A META-ANALYSIS OF ALCOHOL TOXICOLOGY STUDY FINDINGS AMONG HOMICIDE VICTIMS

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Aim: To synthesize the results of alcohol toxicology reports for homicide victims and examine variations in these results across person and setting characteristics.

Methods: We meta-analyzed 61 independent studies from 57 published manuscripts which met the study inclusion criteria and reported alcohol toxicology test results for homicide victims. A total of 71,031 toxicology test results, derived from 78,265 homicide victims across 13 countries (most from the United States), were examined.

*Results***:** On average, 48% of homicide victims tested positive for alcohol and 33% (using the .08 threshold) or 35% (using the .10 threshold) were determined to be intoxicated. The proportion of homicide victims testing positive for alcohol appeared to be decreasing over time. Further, the proportion testing positive increased with age, is higher for female than for male victims, and differs by race. Finally, the overall estimates were relatively stable across study sites.

Conclusion: Alcohol toxicology test results remain an important method for measuring the success of efforts to manage the consequences of alcohol. However, future toxicology studies should focus on collecting information on evidence processing time, establishing measurement standards for reporting data, and ensuring that subgroup estimates are included for purposes of cross-site comparisons.

*Keywords***:** alcohol, intoxication, toxicology, homicide, victimization, meta-analysis

Introduction

This study presents the results of a meta-analysis of alcohol toxicology findings from samples of homicide victims reported in 61 studies. These studies were published from 1953 to 2008, primarily in journals specializing in drugs, medicine, and forensic sciences and included both cross-sectional and longitudinal findings. The focus of this meta-analysis is to understand the variability in the proportion of homicide victims testing positive for alcohol across study and sample characteristics such as testing procedures, study location, years of data collection, and demographic composition of the victim sample. We conclude with some general observations on the role of homicide victim toxicology data as one available method for assessing and understanding the links between alcohol and homicide victimization.

Alcohol and Homicide Victimization

The relationship between alcohol and both violent offending and victimization is firmly established. Previous research has consistently identified and documented the presence of alcohol among homicide victims in varied settings. For example, studies from populations in New York City report that between 30% and 40% of homicide victims tested positive for the presence of alcohol [1, 2]. Among 674 homicide incidents in Allegheny County, PA between 1966 and 1974, about 32% of the victims tested positive for alcohol [3]. Further, within specific settings, alcohol presence among homicide victims also varies across racial [3], gender, [4] and age subgroups [5] in the United States and in other countries, including developing nations [6]. Alcohol-positive rates and intoxication test results among homicide victims also differ depending on the weapon used [7, 8] or the situational context of the homicide, specifically when the victims are adolescents [9] or are involved with domestic violence situations [10].

While considerable research has focused on the correlation between alcohol consumption and the situational and demographic characteristics of homicide offenders and victims, other studies have proposed and explored causal explanations. Recent toxicology research also argues that intoxication rates are comparable between homicide offenders and victims [11], suggesting that the causal mechanisms linking alcohol consumption and violent offending may also contribute to violent victimization. Earlier theoretical perspectives, such as Goldstein's tripartite framework [12], suggested that drug-related violence occurs because of the psychopharmacological effects of the substance (primary factors) or because of the violent nature of illegal drug users and markets (secondary). Other recent research reframes these primary and secondary factors as proximal or distal lifestyle factors [11]. Examples of proximal factors include alcohol-related cognitive impairments [13] and dysfunctions such as disrupted decision-making [14], inability to process perceptual cues accurately [15; 16], and increased reactive aggression [17, 18]. Meanwhile, life-style choices and activities, such as "night-time economies" and "routine activities," are some commonly noted distal factors. Night-time economies suggest that the increased time and length of consumption in particular settings (including those that serve alcohol) increase the risk of involvement in violence [19, 20, 21]. Routine activities theory [22] argues that alcohol consumption reduces the ability to protect oneself and increases the likelihood of being viewed as a suitable target. Considered collectively, these perspectives emphasize the importance of studying why and how alcohol consumption increases the risk for homicide victimization and exploring variations in alcohol-homicide victimization relationships across different populations, settings, times, and homicide event circumstances. This study further examines these relationships within the context of a meta-analysis of alcohol toxicology study findings among homicide victims.

As a point of reference, a 1999 meta-analysis of 65 studies published between 1975 and 1995 relied on post-mortem toxicology data to examine fatal non-traffic injuries involving alcohol [23]. The study focused on three types of violent deaths: unintentional injury deaths (7,459 victims), homicides (28,696 victims), and suicides (19,347 victims). The study found that homicide victims tested positive for alcohol at higher rates (47.1%) than victims of unintentional injury (38.5%) or suicide (29%). The study, however, noted that gender and age breakdowns were rarely reported in previous alcohol toxicology studies, which limits the comparability of toxicology results across demographic categories and research settings. The 1999 meta-analysis only relied on toxicology studies that were conducted within the United States, although alcohol toxicology test results for homicide victims are available from other countries.

The current study examines toxicology findings reported in 61 independent studies of homicide victims. We use the findings from these studies to conduct a meta-analysis that seeks to summarize the findings from this large body of international research. Moreover, we examine the relationships between alcohol toxicology findings and several other variables including the demographic characteristics of victims, toxicology testing procedures, homicide motives, and weapon types. Finally, we conclude with some suggestions for improving the quality and utility of future alcohol toxicology studies.

Data and Methods

This study used meta-analysis to summarize the results of alcohol toxicology tests on homicide victims. This process involved conducting a systematic search and retrieval of the literature for eligible studies, establishing clearly defined eligibility criteria (described below), using a systematic coding process [24], and applying meta-analytic statistical methods to analyze

the pattern of alcohol toxicology test results across studies, both in terms of central tendency and sources of variability.

Study Search and Retrieval Strategy

The following databases were searched for "alcohol" or "ethanol" or "blood alcohol content (BAC)" and "homicide" or "murder" or "violent death": Criminal Justice Abstracts, Criminal Justice Periodical Index, PsychArticles, National Criminal Justice Reference Service, MEDLINE, Criminal Justice Dissertation Abstracts, Social Sciences Citation Index, Sociological Abstracts, Science Citation Index Expanded, PsychInfo, and JSTOR. In some circumstances, searching for the terms in full text was appropriate; however, in other circumstances this method resulted in thousands of initially identified studies. In such cases, the process was limited to those studies for which the search terms were identified in the subject, keyword, or topic field. Furthermore, some databases allowed for further limits to be placed on the search. Such limits included only searching for studies that contained human subjects or that were published in the English language. This resulted in the removal of studies that contained the specified search terms but were either not relevant (due to the use of animal subjects, for example) or were unable to be read because they were not written in English.

The initial searches resulted in a total of 3,162 references. Titles of these references were reviewed to eliminate clearly irrelevant studies, reducing the set to 1,534. The full abstracts of these 1,534 references were then carefully reviewed, yielding 389 potentially eligible studies. Many of the eliminated studies reported information for homicide offenders only (as opposed to victims) or used per capita/aggregate alcohol consumption rates to determine associations between alcohol and homicide. Duplicates across databases were then removed, resulting in a

total of 242 relevant studies, the full-text of which was used to determine final study eligibility. The reference sections of these studies were also cross-checked for additional potentially eligible studies. Copies of these 242 studies were then provided to two teams to review independently for eligibility based on the criteria described below.

Eligibility Criteria

A study was eligible if it met five criteria. First, the study sample consisted of homicide victims, or alcohol toxicology test results were presented separately for homicide victims. Second, the study provided alcohol toxicology test results. These results also needed to be presented in a statistical form that allowed for collection or calculation of the percentage of the sample testing positive for alcohol. Third, the study was available in written form in the English language. Fourth, the overall study sample must not have been restricted by type of weapon (e.g., firearms or sharp instruments) or homicide motive (e.g., domestic or gang-related homicides). If a study sample was comprised only of homicide victims who were killed with a certain weapon type (e.g., firearms) or within certain categories of homicides (gang homicides, for example), the study was excluded. However, if the overall study sample happened to include additional breakout information for weapon and/or homicide motive types, it was potentially included assuming the overall sample was not otherwise restricted or ineligible. Fifth, the sample must not have been restricted by victim type (e.g., only female or juvenile victims). Many of the studies that we excluded examined various sub-populations of homicide victims (adolescent victims, victims only killed with firearms or blunt weapons, male versus female victims, etc.).

We did not restrict inclusion based on the type of testing protocol used. Test methods and testing thresholds have changed over time, and testing protocols may vary depending on the study location or sample available (blood, urine, or other biological samples). Further, some testing equipment may have been readily available in certain areas or countries but unavailable in others. We placed no restrictions on the geographic location of the study and therefore included studies conducted from all available nations and locations. Our English language restriction, however, is likely to have limited the international breadth of this review. The only restriction placed on the year of publication was that the study was published after 1950. However, most of the studies we identified were conducted since the early 1970s and were published as peer reviewed journal articles. A few of the studies were books, book chapters, or technical/governmental reports.

Coding Procedures

The coding forms (available from the first author) captured information on many characteristics of the study such as the years when the homicide data were collected, testing procedures used, weapons and motives, if available, as well as the results of the alcohol toxicology tests. The primary unit of analysis was an independent study sample. Multiple publications based on the same independent study, sample, or dataset were treated as a single study for coding purposes. The protocol allowed for the coding of multiple toxicology results (effect sizes) per study, such as the results for different subgroups within a particular sample. Whenever possible, separate effect sizes were also coded for breakouts of the overall results by gender, race/ethnicity, age group, sample year, and weapon type used in the homicide. All of the

studies were double-coded by independent coders at separate locations and any discrepancies were resolved by a third independent coder (the lead author).

Statistical Analyses

The effect size of interest for this meta-analysis was the proportion (*p*) of homicide victims testing positive for alcohol. The meta-analytic analyses, however, were performed using the *logit* of the proportion given its more desirable statistical properties [25, 26]. If the reported proportion was zero, then the logit was computed based on a proportion equal to 1/*n*. Similarly, if it was one, then the logit was computed as (*n*-1)/*n*. Final results were converted back into proportions for easier interpretation. Meta-analytic analyses, including the mean effect size, estimates of heterogeneity, and moderator analyses, were performed using the inverse variance weight method [25, 27]. We assumed *a priori* that the data conformed to a random-effects model [25, 28]. Under a random effects model, effect sizes are assumed to vary as a result of both within-study sampling error and between-study unobserved random differences. The method-of-moments estimator of the random effects variance component (tau-squared) was used [29]. All analyses were performed in Stata using macros that are available at http://mason.gmu.edu/~dwilsonb/ma.html. Only a single effect size per study was included in a given analysis, thereby maintaining statistical independence among effect sizes. An exception was made for analyses of breakouts. In these analyses, a study could contribute an effect size for each level of a breakout. For example, in the analysis of the breakout by gender, a study could provide one effect size for the mean effect for males and one effect size for the mean effect for females.

Findings

Based on our comprehensive search of the literature, we identified 61 independent studies of alcohol toxicology in homicide victims that were eligible for this meta-analysis [30-90]. Two references reported results separately for different cities (two cities for Cherpitel (1996) and four cities for Harper (1976)) and these were treated as independent studies for our purposes. **Table 1** summarizes the characteristics of these studies, the full-sample rates testing positive for alcohol, and the rates intoxicated at the .08 and/or .10 blood alcohol level. The measurement scale used to report the amount of alcohol detected in the homicide victim was documented for 31 of the 61 studies. However, fourteen different units of measurement were reported: seven studies used mg/mL; four studies used mg/dL; ten studies used g%, g/mL, and mg%; and the remaining ten studies used g/100g, g/100ml, g/100L, gm%, mg/g, mg/ml, or mmol/L. Among the thirty studies that did not indicate a specific unit of measurement, evidence of the presence of alcohol was simply coded as testing positive (yes or no) for purposes of this meta-analysis.

Across these study samples, the meta-analytic random effects mean proportion that tested positive for alcohol was 48% (see **Table 2**). This distribution is highly heterogeneous (Q = 3,995.6, df = 60, $p < .0005$, $tau^2 = 0.26$). With the exception of two outliers at 8% and 86%, the effects ranged from 24% to 76%. The distribution was approximately normal. The mean remained essentially unchanged when the two outliers were removed and when the four largest studies (sample sizes greater than 6,000) were removed. These effect sizes and the overall mean are displayed as a forest plot in **Figure 1**.

Only six studies reported the percentage of homicide victims that were intoxicated at the 80 mg/dl blood alcohol content (BAC) level but 30 studies reported these results at the 100 mg/dl level. The random effects means were roughly similar and predictably less than the overall

percentage testing positive at any level (33% and 35%, respectively). As was the case for the percent testing positive for alcohol, both of these distributions were highly heterogeneous, suggesting large and meaningful differences across the various samples. The remaining analyses explored this variability using only the effect sizes based simply on a positive test for alcohol.

Testing Procedure

The testing procedure had an effect on the percentage positive estimates (see **Table 2**), with tests based on both blood or urine producing the largest overall mean (68%) and tests based on something other than blood or urine (e.g., some other biological sample) producing a lower overall mean (42%; $Q_{between} = 11.56$, $df = 2$, $p = .003$; $tau^2 = 0.21$). Unfortunately, only three studies [40, 47, 63] contributed to the mean for blood and urine, although all three were consistently high (63%, 69%, and 76%).

Year of Data Collection

Alcohol consumption patterns have changed over time [91, 92] and will likely continue to change. As such, the percentage of homicide victims with alcohol in their system at the time of death might have also changed. This issue was first examined by regressing the effect size (the logit of the proportion testing positive) on the year the data were collected. Overall, there was a strong negative linear relationship (B = -.024, $p = 0.0002$, R^2 = .25). One potential complication with this analysis was that some studies reported estimates from data collected over multiple years (see **Table 1**). In those cases, the mid-point was used for these analyses. However, we also performed separate regressions for the six studies that reported data separately across multiple years [32, 34, 39, 46, 69, 87]. The regression coefficient for year was still

negative for five of six of these analyses, consistent with the interpretation of an overall downward trend in the proportion of homicide victims testing positive for alcohol over time.

Victim Age

Although ten studies provided results separately for different age categories, the categories were not consistent across studies, thereby complicating any sub-analysis. As such, we estimated both the linear and curvilinear relationship between the logit for the proportion testing positive for alcohol and age category within each study, except for one study that only provided two age categories. The linear component was positive in all cases, indicating a general increase in the proportion of victims testing positive for alcohol with increasing age (regression coefficients ranged from 1.04 to 5.22). The curvilinear component was negative in all cases (ranging from -.11 to -.63) indicating that the upward trajectory flattens out and decreases for the higher age categories.

Victim Gender and Race

Fifteen studies reported results separately for males and females, with a substantially higher proportion of females testing positive than males (48% versus 28%). This effect is statistically significant and suggests that alcohol is more prevalent among homicides involving female victims.

Nine studies provided separate estimates by race. However, only six studies reported estimates for the four general race categories used in this study: black, white, nonwhite Hispanic, and other. The proportion of victims testing positive for alcohol varied somewhat across certain racial/ethnic groups with a low of 27% for nonwhite Hispanics and a high of 48% for other races.

This difference was statistically significant ($Q_{between} = 10.37$, $df = 3$, $p = .016$; $tau^2 = 0.19$). However, a pairwise comparison between whites and blacks found that the difference was not statistically significant. Thus, similar to gender, victim race does appear to be related to the proportion of homicide victims testing positive for alcohol, although the evidence of a difference is more robust regarding the gender effect.

Geographic Location

Fifteen studies reported data from outside the United States, representing a range of countries including Turkey, South Africa, Denmark, Norway, Finland, Australia, Canada, Sweden, and Thailand. The rest of the studies were conducted within the United States and occurred in various states including Alabama, California, Florida, Georgia, Louisiana, Maryland, Mississippi, North Carolina, New Jersey, Nevada, New York, Ohio, Oklahoma, Pennsylvania, Tennessee, Texas, and Washington. Some of the studies used city-based samples only, while others were collected from county records or a combination of city and county records. Eight studies involved data representing an entire state or from more than just one city/county; these locations were coded as other (see **Table 2)**. Overall, the percentage of homicide victims testing positive was remarkably similar across samples drawn from these different geographic regions, and the difference in means across study locations was not statistically significant.

Discussion

Across 61 independent studies conducted in 16 countries, an average of 48% of homicide victims tested positive for alcohol. About a third of the victims were intoxicated in study locations that used a quantitative threshold of 80 mg/dl, compared with 35% for countries that

used a threshold of 100 mg/dl level to define intoxication. These broad findings were remarkably consistent with the results of a previous meta-analysis [23] that was based only on studies conducted in the United States (47.1% testing positive and 31.5% intoxicated at the 100 mg/dl threshold).

However, the current study goes beyond the previous meta-analysis and provides additional information that furthers our understanding of alcohol toxicology results, with a specific focus on homicide victims. First, the results suggest that testing procedure and the specific tested sample (blood, urine, and/or other body tissue) appear to impact overall estimates for testing positive and for intoxication. This finding should be particularly important for crime scene technicians, forensic scientists and pathologists to consider as they collect and analyze homicide victim and offender samples. In fact, the three studies that included both blood and urine yielded much higher estimates of the proportion of victims testing positive for alcohol. The implications, although speculative, suggest that many toxicology studies could be systematically reporting lower-bound estimates.

Second, the mean percentage of homicide victims testing positive for alcohol appears to be dropping slightly over time within samples from the United States (most other countries only had one study included). These results may indicate some effectiveness of North American alcohol policies developed to manage consumption rates, discourage binge or excess drinking, delay the age of onset of drinking, treat alcoholism, or other similar goals, but this is merely speculation. A follow up study that examines the rates of alcohol use among homicide offenders would be particularly useful as a basis of comparison, and as a means of gauging the impact of alcohol policies within the broader population of violent offenders.

Third, the proportion of homicide victims testing positive for alcohol increases with age to a point, and then levels off in later adulthood. Further, female victims were more likely to test positive for alcohol than male victims, and some racial differences also emerged. These are important findings that were unavailable in an earlier meta-analysis [23]. However, to the extent that alcohol consumption patterns are prevalent and/or changing within certain demographic subgroups, the corresponding impact on homicide offending and victimization should be carefully monitored [91. 92].

Finally, despite the inclusion of 15 studies conducted outside of the United States, the average rate of homicide victims testing positive was fairly consistent regardless of the study location. This finding is particularly interesting considering variations in minimum drinking ages [93], access to alcohol, and cultural patterns of alcohol use. Nevertheless, it seems clear that improved and effective alcohol-consumption and control policies are needed in a wide variety of settings. Recent promising approaches have focused on training bar staff to manage aggression within drinking establishments in Canada [94], using problem-oriented policing and problem-solving approaches to reduce assaults in and around bars in the United States [95], and limiting operating hours among drinking establishments [96] or instituting alcohol dry laws in Brazil [97].

Improving the Future of Toxicology Studies

Most of the studies reviewed here did not contain measures of processing time. Specifically, future studies should begin to capture the timeframe between alcohol consumption, lethal outcome and evidence collection, preservation and testing. This is particularly important for ensuring the validity of alcohol toxicology test results since alcohol continues to evaporate

and deteriorate over time following death [98, 99, 100]. In addition, we found wide variations in the units of measurement reported in alcohol toxicology studies (mg/mL, mg/dL, g%, g/mL, mg%, g/100g, g/100ml, g/100L, gm%, mg/g, mg/ml, and mmol/L). It would be helpful for forensic scientists to standardize such reporting in the future. In this review, most of the studies used g%, g/mL as the reported measurement.

Finally, dozens of studies did not report toxicology results within demographic subgroups. We consider this particularly important for the future of toxicology science given documented variations in metabolism rates [101], community characteristics, alcohol preferences [91] and availability [93], and laws that attempt to minimize the harms associated with alcohol use and abuse [96, 97].

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Figure 1 – Proportion Testing Positive for Alcohol with 95% Confidence Interval

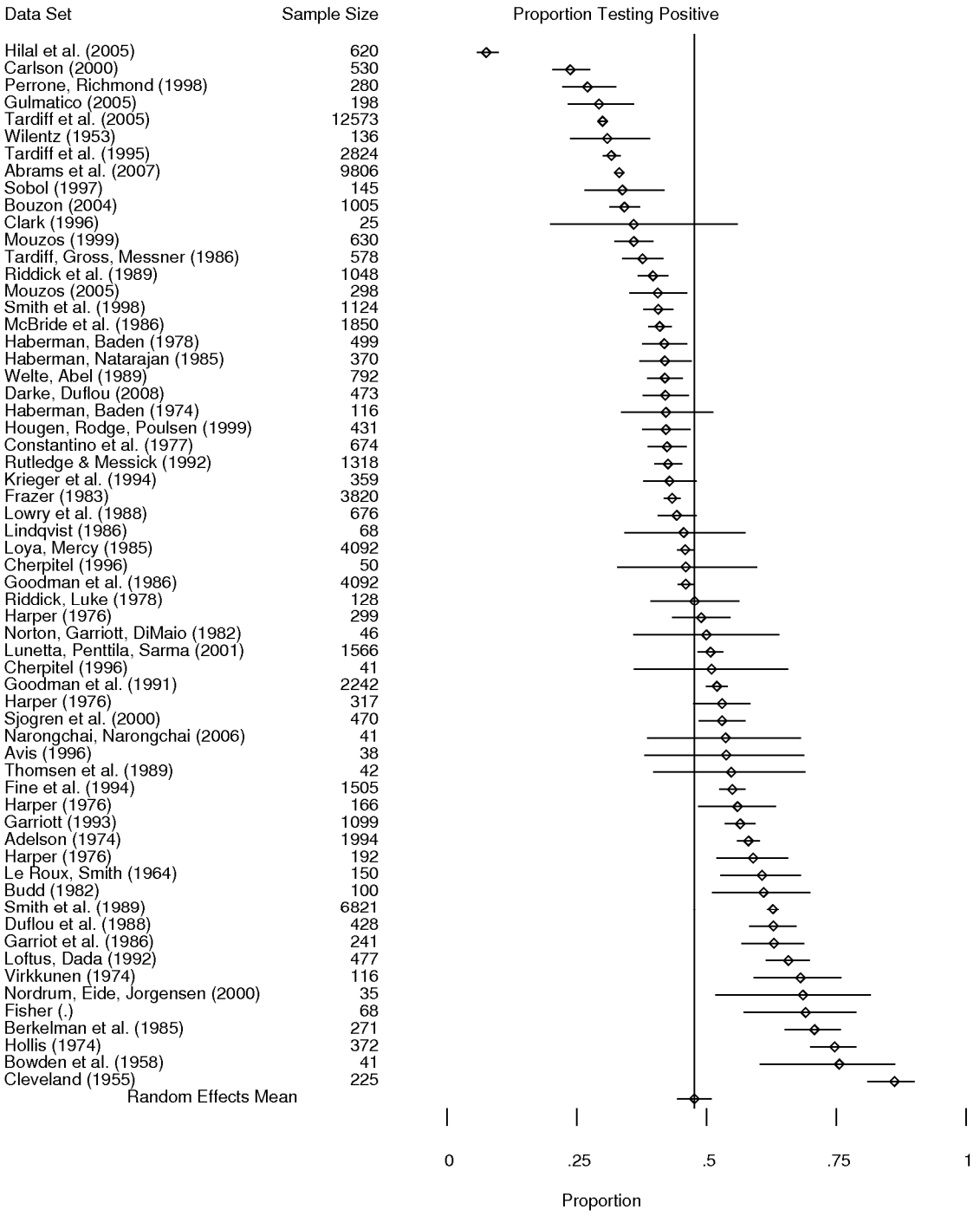

Table 1 – Study Characteristics and Full Sample Alcohol Toxicology Results

(Study ID) Citation	Place	Years	N	Positive for Alcohol	Intoxicated at .08	Intoxicated at .10
Avis (1996)	Newfoundland, Canada	1985-1993	38	53.8	NA	NA
Abrams, Leon, Tardiff, Marzuk, & Sutherland (2007)	New York City, NY	1990-1998	9806	33.2	NA	NA
Adelson (1974)	Cuyahoga County, OH	1958-1971	1994	58.1	39.3	39.3
Berkelman, Herndon, Callaway, Stivers, Howard, Bezjak, & Sikes (1985)	Fulton County, GA	1981-1982	271	70.8	NA	50.9
Bouzon (2004)	Orleans Parish, LA	1980, 1985, 1990, 1995, 2000	1005	34.2	13	5
Bowden, Wilson, & Turner (1958)	Victoria, Melbourne	1951-1956	41	75.6	NA	53.7
Budd (1982)	Los Angeles, CA	1980	100	61	43	36
Carlson (2000)	Dallas County, TX	1997-1998	530	23.8	23.8	NA
Clark (1996)	St. John Parish, LA	1992-1995	25	36	NA	NA
Cleveland (1955)	Hamilton County, OH	1947-1953	225	86.2	NA	NA
Constantino, Kuller, Perper, & Cypress (1977)	Allegheny County, PA	1966-1974	674	42.4	NA	31.8
Darke & Duflou (2008)	New South Wales	1996-2005	473	42.1	NA	NA
Duflou, Lamont, & Knobel (1988)	Cape Town, South Africa	1986	428	62.9	53.3	53.3
Fine, Roseman, Constandinou, Brissie, Glass, & Wrigley (1994)	Jefferson County, AL	1978-1989	1505	55	NA	NA
Fisher (n.d.)	Baltimore, MD	1949	68	69.1	NA	57.4
Frazer (1983)	Cuyahoga County, OH	1969-1980	3820	43.4	NA	NA
Garriott (1993)	Bexar County, TX	1985, 1987, 1990, 1991	1099	56.5	NA	NA
Garriott, Di Maio, & Rodriguez (1986)	Bexar County, TX	1985	241	63	NA	NA
Goodman, Istre, Jordan, Herndon, & Kelaghan (1991)	Oklahoma	1978-1984	2242	52	NA	34
Goodman, Mercy, Loya, Rosenberg, Smith, Allen, Vargas, & Kolts (1986)	Los Angeles, CA	1970-1979	4092	46	NA	30
Haberman & Baden (1974)	New York City, NY	1972	116	42.2	NA	42.2
Haberman & Baden (1978)	New York City, NY	1974-1975	499	41.9	NA	26.7
Haberman & Natarajan (1986)	Essex County, NJ	1981-1984	370	42	NA	27
Hilal,Çekin, Gülmen, Özdemir, & Karanfil (2005)	Adana, Turkey	1997-2001	620	7.6	NA	NA
Hollis (1974)	Shelby County, TN	NA	372	74.7	NA	NA

Hougen, Rodge, & Poulsen (1999)	Copenhagen, Denmark	1985-1994	431	42.2	NA	NA
Le Roux & Smith (1964)	Cape Peninsula, South Africa	1962	150	60.7	NA	54.7
Lindqvist (1986)	Northern Sweden	1970-1980	68	45.6	NA	NA
Lowry, Hassign, Gunn, & Mathison (1988)	New Orleans, LA	1979, 1982, 1985, 1986	676	44.3	NA	24.2
Lunetta, Penttilä, & Sarna (2001)	Finland	1987-1996	1566	50.8	NA	NA
McBride, Burgman-Habermehl, Alpert, & Chitwood (1986)	Miami-Dade County, FL	1978-1982	1850	41	NA	19.2
Mouzos (1999)	Australia	1996-1998	630	36	NA	NA
Mouzos (2005)	Australia	2003-2004	298	40.6	NA	NA
Nordrum, Eide, Jørgensen (2000)	Northern Norway	1973-1992	35	68.6	NA	65.7
Norton, Garriott, Di Maio (1982)	Dallas, TX	1978	46	50	NA	37
Perrone & Richmond (1998)	Hawaii	1992-1997	280	27.1	NA	NA
Riddick & Luke (1978)	District of Columbia	1974-1975	128	47.7	NA	42.8
Rutledge & Messick (1992)	North Carolina	1986-1988	1318	42.6	NA	42.6
Sjögren, Erriksson, Ahlm (2000)/ Sjögren, Valverius, & Eriksson (2006)	Sweden	1992-1996	470	53	NA	NA
Smith, Kuller, Perper, Brent, Moritz, & Constantino (1998)	Allegheny County, PA	1966-1974, 1984-1990, 1992-1993	1124	40.7	NA	23.3
Smith, Goodman, Thacker, Burton, Parsons, & Hudson (1989)	North Carolina	1973-1983	6821	62.8	NA	52.1
Sobol (1997)	Buffalo, NY	1992-1993	145	33.8	NA	NA
Tardiff, Gross, & Messner (1986)	Manhattan, NY	1981	578	37.7	NA	NA
Tardiff, Marzuk, Leon, Hirsch, Sta1jic, Portera, & Hartwell (1995)	New York City, NY	1990-1991	2824	31.7	NA	NA
Tardiff, Wallace, Tracey, Piper, Vlahov, Galea (2005)	New York City, NY	1990-1998	12573	30	NA	NA
Virkunnen (1974)	Helsinki, Finland	1963-1968	116	68.1	NA	NA
Welte & Abel (1989)	Erie County, NY	1972-1984	792	42	NA	33
Cherpitel (1996a)	Contra Costa County, CA	1987-1988	50	46	NA	30
Cherpitel (1996b)	Hinds County, MS	1992-1993	41	51	NA	27
Gulmatico (2007)	Clark County, NV	2005	198	29.3	NA	NA
Harper (1976a)	Atlanta, GA	1974	192	59	NA	NA
Harper (1976b)	Cleveland, OH	1974	317	53	NA	NA
Harper (1976c)	Miami, FL	1974	166	56	NA	NA

Harper (1976d)	District of Columbia	1974	299	49	NA	NA
Riddick, Brissie, Embry, Cumberland, Gilchrist, Glass, & Rabren (1989)	Alabama	1980-1982	1048	39.7	NA	39.7
Krieger, Song, Heck, Talltree, & Allen (1994)	King County, WA	1988-1992	359	42.9	NA	NA
Thomsen, Albrektsen, Aalund, Breiting, Danielsen, Helweg-Larsen, Jacobsen, Kjaerulff, & Staugaard (1989)	Copenhagan, Denmark	1985-1986	42	54.8	NA	45.2
Loya & Mercy (1985)	Los Angeles, CA	1970-1979	4092	45.9	NA	30.2
Loftus & Dada (1992)	South Africa	1985-1989	477	65.8	NA	NA
Narongchai & Narongchai (2006)	Thailand	2003	41	53.7	39	31.7
Wilentz (1953)	Unknown	1933-1951	136	30.9	NA	14

Table 2: Random Effects Mean Percent Testing Positive for Alcohol

Analysis	Mean Percent	95% C.I. Lower	95% C.I. Upper	*Q*	*p*	Tau^2	*k*
Alcohol (any)	48%	44%	51%	3994.6	0.000	0.26	61
BAC above .08	33%	21%	48%	294.2	0.000	0.57	6
BAC above .10	35%	30%	39%	1693.6	0.000	0.29	30
Test Sample				11.56[a]	0.003	0.21	
Blood	50%	46%	54%				40
Blood and Urine	68%	54%	80%				3
Other	42%	35%	48%				12
Geographic Location				3.67[a]	0.299	0.19	
U.S. City	44%	39%	50%				15
U.S. County	50%	45%	54%				21
Foreign Country	50%	44%	55%				17
Other	44%	36%	52%				8
Victim Sex				18.72[a]	0.000	0.25	
Female	48%	41%	55%				15
Male	28%	22%	34%				15
Victim Race				10.37[a]	0.016	0.19	
African American	41%	32%	49%				6
Caucasian	32%	25%	41%				6
Non-White Hispanic	27%	18%	38%				6
Other	48%	39%	58%				6

Note: Meta-analyses performed on logged odds (logits) and converted back into percentages.
a. Represents a Q-between that is analogous to a one-way F and tests whether the means differ across categories