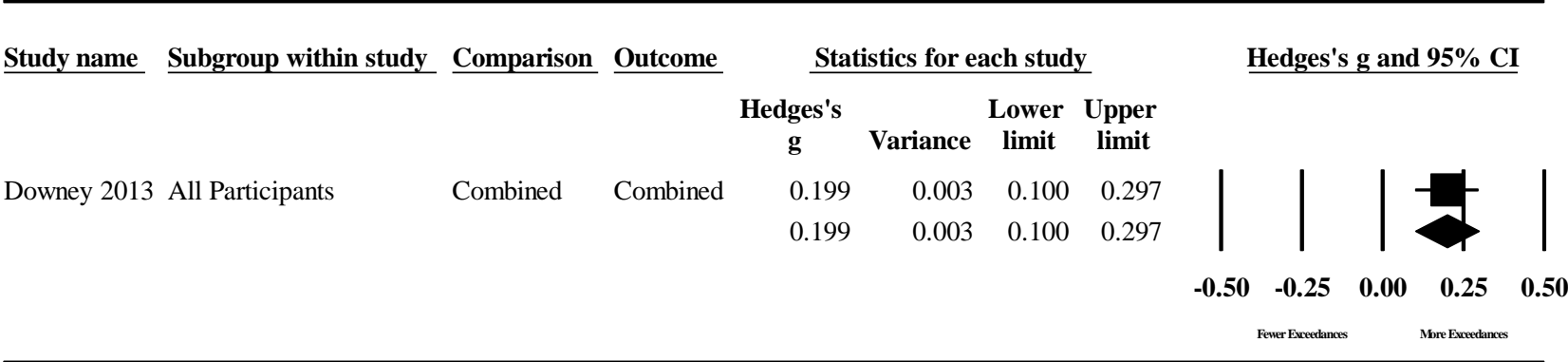


Figure C164. Forest plot illustrating *Combination v. Cannabis: Speed Exceedances* (missing pre-post correlations set to $r = 0.9$).

Appendix D: Forest Plots (Subgroup Analyses)

Alcohol/Cannabis v. Baseline: Crashes (Subgroup Analysis)

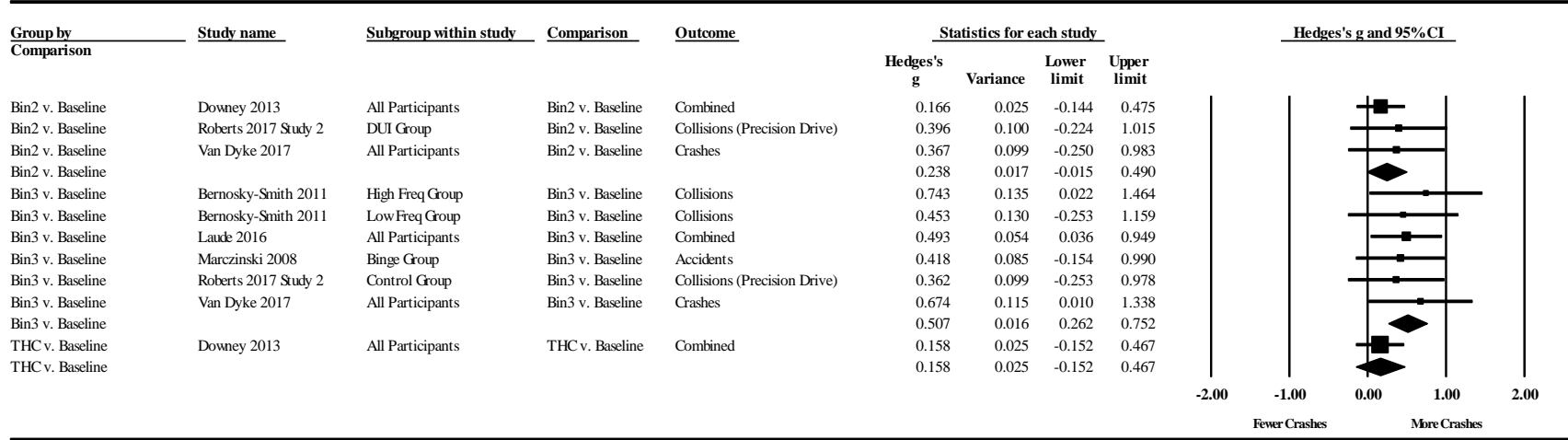


Figure D1. Forest plot illustrating the effects of varying levels of alcohol, and THC, on crashes. Missing pre-post correlations set to $r = \text{zero}$.

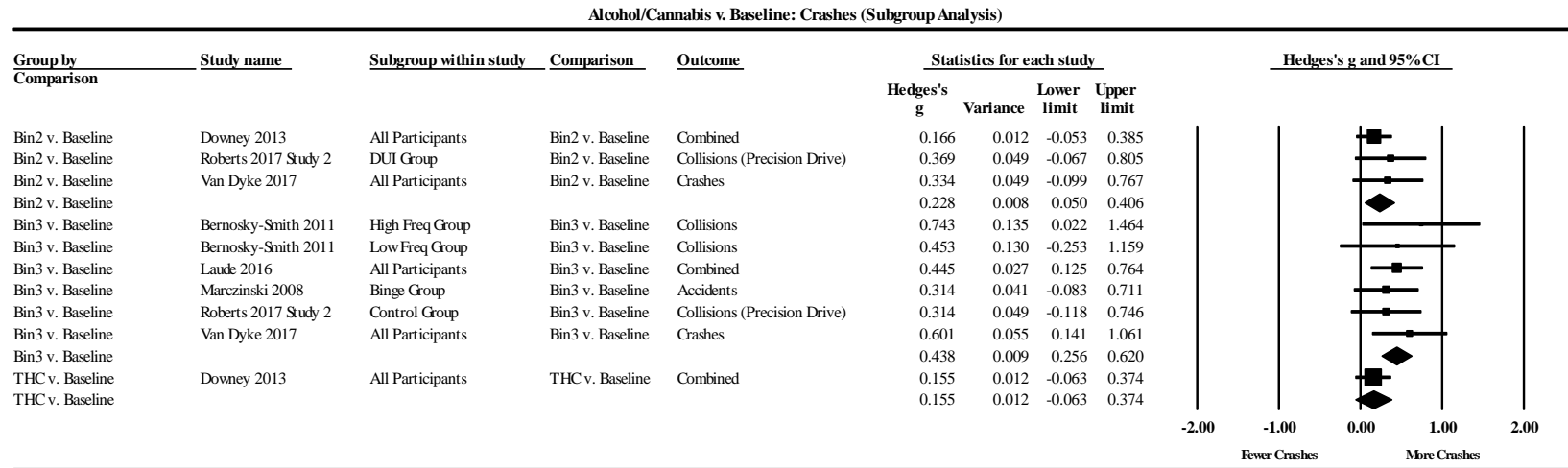


Figure D2. Forest plot illustrating the effects of varying levels of alcohol, and THC, on crashes. Missing pre-post correlations set to $r = 0.5$.

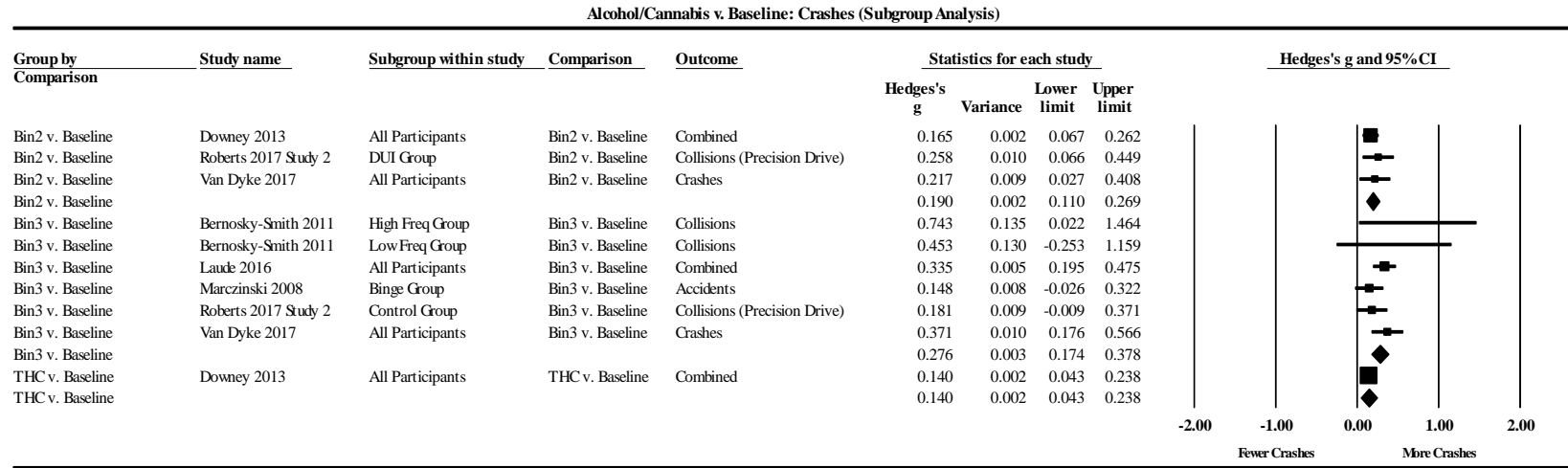


Figure D3. Forest plot illustrating the effects of varying levels of alcohol, and THC, on crashes. Missing pre-post correlations set to $r = 0.9$.

Alcohol/Cannabis v. Baseline: Hazard RT (Subgroup Analysis)

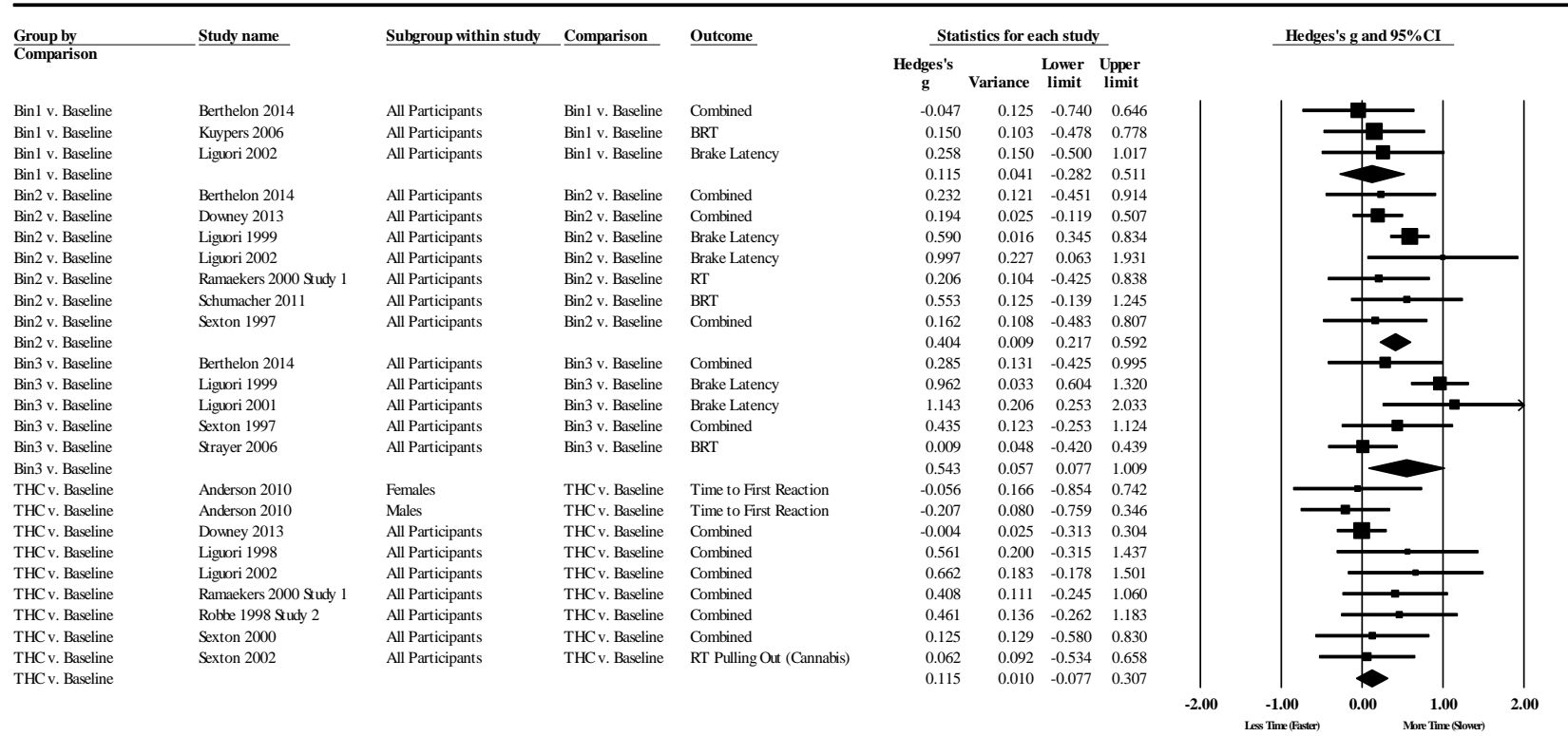


Figure D4. Forest plot illustrating the effects of varying levels of alcohol, and THC, on hazard RT. Missing pre-post correlations set to $r = \text{zero}$.

Alcohol/Cannabis v. Baseline: Hazard RT (Subgroup Analysis)

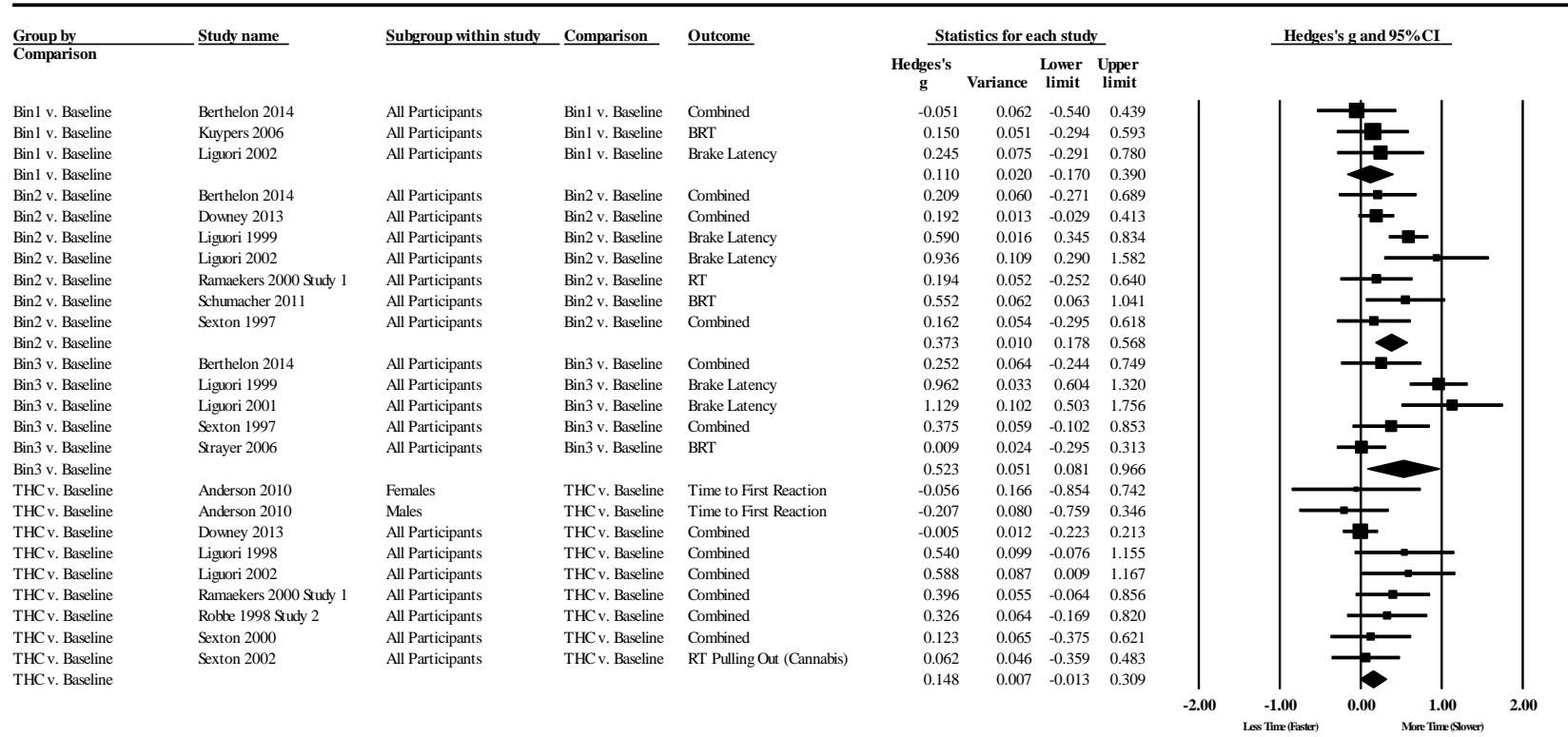


Figure D5. Forest plot illustrating the effects of varying levels of alcohol, and THC, on hazard RT. Missing pre-post correlations set to $r = 0.5$.

Alcohol/Cannabis v. Baseline: Hazard RT (Subgroup Analysis)

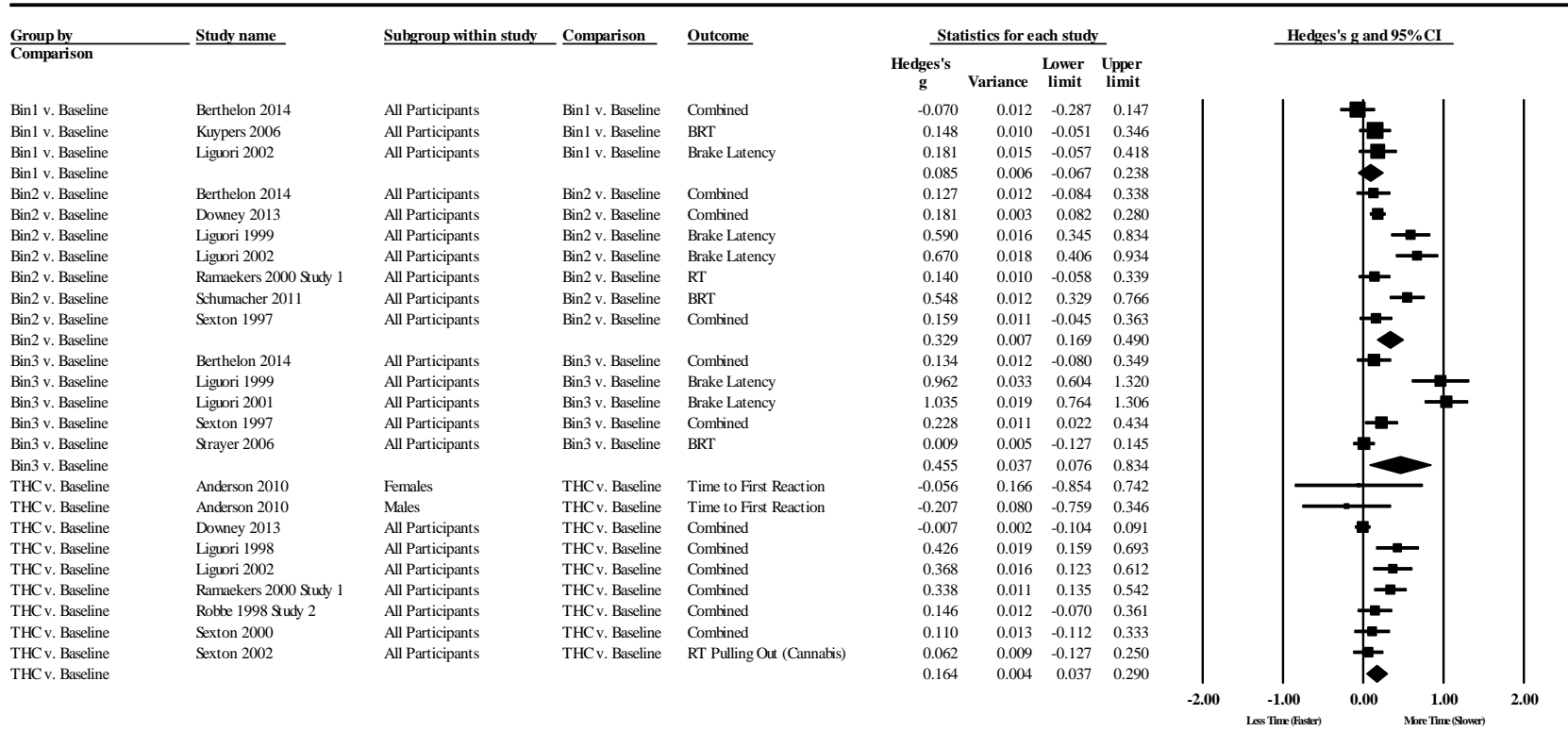


Figure D6. Forest plot illustrating the effects of varying levels of alcohol, and THC, on hazard RT. Missing pre-post correlations set to $r = 0.9$.

Alcohol/Cannabis vs. Baseline: Lateral Position Variability (Subgroup Analysis)

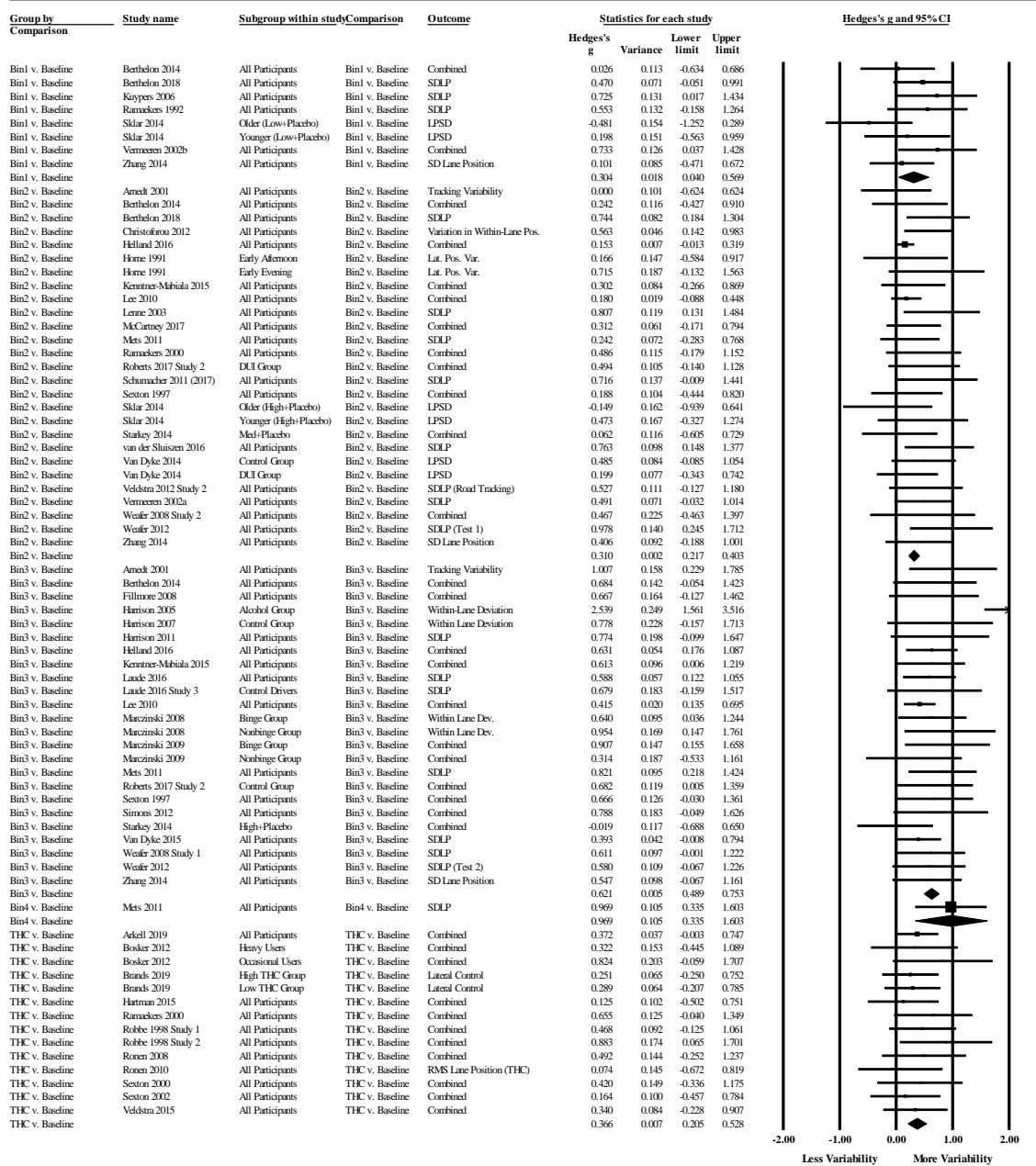


Figure D7. Forest plot illustrating the effects of varying levels of alcohol, and THC, on lateral position variability. Missing pre-post correlations set to $r = 0$.

Alcohol/Cannabis vs. Baseline: Lateral Position Variability (Subgroup Analysis)

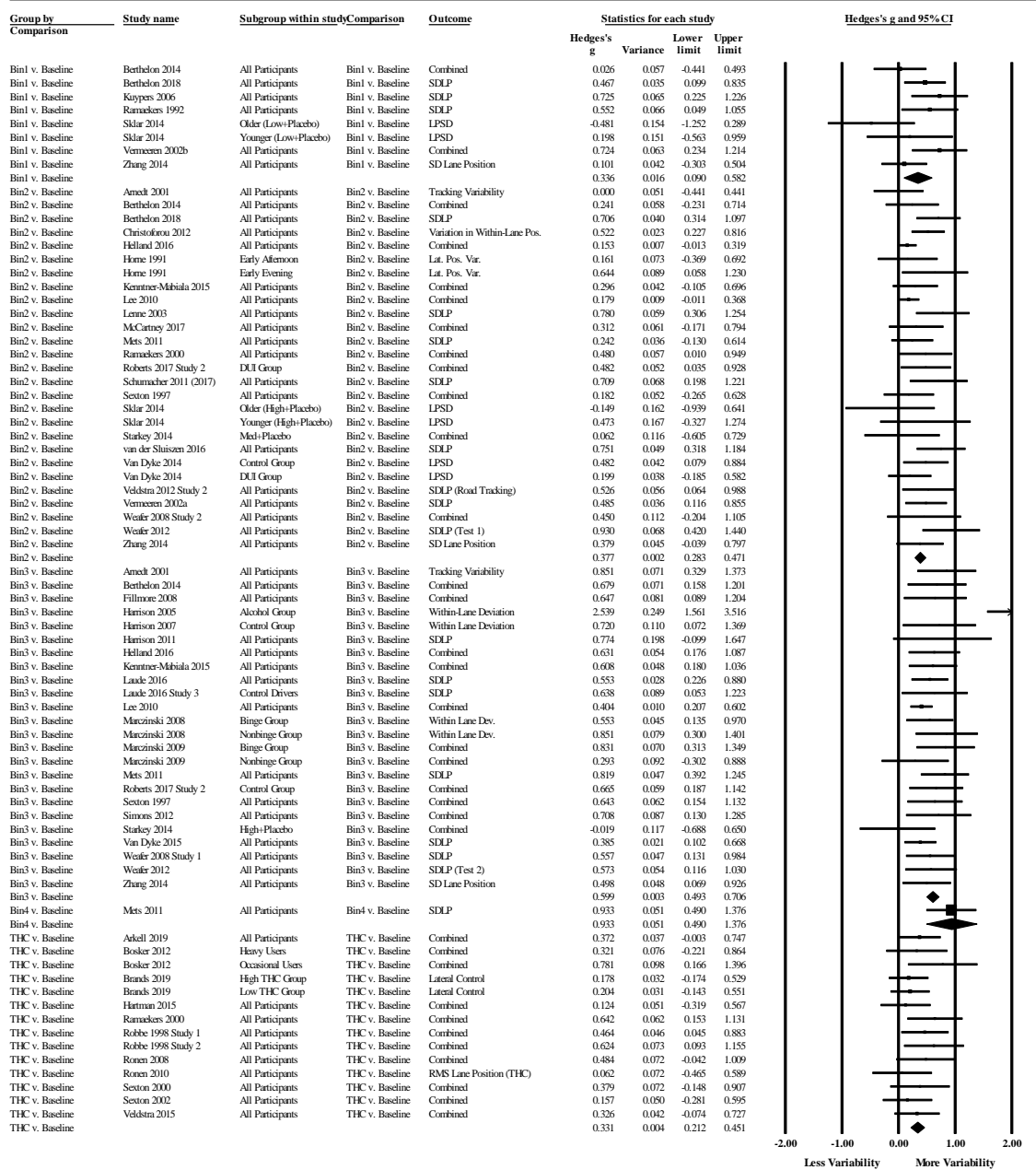


Figure D8. Forest plot illustrating the effects of varying levels of alcohol, and THC, on lateral position variability. Missing pre-post correlations set to $r = 0.5$.

Alcohol/Cannabis vs. Baseline: Lateral Position Variability (Subgroup Analysis)

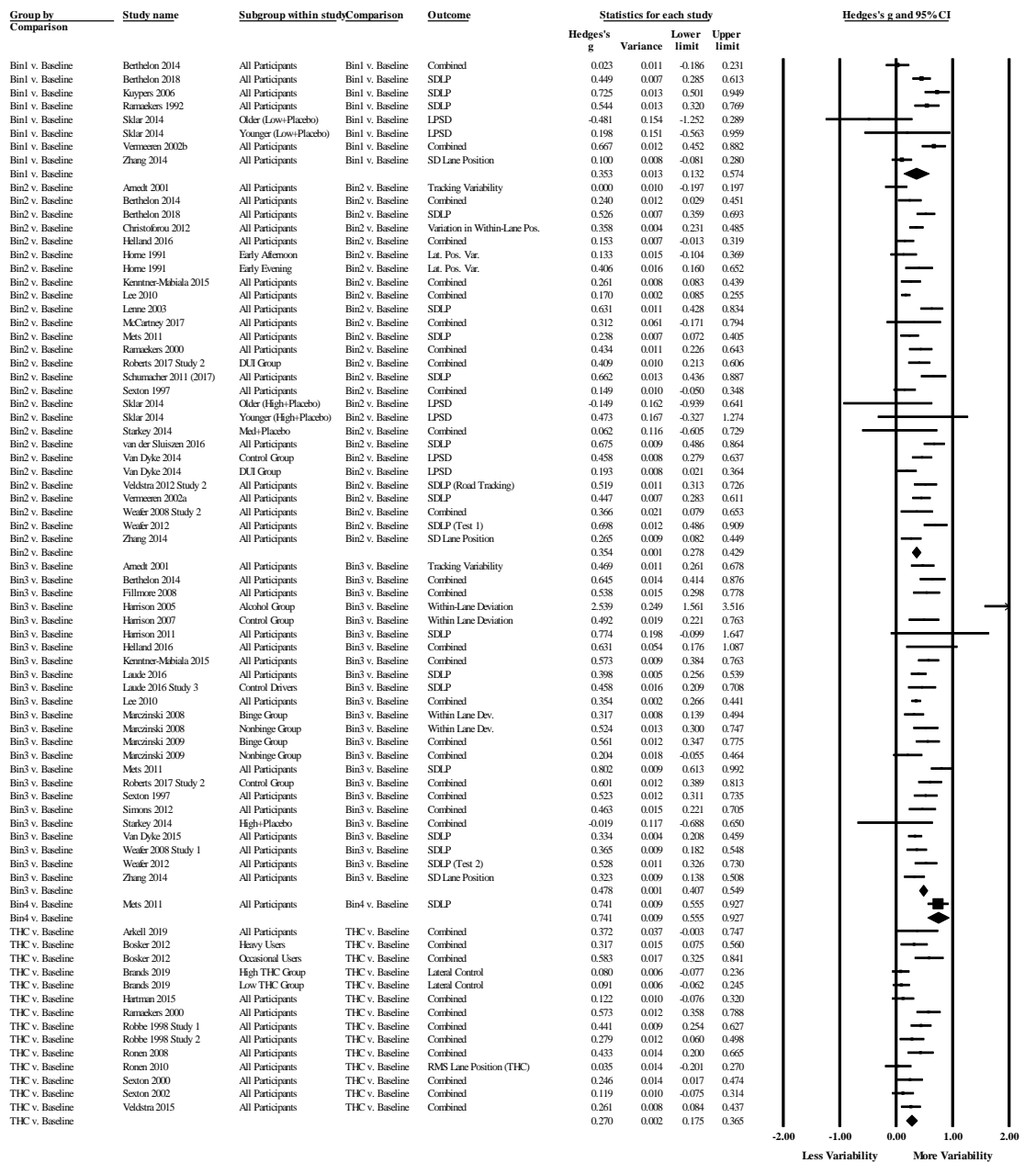


Figure D9. Forest plot illustrating the effects of varying levels of alcohol, and THC, on lateral position variability. Missing pre-post correlations set to $r = 0.9$.

Alcohol/Cannabis v. Baseline: Lane Excursions (Subgroup Analysis)

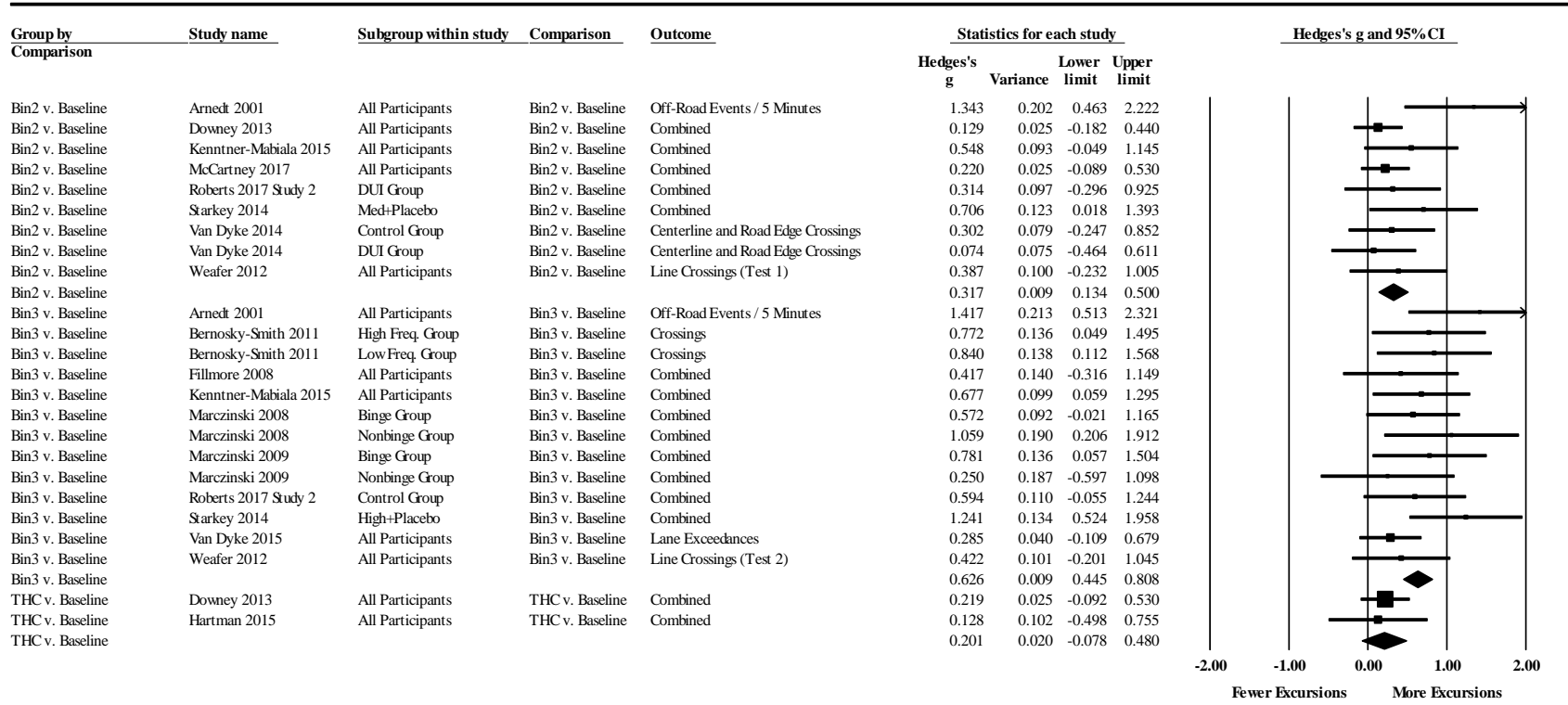


Figure D10. Forest plot illustrating the effects of varying levels of alcohol, and THC, on lane excursions. Missing pre-post correlations set to $r = \text{zero}$.

Alcohol/Cannabis v. Baseline: Lane Excursions (Subgroup Analysis)

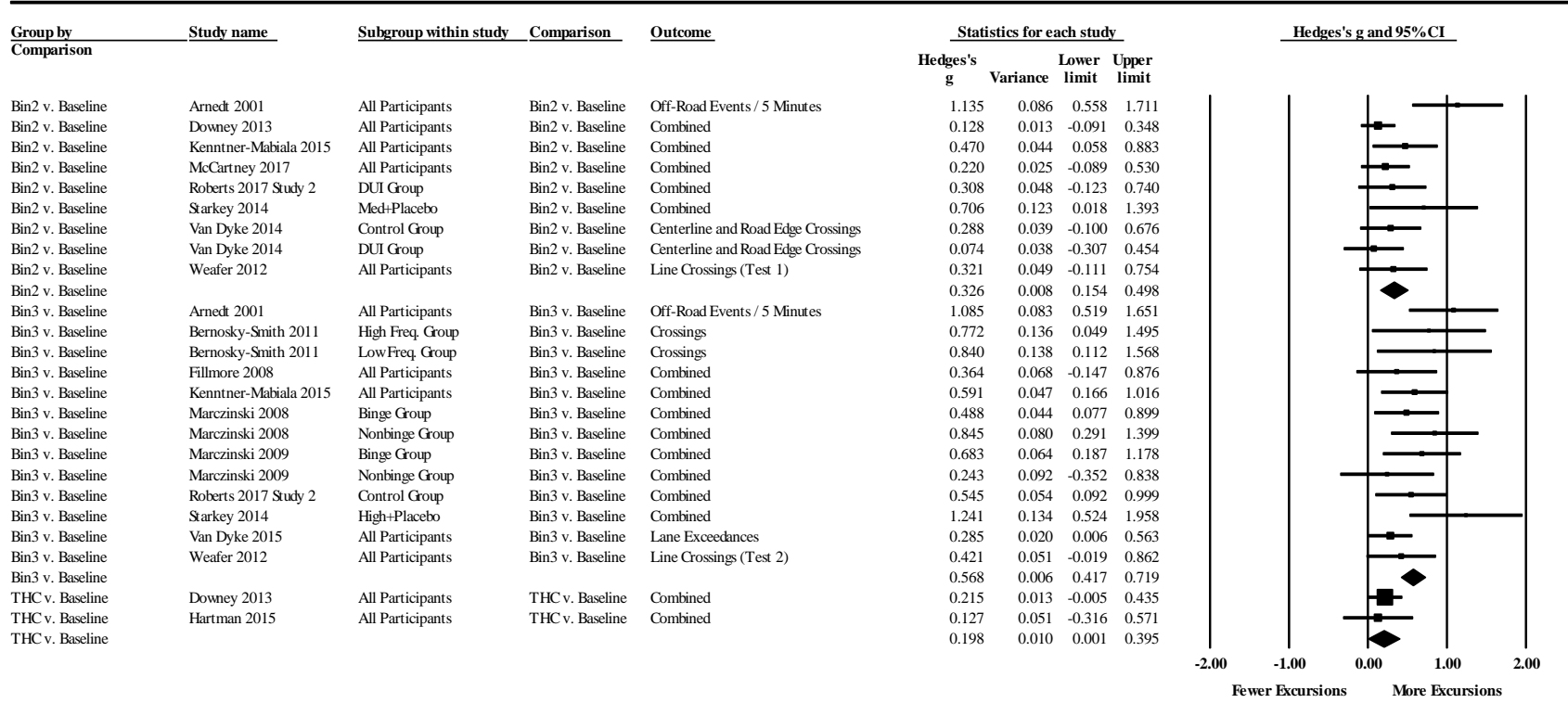


Figure D11. Forest plot illustrating the effects of varying levels of alcohol, and THC, on lane excursions. Missing pre-post correlations set to $r = 0.5$.

Alcohol/Cannabis v. Baseline: Lane Excursions (Subgroup Analysis)

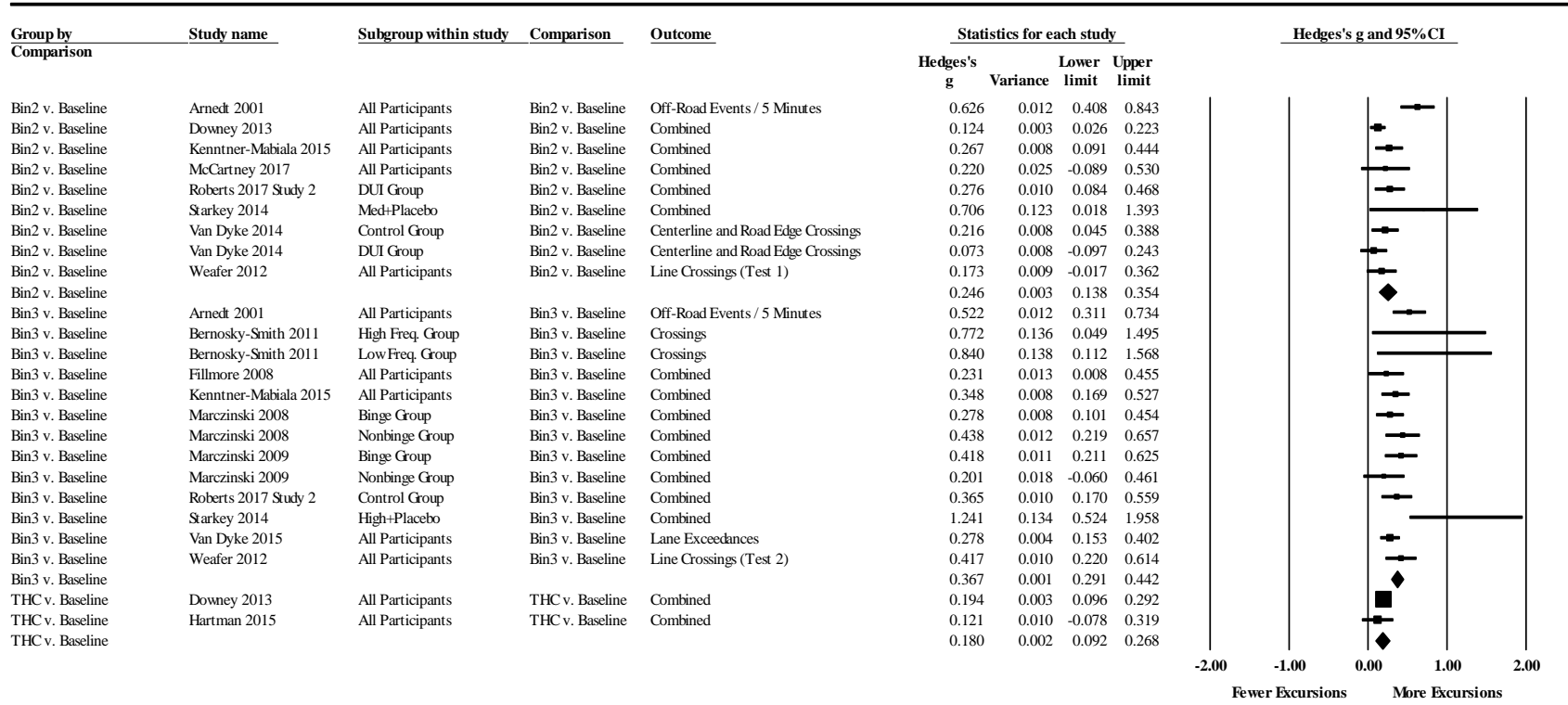


Figure D12. Forest plot illustrating the effects of varying levels of alcohol, and THC, on lane excursions. Missing pre-post correlations set to $r = 0.9$.

Alcohol/Cannabis v. Baseline: Speed (Subgroup Analysis)

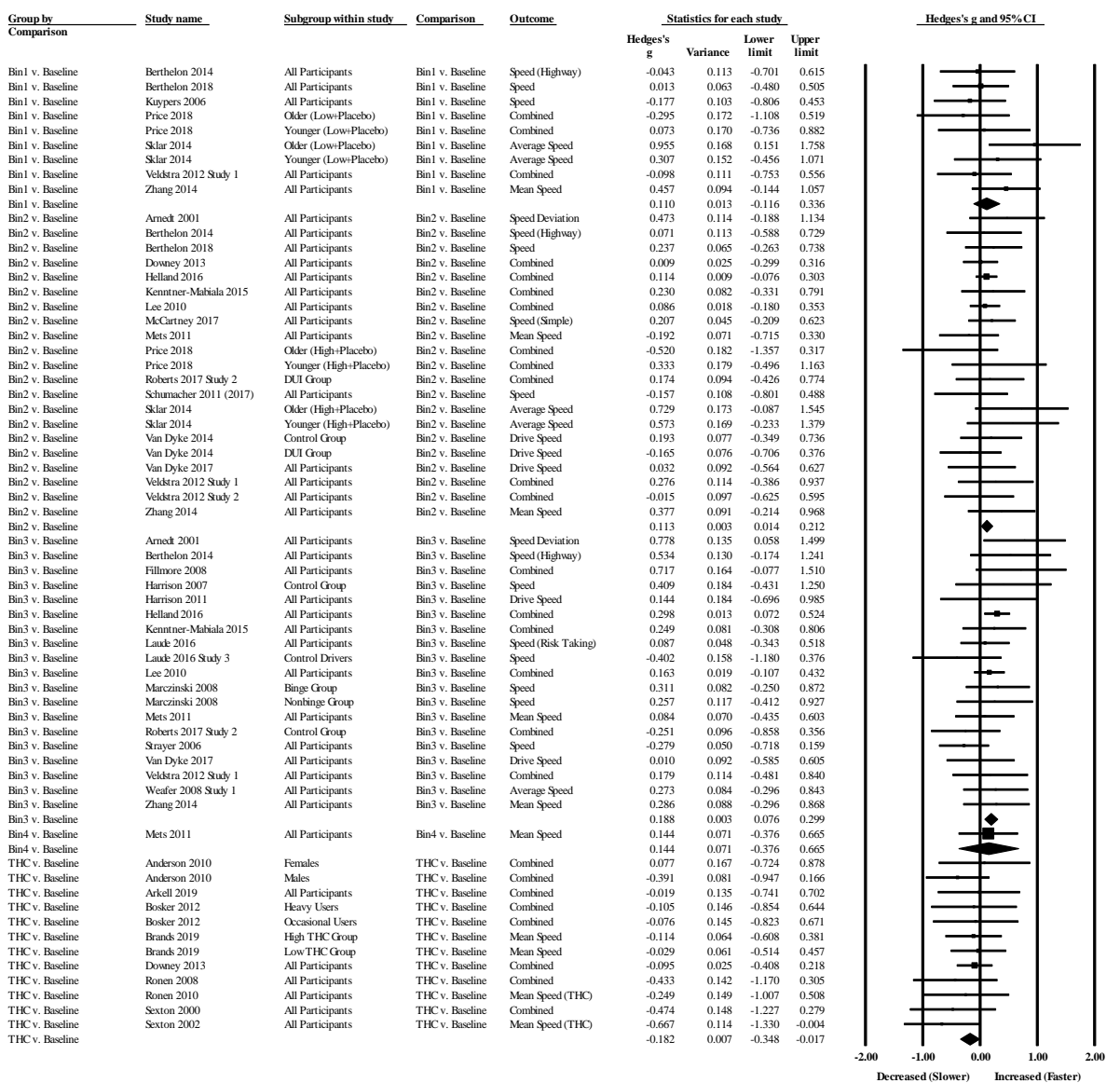


Figure D13. Forest plot illustrating the effects of varying levels of alcohol, and THC, on speed. Missing pre-post correlations set to $r = \text{zero}$.

Alcohol/Cannabis v. Baseline: Speed (Subgroup Analysis)

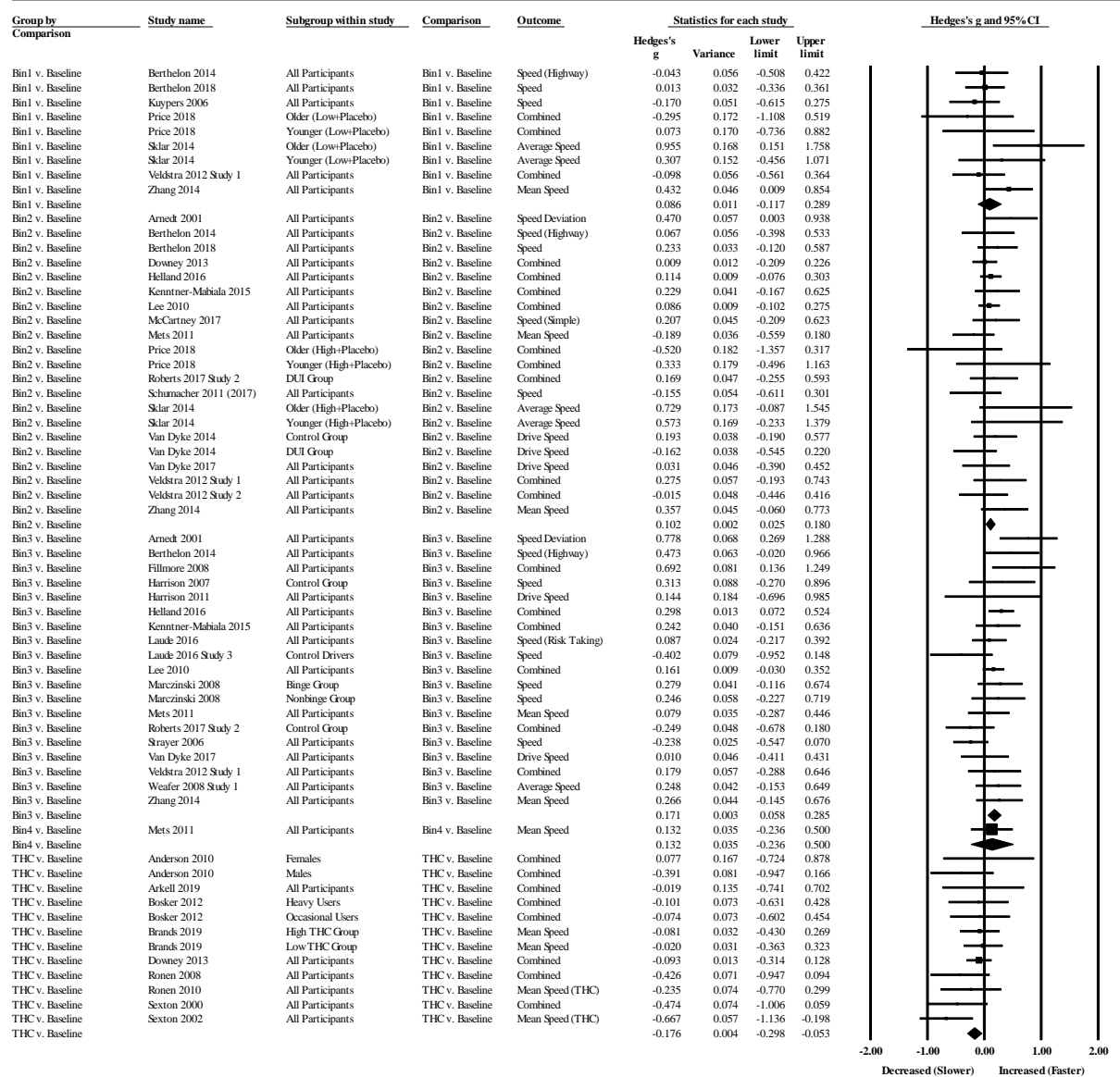


Figure D14. Forest plot illustrating the effects of varying levels of alcohol, and THC, on speed. Missing pre-post correlations set to $r = 0.5$.

Alcohol/Cannabis v. Baseline: Speed (Subgroup Analysis)

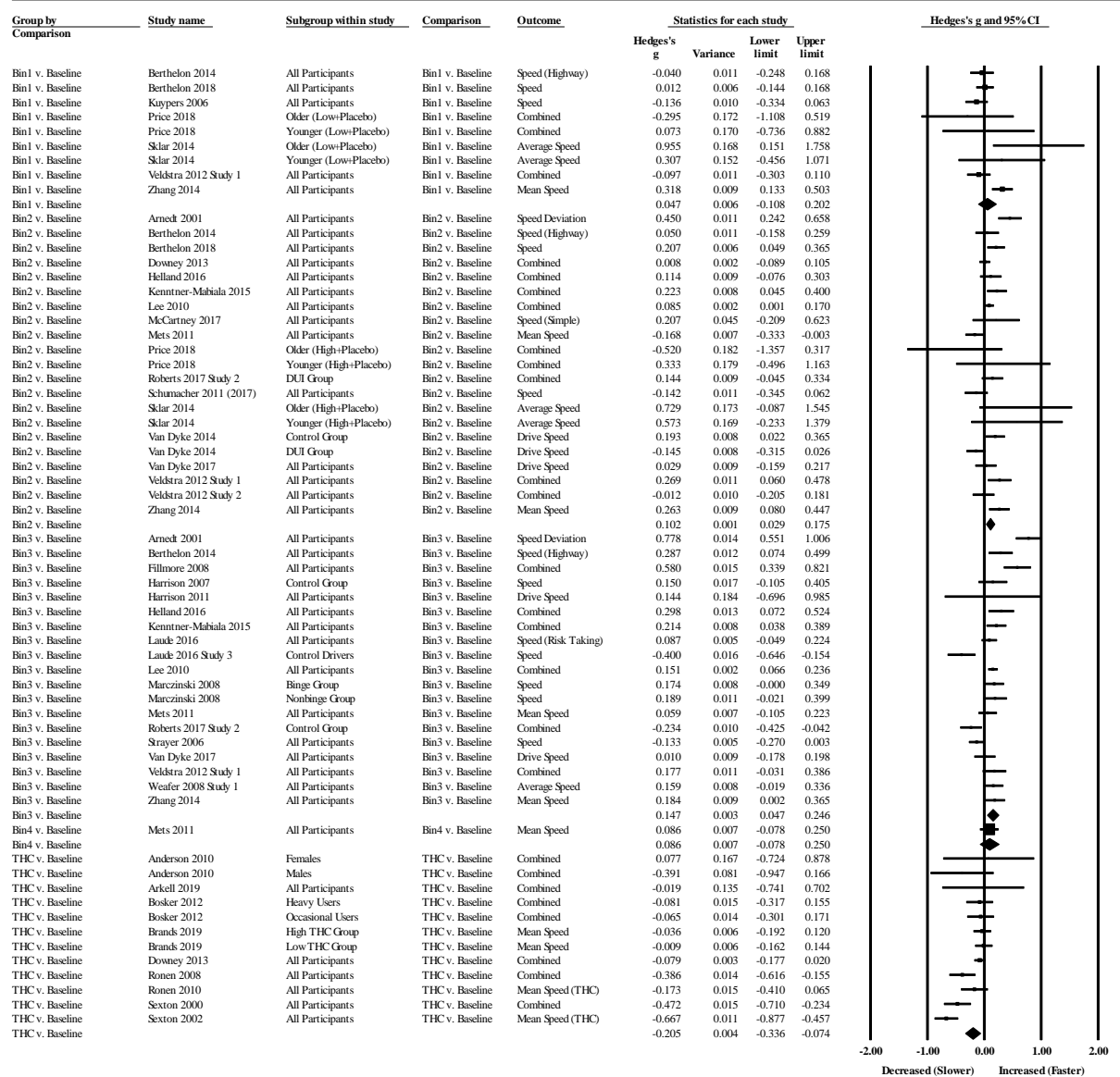


Figure D15. Forest plot illustrating the effects of varying levels of alcohol, and THC, on speed. Missing pre-post correlations set to $r = 0.9$.

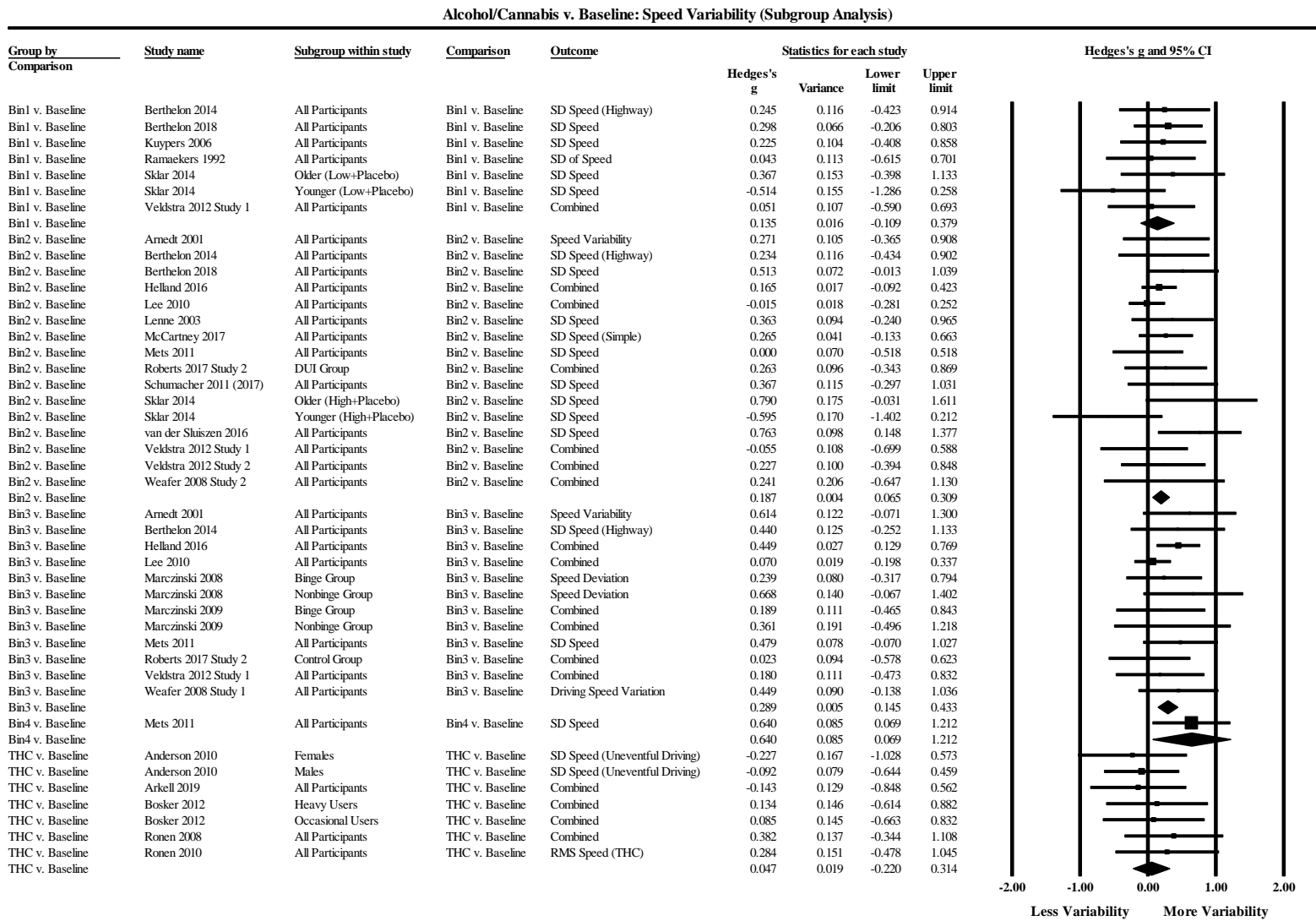


Figure D16. Forest plot illustrating the effects of varying levels of alcohol, and THC, on speed variability. Missing pre-post correlations set to $r = \text{zero}$.

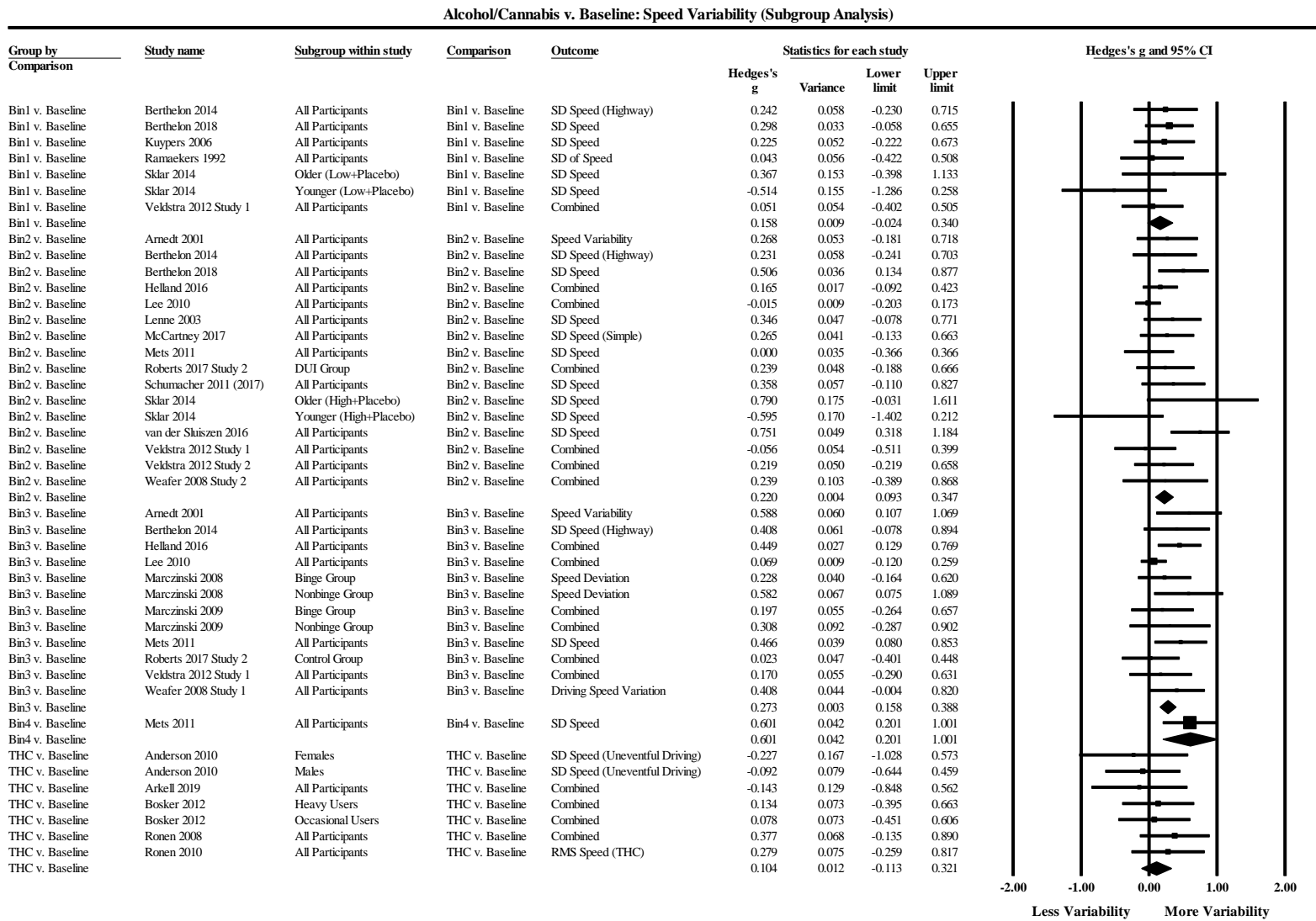


Figure D17. Forest plot illustrating the effects of varying levels of alcohol, and THC, on speed variability. Missing pre-post correlations set to $r = 0.5$.

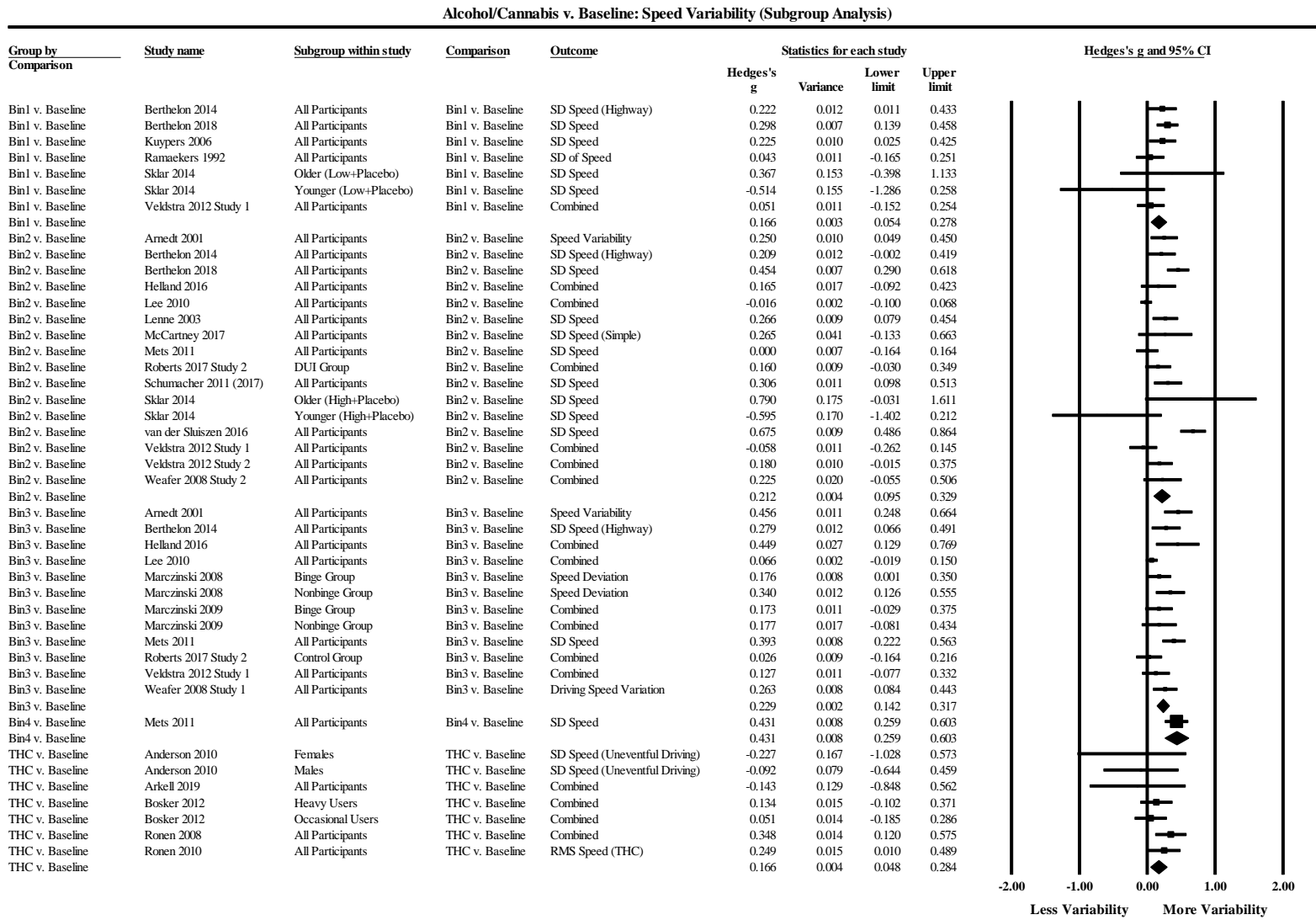


Figure D18. Forest plot illustrating the effects of varying levels of alcohol, and THC, on speed variability. Missing pre-post correlations set to $r = 0.9$.

Appendix E: Funnel Plots

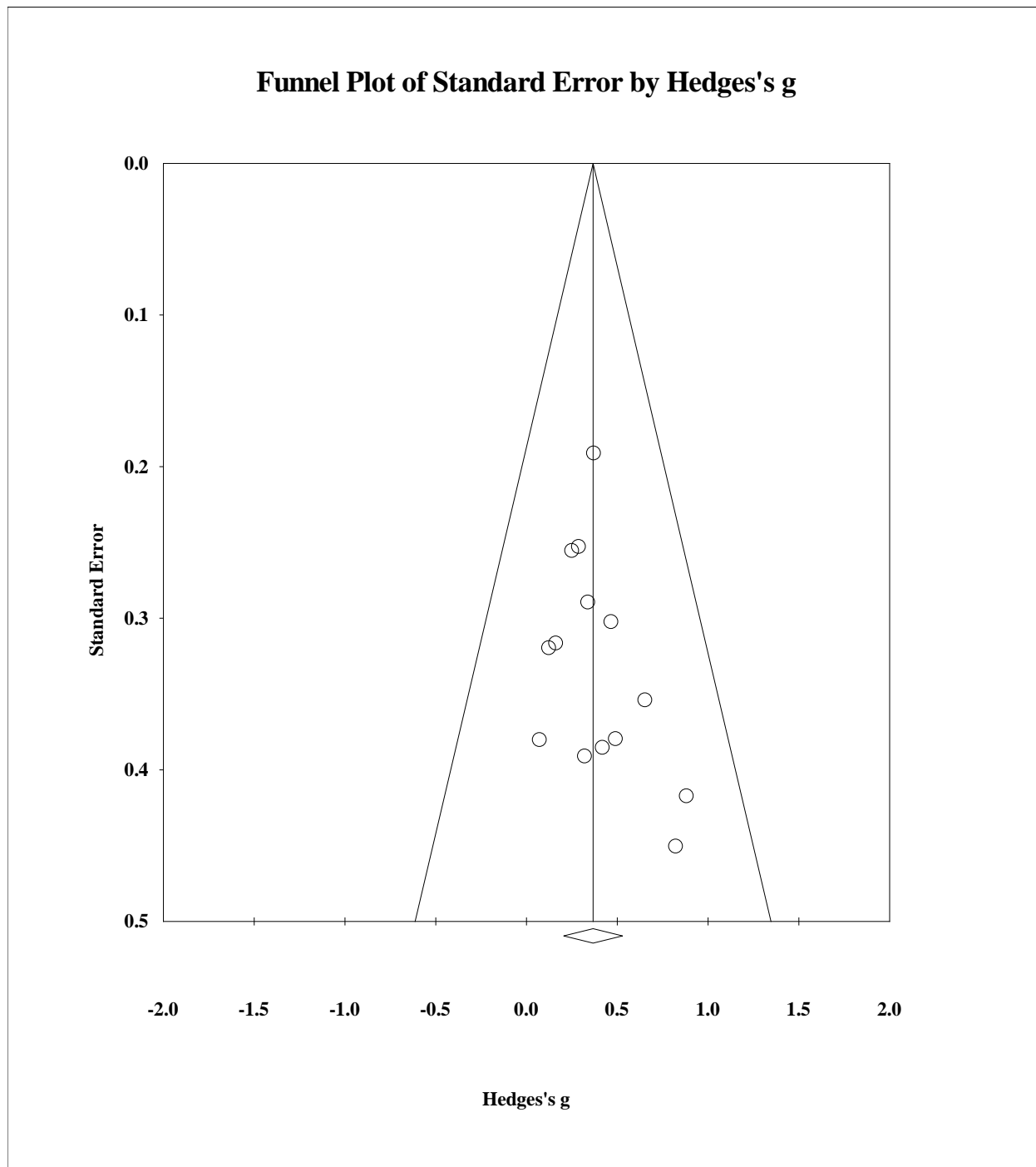


Figure E1. Funnel plot illustrating *Cannabis v. Baseline: Lateral Position Variability* (missing pre-post correlations set to $r = \text{zero}$).

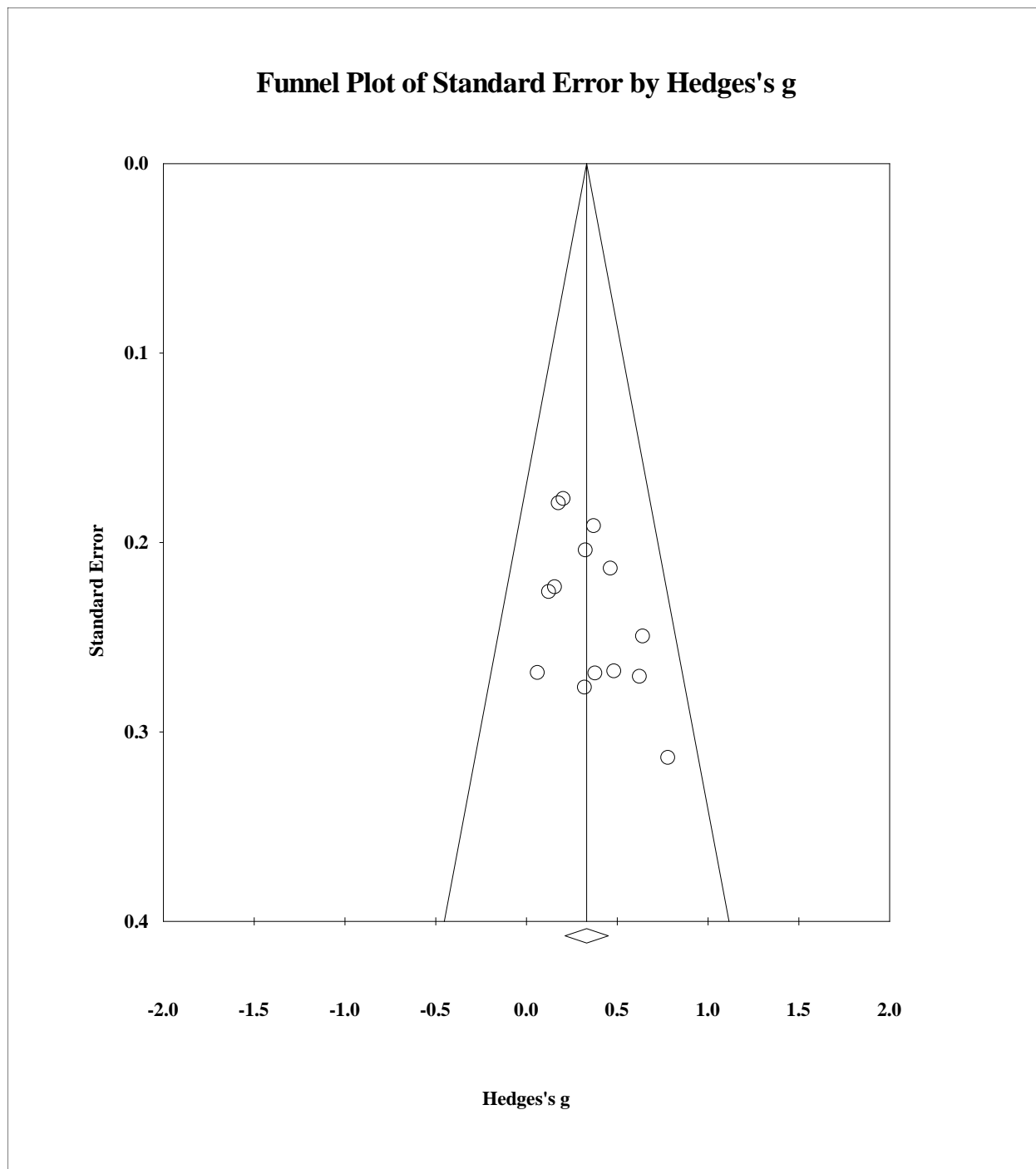


Figure E2. Funnel plot illustrating *Cannabis v. Baseline: Lateral Position Variability* (missing pre-post correlations set to $r = 0.5$).

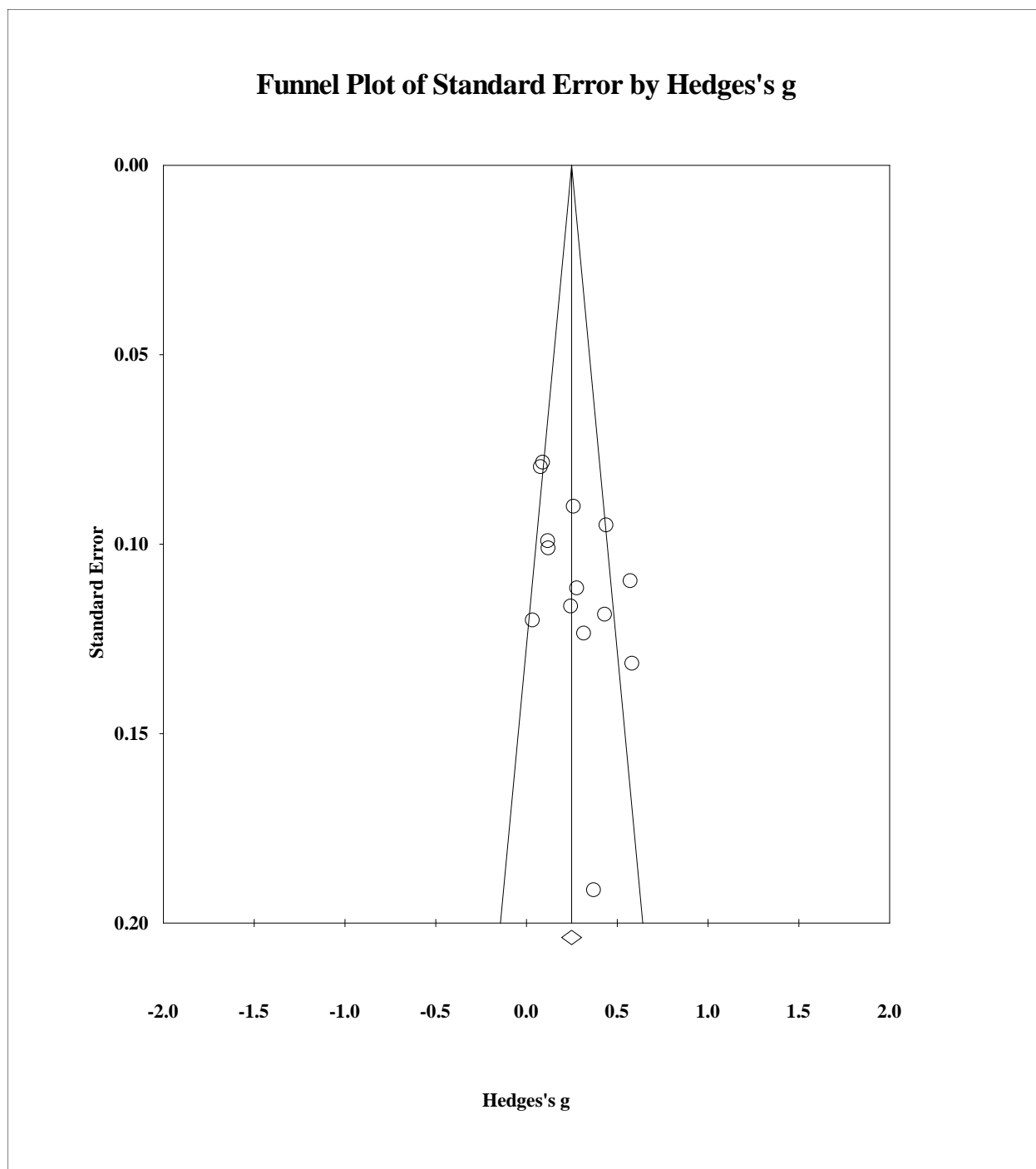


Figure E3. Funnel plot illustrating *Cannabis v. Baseline: Lateral Position Variability* (missing pre-post correlations set to $r = 0.9$).

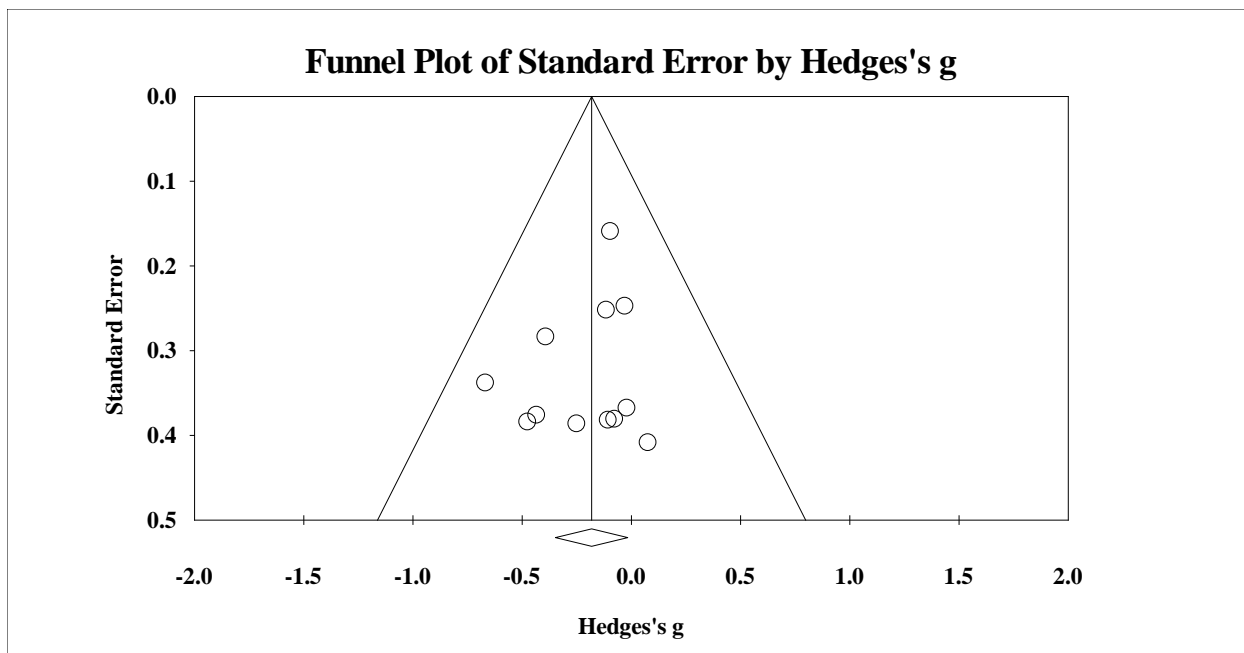


Figure E4. Funnel plot illustrating *Cannabis v. Baseline: Speed* (missing pre-post correlations set to $r = 0$).

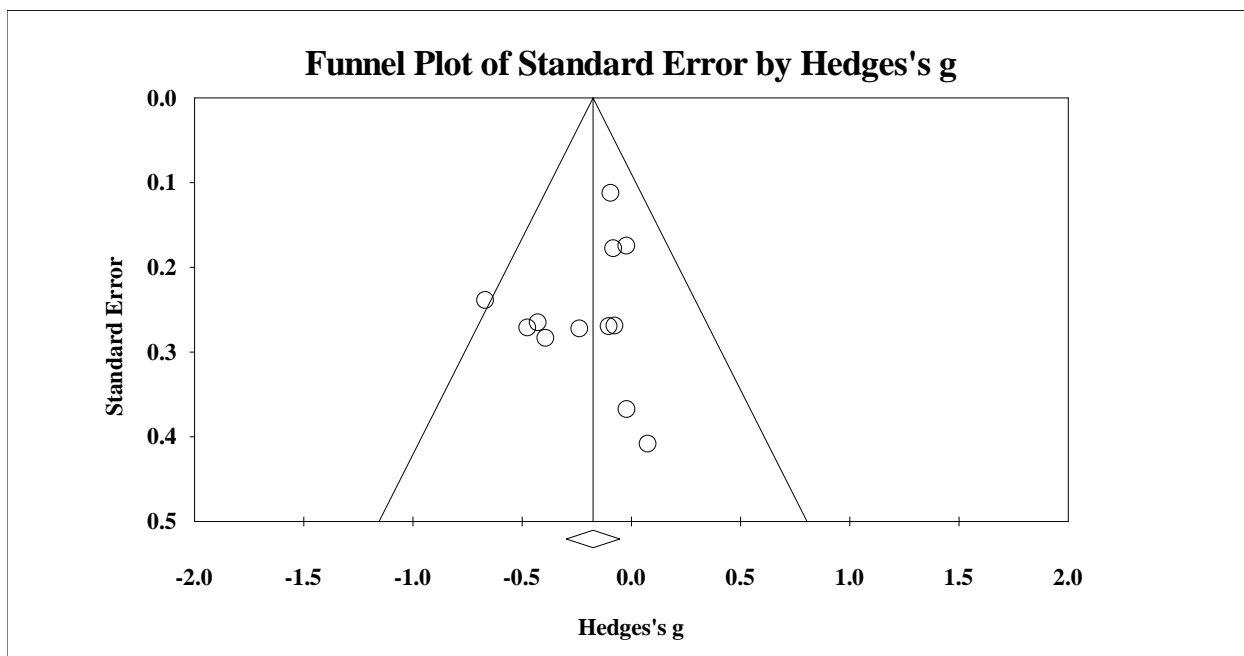


Figure E5. Funnel plot illustrating *Cannabis v. Baseline: Speed* (missing pre-post correlations set to $r = 0.5$).

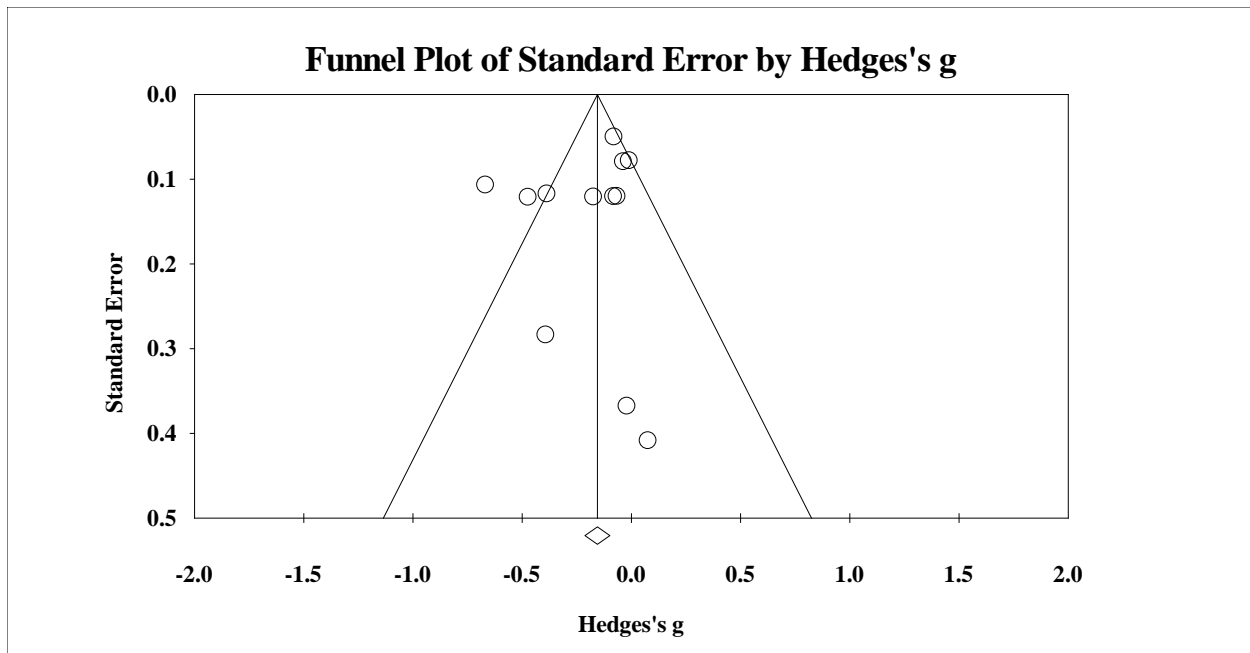


Figure E6. Funnel plot illustrating *Cannabis v. Baseline: Speed* (missing pre-post correlations set to $r = 0.9$).

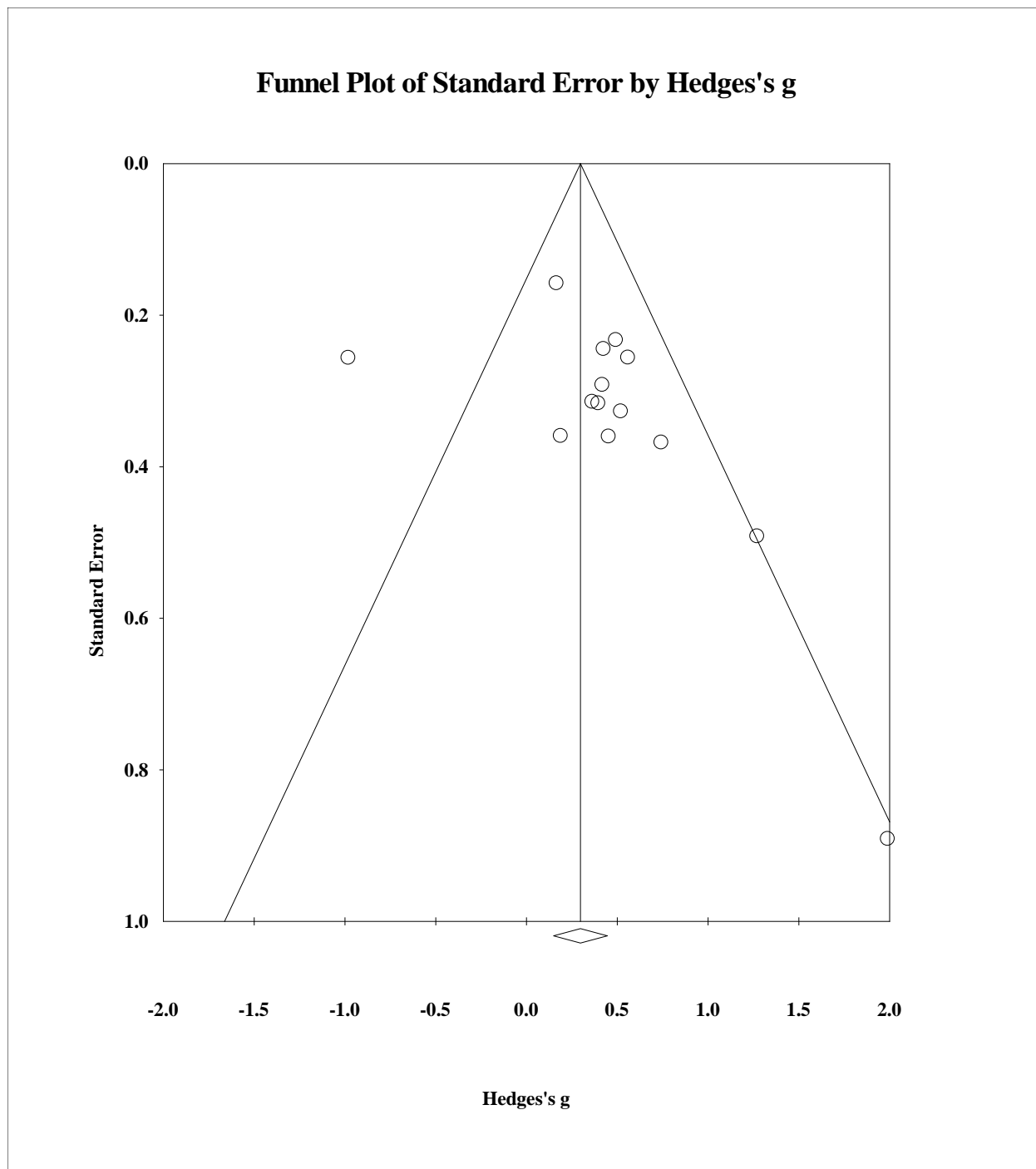


Figure E7. Funnel plot illustrating *Alcohol v. Baseline: Crashes* (missing pre-post correlations set to $r = \text{zero}$). Includes Bernosky-Smith et al., 2012.

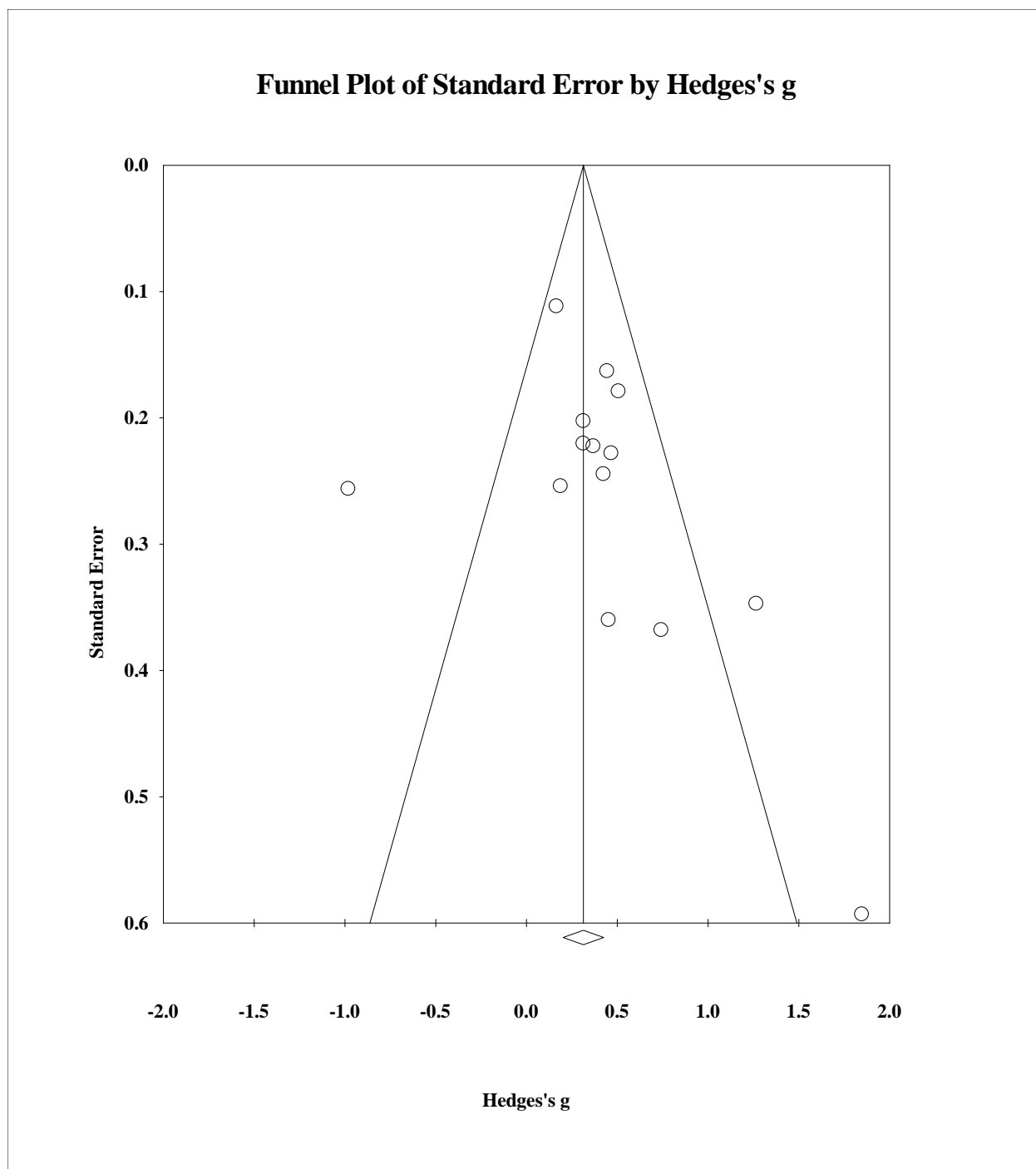


Figure E8. Funnel plot illustrating *Alcohol v. Baseline: Crashes* (missing pre-post correlations set to $r = 0.5$). Includes Bernosky-Smith et al., 2012.

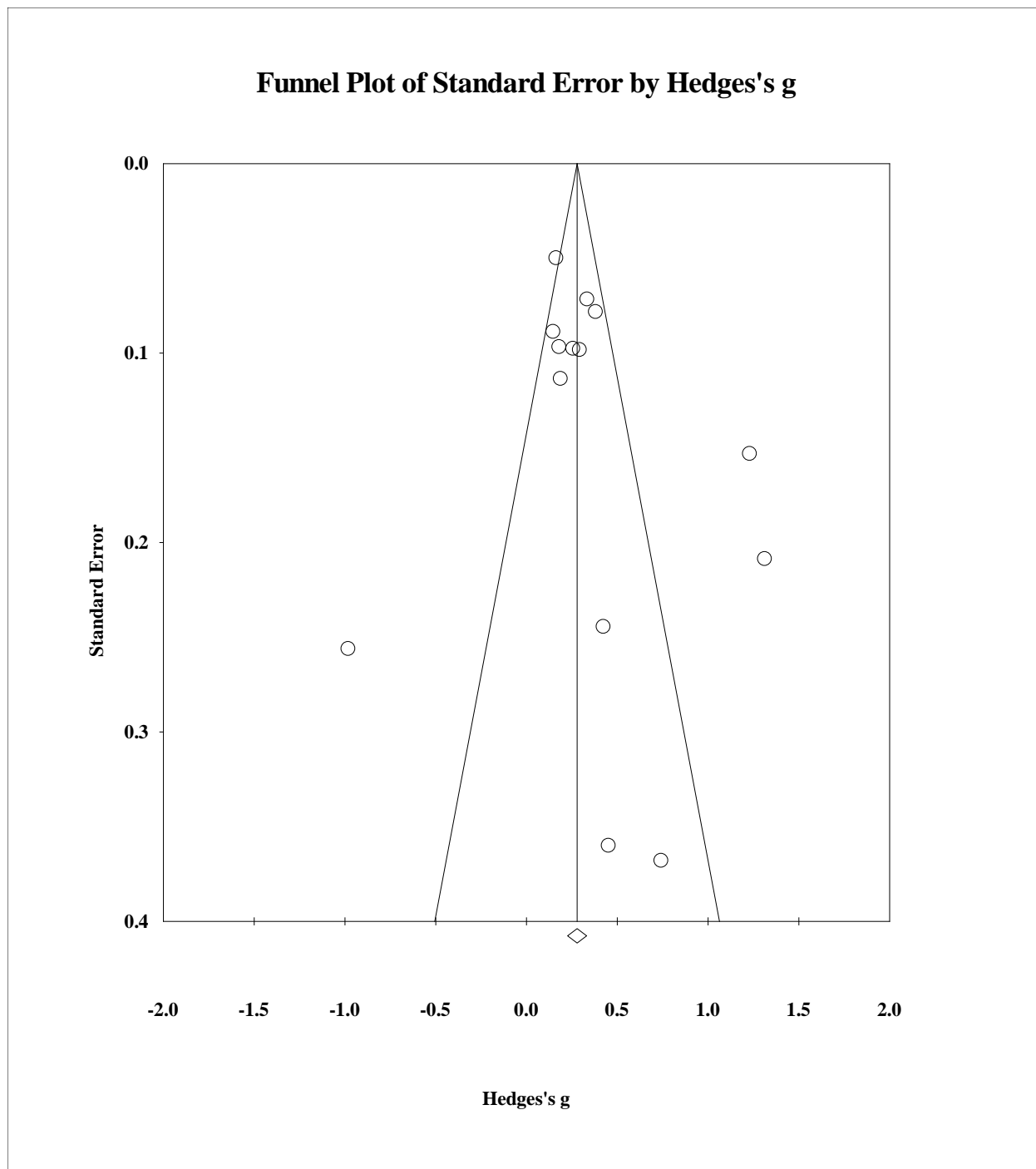


Figure E9. Funnel plot illustrating *Alcohol v. Baseline: Crashes* (missing pre-post correlations set to $r = 0.9$). Includes Bernosky-Smith et al., 2012.

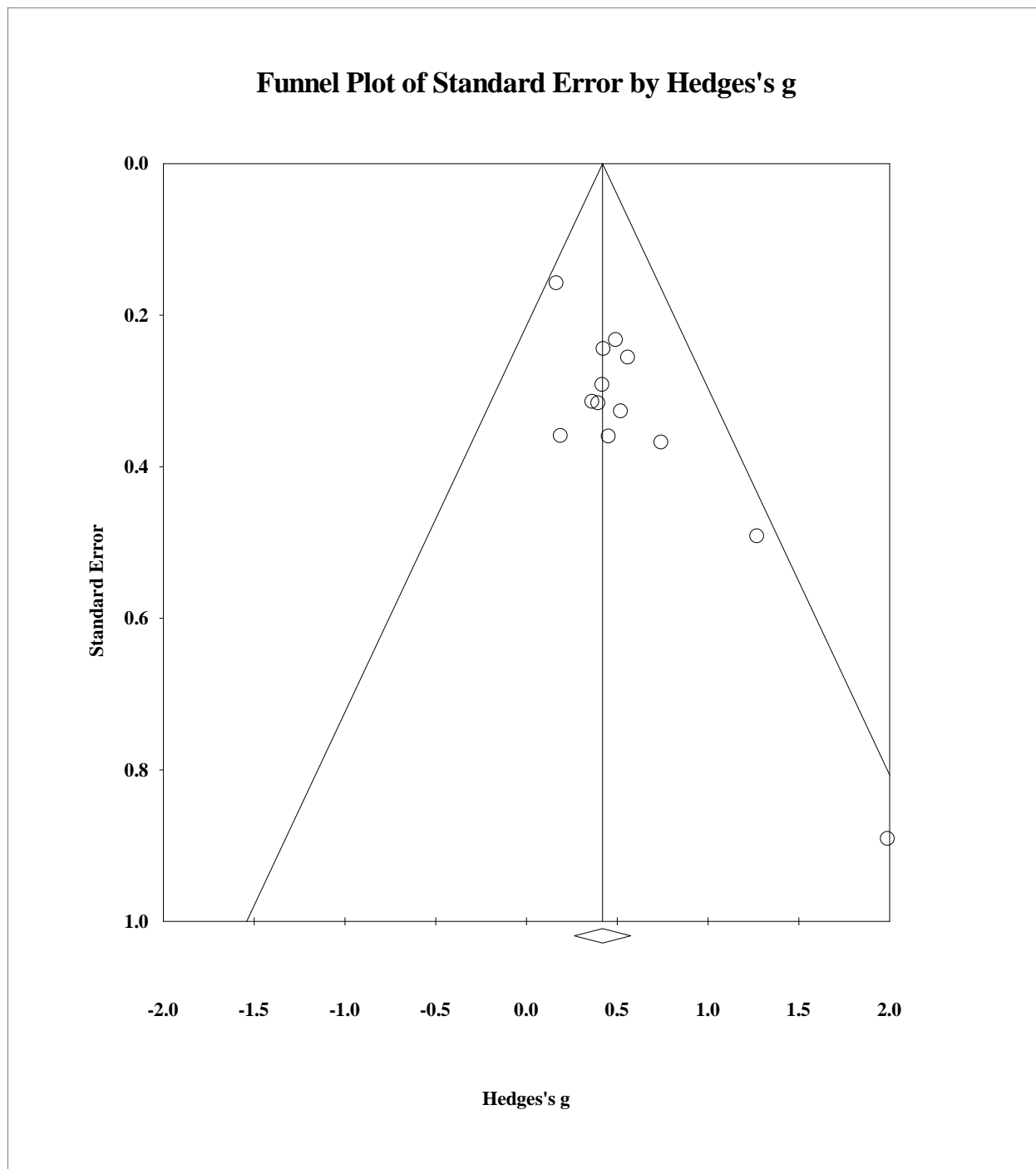


Figure E10. Funnel plot illustrating *Alcohol v. Baseline: Crashes* (missing pre-post correlations set to $r = \text{zero}$). Excludes Bernosky-Smith et al., 2012.

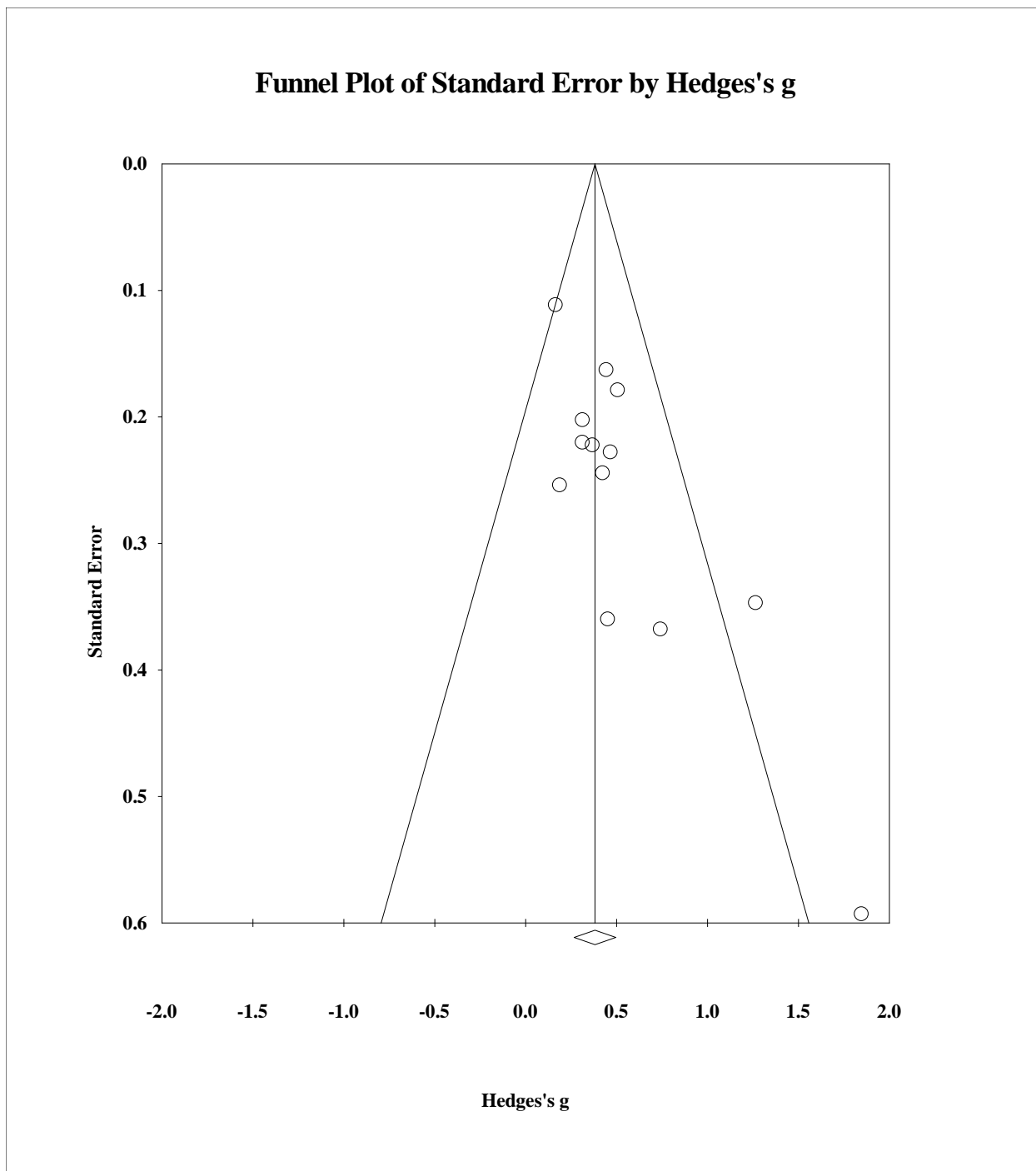


Figure E11. Funnel plot illustrating *Alcohol v. Baseline: Crashes* (missing pre-post correlations set to $r = 0.5$). Excludes Bernosky-Smith et al., 2012.

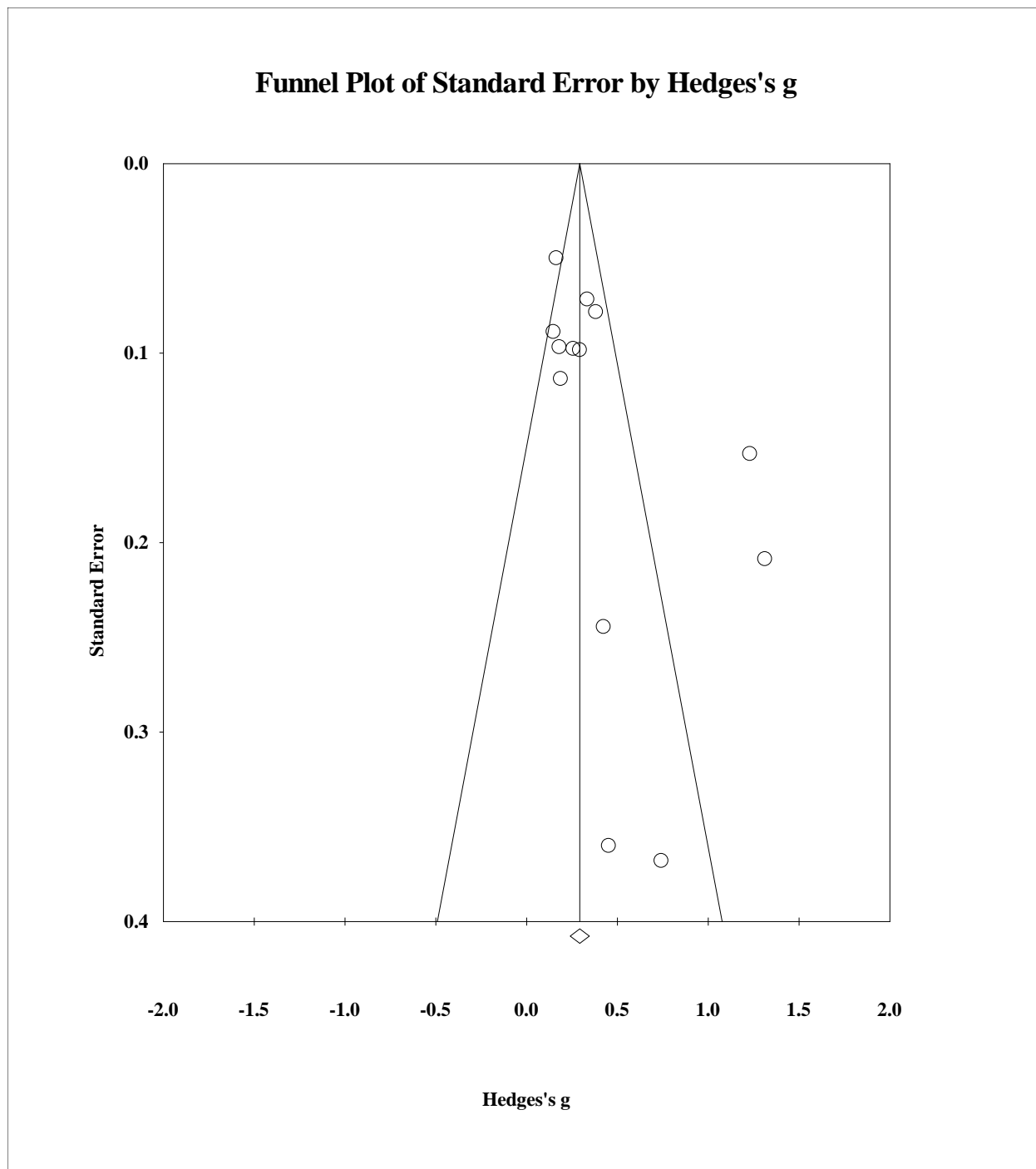


Figure E12. Funnel plot illustrating *Alcohol v. Baseline: Crashes* (missing pre-post correlations set to $r = 0.9$). Excludes Bernosky-Smith et al., 2012.

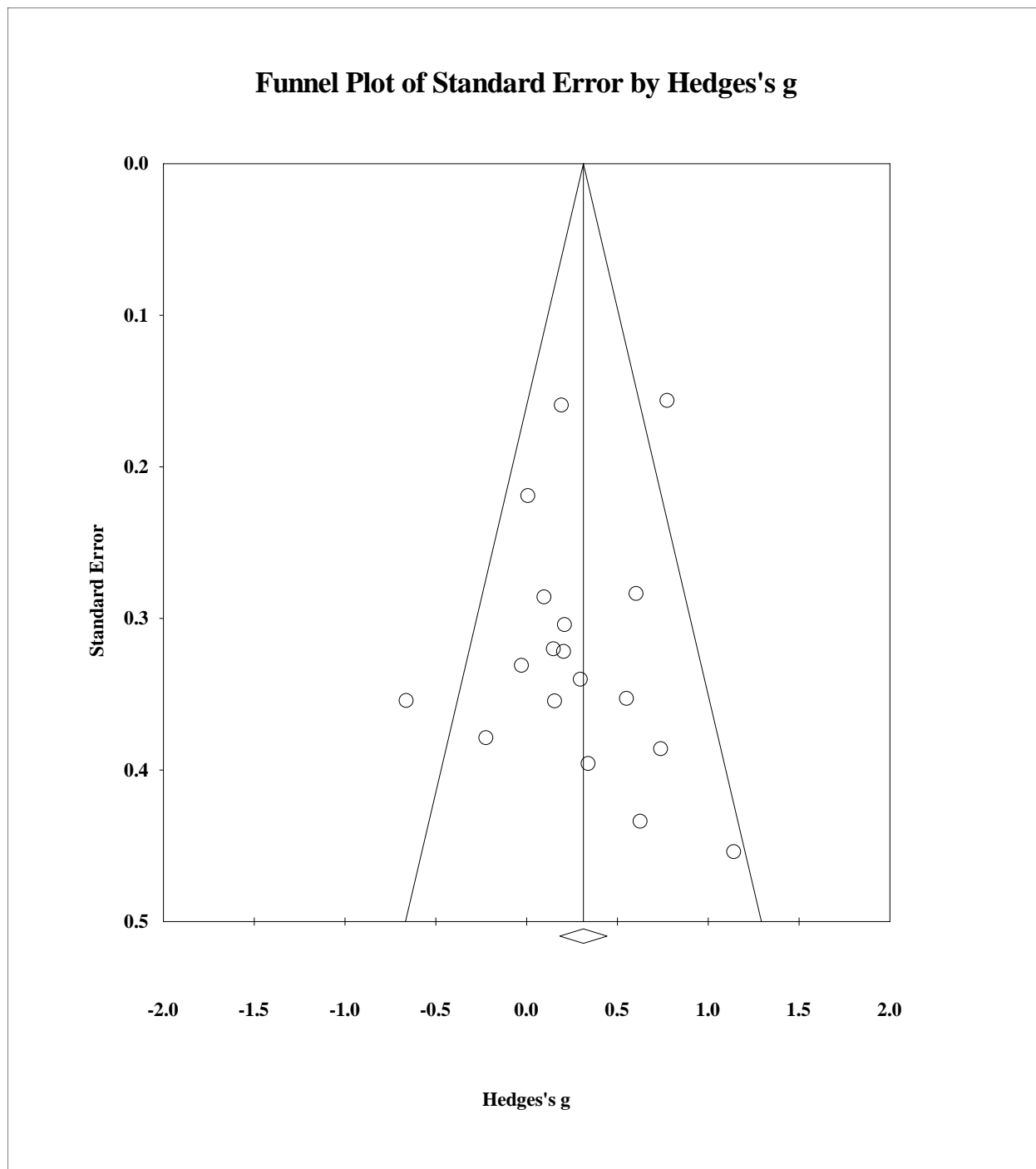


Figure E13. Funnel plot illustrating *Alcohol v. Baseline: Hazard RT* (missing pre-post correlations set to $r = \text{zero}$).

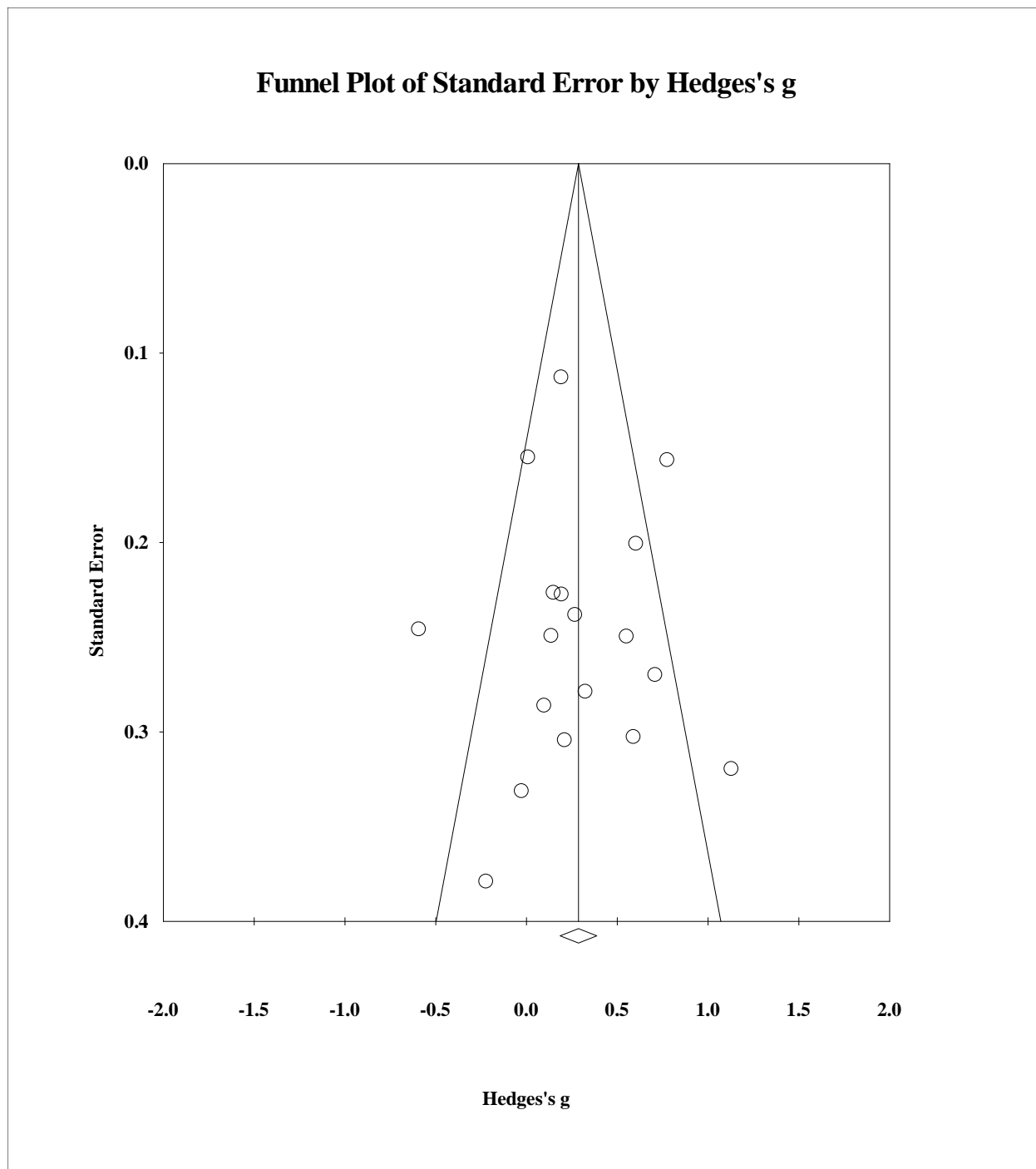


Figure E14. Funnel plot illustrating *Alcohol v. Baseline: Hazard RT* (missing pre-post correlations set to $r = 0.5$).

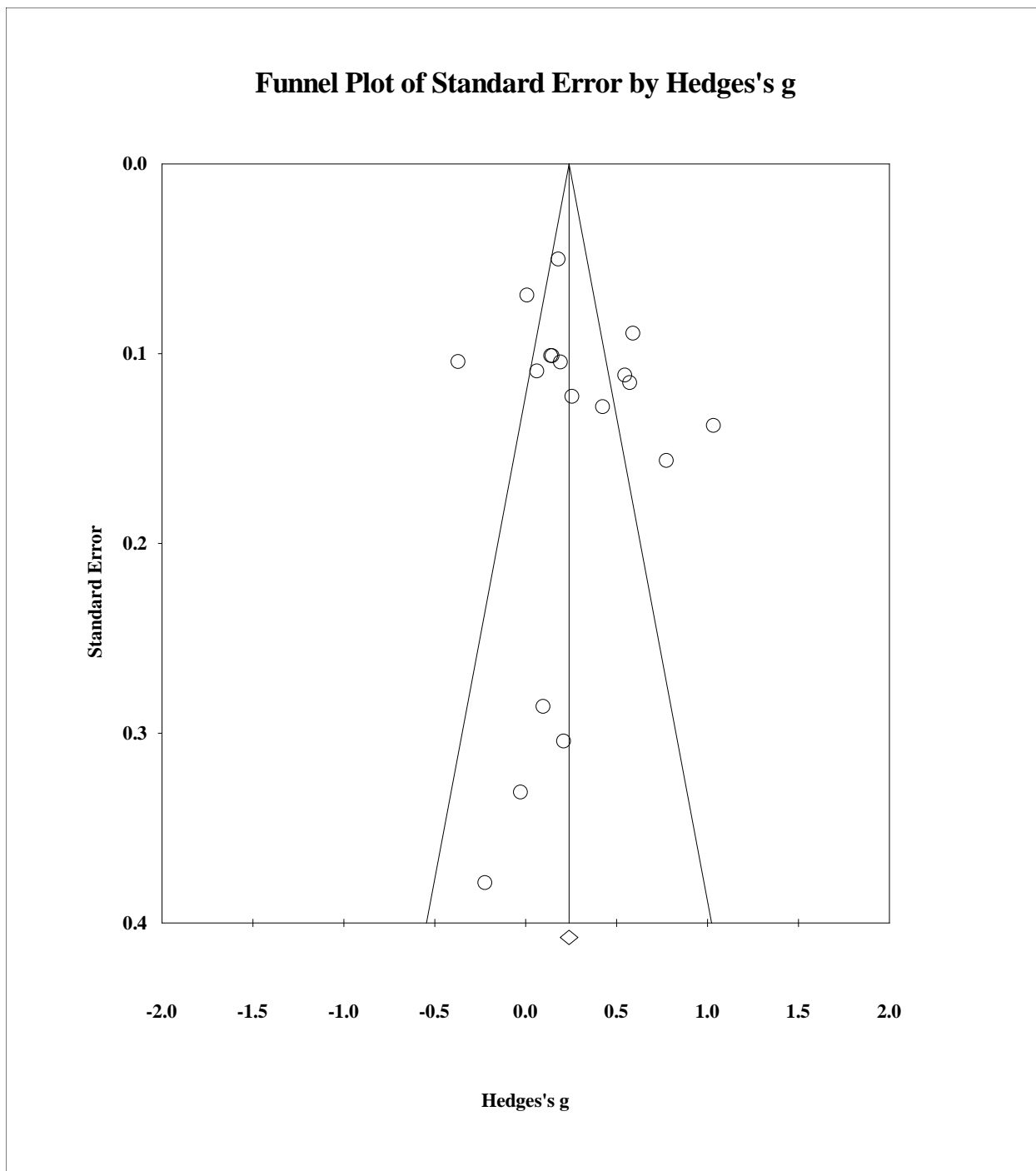


Figure E15. Funnel plot illustrating *Alcohol v. Baseline: Hazard RT* (missing pre-post correlations set to $r = 0.9$).

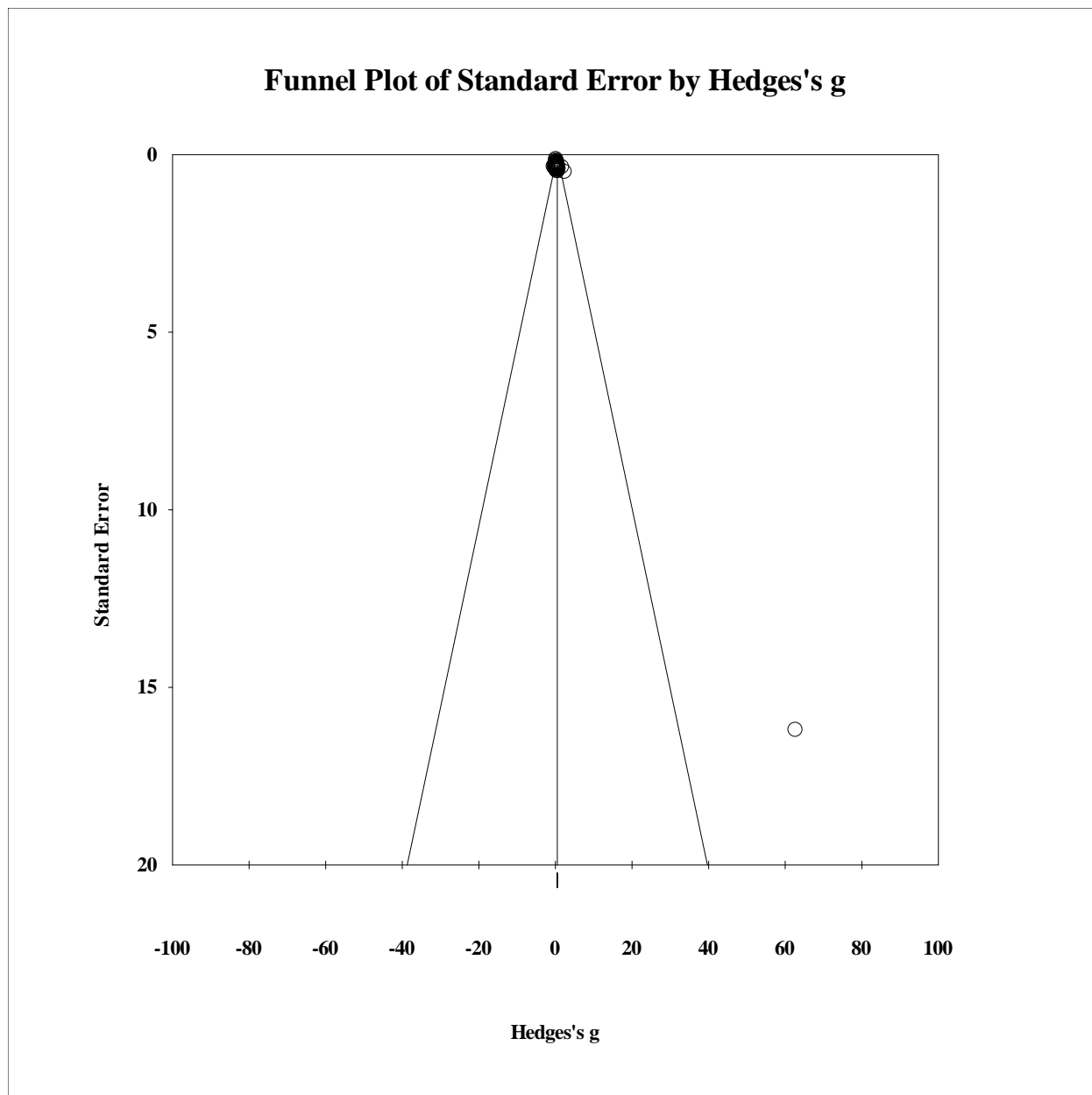


Figure E16. Funnel plot illustrating *Alcohol v. Baseline: Lateral Position Variability* (missing pre-post correlations set to $r = \text{zero}$). Includes Study 1 from Veldstra et al. (2012).

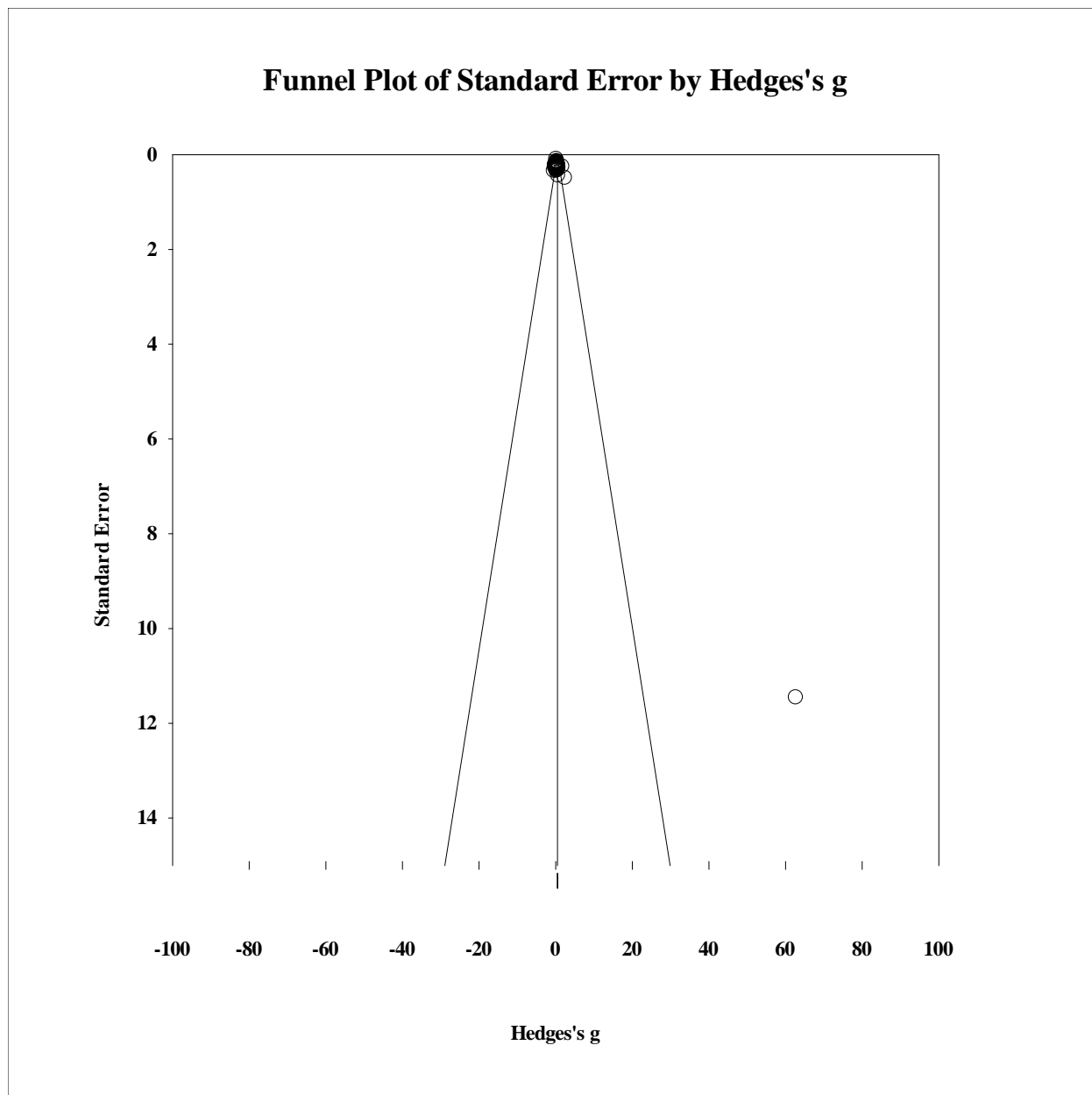


Figure E17. Funnel plot illustrating *Alcohol v. Baseline: Lateral Position Variability* (missing pre-post correlations set to $r = 0.5$). Includes Study 1 from Veldstra et al. (2012).

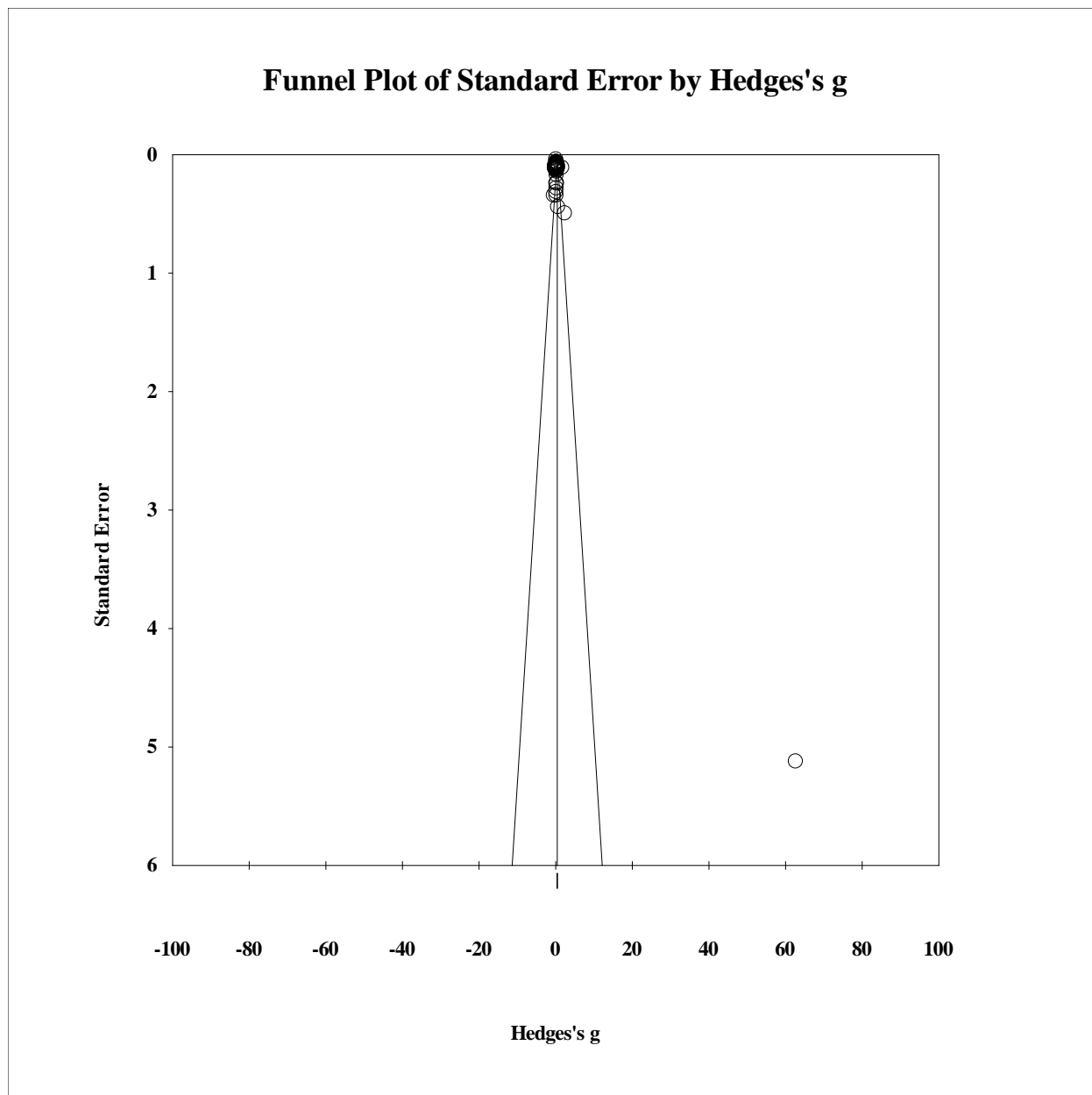


Figure E18. Funnel plot illustrating *Alcohol v. Baseline: Lateral Position Variability* (missing pre-post correlations set to $r = 0.9$). Includes Study 1 from Veldstra et al. (2012).

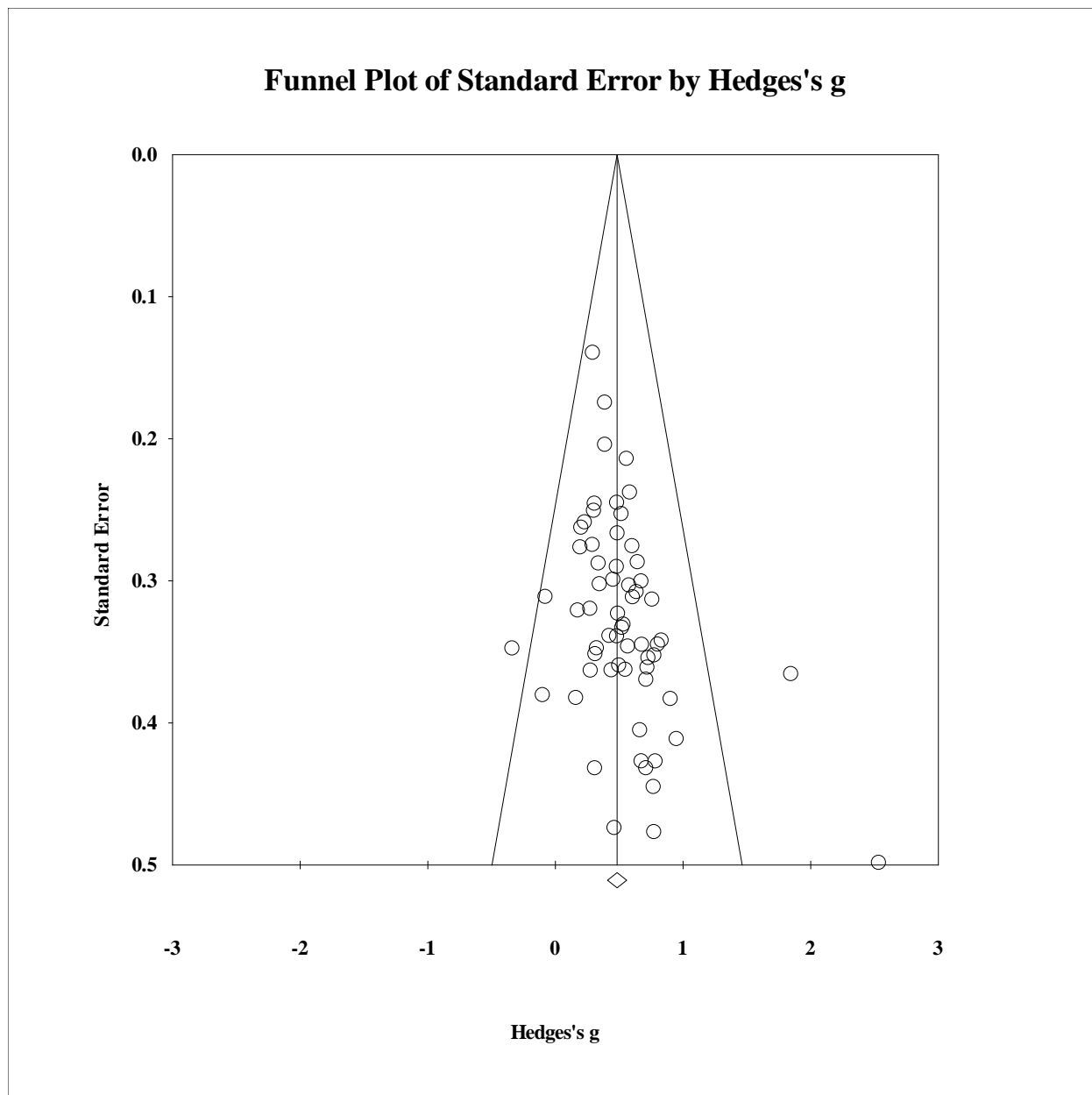


Figure E19. Funnel plot illustrating *Alcohol v. Baseline: Lateral Position Variability* (missing pre-post correlations set to $r = \text{zero}$). Excludes Study 1 from Veldstra et al. (2012).

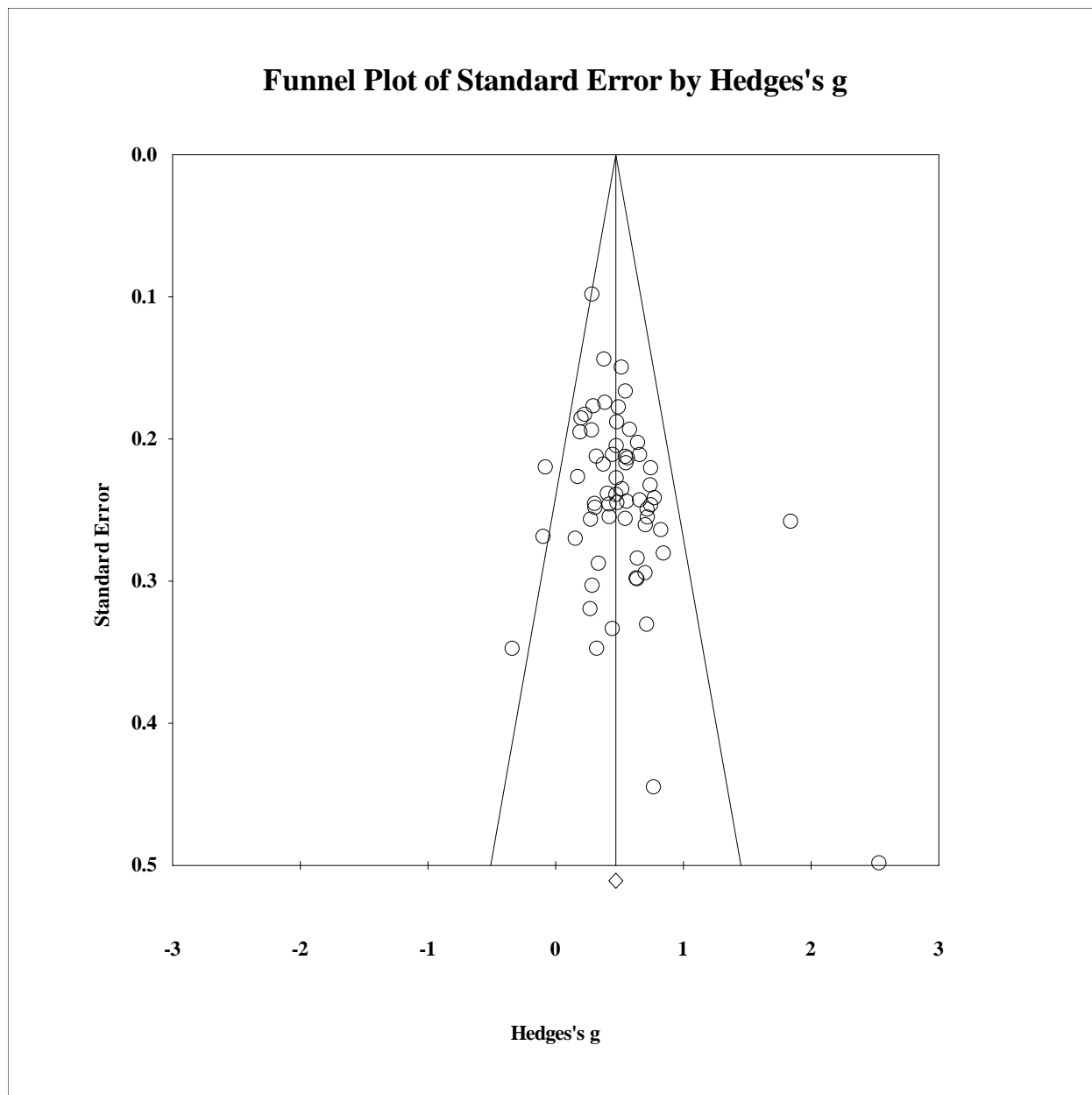


Figure E20. Funnel plot illustrating *Alcohol v. Baseline: Lateral Position Variability* (missing pre-post correlations set to $r = 0.5$). Excludes Study 1 from Veldstra et al. (2012).

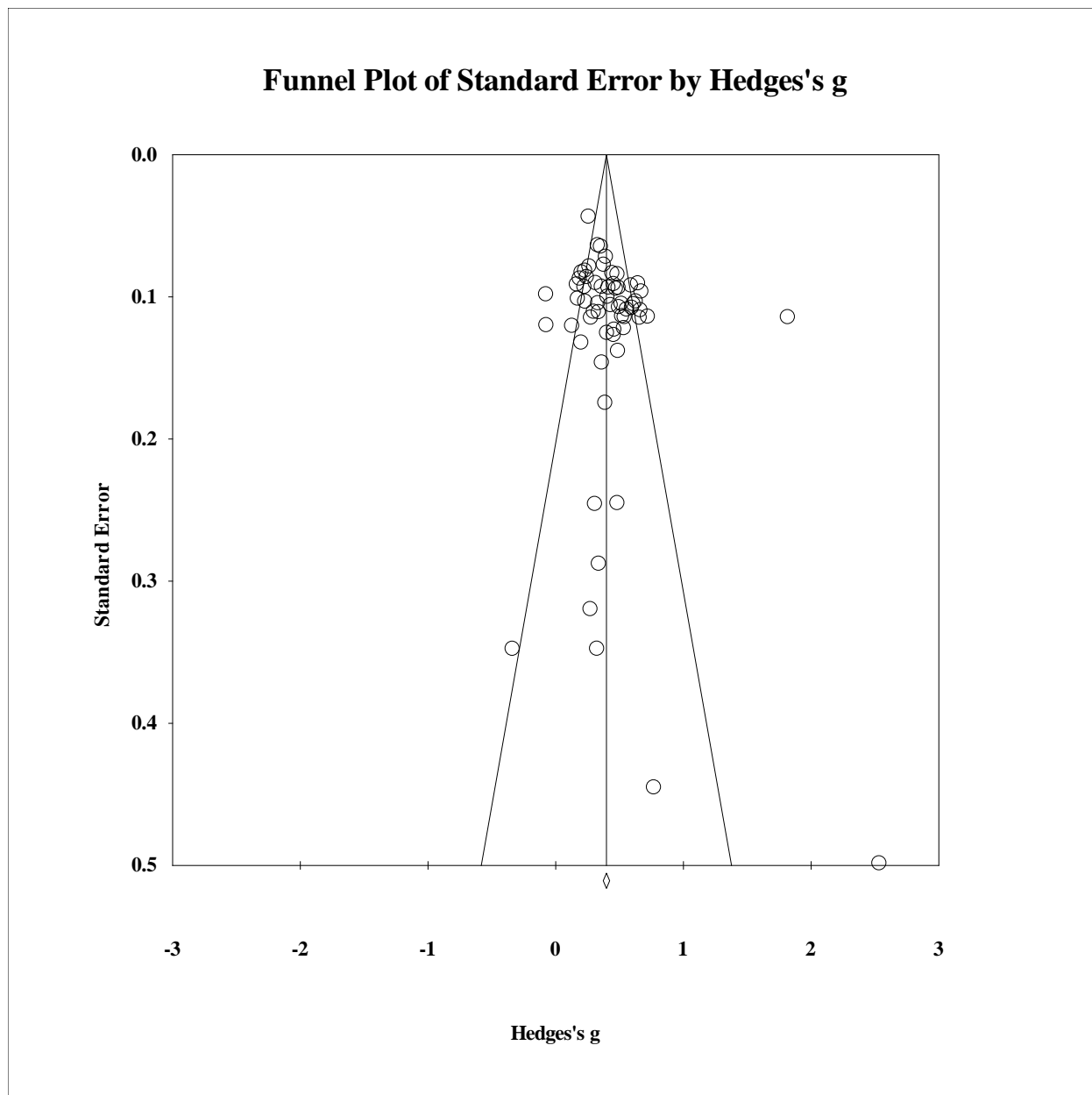


Figure E21. Funnel plot illustrating *Alcohol v. Baseline: Lateral Position Variability* (missing pre-post correlations set to $r = 0.9$). Excludes Study 1 from Veldstra et al. (2012).

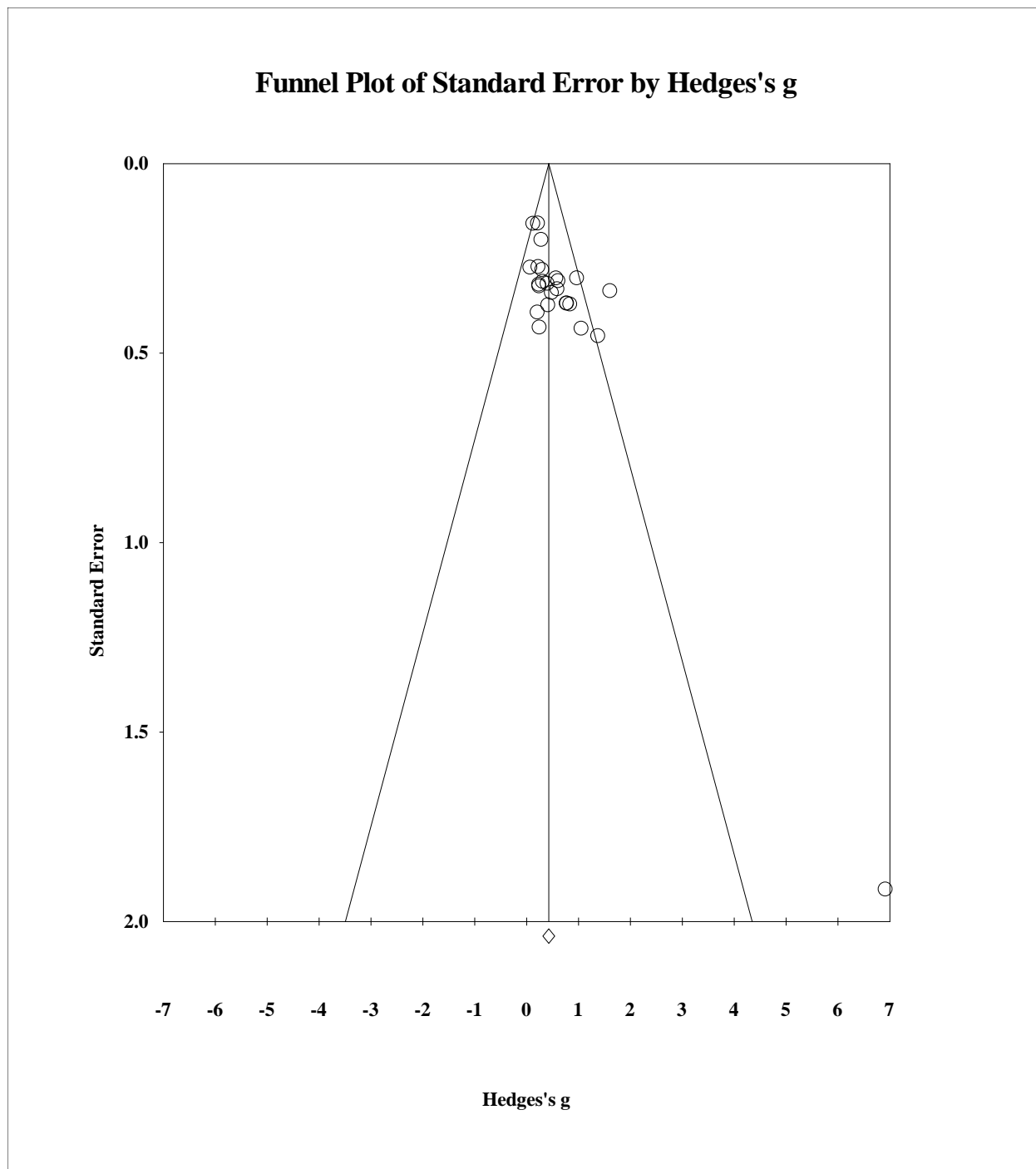


Figure E22. Funnel plot illustrating *Alcohol v. Baseline: Lane Excursions* (missing pre-post correlations set to $r = \text{zero}$). Includes Berthelon and Galy (2014) and Weiler et al. (2000).

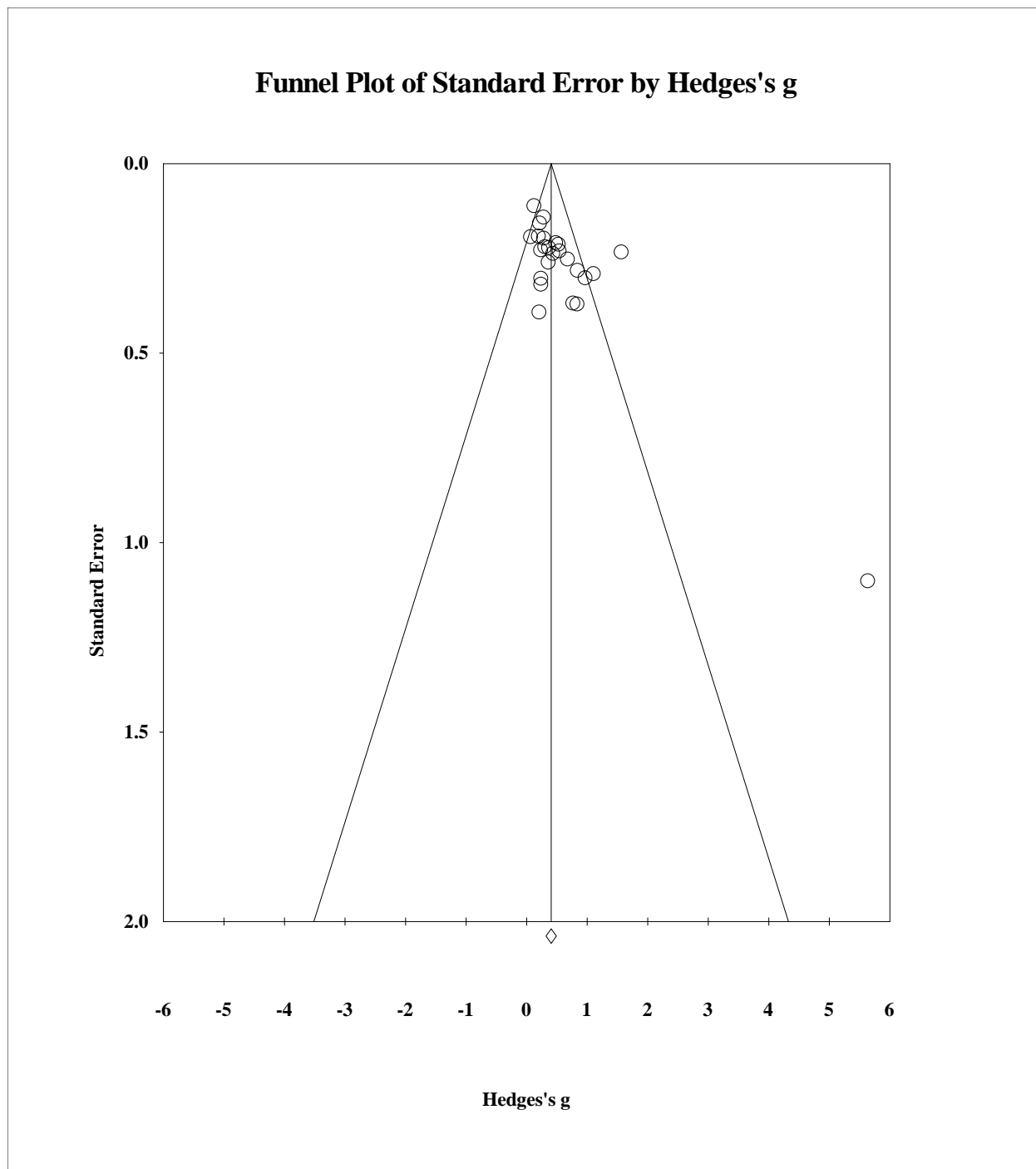


Figure E23. Funnel plot illustrating *Alcohol v. Baseline: Lane Excursions* (missing pre-post correlations set to $r = 0.5$). Includes Berthelon and Galy (2014) and Weiler et al. (2000).

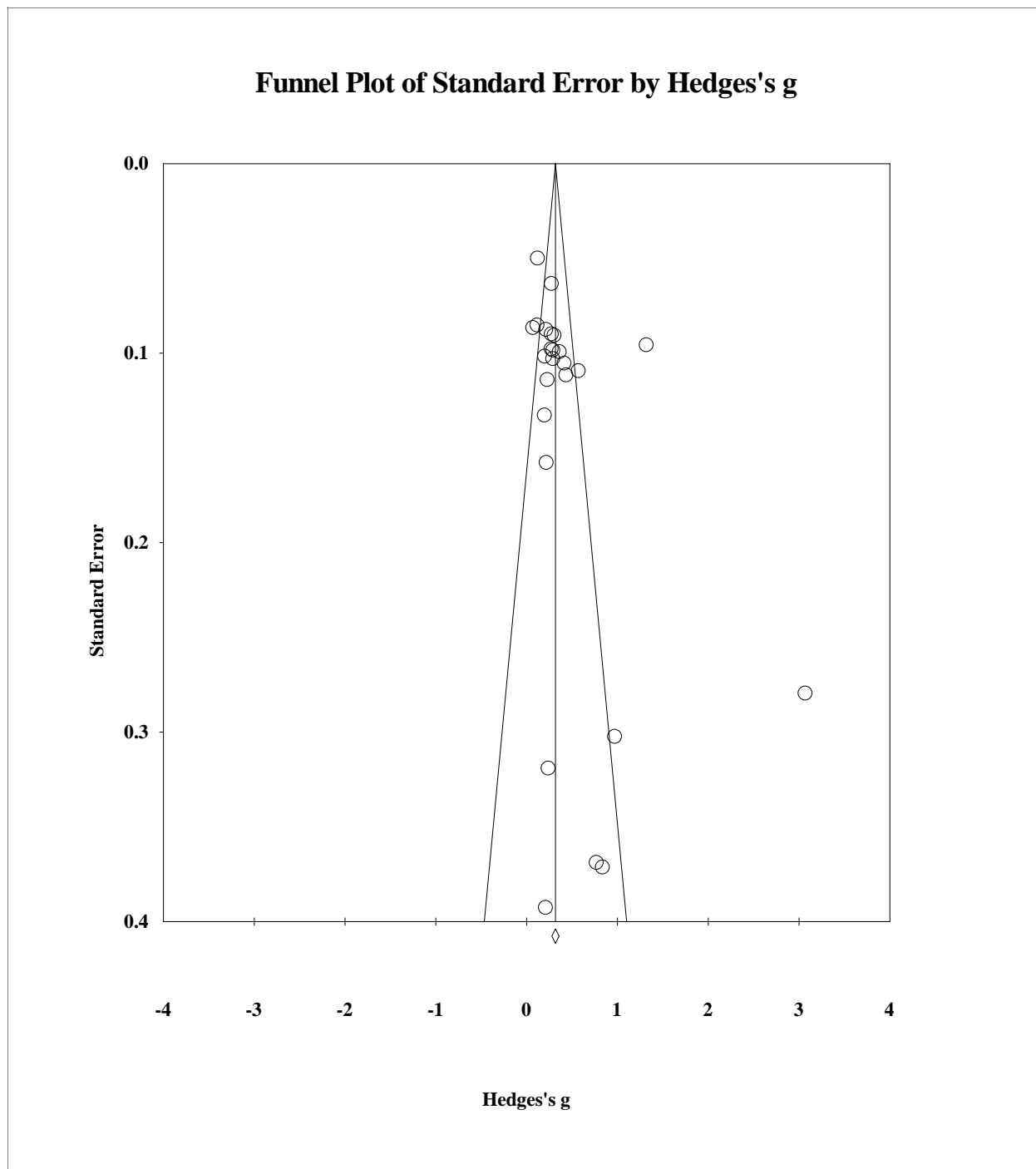


Figure E24. Funnel plot illustrating *Alcohol v. Baseline: Lane Excursions* (missing pre-post correlations set to $r = 0.9$). Includes Berthelon and Galy (2014) and Weiler et al. (2000).

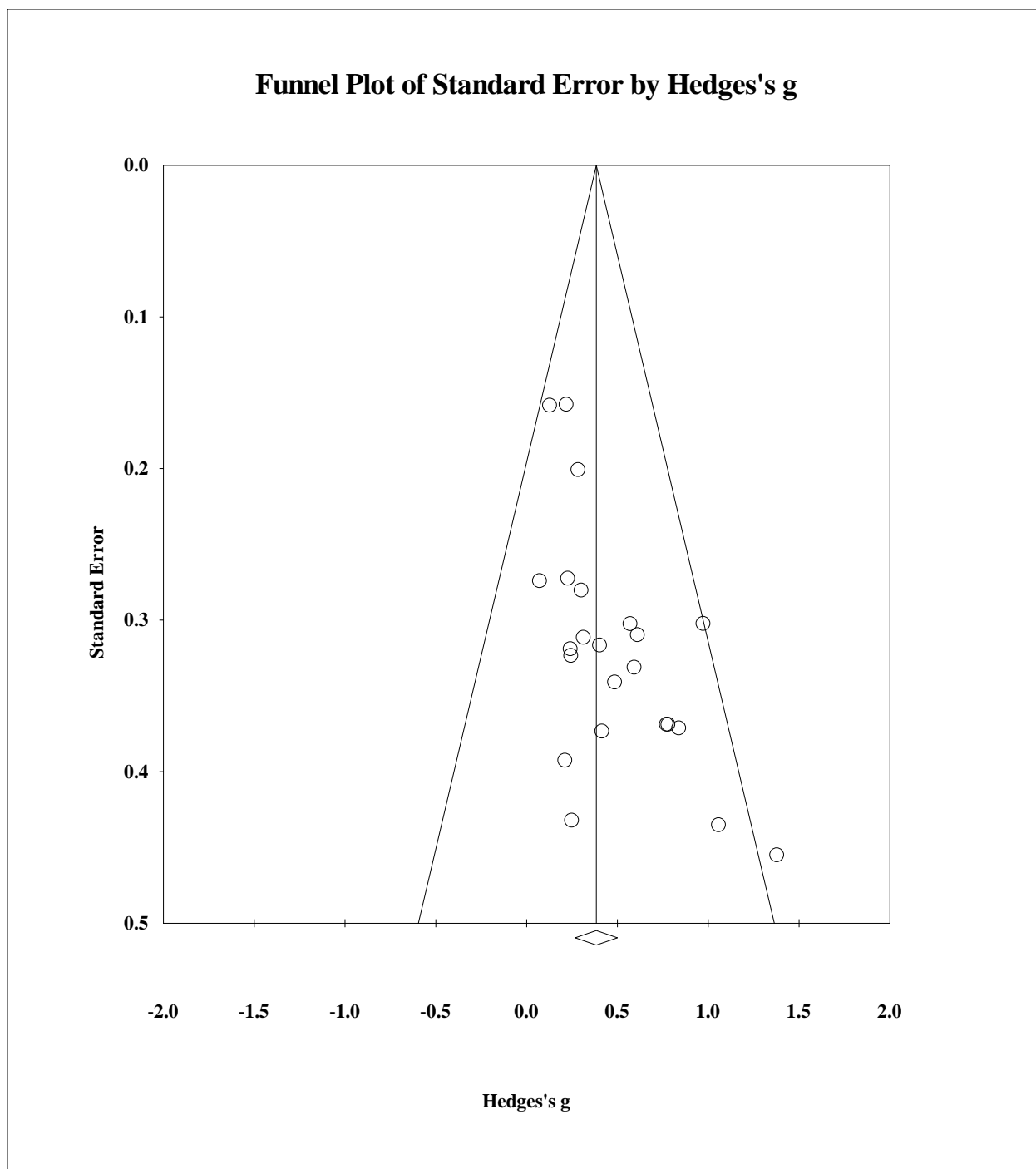


Figure E25. Funnel plot illustrating *Alcohol v. Baseline: Lane Excursions* (missing pre-post correlations set to $r = \text{zero}$). Excludes Berthelon and Galy (2014) and Weiler et al. (2000).

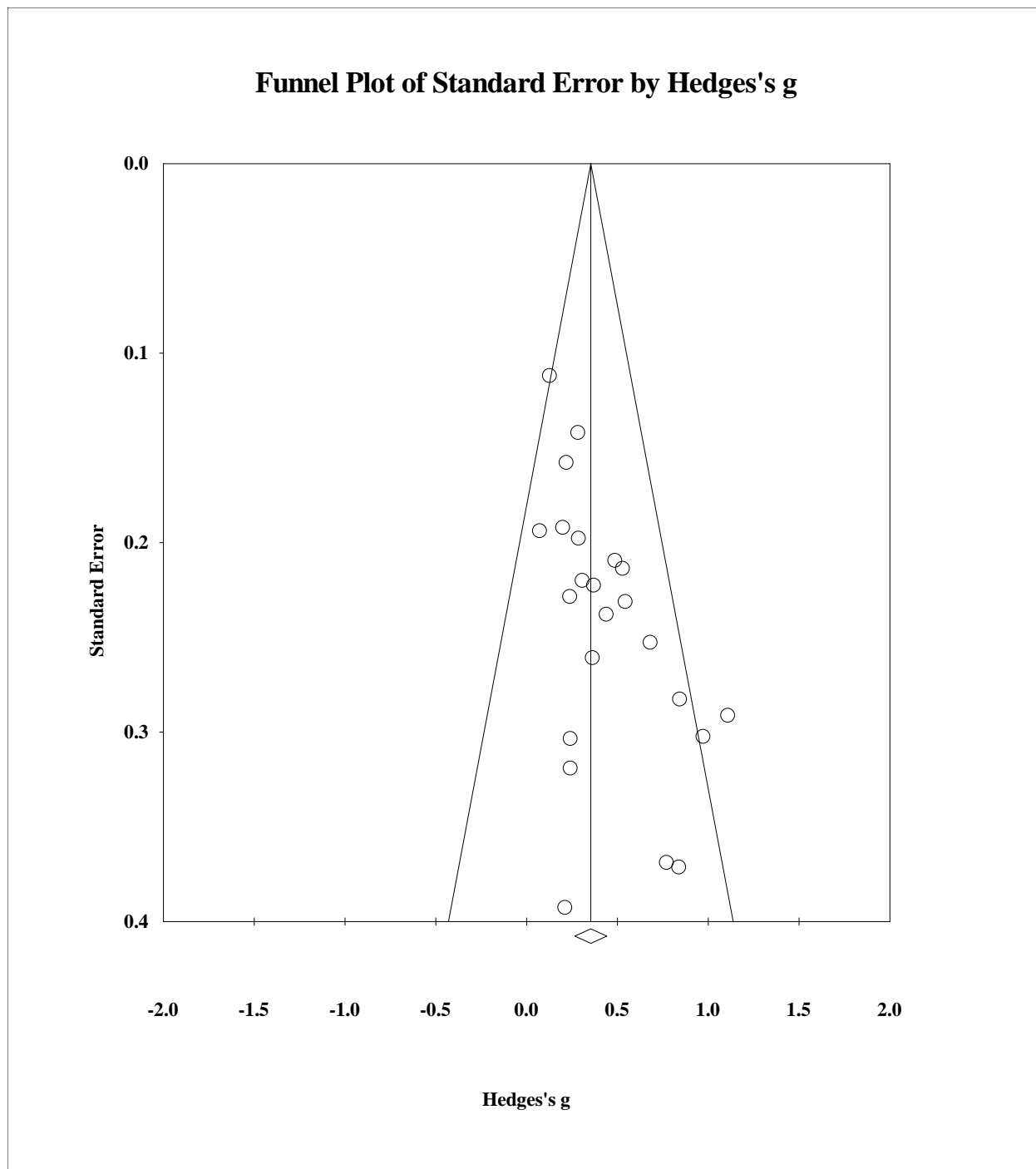


Figure E26. Funnel plot illustrating *Alcohol v. Baseline: Lane Excursions* (missing pre-post correlations set to $r = 0.5$). Excludes Berthelon and Galy (2014) and Weiler et al. (2000).

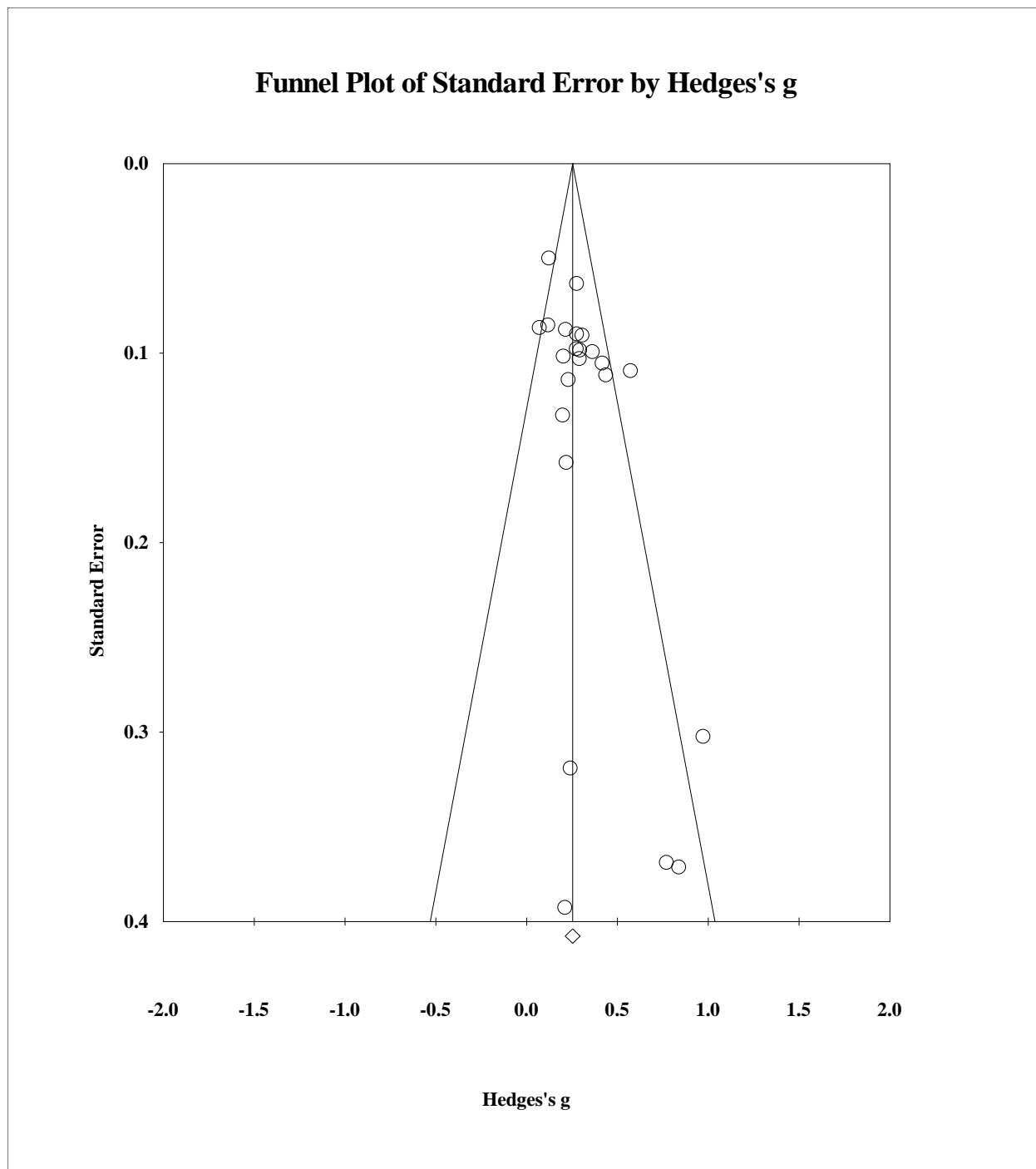


Figure E27. Funnel plot illustrating *Alcohol v. Baseline: Lane Excursions* (missing pre-post correlations set to $r = 0.9$). Excludes Berthelon and Galy (2014) and Weiler et al. (2000).

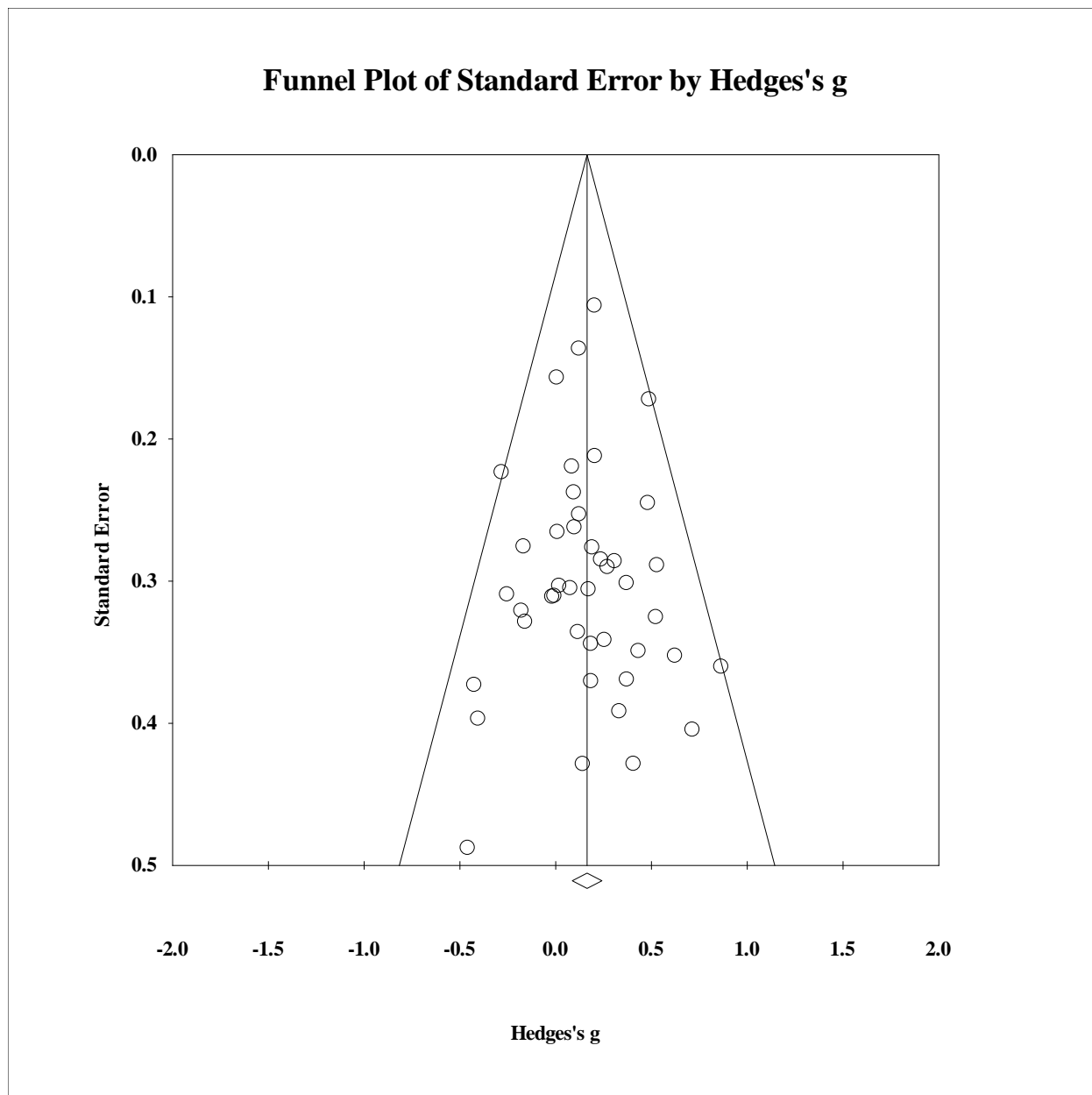


Figure E28. Funnel plot illustrating *Alcohol v. Baseline: Speed* (missing pre-post correlations set to $r = \text{zero}$).

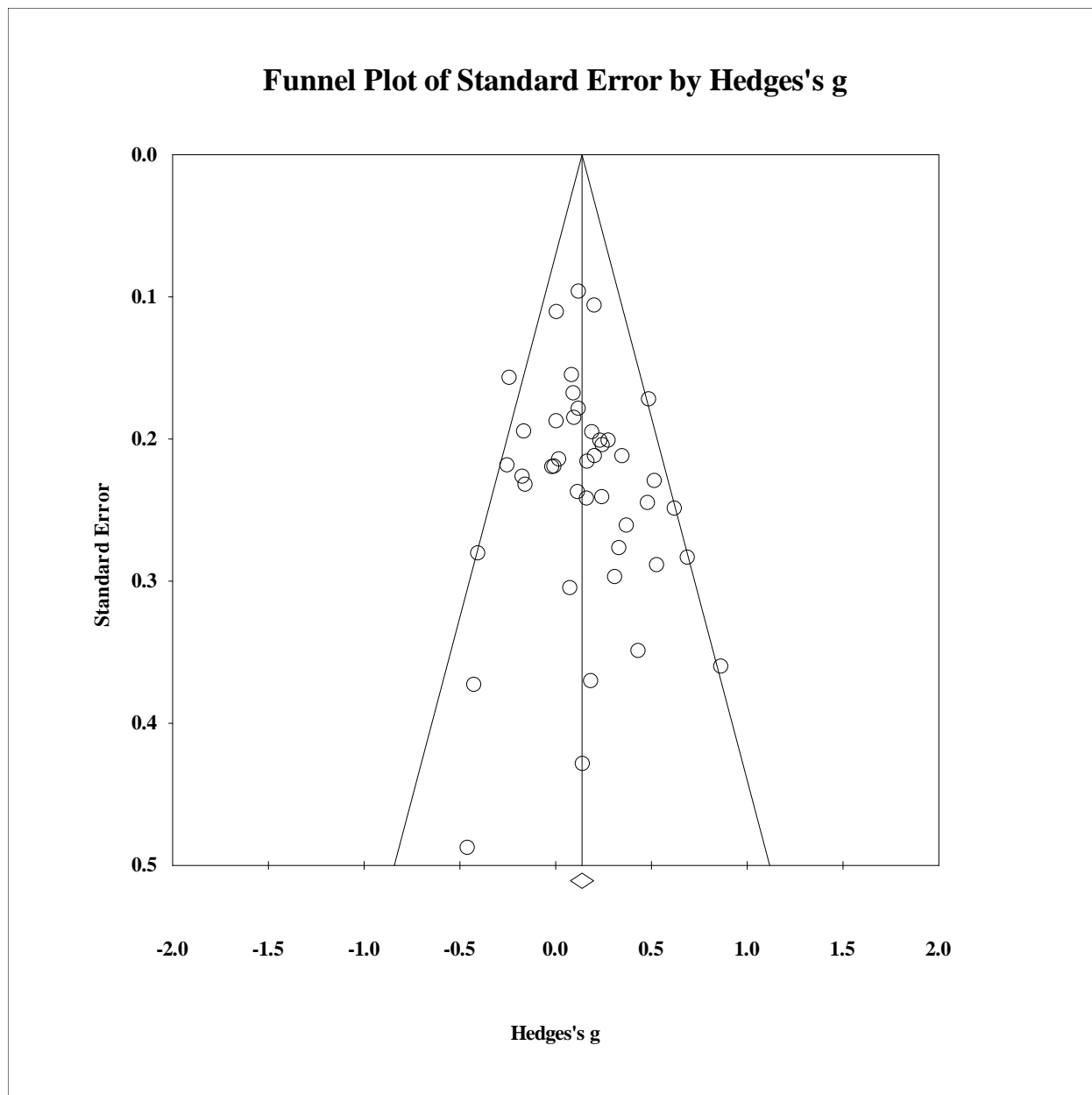


Figure E29. Funnel plot illustrating *Alcohol v. Baseline: Speed* (missing pre-post correlations set to $r = 0.5$).

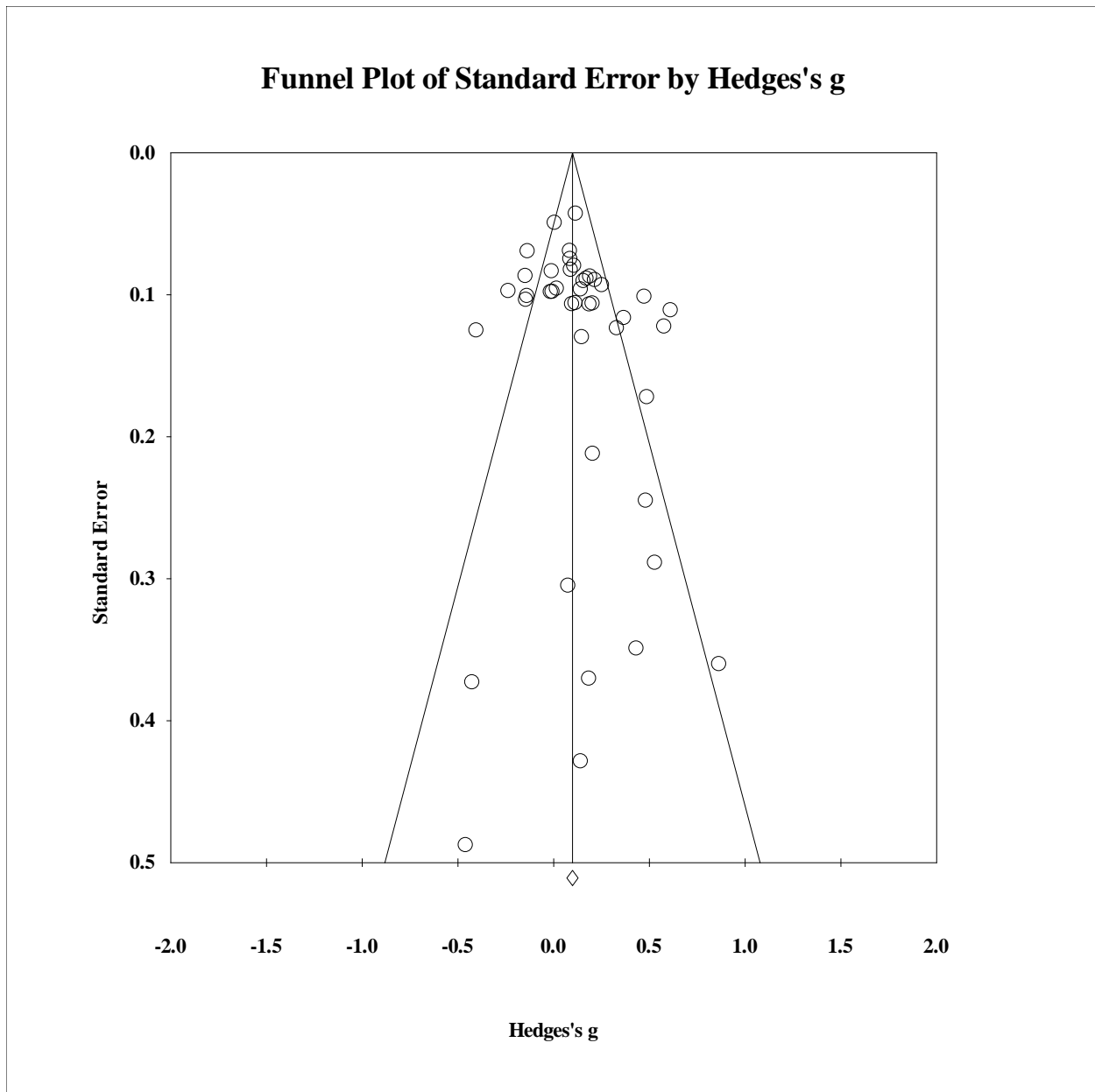


Figure E30. Funnel plot illustrating *Alcohol v. Baseline: Speed* (missing pre-post correlations set to $r = 0.9$).

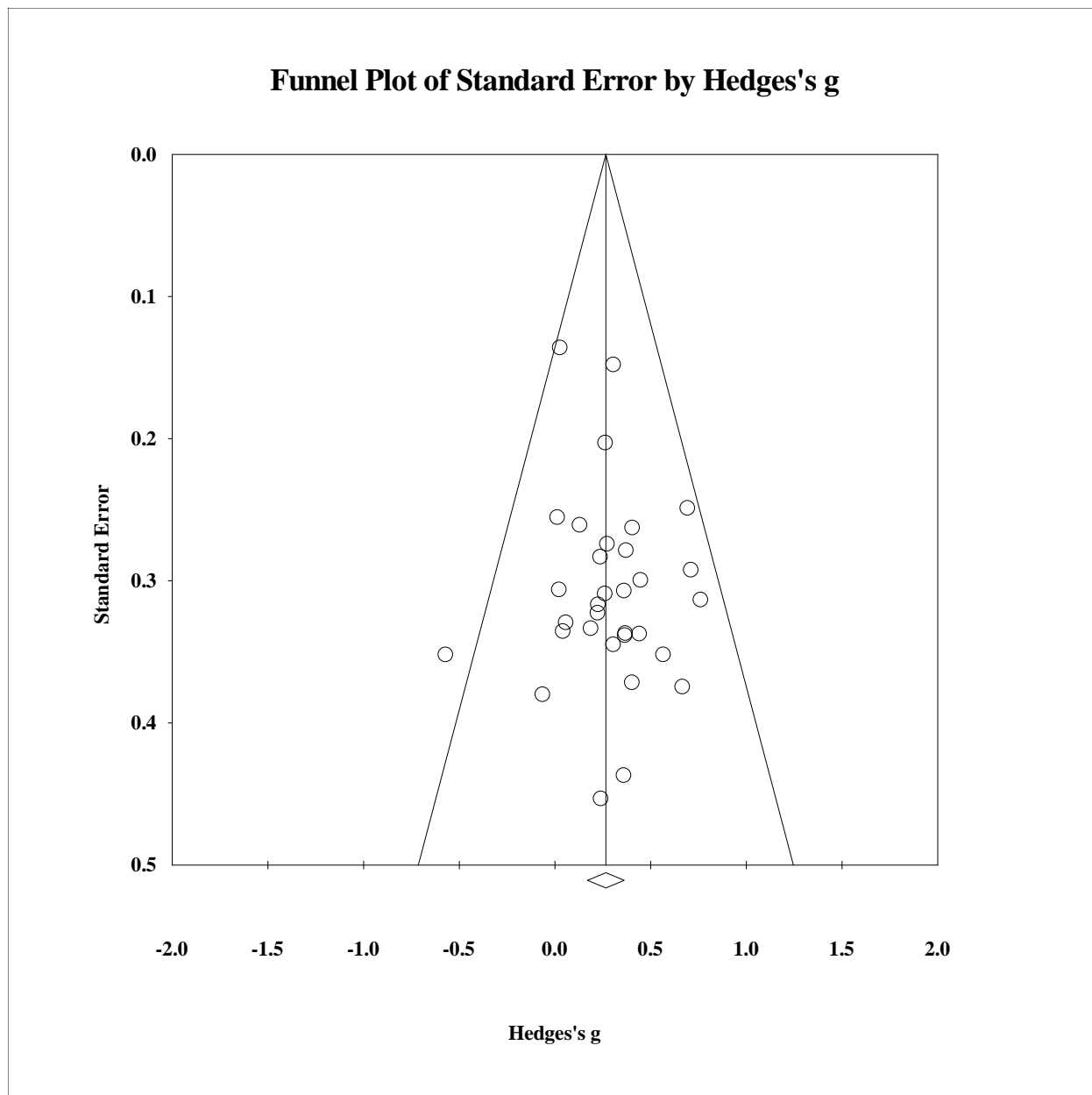


Figure E31. Funnel plot illustrating *Alcohol v. Baseline: Speed Variability* (missing pre-post correlations set to $r = \text{zero}$).

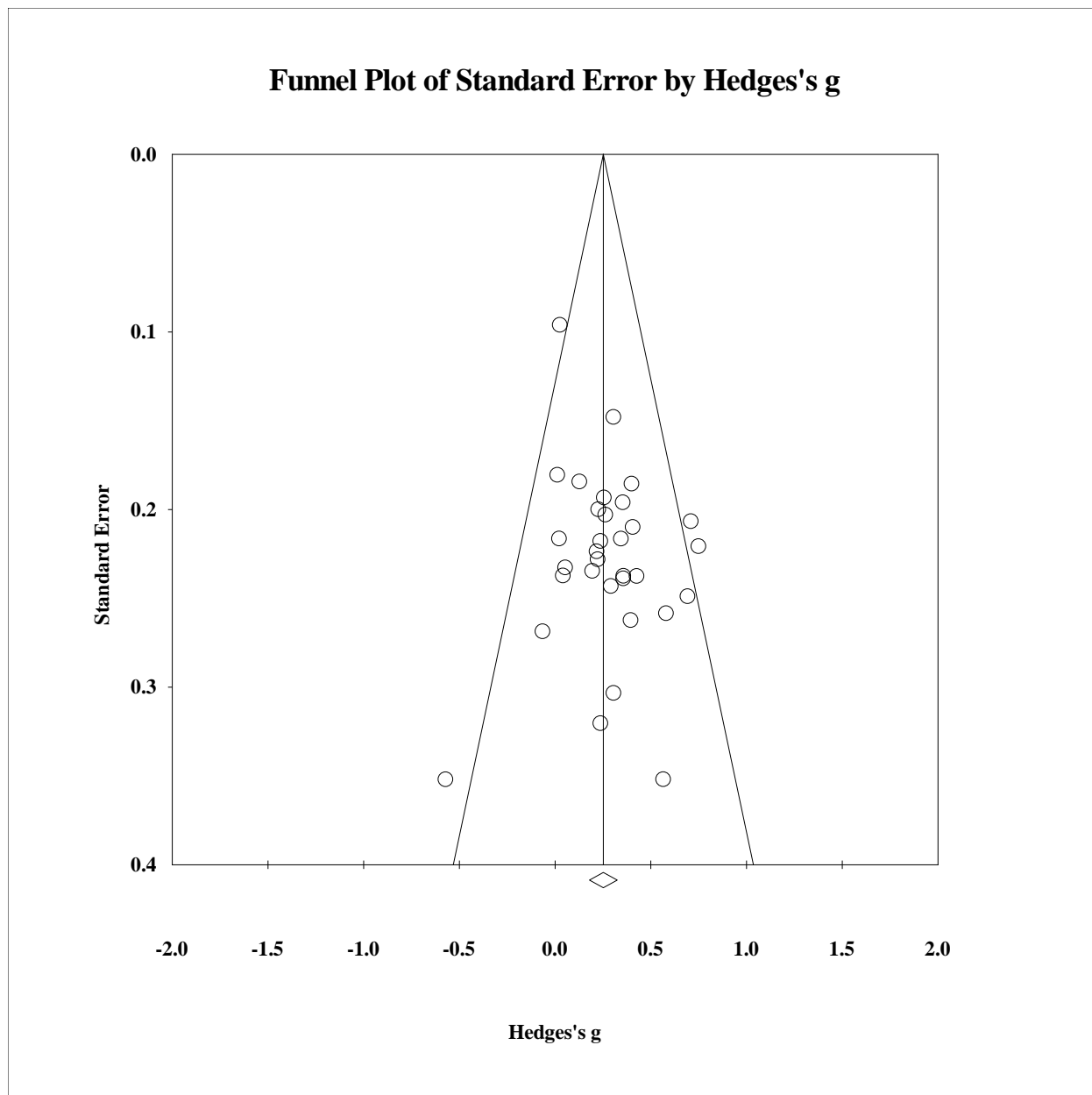


Figure E32. Funnel plot illustrating *Alcohol v. Baseline: Speed* (missing pre-post correlations set to $r = 0.5$).

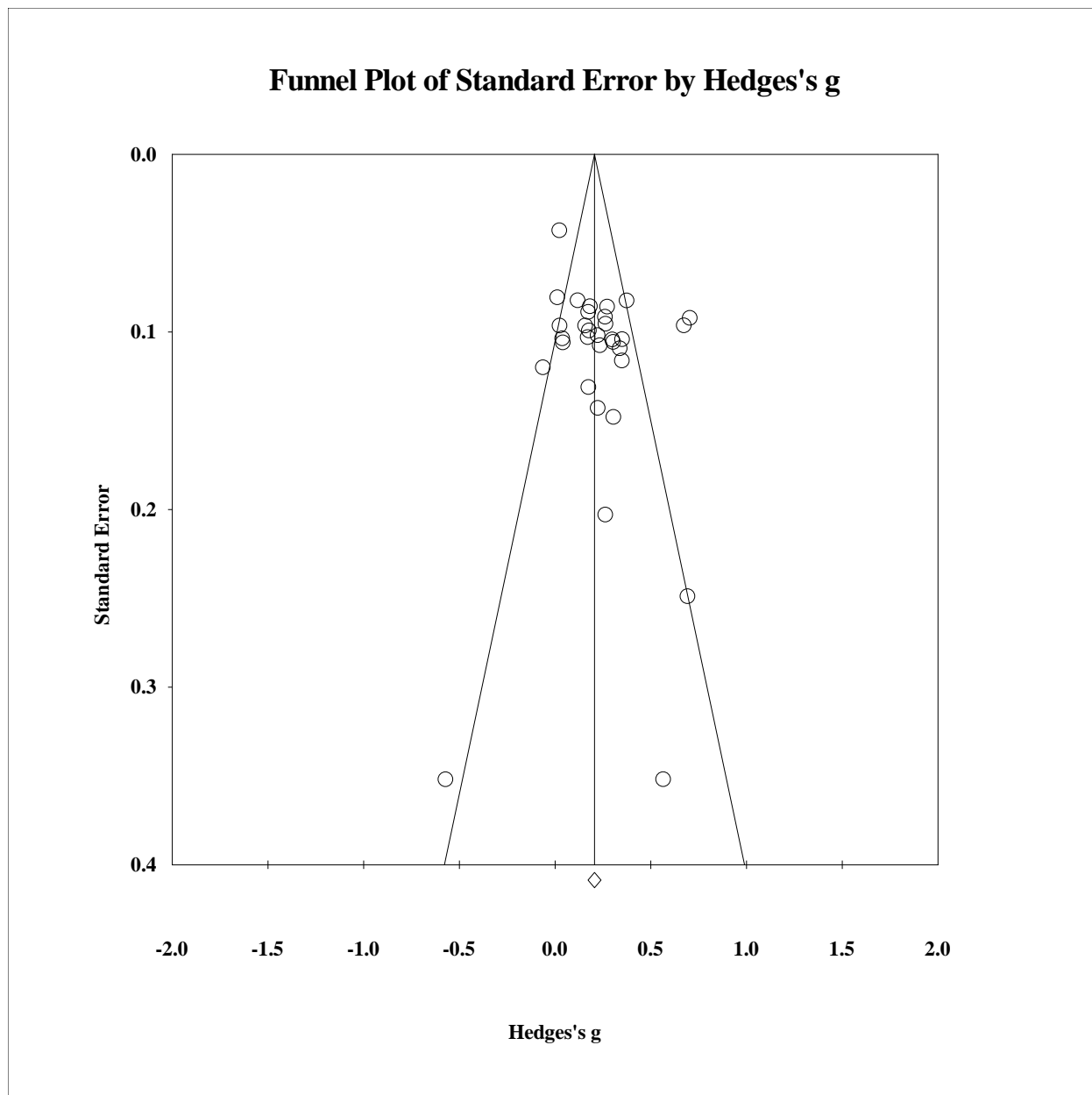


Figure E33. Funnel plot illustrating *Alcohol v. Baseline: Speed* (missing pre-post correlations set to $r = 0.9$).

Appendix F: Study Quality and Risk of Bias Assessment

Table F1. Study quality and risk of bias judgements.

Study	Representative Sample?	Participation Agreement Rate?	Drug Conditions Described as Randomized?	If Repeated Measures, Counterbalancing or Randomization?	Important Differences Between Groups Before Drive?	Driving Performance Assessors Aware of Drug Condition?	Participants Aware of Research Question?	Reliable Driving Data Collection?	Numbers and Reasons for Withdrawals and Drop-Outs Reported?	Percentage of Sample Completing Study?	Consistent Treatments?	Possible Treatment Contamination?	Risk of Reporting Bias?	Sample Size Based on Power Calculation?
Anderson et al., 2010	Not Likely	N/A	Yes	N/A	No	No	Can't Tell	Yes	Yes	73/85	Yes	No	Low	No
Arkell et al., 2019	Not Likely	N/A	Yes	Yes	N/A	No	N/A (Expl.)	Yes	Yes	14/17	Yes	No	Low	Yes
Arndt et al., 2001	Not Likely	N/A	Can't Tell	Yes	N/A	Yes	Can't Tell	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Beard, 2012	Not Likely	N/A	Yes ¹	N/A	Can't Tell	Can't Tell	N/A (Expl.)	Yes	Can't Tell ²	Can't Tell ²	Yes	No	Low	No
Bernosky-Smith et al., 2011	Not Likely	N/A	Yes	N/A	Possibly	No	Can't Tell	Yes	No	59/60	Yes	No	Unclear	No
Bernosky-Smith et al., 2012	Not Likely	N/A	No (Fixed)	N/A (Fixed)	N/A	Can't Tell	Can't Tell	Yes	N/A	100%	Yes	No	Low	No
Berthelon & Galy, 2018	Can't Tell	Can't Tell	Yes	Yes	N/A	Can't Tell	Can't Tell	Yes	Can't Tell	Can't Tell	Can't Tell	No	High	No
Berthelon & Gineyt, 2014	Can't Tell	Can't Tell	Can't Tell	Yes	N/A	Yes	Can't Tell	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Bosker et al., 2012	Can't Tell	Can't Tell	Yes	Yes	N/A	No	N/A (Expl.)	Yes	Can't Tell	Can't Tell	Yes	No	Low	Yes
Brands et al., 2019	Not Likely	N/A	Yes	N/A	Possibly	No	N/A (Expl.)	Yes	Yes	91/96	Yes	No	Low	Yes
Burns et al., 2002	Likely	Can't Tell	Can't Tell	Can't Tell	N/A	Can't Tell	No	Yes	Can't Tell	Can't Tell	Yes	No	Low	No

Study	Representative Sample?	Participation Agreement Rate?	Drug Conditions Described as Randomized?	If Repeated Measures, Counterbalancing or Randomization?	Important Differences Between Groups Before Drive?	Driving Performance Assessors Aware of Drug Condition?	Participants Aware of Research Question?	Reliable Driving Data Collection?	Numbers and Reasons for Withdrawals and Drop-Outs Reported?	Percentage of Sample Completing Study?	Consistent Treatments?	Possible Treatment Contamination?	Risk of Reporting Bias?	Sample Size Based on Power Calculation?
Charlton & Starkey, 2015	Not Likely	N/A	Yes	N/A	Can't Tell	Can't Tell	N/A (Expl.)	Yes	Yes	44/71	Yes	No	Unclear	No
Chen et al., 2016	Not Likely	Can't Tell	Can't Tell	N/A	Can't Tell	Can't Tell	N/A (Expl.)	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Christoforou et al., 2012	Can't Tell	Can't Tell	No (Fixed)	N/A (Fixed)	N/A	Can't Tell	Can't Tell	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Downey et al., 2013	Can't Tell	Can't Tell	Can't Tell	Yes	N/A	No	N/A (Expl.)	Yes	Can't Tell	Can't Tell	Yes	No	Unclear	No
Fillmore et al., 2008	Not Likely	N/A	Can't Tell	Yes	N/A	Can't Tell	No	Yes	Can't Tell	Can't Tell	Yes	No	Unclear	No
Freydier et al., 2014	Can't Tell	Can't Tell	Can't Tell	Yes	N/A	Yes	Can't Tell	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Harrison & Fillmore, 2005	Not Likely	N/A	No (Fixed)	N/A (Fixed)	N/A	Can't Tell	No	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Harrison & Fillmore, 2011	Not Likely	N/A	Yes	N/A	No	Can't Tell	No	Yes	Can't Tell	Can't Tell	Yes	No	Unclear	No
Harrison et al., 2007	Not Likely	N/A	No (Fixed)	N/A (Fixed)	N/A	Can't Tell	No	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Hartman et al., 2015	Can't Tell	Can't Tell	Yes	Yes	N/A	No	Can't Tell	Yes	Yes	18/19	Yes	No	Low	No
Helland et al., 2016	Can't Tell	Can't Tell	Yes	Yes	N/A	Yes	N/A (Expl.)	Yes	Yes	18/20	Yes	No	Low	Yes
Horne & Baumber, 1991	Not Likely	N/A	Can't Tell	Yes	N/A	No	Can't Tell	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Howard et al., 2007	Can't Tell	Can't Tell	Yes	Yes ³	N/A	Can't Tell	Can't Tell	Yes	Yes	16/19	Yes	No	Unclear	No

Study	Representative Sample?	Participation Agreement Rate?	Drug Conditions Described as Randomized?	If Repeated Measures, Counterbalancing or Randomization?	Important Differences Between Groups Before Drive?	Driving Performance Assessors Aware of Drug Condition?	Participants Aware of Research Question?	Reliable Driving Data Collection?	Numbers and Reasons for Withdrawals and Drop-Outs Reported?	Percentage of Sample Completing Study?	Consistent Treatments?	Possible Treatment Contamination?	Risk of Reporting Bias?	Sample Size Based on Power Calculation?
Howland et al., 2010	Not Likely	N/A	Yes	N/A	No	No	N/A (Expl.)	Yes	Yes	121/154	Yes	No	Low	Yes
Huemer & Vollrath, 2010	Not Likely	N/A	No (Fixed)	N/A (Fixed)	N/A	Can't Tell	N/A (Expl.)	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Jelen et al., 2011	Can't Tell	Can't Tell	No (Fixed)	N/A (Fixed)	N/A	Can't Tell	N/A (Expl.)	Yes	Can't Tell	Can't Tell	Yes	No	High	No
Kay et al., 2013	Can't Tell	Can't Tell	Yes	Yes	N/A	Can't Tell	Can't Tell	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Kenntner-Mabiala et al., 2015	Can't Tell	Can't Tell	Yes	Yes	N/A	No	No	Yes	N/A	100%	Yes	No	Low	Yes
Kuypers et al., 2006	Not Likely	N/A	Yes	Yes	N/A	No	N/A (Expl.)	Yes	N/A ⁴	100% ⁴	Yes	No	Low	No
Laude & Fillmore, 2015	Not Likely	N/A	Can't Tell	Yes	N/A	Can't Tell	Can't Tell	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Laude & Fillmore, 2016	Not Likely	N/A	Can't Tell	Yes	N/A	Can't Tell	Can't Tell	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Laude, 2016 (Study 3)	Not Likely	N/A	Can't Tell	Yes	N/A	Can't Tell	Can't Tell	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Lee et al., 2010	Not Likely	N/A	Can't Tell	Yes	N/A	Can't Tell	N/A (Expl.)	Yes	No	108/130	Yes	No	Low	No
Lenne et al., 1999	Can't Tell	Can't Tell	Can't Tell	Yes ⁵	N/A	Can't Tell	N/A (Expl.)	Yes	Can't Tell	Can't Tell	Yes	No	High	No
Lenne et al., 2003	Can't Tell	Can't Tell	Can't Tell	Yes	N/A	Can't Tell	Can't Tell	Yes	Can't Tell	Can't Tell	Yes	No	Unclear	No
Leung et al., 2012	Not Likely	N/A	No (Fixed)	N/A (Fixed)	N/A	Can't Tell	N/A (Expl.)	Yes	Can't Tell	Can't Tell	Yes	No	High	No

Study	Representative Sample?	Participation Agreement Rate?	Drug Conditions Described as Randomized?	If Repeated Measures, Counterbalancing or Randomization?	Important Differences Between Groups Before Drive?	Driving Performance Assessors Aware of Drug Condition?	Participants Aware of Research Question?	Reliable Driving Data Collection?	Numbers and Reasons for Withdrawals and Drop-Outs Reported?	Percentage of Sample Completing Study?	Consistent Treatments?	Possible Treatment Contamination?	Risk of Reporting Bias?	Sample Size Based on Power Calculation?
Liguori & Robinson, 2001	Not Likely	N/A	Yes	Yes	N/A	No	N/A (Expl.)	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Liguori et al., 1998	Not Likely	N/A	Can't Tell	Can't Tell	N/A	Can't Tell	N/A (Expl.)	Yes	Yes	10/24	Yes	No	Unclear	No
Liguori et al., 1999	Not Likely	N/A	Yes	Yes	N/A	No	Can't Tell	Yes	Can't Tell	Can't Tell	Yes	No	Unclear	No
Liguori et al., 2002	Not Likely	N/A	Yes	Yes	N/A	No	Can't Tell	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Louwerens et al., 1987	Can't Tell	Can't Tell	Can't Tell	Can't Tell	N/A	Yes	N/A (Expl.)	Yes	N/A	100%	Yes	No	Low	No
Marczinski & Fillmore, 2009	Not Likely	N/A	Yes	Yes	N/A	No	No	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Marczinski et al., 2008	Not Likely	N/A	Yes	Yes	N/A	No	No	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
McCartney et al., 2017	Can't Tell	Can't Tell	Yes	Yes	N/A	Can't Tell	Can't Tell	Yes	Yes	22/25	Yes	No	Low	Yes
Mets et al., 2011	Not Likely	N/A	Yes	Yes	N/A	Yes	Can't Tell	Yes	Yes	27/36	Yes	No	Low	No
Price et al., 2018	Not Likely	N/A	Yes	N/A	No	No	Can't Tell	Yes	Can't Tell	Can't Tell	Yes	No	High	No
Ramaekers et al., 1992	Can't Tell	Can't Tell	Yes	Yes	N/A	No	N/A (Expl.)	Yes	Can't Tell	Can't Tell	Yes	No	Unclear	No
Ramaekers et al., 2000	Not Likely	N/A	Can't Tell	Yes	N/A	No	N/A (Expl.)	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Ramaekers et al., 2000 (Study 1)	Can't Tell	Can't Tell	Can't Tell	Yes	N/A	No	N/A (Expl.)	Yes	Can't Tell	Can't Tell	Yes	No	Low	No

Study	Representative Sample?	Participation Agreement Rate?	Drug Conditions Described as Randomized?	If Repeated Measures, Counterbalancing or Randomization?	Important Differences Between Groups Before Drive?	Driving Performance Assessors Aware of Drug Condition?	Participants Aware of Research Question?	Reliable Driving Data Collection?	Numbers and Reasons for Withdrawals and Drop-Outs Reported?	Percentage of Sample Completing Study?	Consistent Treatments?	Possible Treatment Contamination?	Risk of Reporting Bias?	Sample Size Based on Power Calculation?
Robbe, 1998 (Study 1)	Can't Tell	Can't Tell	Can't Tell	Yes	N/A	No	N/A (Expl.)	Can't Tell	Yes	23/24	Yes	Yes	Low	No
Robbe, 1998 (Study 2)	Can't Tell	Can't Tell	No (Fixed)	N/A (Fixed)	N/A	No	N/A (Expl.)	Can't Tell	Yes	15/16	Yes	Yes	High	No
Roberts, 2016 (Study 2)	Not Likely	N/A	Yes	Yes	N/A	Can't Tell	Can't Tell	Yes	Can't Tell ⁶	Can't Tell ⁶	Yes	No	Unclear	No
Ronen et al., 2008	Can't Tell	Can't Tell	Can't Tell	Yes	N/A	No	N/A (Expl.)	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Ronen et al., 2010	Can't Tell	Can't Tell	Can't Tell	Yes	N/A	Can't Tell	N/A (Expl.)	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Rupp et al., 2007	Can't Tell	Can't Tell	Yes	Yes	N/A	Can't Tell	Can't Tell	Yes	Yes	26/29	Yes	No	Low	No
Schumacher et al., 2017	Can't Tell	Can't Tell	No (Fixed)	N/A (Fixed)	N/A	Can't Tell ⁷	Can't Tell	Yes	Yes	17/19	Yes	No	Low	Yes
Sexton, 1997	Can't Tell	Can't Tell	Yes	Yes	N/A	Can't Tell	Can't Tell	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Sexton et al., 2000	Not Likely	Can't Tell	Yes	Yes	N/A	No	N/A (Expl.)	Yes	Yes	Can't Tell ⁸	Yes ⁸	No	Low	Yes
Sexton et al., 2002	Not Likely	Can't Tell	Yes	Yes	N/A	No	N/A (Expl.)	Yes	Yes*	Can't Tell ⁸	Yes ⁸	No	Unclear	Yes
Simons et al., 2012	Can't Tell	Can't Tell	Yes	Yes	N/A	No	N/A (Expl.)	Yes	Yes	13/18 ⁹	Yes	No	High	No
Sklar et al., 2014	Can't Tell	Can't Tell	Yes	N/A	No	Can't Tell	Can't Tell	Yes	No ⁹	Can't Tell ⁹	Yes	No	Low	No
Starkey & Charlton, 2014	Not Likely	N/A	Yes	N/A	Can't Tell	Can't Tell	Can't Tell	Yes	Yes	11/12	Yes	No	High	No

Study	Representative Sample?	Participation Agreement Rate?	Drug Conditions Described as Randomized?	If Repeated Measures, Counterbalancing or Randomization?	Important Differences Between Groups Before Drive?	Driving Performance Assessors Aware of Drug Condition?	Participants Aware of Research Question?	Reliable Driving Data Collection?	Numbers and Reasons for Withdrawals and Drop-Outs Reported?	Percentage of Sample Completing Study?	Consistent Treatments?	Possible Treatment Contamination?	Risk of Reporting Bias?	Sample Size Based on Power Calculation?
Strayer et al., 2006	Not Likely	N/A	Can't Tell	Yes	N/A	Can't Tell	N/A (Expl.)	Yes	Can't Tell	Can't Tell	Yes	No	Unclear	No
Subramaniam et al., 2018	Can't Tell	Can't Tell	Can't Tell	Can't Tell	N/A	Can't Tell	Can't Tell	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Tremblay et al., 2015	Can't Tell	Can't Tell	Can't Tell	N/A	Can't Tell	Can't Tell	N/A (Expl.)	Yes	Yes	16/20	Yes	No	Low	No
van der Sluiszen et al., 2016	Not Likely	N/A	No (Fixed)	N/A (Fixed)	N/A	Yes ¹⁰	N/A (Expl.)	Yes	Yes	25/31 ¹¹	Yes	No	Low	Yes
Van Dyke & Fillmore, 2014	Not Likely	N/A	Can't Tell	Yes	N/A	Can't Tell	No	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Van Dyke & Fillmore, 2015	Not Likely	N/A	Can't Tell	Yes	N/A	Can't Tell	Can't Tell	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Van Dyke & Fillmore, 2017	Not Likely	N/A	Can't Tell	Yes	N/A	Can't Tell	Can't Tell	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Veldstra et al., 2012 (Study 1)	Can't Tell	Can't Tell	Can't Tell	Yes	N/A	No	N/A (Expl.)	Yes	Yes	17/19	Yes	No	Low	No
Veldstra et al., 2012 (Study 2)	Not Likely	N/A	Yes	Yes	N/A	mp	N/A (Expl.)	Yes	Yes	19/20	Yes	No	Low	No
Veldstra et al., 2015	Can't Tell	Can't Tell	Can't Tell	Yes	N/A	No	N/A (Expl.)	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Vermeeren & O'Hanlon, 1998	Not Likely	N/A	No (Fixed)	N/A (Fixed)	N/A	Can't Tell ¹²	No (Expl.)	Yes	Yes	24/25	Yes	No	Low	No
Vermeeren et al., 2002a	Not Likely	N/A	No (Fixed)	N/A (Fixed)	N/A	Can't Tell ¹²	N/A (Expl.)	Yes	Yes	19/21	Yes	No	Low	No
Vermeeren et al., 2002b (Part 1)	Not Likely	N/A	Yes	Yes	N/A	Yes	Can't Tell	Yes	Can't Tell	Can't Tell	Yes	No	Low	No

Study	Representative Sample?	Participation Agreement Rate?	Drug Conditions Described as Randomized?	If Repeated Measures, Counterbalancing or Randomization?	Important Differences Between Groups Before Drive?	Driving Performance Assessors Aware of Drug Condition?	Participants Aware of Research Question?	Reliable Driving Data Collection?	Numbers and Reasons for Withdrawals and Drop-Outs Reported?	Percentage of Sample Completing Study?	Consistent Treatments?	Possible Treatment Contamination?	Risk of Reporting Bias?	Sample Size Based on Power Calculation?
Verster et al., 2002 (Part 1)	Not Likely	N/A	Yes	Yes	N/A	Yes	Can't Tell	Yes	Yes	29/30	Yes	No	Low	No
Vollrath & Fischer, 2017 (Study 1)	Can't Tell	Can't Tell	Yes	N/A	Can't Tell	Can't Tell	No	Yes	No ¹³	Can't Tell	Yes	No	Low	No
Vollrath & Fischer, 2017 (Study 2)	Can't Tell	Can't Tell	Yes	N/A	Can't Tell	Can't Tell	No	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Wan et al., 2017	Not Likely	N/A	Yes	Yes	N/A	Can't Tell	Can't Tell	Yes	Can't Tell	Can't Tell	Yes	No	High	No
Weafer & Fillmore, 2012	Not Likely	N/A	Yes	Yes	N/A	Can't Tell	No	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Weafer et al., 2008 (Study 1)	Not Likely	N/A	Can't Tell	Yes	N/A	Can't Tell	Can't Tell	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Weafer et al., 2008 (Study 2)	Not Likely	N/A	Yes	Yes	N/A	Can't Tell	Can't Tell	Yes	Can't Tell	Can't Tell	Yes	No	Low	No
Weiler et al., 2000	Can't Tell	Can't Tell	Yes	Yes	N/A	No	N/A (Expl.)	Can't Tell	Yes	40/41	Yes	No	High	No
Zhang et al., 2014	Can't Tell	Can't Tell	Yes	Yes	N/A	Can't Tell	N/A (Expl.)	Yes	No	22/25	Yes	No	Low	No

1. Randomly allocated to doses, but then reallocated to bins based on BAC resulting from dose.
2. Numbers and reasons for exclusion of specific datapoints reported.
3. Different levels of alcohol within the alcohol condition (i.e., fixed order).
4. Some missing data.
5. Fluctuating BAC studied at multiple time points (i.e., fixed order).
6. Numbers and reasons for withdrawals of specific datapoints reported in irrelevant measure.
7. Blinding not described. However, it seems unlikely that driving assessors could be blinded to treatments because the order of treatments was fixed.
8. The number of participants represented throughout parts of the study is not entirely clear.
9. Reasons and numbers are provided, but they are not reported clearly. Consequently, it is difficult to track participants' trajectories throughout the study.
10. The study is described as double-blind, but it is unclear how researcher blinding to the alcohol condition could have been achieved.

11. Additional driving data loss from two participants occurred in conditions not relevant to the meta-analysis.
12. The study is described as double-blind, but it is unclear how researcher blinding to the alcohol condition could have been achieved.
13. Unclear which group the attrition occurred in.

Table F2. Interrater agreement for study quality and risk of bias judgements.

	Representative Sample?	Participation Agreement Rate?	Drug Conditions Described as Randomized?	If Repeated Measures, Counterbalancing or Randomization?	Important Differences Between Groups Before Drive?	Driving Performance Assessors Aware of Drug Condition?	Participants Aware of Research Question?	Reliable Driving Data Collection?	Numbers and Reasons for Withdrawals and Drop-Outs Reported?	Percentage of Sample Completing Study?	Consistent Treatments?	Possible Treatment Contamination?	Risk of Reporting Bias?	Sample Size Based on Power Calculation?
Number of Items	77*	78	78	78	78	77*	78	78	78	N/A**	78	78	78	78
Kappa Score	0.87	0.82	0.91	0.83	0.83	0.85	0.59	-0.02	0.78	N/A**	0.66	0.00	0.43	0.64
Percent Agreement	94%	91%	95%	91%	95%	91%	74%	94%	88%	N/A**	99%	96%	77%	95%

Note that this analysis is based on judgements made between two coders: SS and DSL. It represents approximately 94% of the sample of judgements.

* Reflects erroneous omission of a judgement by one of two coders. When this occurred, the judgement was omitted from analysis.

** This item involved both categorical responses (e.g., *Can't Tell*), as well as continuous responses (e.g., *100%*), which precluded the calculation of inter-rater agreement.

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