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The Prevalence of Alcohol-Involved Homicide Offending: A Meta-Analytic Review

Joseph B. Kuhns[1], M. Lyn Exum[1], Tammatha A. Clodfelter[2] and Martha Cecilia Bottia[3]

Abstract

This study meta-analyzes 23 independent studies that included information from 28,265 homicide offenders across nine countries. On average, 48% of homicide offenders were reportedly under the influence of alcohol at the time of the offense and 37% were intoxicated. We found no demographic variations across age, gender, or race, although the proportion testing positive within the United States appears to be decreasing over time. Further, the proportion of offenders who were under the influence of alcohol was lower among those who committed the homicide with a firearm. Communities that have high homicide rates should work to reduce alcohol consumption rates.

Keywords

alcohol, intoxication, homicide, offender, meta-analysis

Introduction

Although the relationship between alcohol and violent crime has been studied extensively (Collins, 1981; Dingwall, 2006; Felson, Savolainen, Aaltonen, & Moustgaard, 2008; Felson & Staff, 2010; Felson, Teasdale, & Burchfield, 2008; Galanter, 1997; Martin, 1993; McMurran, 2013; Parker & McCaffree, 2013; Parker & Rebhun, 1995; Pernanen, 1991), the percentage of homicide offenders who are under the influence of

[1]Department of Criminal Justice & Criminology, University of North Carolina at Charlotte, Charlotte, NC, USA
[2]Appalachian State University, Boone, NC, USA
[3]Department of Sociology, University of North Carolina at Charlotte, Charlotte, NC, USA

Corresponding Author:
Joseph B. Kuhns, Department of Criminal Justice & Criminology, University of North Carolina at Charlotte, 9201 University City Blvd, Colvard 5072, Charlotte, NC 28223, USA.
Email: jbkuhns@uncc.edu

alcohol (at any level) at the time of the offense remains subject to debate. Prevalence estimates vary widely across studies, ranging from a high of 83% (Shupe, 1954) to a low of 15% (Varano & Cancino, 2001). This variability can be attributed not only to fixed differences across studies (e.g., sample size and composition), but also to unobserved random differences. Such differences make narrative reviews of this literature especially difficult to conduct.

Meta-analytical techniques were designed to synthesize individual study findings from a given literature, mindful of the inherent differences across studies (Cooper, Hedges, & Valentine, 2009; Lipsey & Wilson, 2001). In doing so, meta-analyses provide several advantages over the typical narrative summation of research studies (Pratt, Cullen, Blevins, Daigle, & Madensen, 2008). For example, meta-analyses offer more precise accounts of aggregate effects by calculating a weighted mean effect across studies. In addition, meta-analyses allow these estimates to be disaggregated across various factors that may moderate the overall effect (e.g., sample demographics, the research design, etc.). Finally, meta-analytic databases can be routinely updated as new literature emerges, thereby allowing the results to be reassessed as necessary in order to grow with the literature.

To our knowledge, the current study is the only meta-analysis to date that systematically examines a cross-national collection of homicide offenders and their alcohol and intoxication status at the time of their crimes. The analysis was based on 23 studies conducted over the past 60 years; these studies often appeared in journals specializing in drugs, violence, medicine, and forensic sciences. The primary purpose of this meta-analysis was to estimate the proportion of homicide offenders who were positive for alcohol and/or intoxicated at the time of the offense.[1] Furthermore, we disaggregated this global estimate across a variety of study-, offender- and offense-characteristics such as the country of origin, the years in which the homicides occurred, offender demographics, and the use of various types of weapons. We conclude with some general observations about the prevalence of alcohol use among homicide offenders, and discuss the potential violence-reduction benefits from alcohol control policies.

Alcohol, Aggression, and Homicide

Several theories purport to explain the relationship between alcohol and aggression—of which homicide is but one manifestation (Chermack & Giancola, 1997; Gustafson, 1994; McMurran, 2013; Parker & Auerhahn, 1998; Parker & McCaffree, 2013). Most of these theories can be organized into different groups or "models" based on similarities in their founding assumptions. Bushman (1997) identified three such groupings of theories: the disinhibition model, the expectancy model, and the indirect causal model.

The disinhibition model assumes cognitive structures in the frontal cortex inhibit aggressiveness. According to this perspective, when alcohol is consumed the pharmacological properties of ethanol directly weaken cortical inhibitory processes, thereby allowing aggression to flow unchecked (Assaad & Exum, 2002; Taylor & Leonard, 1983). In its most basic form, the disinhibition model is heavily deterministic and largely assumed to be invariant; yet, research suggests that intoxicated aggression is

probabilistic and moderated by various individual and situational factors (Chermack & Giancola, 1997; Exum, 2006). While some have therefore come to view the disinhibition model as an insufficient explanation for intoxication aggression (Bushman, 1997; Giancola, 2013), others have sought to modify the perspective. For example, Parker and Rebhun (1995) offer a theory of *selective* disinhibition in which the pharmacological properties of alcohol interact with social norms, which may vary across social situations. According to this perspective, the pharmacological properties of alcohol are no longer assumed to be invariant but are seen as conditional upon various contextual factors such as the perceived normative expectations for behavior in a given setting (see also Parker & McCaffree, 2013).

The expectancy model states that a person's learned beliefs about alcohol (rather than the pharmacological properties of ethanol) foster aggressive behavior (Assaad & Exum, 2002; Giancola, 2013). According to this perspective, if a person holds a strong belief that alcohol promotes aggression, then these expectations will be primed during a drinking episode. Much like a self-fulfilling prophecy, the drinker will become more aggressive because that is what he expects will happen. Although there is some empirical evidence to suggest expectancy effects contribute to the problem of intoxicated aggression (Lang, Goeckner, Adesso, & Marlatt, 1975), these effects are generally weak and have little explanatory power (Chermack & Taylor, 1995; Duke, Giancola, Morris, Holt & Gunn, 2011; Exum, 2006; Hull & Bond, 1986; Lipsey, Wilson, Cohen & Cerzon, 1997).

A commonly touted model for explaining intoxicated aggression is the indirect causal model (Assaad & Exum, 2002; Giancola, 2013). Much like the notion of selective disinhibition (Parker & Rebhun, 1995), the indirect causal model focuses on the pharmacological properties of alcohol and considers their impact on aggression to be indirect and probabilistic. In other words, the indirect causal model assumes the alcohol–aggression relationship is moderated by various cognitive, emotional, and/or physiological changes that may result after drinking—changes that then interact with social/situational cues present in the drinking setting (Bushman, 1997; Pihl, Peterson, & Lau, 1993). For example, alcohol may affect the portion of the brain associated with reasoning, problem solving, and formulating strategies in response to threats. In certain settings (e.g., when victim provocation is present), alcohol may alter the way the brain processes such situational cues and/or reshape our behavioral repertoire of how to respond, thereby making aggressive behavior more likely (Assaad & Exum, 2002; Giancola, 2004, 2013; Giancola, Josephs, Parrott & Duke, 2010; Lau, Pihl, & Peterson, 1995; Pernanen, 1976; Pihl et al., 1993; Steele & Josephs, 1990; Taylor & Leonard, 1983).

Many studies have examined the relationship between violent crime and alcohol consumption—be it consumption by the victim, the offender, or both (e.g., Brecklin & Ullman, 2002; Collins & Schlenger, 1988; Greenfeld & Henneberg, 2001; Pridemore & Eckhart, 2008) . A recent meta-analysis of homicide victims suggested that approximately 48% are positive for alcohol and about a third are intoxicated at the time of death (Kuhns, Wilson, Clodfelter, Maguire, & Ainsworth, 2011). Some scholars contend that the intoxication rates for homicide victims may mirror those of the offenders (Darke, 2010), thereby suggesting that the mechanisms linking alcohol to violence may be similar for both parties. To date, however, no meta-analysis examining the

alcohol status of homicide offenders exists, so the degree to which the prevalence rates of victims and offenders are similar remains open to debate. The current study will address this gap in the literature.

Note that while toxicology tests are often available for homicide victims, such forensic evidence is typically unavailable for homicide offenders. The passage of time between the commission of the crime and the apprehension of the offender—coupled with the rate at which alcohol is metabolized—adversely affects researchers' abilities to document blood alcohol levels at the time of the crime. To determine the alcohol status of homicide offenders, researchers have therefore generally relied on either self-report data or second-hand observations, such as those recorded in the offenders' case files. In the current study, we merged the results from these various sources of data to generate a composite estimate of the prevalence of alcohol-involved homicide offending.

Method

This study used meta-analysis to summarize the alcohol and intoxication status of homicide offenders at the time of the offense, as reported in prior studies. This process involved conducting a systematic search and retrieval of the literature for eligible studies, establishing a clearly defined eligibility criteria (described below), using a systematic coding process (Wilson, 2009), and applying meta-analytic statistical methods to analyze the alcohol prevalence rates from previous 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 offending" 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, Google Scholar, and JSTOR. In some circumstances, searching for the terms in full text was useful for identifying a core set of relevant studies; however, in other circumstances this method resulted in thousands of initially identified studies. In such cases, the process was limited to those studies in 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 were published in the English language.

The initial searches resulted in a total of several thousand references. Titles of these references were reviewed to eliminate clearly irrelevant studies (e.g., those focused on victims), reducing the set to approximately 200 studies. The full abstracts of these references were then carefully reviewed, yielding fewer potentially eligible studies. Many of the eliminated studies reported information for homicide victims only (as opposed to offenders) or used per capita/aggregate alcohol consumption rates to determine associations between alcohol and homicide rates. Duplicates across databases were also

removed, resulting in a total of 49 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 studies were provided to two members of the research team to review independently for eligibility based on the criteria described below.

Eligibility Criteria

A study was eligible if it met five inclusion criteria. First, the study must have contained self-report, case file, or biological (toxicology) data obtained from the homicide offenders themselves or from case studies of homicide offenders. Second, the study must have reported the offenders' alcohol use immediately prior to or during a homicide event. These results needed to be presented in a statistical form that allowed for the collection or calculation of the percentage of the sample or subsample that had: (a) a positive biological test, (b) self-reported using alcohol immediately prior to or during the homicide event, or (c) evidence of such alcohol use documented in a case file such as a police, prosecutor, or psychiatric file. If available, a determination of an offender's *intoxication* status was also made through either toxicology, self-report, or case file data.[2] Third, the study needed to be available in written form in the English language. Fourth, the overall study sample must not have been restricted by type of weapon or homicide motive. If a study sample was comprised only of homicide offenders killed with a certain weapon type (e.g., firearms only) or offenders within certain categories of homicides (only gang-related homicides, for example), the study was excluded. However, if a study disaggregated its alcohol findings by weapon or motive type, it was included on the condition that the overall sample or study was not otherwise restricted or ineligible. Finally, the sample must not have been targeted to a specific homicide offender type (e.g., only juvenile offenders). Note, however, that adult-only samples were typical in this review and were not excluded. Many of the studies that we included examined various subpopulations of homicide offenders (adolescents, those who only killed with firearms or blunt weapons, males, etc.), but the overall sample was not restricted.

We placed no restrictions on the geographic location of the study and therefore included studies conducted within and outside of the United States; however, our English language restriction is likely to have limited the international breadth of this review to some degree (yet, we found only one study written in another language during our search). There were no restrictions based on the year of publication or publication type. However, approximately half of the studies we identified were published after 1990 (n = 13) and/or appeared as peer reviewed journal articles (n = 12). The remaining studies were available as books, book chapters, or technical/governmental reports.[3]

Coding Procedures

The coding forms (available from the first author) captured information on many characteristics of the study such as the year(s) in which the homicides occurred,

the location of the study sample, the demographic composition of the sample, the homicide weapons and motives (if available), as well as the results of the alcohol toxicology tests, self-report data, or case file data. The primary unit of analysis was an independent study sample. Multiple publications based on the same data set were treated as a single study for coding purposes. The protocol allowed for the coding of multiple alcohol status results (effect sizes) per study, such as the results for different subgroups (e.g., by race, sex, weapon type, etc.) within a particular sample. The studies were double-coded by independent coders and the first and second authors resolved any discrepancies.

Statistical Analyses

We focused our analyses on two primary effect sizes: (a) the proportion of homicide offenders who were positive for alcohol at the time of their offense, and (b) the proportion who were intoxicated. Prior to analyses, we first converted all proportions to their logit values in order to minimize problems associated with underestimated confidence intervals and overestimated heterogeneity estimates (Lipsey & Wilson, 2001). Note that no proportion reported in the set of eligible studies was found to be zero or one, and therefore, we did not need to correct for such extreme outcomes prior to calculating the logits (Kuhns et al., 2011). For ease of interpretation, all findings were converted back into proportions/percentages, and are reported here as such.

Our analytic approach mirrored that of Kuhns et al. (2011). In all meta-analytic calculations we adopted the inverse variance weight method to compensate for the different sample sizes across studies. We also analyzed the data using a random effects model that assumes the differences in effect sizes are attributed, in part, to unobserved random differences across studies. Using this technique, we estimated the weighted mean proportion (or percentage) of homicide offenders who tested positive for alcohol, as well as the lower and upper bounds of the 95% confidence interval around this mean. To test for the presence of heterogeneity across the individual effect sizes (i.e., to see if there is variability in the mean estimate that cannot be easily attributed to sampling error) the Q-statistic and its corresponding p-value were calculated. If the p-value for Q was found to be less than our alpha of .05, then the individual effect sizes that make up the mean estimate were considered to be heterogeneous and not derived from a single population of offenders. Finally, using the method of moments (the DerSimonian and Laird method), we estimated tau^2 as an indicator of the amount of between-studies variance (Borenstein, Hedges, Higgins, & Rothstein, 2009; Hedges & Olkin, 1985; Lipsey & Wilson, 2001; Overton, 1998). All mean effect sizes, estimates of heterogeneity, and subsequent moderator analyses were computed in SPSS using macros available at http://mason.gmu.edu/~dwilsonb/ma.html.

Results

Table 1 summarizes all studies that met the eligibility criteria. Note that while the table displays information for 23 different samples, a total of 26 references were used to

Table 1. Study Characteristics and Alcohol Involvement Status among Homicide Offenders (full sample).

Study citation	Country of study	Data years	Number of homicide offenders[a]	Percent positive for alcohol	Percent reportedly intoxicated
James & Carcach (1997)	Australia	1989-1996	1,673	53.9	NA
Mouzos (2000)	Australia	1996-1999	945	47.2	NA
Dearden & Payne (2009)	Australia	2000-2006	1,565	46.6	NA
Makkai & Payne (2003)	Australia	2001	113	NA	44.0
Dearden & Jones (2008)	Australia	2006-2007	115	61.7	NA
Virueda & Payne (2010)	Australia	2007-2008	260	35.0	NA
Scott (1968)	England	Unknown	50	22.0	16.0
Shaw et al. (2006)	England & Wales	1996-1999	1,168	45.0	6.0
Virkkunen (1974)	Finland	1963-1968	114	66.4	NA
Dooley (1995)	Ireland	1972-1991	582	NA	46.5
Dooley (2001)	Ireland	1992-1996	278	NA	28.7
Pridemore (2006)[b]	Russia	1989-1991, 1998	246	66.0	40.0
Gillies (1976)	Scotland	1953-1974	400	NA	55.8
Lindquist (1986)	Sweden	1970-1980	64	NA	66.0
Wolfgang (1958)	United States	1948-1952	621	55.2	NA
Shupe (1954)	United States	1951-1953	30	83.0	67.0
State of California (1960)	United States	1959	74	70.3	37.8
Criminal Justice Commission (1967)	United States	1960-1964	624	36.2	NA
Varano & Cancino (2001)	United States	1975-1995	8,028	14.7	NA
Holcomb & Anderson (1983)	United States	1976-1979	110	40.0	NA
Wieczorek, Welte, & Abel (1990)	United States	1978-1979	1,844	49.0	NA
Spunt et al. (1994)[c]	United States	1984	268	33.0	32.0
Bureau of Justice Statistics (1999)	United States	1996	9,093	48.6	NA

Note: NA: Not available.

[a]We chose to report the number of offenders for whom alcohol (or intoxication) status could be determined. In many cases, this is the total number of homicide offenders mentioned in the sample description from the original study. In other cases, and because of missing or unknown alcohol involvement, this number is a subset of the total number of offenders reported in the original study.

[b]Information about this sample also came from Pridemore & Eckhart (2008).

[c]Information about this sample also came from Spunt et al., (1995) and Fendrich et al. (1995).

construct the data set. Information on the sample in one study (Pridemore, 2006) was supplemented with information about the same sample appearing in a separate publication (Pridemore & Eckhart, 2008). Similarly, information about a sample in a different study (Spunt, Goldstein, Brownstein, Fendrich, & Langley, 1994) was supplemented with information taken from two separate publications (Fendrich, Mackesy-Amiti, Goldstein, Spunt, & Brownstein, 1995; Spunt, Brownstein, Goldstein, Fendrich, & Liberty, 1995). For simplicity, we refer to each of the 23 data sets in Table 1 with a single reference, thereby constituting 23 studies in our analysis.[4]

Nine of the studies examined homicide offenders from the United States, whereas the remaining 14 examined offenders in Australia, England, England and Wales (collectively), Finland, Ireland, Scotland, Sweden, and Russia. All studies were published between the years of 1954 and 2010, and describe homicides that occurred between 1948 and 2008. Note that the studies conducted within the United States generally

describe more dated homicides, with just two studies including alcohol information on homicide offenders occurring after 1990 (compared to 10 of the non-U.S. studies). Across all studies, information about alcohol usage was available for 28,265 homicide offenders. Eighteen studies reported the percentage of offenders who were positive for alcohol at the time of their crimes, while 11 reported the percent intoxicated. Most studies determined the offender's alcohol status through a review of criminal justice records such as police case files or court documents, although some determined alcohol status through psychological evaluations, face-to-face interviews with researchers, self-reported questionnaires, urinalysis results, or some combination of these methods.

Where possible, we coded information on offender demographics and offense-characteristics. For example, some studies reported the alcohol status for their entire sample as well as for men and women. When available, we coded the results for male and female offenders separately and included the results in a moderator analysis of gender. Some studies were based exclusively on male offenders, and as such, those alcohol percentages were included in the estimates for the total effect size estimate as well as for the effect size for men. Similarly, although most studies focused exclusively on an all-adult sample of offenders, some also included alcohol estimates for a subsample of juvenile offenders (i.e., those less than 18 years old). We therefore calculated effect size estimates for adult and juvenile offenders separately for purposes of comparison. A few studies reported alcohol rates for different racial/ethnic categories, allowing us to compute and compare effect sizes across these groups as well.[5] Unfortunately, little information was provided regarding the prevalence of alcohol use across differentially motivated homicides (e.g., gang-related, drug-related, domestic, revenge, etc.). More information was available regarding the type of weapon used in the event (e.g., guns, knives, blunt objects); however, given the functional similarity of certain weapons (i.e., both knives and blunt objects require attacking the victim with direct physical force during hand-to-hand combat), we eventually dichotomized this variable to distinguish between homicides committed with a firearm from those that were committed with some other weapon.

Table 2 summarizes the random-effects mean percentage of homicide offenders who were positive for alcohol (48%) and intoxicated (37%) at the time of their crimes. (Note that in the table, *k* refers to the number of effect sizes upon which each mean is based). For both estimates, the distributions of the individual effect sizes are heterogeneous, and the *Q* estimates are statistically significant (*p*s = .00). Figures 1 and 2 present forest plots for the individual study findings that comprise the mean estimates for those who are positive for alcohol and who are intoxicated, respectively.

In order to better understand the variability in the effect size estimates across studies, we conducted a series of moderator analyses that examined various characteristics of the study (source of alcohol information, country of study, data years), of the offender (age, sex, race), and of the homicide event itself (weapon used). To maximize the number of studies included in these analyses, we focused our attention on the percentage of offenders who were positive for alcohol, as opposed to only those who were identified as being intoxicated. Note that all between-groups *Q* estimates in these analyses represent a difference-of-means test that is analogous to a one-way *F* test.

Table 2. Random-Effects Mean Percentage of Homicide Offenders Positive for Alcohol or Reportedly Intoxicated, With Moderator Analyses.

		95% CI					
Analysis	Mean %	Lower %	Upper %	*Q*	*p*	τ^2	*k*
Alcohol estimate							
Positive for alcohol	48	39	57	2595.03	.00	0.58	18
Reportedly intoxicated	37	25	51	457.67	.00	0.95	11
Positive for alcohol, by study characteristic							
Source of alcohol information				5.41	.37	0.70	
Criminal justice records	47	35	60				10
Psychiatric evaluations	33	13	63				2
Researcher interviews	43	23	67				3
Self-report questionnaires	70	30	93				1
Urine tests	83	42	97				1
Combination of methods	40	11	78				1
Country of study				0.12	.73	0.62	
United States	46	33	59				9
Other	49	37	62				9
Positive for alcohol, by offender characteristic							
Age status				0.02	.87	0.09	
Juvenile (<18 years)	44	31	58				3
Adult (18+ years)	43	38	47				16
Sex				0.66	.42	0.14	
Male	49	42	55				11
Female	44	37	52				7
Race/ethnicity				0.59	.90	0.33	
Black	50	31	69				2
White	48	34	62				4
Hispanic	54	27	79				1
Other	55	41	69				4
Positive for alcohol, by offense characteristic							
Weapon used				7.72	.01	0.12	
Gun	34	22	49				2
Other	59	50	67				5

Study Characteristics

As seen in Table 2, the proportion of homicide offenders who were positive for alcohol at the time of the offense did not vary significantly across the sources of alcohol information ($Q_{\text{between}} = 5.41$, $df = 5$, $p = .37$), although the tau^2 estimate was relatively large. Alcohol involvement rates derived from criminal justice records, psychiatric interviews, face-to-face interviews with researchers, and some "combination" of methods were all fairly similar and ranged between 33% and 47%. The estimates based on

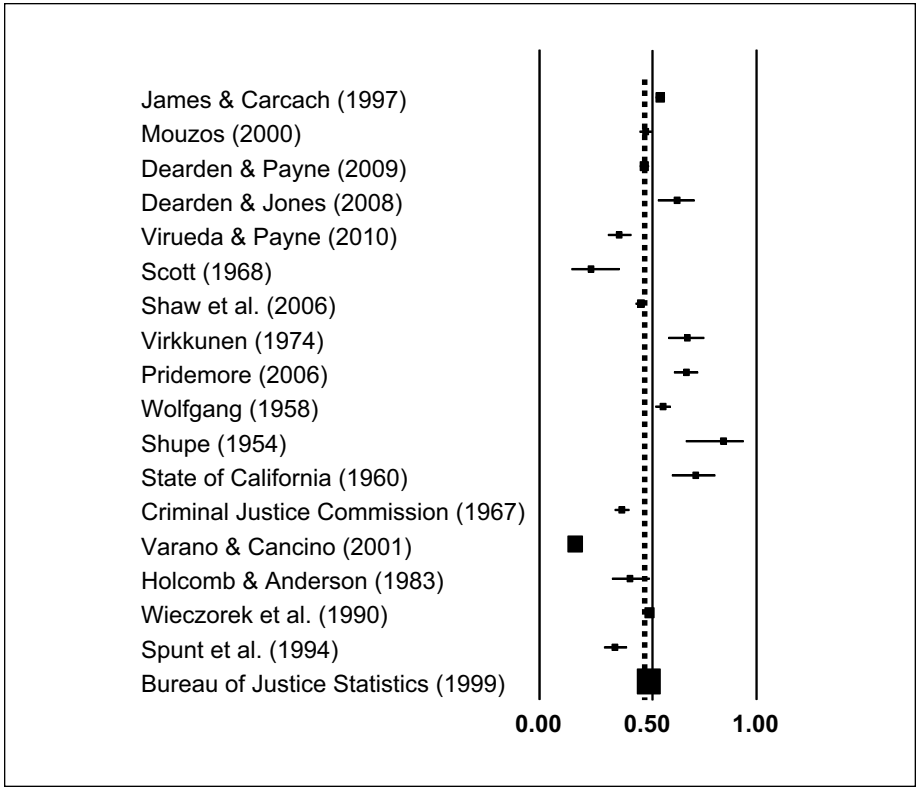

Figure 1. Forrest plot: Proportion of homicide offenders who were positive for alcohol, with random-effects mean (broken vertical line).

self-reported surveys (70%) and from urine tests (83%) tended to be higher; however, as these are each based on a single study they are potentially volatile estimates and should be interpreted with caution. Thus, on the whole, our analysis uncovered no meaningful differences in the alcohol status estimates across data source.

As also seen in Table 2, the percentage of homicide offenders positive for alcohol in the United States (46%) and in other countries (49%) was statistically equivalent ($Q_{\text{between}} = 0.12$, $df = 1$, $p = .73$), although the tau^2 was again relatively large.[6] In a separate analysis (not shown in the table), we examined the mean effect size estimates among just those studies conducted in the United States to see if there was significant variability across studies of homicides offenders in U.S. cities versus U.S. states versus the nation as a whole. The mean estimates were 43%, 47%, and 49%, respectively, with no significant variability across the groups ($Q_{\text{between}} = 0.16$, $df = 2$, $p = .92$).

Given that alcohol consumption patterns have changed over time (Greenfield & Kerr, 2009; Nephew, Williams, Stinson, Nguyen, & Dufour, 2000), we next sought to determine if the estimates of homicide offenders who were positive for alcohol have

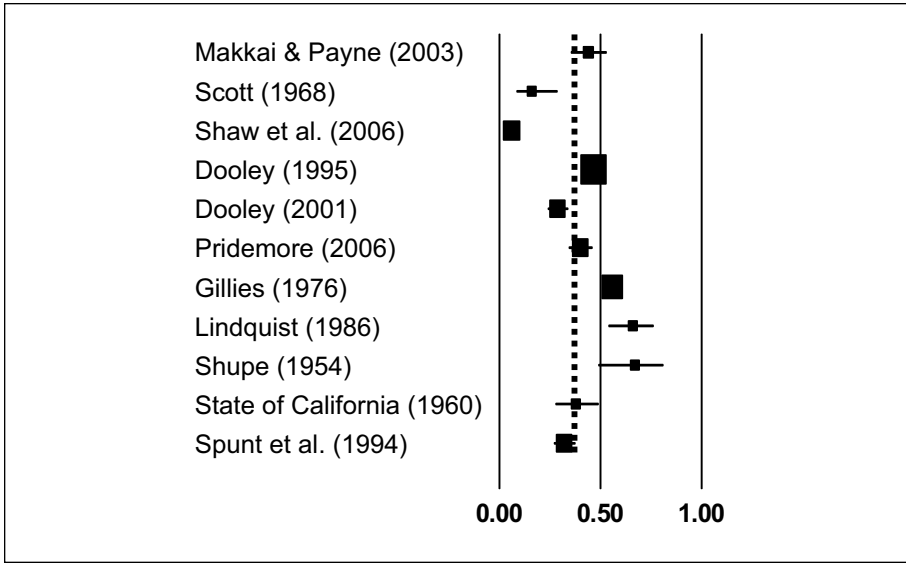

Figure 2. Forrest plot: Proportion of homicide offenders who were reportedly intoxicated, with random-effects mean (broken vertical line).

changed over time as well. We therefore regressed the effect size (i.e., the logit of the proportion of offenders who were positive for alcohol) on the study's data year (i.e., the year in which the homicides occurred). In the event that a study examined multiple years of homicide data (e.g., from 1960-1964), we determined the midpoint of this range and used that value as the data year. The results of this analysis showed a weak and nonsignificant relationship between the data year and the proportion of offenders positive for alcohol ($b = 0.0006$, $p = .17$, $R^2 = 0.12$).

This null effect is perhaps not surprising given (a) the null findings reported earlier across country of study, and (b) the relationship between country and data year (i.e., virtually all U.S. studies have older data years). We therefore reran the regression separately for studies based in the United States and those in non-U.S. countries. Collectively across all studies conducted outside the United States, the coefficient for data year was positive and significant ($b = 0.006$, $p = .0036$, $R^2 = 0.35$). We are cautious, however, about attaching any interpretation to this finding given the differences in alcohol access, drinking contexts, and social culture that may exist across the eight countries upon which this analysis is based (and with some countries represented by a single study). In short, we cannot say with confidence that the percentage of homicide offenders who are positive for alcohol has changed over time outside the United States.

The interpretation of findings within the United States is less problematic. Although there is variability in the scope of the studies conducted within the United States (i.e., some used city-based samples whereas others used state- or national-samples of

offenders), recall that we found no differences in prevalence estimates across these different sampling units. When we therefore enter all U.S. studies in a regression, we find that there is a negative and statistically significant relationship between data year and the percentage of offenders under the influence ($b = –0.05$, $p = .01$, $R^2 = 0.56$). Mindful of the fact that the U.S. studies are based on homicides occurring between 1951 and 1996 (noninclusively; see Table 1), this linear trend in the data suggests the percentage of homicide offenders in the United States who are positive for alcohol at the time of the offense has declined—at least during this window of time.

Offender Characteristics

As seen in Table 2, the percentages of juvenile and adult offenders who were positive for alcohol at the time of their homicides were nearly identical and did not differ significantly ($Q_{between} = 0.02$, $df = 1$, $p = .87$; $\tau^2 = 0.09$). A similar set of results was also found when examining differences in the estimates across gender ($Q_{between} = 0.66$, $df = 1$, $p = .42$; $\tau^2 = 0.14$) and race/ethnicity ($Q_{between} = 0.59$, $df = 3$, $p = .90$; $\tau^2 = 0.33$). Therefore, we find no evidence to suggest that these basic demographic characteristics moderate the likelihood of committing a homicide while positive for alcohol.

Offense Characteristic

Table 2 also summarizes the percentage positive for alcohol among offenders committing homicide with firearms (34%) and for those using some other type of weapon (59%). Despite the limitations associated with having a relatively small number of cases in the analysis ($n = 7$), this difference in percentages was statistically significant ($Q_{between} = 7.72$, $df = 1$, $p = .01$; $\tau^2 = 0.12$). In a separate analysis (not shown in the table), we also examined the mean effect size estimates across just the nonfirearm weapons, crudely trichotomized into categories of blunt objects ($k = 1$), sharp objects ($k = 2$), and "other" weapons ($k = 2$). These estimates were 52%, 60%, and 62%, respectively, with no significant variability across the groups ($Q_{between} = 0.23$, $df = 2$, $p = .89$). Thus, compared to homicides committed with a firearm, those committed with some other type of weapon were significantly more likely to involve an offender who had been drinking.

Discussion

Across 23 independent studies conducted in one of nine countries, an average of 48% of homicide offenders were reportedly positive for alcohol and an average of 37% were intoxicated at the time of the offense. These results are based primarily on secondary data derived from case study files and interviews with offenders. In contrast to studies of alcohol use among homicide victims (Kuhns et al., 2011), only one of the studies identified in this review of homicide offenders included a biological sample (i.e., Shupe, 1954). The paucity of such tests is understandable given the difficulties associated with the timely collection of biological samples from offenders.

Nevertheless, it seems apparent that we have a relatively limited understanding of the relationship between alcohol consumption and homicide offending beyond what we can learn from offender's self-report or from observations as recorded in case records.

Despite these limitations, the meta-analytic estimates for homicide offenders are remarkably similar to the meta-analysis results of alcohol use among homicide victims (48% of victims tested positive and 33-35% met the threshold for intoxication; Kuhns et al., 2011). Considered collectively, and recognizing that the samples of offenders and victims were not selected from the same locations, during the same timeframes, or within the same criminal events, these two sets of meta-analytic findings suggest that a substantial proportion of homicides will likely include alcohol as a contributing factor to either the offender's proclivity or the victim's vulnerability. This finding, in and of itself, is not particularly surprising because the link between alcohol and violence has been well documented (e.g., Galanter, 1997; Martin, 1993; Parker & McCaffree, 2013; Pernanen, 1991). However, if an average of nearly half of all homicide offenders and half of all homicide victims are under the influence of alcohol, and about a third of each group is intoxicated, it seems clear that managing alcohol consumption rates is an important public health step toward reducing lethal violence outcomes.

Importantly, these estimates were relatively stable across different age, gender, and racial groups and across varied geographic locations. The lack of demographic differences in this offender-based study differs from the results in the victim-based analysis (Kuhns et al., 2011), which found age, gender, and racial variations in alcohol use (although alcohol use did not vary across geographic location). Thus, these sets of results suggest that across a broad range of countries and cultures, alcohol's role among homicide offenders may be more uniform than its role among homicide victims. Stated differently, some victims may be more at risk for homicide depending on alcohol consumption, but there may be different risk factors for offenders. Therefore, while policies that focus on managing alcohol consumption and reducing alcohol-related violence may be equally effective (or ineffective) across heterogeneous communities and populations, such policies may need to be more specifically developed for and targeted to potential victims.

A second important finding, also consistent with meta-analytic results of homicide victims (Kuhns et al., 2011), is that the mean percentage of homicide offenders who were positive for alcohol during the offense has declined over time in the United States—at least, across the data years examined here (roughly the 1950s through the mid-1990s). This finding is also consistent with data from the National Crime Victimization Survey, which show that while the number of violent victimizations in the United States declined by 23% in the 1990s, the number of violent victimizations committed by an offender *thought to be under the influence of alcohol* declined by 34% (Greenfeld & Henneberg, 2001).[7]

To the extent that alcohol use is but one of many potential factors contributing to homicide (which are beyond the scope of the current study), the decline in prevalence rates may indicate that one or more American alcohol control policies are impacting lethal violence outcomes. We draw the reader's attention to one policy in particular, which increased the minimum drinking age from 18 to 21 years in 1984. Evidence

suggests that the risk for homicide increases 5% during the age that access to alcohol becomes legal (e.g., in the United States, age 18 prior to 1984, but age 21 thereafter), and that delaying the legal drinking age can reduce the risk of a wide variety of forms of violent deaths, including homicide (Jones, Pieper, & Robertson, 1992; Parker & Rebhun, 1995). Thus, the change in the U.S. legal drinking age in 1984 may have paid additional dividends beyond the substantial reductions in alcohol use and alcohol-related traffic accidents (Wagenaar & Toomey, 2002), and may also help explain the decline in the prevalence of alcohol-involved homicide offenders we find extending into the mid-1990s. To be clear, the 1984 National Minimum Drinking Age Act that increased the legal drinking age in the United States to 21 years old was not specifically focused on violence reduction or homicide offenders (or victims for that matter). However, we are suggesting, as others have observed, that delaying the onset of the legal drinking age can contribute to a reduction in the role of alcohol as a contributor to homicides, in general (Jones et al., 1992; Parker & Rebhun, 1995). To the extent that offender alcohol use directly or indirectly contributes to homicide, limiting or delaying access to alcohol, particularly to those in crime-prone ages, should assist our efforts in reducing violence. Recognizing that our study cannot speak directly to the relationship of specific alcohol control policies and violent crime (but see Parker & Cartmill [1998] for an excellent discussion), we believe the pattern of meta-analytic results reported here and elsewhere (Kuhns et al., 2011) warrant future research on the impact of minimum drinking ages on alcohol-related homicides.

There is another possible—although, less encouraging—explanation for the apparent decline in the prevalence of alcohol-involved homicide offenders in the United States. Recall that the current study finds that alcohol involvement is *less common* among homicide offenders who use firearms as opposed to those who use some other type of weapon. Thus, if the percentage of homicides involving firearms were to increase over time in the United States (and, again, assuming these offenders are less likely to have been drinking), then the percentage of homicide offenders who are positive for alcohol will, by definition, decrease. Interestingly, findings from the U.S. Bureau of Justice Statistics indicate that the number of handgun-related homicides (which less often involved offender alcohol use in this meta-analysis) rose from approximately 10,000 in the early1980s to approximately 14,000 in the early 1990s; yet, during this same time the number of homicides committed with knives, blunt objects, or other weapons (which significantly more often involved offender alcohol use in this meta-analysis) experienced a decline or remained relatively stable (Cooper & Smith, 2011). Therefore, the decline in alcohol-involved homicide offenders in the United States uncovered in this study may, in part, be related to a shift in the type of weapon used (i.e., more homicides are being committed with firearms). Firearms-related homicides are also directly linked to some homicide motives (e.g., gang-related homicides are more likely to involve firearms; Maxson, Gordon, & Klein, 1985, see also National Institute of Justice, 2013). Thus, additional research is needed to better understand the various reasons why alcohol-involved offending in the United States may have declined between the 1950s and the 1990s, and whether this trend has continued to present day.

In conclusion, the fact that approximately half of all homicide offenders are positive for alcohol sheds additional light on the alcohol/violence nexus in general, and on potential strategies for reducing homicide more specifically. The findings reported here are generally consistent with prior research that finds alcohol consumption rates (in the aggregate) are positively related to homicide rates in the United States (Parker et al., 2011), in Europe (Rossow, 2001), and in Russia (Pridemore, 2002). Additional research has shown that reductions in alcohol consumption rates lead to reductions in homicide (and suicide) rates (Darke, 2010; Lester, 1995; Parker & Cartmill, 1998; Rossow, 2001) across varied geographical settings. Therefore, communities that suffer from high homicide rates may very well benefit from implementing more restrictive alcohol control policies.

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Notes

1. In order to determine if alcohol is present in an offender's bloodstream, researchers must typically rely on the offender's self-reported account of his/her alcohol consumption immediately before or during the commission of the homicide. Thus, the phrase "positive for alcohol" used here describes those offenders who were thought to have alcohol present in their system at the time of their crimes, and does not necessarily reflect positive findings from biological tests.
2. Seven studies reported case file information (from criminal justice records of psychological evaluations) that allowed us to infer that the offender was intoxicated at the time of the homicide. Two studies determined intoxication status through face-to-face interviews with offenders, while one used a self-report questionnaire and the other used urinalysis results. Thus, as with our estimates of alcohol status, our estimates of intoxication status are based on different sources of information. As discussed below, we did not find significant differences in the effect size estimates across these various sources.
3. Publication bias within meta-analyses can assume many forms (Rothstein, Sutton, & Borenstein, 2005). Editorial bias is a concern for journal articles, but our review included studies that were published in other forms or outlets. Significance testing bias and outcome bias, perhaps the most prominent forms of bias, were not primary concerns in this meta-analysis because our review focused on alcohol prevalence studies that do not require significance testing (the studies simply reported percentages of homicide offenders who used alcohol and/or were intoxicated at the time of the offense). Our restriction to studies available in the English language may introduce a language bias (but see Song, Khan, Dinnes, & Sutton, 2002). At the same time, studies from non-English speaking countries were included in this review (as long as the research was published in English). Availability, cost, and familiarity bias were lesser concerns given our free university access to sophisticated

search engines and academic and governmental materials from a wide variety of disciplines including medicine, psychology, and criminal justice. In short, we find little reason to believe that any of these potential biases had a meaningful effect on the results presented here.

4. Most of the studies of Australian homicides in Table 1 are based on data collected through the Australian Institute of Criminology's (AIC) National Homicide Monitoring Program (NHMP). We found several AIC reports that summarize NHMP data for a given period of time (e.g., from 2001-2002). If a report spanning a longer period of time (e.g., from 2001-2006; see Dearden & Payne, 2009) was found to subsume the NHMP data published in an earlier report, then we included only the former in our review in order to maintain independence across studies.
5. Studies of Australian homicide offenders often reported the alcohol status for Indigenous/Aboriginal/Torres Strait Islander offenders versus all non-Indigenous offenders (e.g., Dearden & Payne, 2009). The latter group consisted of offenders who were Caucasian, Asian, or some "other" race (James & Carcach, 1997). Given our inability to isolate the alcohol status of these specific types of non-Indigenous races, we excluded the Australian studies in our analysis of race/ethnicity.
6. We also trichotomized the country of study in order to distinguish between those studies conducted in the United States, Australia, and all "other" countries. We again found no significant differences ($Q_{between} = 0.11$, $df = 2$, $p = .94$).
7. Of course, while the NCVS can speak to trends in violent offending, it cannot address homicide specifically.

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Author Biographies

Joseph B. Kuhns is an Associate Professor in the Department of Criminal Justice and Criminology at UNC Charlotte. His research focuses on community policing in small cities/towns, managing the growth of police agencies, and drug and violent crime relationships.

M. Lyn Exum is an Associate Professor in the Department of Criminal Justice and Criminology at UNC Charlotte. His research interests include criminal decision making and research methodology.

Tammatha A. Clodfelter is an Assistant Professor at Appalachian State University. Her current research interests include policing, interpersonal violence, and the relationship between drug and alcohol consumption and crime.

Martha Cecilia Bottia is an Assistant Research Professor of Sociology at the University of North Carolina at Charlotte. Her current research focuses on the role of structural characteristics of K-12 schools on the decision of students to major and graduate from a STEM major. Her other research interests include illicit drugs and terrorist organizations.