Published in final edited form as:
Psychol Bull. 2009 January ; 135(1): 142–156. doi:10.1037/a0014414.

Binge Drinking in Young Adults: Data, Definitions, and Determinants

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Abstract

Binge drinking is an increasingly important topic in alcohol research, but the field lacks empirical cohesion and definitional precision. The present review summarizes findings and viewpoints from the scientific binge-drinking literature. Epidemiological studies quantify the seriousness of alcohol-related problems arising from binge drinking, with a growing incidence reported in college-age men over the last 2 years. Experimental studies have found neurocognitive deficits for frontal lobe processing and working memory operations in binge-drinking compared with nonbinge alcohol drinkers. The findings are organized with the goals of providing a useful binge-drinking definition in the context of the empirical results. Theoretical implications are discussed on how binge drinking may alter neurophysiological and neurocognitive function.

Keywords

binge drinking; epidemiology; neuropsychological; review; definitions

Alcohol consumption in humans is the third leading preventable cause of death in the United States (McGinnis & Foege, 1993). A common abuse pattern called *binge drinking* contributes to a substantial portion of alcohol-related deaths (Chikritzhs, Jonas, Stockwell, Heale, & Dietze, 2001). This type of drinking also is associated with alcohol poisoning, unintentional injuries, suicide, hypertension, pancreatitis, sexually transmitted diseases, and meningitis, among other disorders. As binge drinking is relatively common, it underlies many negative social costs, including interpersonal violence, drunk driving, and lost economic productivity, as reported by the National Institute on Alcohol Abuse and Alcoholism (NIAAA, 2000). These statistics have attracted increased attention from a variety of perspectives.

The term “binge” originated as a clinical description of alcoholics and was defined by periods of heavy drinking followed by abstinence (Tomsovic, 1974). The word is distinct from the expression “binge drinking” that, since its conception, has engendered a wide array of definitional elements. This definitional difficulty originates from two different but related uses of the phrase: (1) epidemiological studies that emphasize isolated excessive drinking episodes, and (2) experimental studies that evaluate behavioral drinking patterns (Lange & Voas, 2000a). The present review was undertaken to bridge these approaches and to provide a comprehensive, integrative, and useful portrait of the binge-drinking literature with a focus on

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young adult humans. We obtained studies through literature searches using “binge drinking,” “alcohol binging,” and “college drinking.” Ancillary terms, such as light or social drinking and alcohol dependence, were included when they occurred within the binge framework (Boyd, McCabe, & Morales, 2005). The goals were to characterize the primary data and definitional attributes of binge drinking as delineated by current scientific findings.

Table 1 summarizes the binge-drinking studies identified. Although the conceptual and empirical views of an operational definition have been slow to coalesce, technical agreement about binge drinking has evolved appreciably over the last 10 years. Specific reports are used to illustrate how the definition, its rationale, and utility have developed. The approach considers both quantity and frequency of consumption as defining characteristics of binge drinking. The review is organized into three sections: (1) Issues underlying the concept of binge drinking are outlined; (2) the relationship of alcohol consumption to binge drinking is highlighted; (3) binge drinking and its cognitive, physiological, and withdrawal effects are examined, with the influence of alcoholism, family history for alcoholism, and other determinants sketched. In the Discussion section, we review the implications of the findings and suggest future research directions.

Definitional Background

Quantity

An initial view defined binge drinking as at least five alcoholic drinks consumed during the same session (Cahalan, Cisin, & Crossley, 1969). However, the comprehensive College Alcohol Study (CAS) conducted by the Harvard School of Public Health characterized binge drinking as five drinks for men and four drinks for women on a single occasion within the past 2 weeks (Wechsler, Davenport, Dowdall, Moeykens, & Castillo, 1994). The adjustment to the four-drink cutoff for women was based on their lower rate of gastric metabolism for alcohol, which leads to higher blood alcohol levels compared with men for the same quantity (Wechsler, Dowdall, Davenport, & Rimm, 1995). The 5/4 definition is consistent with findings that after consumption of this amount or more, individuals are at greater risk for exhibiting serious alcohol-related problems (e.g., vandalism, fights, injuries, drunk driving, trouble with police, etc.) and subsequent negative health, social, economic, or legal consequences (Wechsler, 2000).

Despite the intended practicality of the CAS and other large scale survey definitions, characterizing binge drinking using only a “single occasion” within a specified time-frame may conflate the estimates of binge drinkers as defined by a pattern of behavior (Naimi et al., 2003; Substance Abuse and Mental Health Services Administration [SAMHSA], 2007; Wechsler et al., 1994), as both drinking quantity and frequency have been shown to be important indictors of risky drinking in college students (Presley & Pimentel, 2006). Additional issues include how a single “drink” is defined, consumption amount, and alcohol tolerance contribute to individual inebriation levels (Jaccard & Turrisi, 1987).

One attempt to quantify behavioral drinking employed blood alcohol concentration (BAC) level, such that a 0.08 gram percent—now the legal intoxication level in all 50 states (Alcohol Policy Information System, 2007)—for a given occasion indicated binge-drinking patterns (Lange & Voas, 2000b). Another approach developed a Binge-Drinking Score from three questions of the Alcohol Use Questionnaire (Mehrabian & Russell, 1978; Townshend & Duka, 2002). This method used quantifiable assessments of drinks per hour, times drunk within the last 6 months, and percentage of time being intoxicated when drinking to calculate a summary score unrelated to the weekly consumption of alcohol (Townshend & Duka, 2005).

A standardized conceptual definition of binge drinking was proposed by the NIAAA in 2004:

> A "binge" is a pattern of drinking alcohol that brings BAC to 0.08 gram percent or above. For the typical adult, this pattern corresponds to consuming five or more drinks (male), or four or more drinks (female), in about two hours. (p. 3)

A standard drink equals 0.5 oz of alcohol as is found in one 12-oz beer, one 5-oz glass of wine, or one 1.5-oz shot of distilled spirits (NIAAA, 2004). This definition of binge drinking is similar to many used in epidemiological studies, which employ quantity (BAC), consumption amounts, and episode duration. The definition does not specify, however, the time period or number of binging occurrences that would describe a long-term binge-drinking practice. Thus, NIAAA's definition characterizes single binge episodes but does not capture the consumption pattern associated with serious health and social consequences.

Time-Frame

The inclusion of a past time-frame to quantify frequency of binging episodes is necessary to differentiate "binge drinking" from "alcoholism" or "alcohol dependence." This temporal aspect of a binge-drinking pattern has been variably defined as the *past week* (Kokavec & Crowe, 1999), *past 2 weeks* (Wechsler et al., 1994), past *30 days/month* (Okoro et al., 2004; SAMHSA, 2007; Zeigler et al., 2005), *past 6 months* (Hartley, Elsabagh, & File, 2004; Townshend & Duka, 2002, 2005; Weissenborn & Duka, 2003), and *past year* (Cranford, McCabe, & Boyd, 2006). These different time-frames emphasize various aspects of binge-drinking patterns, but their use inhibits direct comparison among findings.

The most informative time-frame appears to be within the past 6 months, as it is an optimal period to link alcohol consumption and alcohol-related problems (Hartley et al., 2004; Townshend & Duka, 2002, 2005; Weissenborn & Duka, 2003). Longitudinal studies of binge drinking have established that college students inconsistently report heavy episodic drinking across time (Schulenberg, O'Malley, Bachman, Wadsworth, & Johnston, 1996; Weingardt et al., 1998), so that a 2-week time-frame would underestimate binging prevalence (Vik, Tate, & Carrello, 2000). A recent study found that nearly one third of those classified as nonbinge drinkers (<5/4 drinks) during a 2-week time period in the middle of the month were classified as either binge drinkers (≥5/4 drinks, 1 or 2 times during the past 2 weeks) or frequent binge drinkers (≥5/4 drinks, ≥3 times in past 2 weeks) during the first 2 weeks of the month (LaBrie, Pedersen, & Tawalbeh, 2007). Use of a 2-week time period, therefore, would yield approximately 30% of heavy binge drinkers being excluded. A *past 6 months* time-frame for college samples captures the vacation time of the academic calendar during which students would be more apt to binge drink. Although longer time frames have yet to be analyzed, the ability to recall consumption amounts and frequencies accurately (e.g., recall bias) would seem to diminish with extended time frames. The goal in selecting an optimal time frame associated with a binge-drinking pattern is to optimize the accuracy of self-reported drinking amounts, while also capturing an accurate representation of this problematic drinking pattern. Further, employing a *multiple binging occurrences* evaluation strengthens the definition as these attributes together integrate the quantifiable dimensions of binge drinking.

Epidemiology

The age of onset of regular (> once a month) drinking has been reported to be "15.2 ± 1.2 years old ($M \pm SD$) for high-risk children and 16.5 ± 1.2 years old for low-risk children" on the basis of a sample of 125 children (Hill, Shen, Lowers, & Locke, 2000, p. 269). Of the total 10.8 million underage Americans (12–20 years) who reported consuming alcohol in the past 30 days, 7.2 million (or 19%) were binge drinkers (≥5 drinks on the same occasion on ≥1 day in past 30 days) as defined by National Survey on Drug Use and Health (SAMHSA, 2007). Early onset of binge drinking or exposure to binging has been linked to the increased risk of binging in adulthood (Wechsler, Dowdall, Davenport, & Castillo, 1995; Weitzman, Nelson, &

Wechsler, 2003). Other factors that predict binging include the following: never being married, having a grade point average of B or less, and placing little importance on religion.

The CAS study found that for a sample of 140 colleges nationwide, 44% of the responding students were binge (≥5/4 successive drinks) drinkers (Wechsler et al., 1994). The Behavioral Risk Factor Surveillance System (BRFSS) study assessed adults who were 18 years of age or greater through a random-digit telephone survey across the United States between 1993 and 2001 (Naimi et al., 2003). The number of binge episodes (≥5 alcoholic beverages in one sitting) among adults in the United States increased from about 1.2 billion to 1.5 billion. The younger adults in this sample (18–25 years) evinced the highest rate of binge-drinking episodes in the year 2001, whereas individuals older than 55 years had the lowest rate of binge-drinking episodes (Naimi et al., 2003). Differences in the prevalence estimates (CAS vs. BRFSS) may be due to different populations, with the CAS targeting college students and the BRFSS targeting the general community.

Most epidemiological reports indicate that men account for the majority of binge drinkers (Cranford et al., 2006; Wechsler et al., 1994; Wechsler, Dowdall, Davenport, & Castillo, 1995). The CAS study found that approximately 50% of the male and 39% of the female students were binge drinkers, with the BRFSS study concluding that men accounted for 81% of all binge-drinking episodes (Naimi et al., 2003). Furthermore, bingers in the BRFSS study were less likely to report any college education compared with nonbingers, although the opposite outcome also has been reported (Dawson, Grant, Stinson, & Chou, 2004; Slutske, 2005).

Racial differences were reported. Being White accounted for 78% of all binge-drinking episodes, and Hispanics demonstrated the highest rate of binge-drinking episodes per person for most of the years examined. African Americans constituted the lowest binge-drinking racial group, with fewer than five episodes per person per year (Naimi et al., 2003). Another large scale survey (N = 4,580) found a 33.2% prevalence estimate for binging (≥5/4 drinks in a row during past 2 weeks) for Asians compared with a 60.7% prevalence estimate for Whites (Cranford et al., 2006). The high frequency of a “flushing response” after alcohol ingestion has been theorized to account for the lower binging rates in Asians. The aldehyde dehydrogenase gene (ALDH2, Chromosome 12) that is prevalent in Asian populations fosters severe and predominately negative reactions to a moderate dose of alcohol compared with a heterozygous or individual without the allele (Cook et al., 2005).

Alcohol Consumption

Alcohol's effect on individuals stems from a variety of cognitive, biological, and social factors. The propensity to binge drink may arise from a combination of these factors, which could contribute to the underlying “cause” of binge drinking. Studies of these factors typically employ drinking definitions that are specialized for the particular variable or measure used, so that result comparisons need to be made from this perspective. However, these variables taken in the context of their roles as mediators and moderators of alcohol consumption are potentially important indices of future binge drinking and are reviewed here to provide appropriate background for their effects.

Alcohol Expectancies

Alcohol impairs the functioning of a variety of domains, including memory, judgment, and behavior (Nelson et al., 1998; Sayette, 1999). It diminishes eye movements (Blekher et al., 2002; Holdstock & de Wit, 1999; Moser, Heide, & Kömpf, 1998), short-term memory (Chait & Perry, 1994; Heishman, Arasteh, & Stitzer, 1997; Mattila et al., 1996), and motor performance (Fogarty & Vogel-Sprott, 2002). These direct influences of alcohol consumption,

however, vary in magnitude as a function of amount ingested and individual differences in alcohol expectancies. A study of 302 undergraduates found that mood was affected by alcohol intake: Men more often reported social-situational enhancements (e.g., meeting people), whereas women often reported physical (e.g., falling asleep) effects (Goldstein, Wall, McKee, & Hinson, 2004). Alcohol-related memories can account for as much as 50% of the variance in predicting concurrent and prospective drinking (Wiers et al., 2002), and expectances can predict as well as demographic variables, such as social and problem drinking (Christiansen & Goldman, 1983).

Expectancy effects can be manipulated: Drinkers instructed to “try and stay sober” demonstrated superior hand coordination and recall memory performance compared with those not so motivated (Young & Pihl, 1980). Lower numbers of positive alcohol expectancies and reduced consumption have been linked to fewer binge-drinking episodes, whereas negative expectancies were not (Blume, Schmaling, & Marlatt, 2003). Alcohol expectancies and drinking refusal self-efficacy have been proposed to be significant predictors of drinking styles. Binge drinkers (≥6/4 drinks per drinking period) were characterized as either having positive (are able to refuse drinks easily) or negative (unable to stop drinking) drinking refusal self-efficacy. A model derived from these observations “predicts that social and binge drinkers can be discriminated on the basis of their alcohol expectancies, while binge drinkers and alcoholics can be discriminated on the basis of drinking refusal self-efficacy” (Oei & Morawska, 2004, p. 173). Thus, beliefs about alcohol effects appear to contribute to the experience of drinking.

Perception of Drunkenness

Inebriation is another important factor related to binge drinking, and it is often reported as the basis for binging (Wechsler et al., 1994). However, alcohol drinkers misbelieve that standard mixed drinks are more potent than standard servings of wine or beer. These individuals also believe that each additional drink they consumed had a decreasing impact on BAC (Jaccard & Turrisi, 1987). Sober adolescents were asked to estimate their perceived level of simulated drunkenness as quantified by whether their BAC was under or over the legal limit while they were exposed to external cues that systematically described drinking scenarios (Turrisi & Wiersma, 1999). The young people underestimated their “perceived” level of inebriation during 19% of the experimental scenarios, suggesting that their judgment was affected by the cues.

Induced public self-awareness (stimulated by exposure to mirrors and a camera) was hypothesized to increase salience of the situational behavioral standard (i.e., sober comportment), which increased motivation toward effortful performance. Shorter response time was obtained for the self-aware compared with the control group on a task that required the participant to identify correct and misspelled words (Ross & Pihl, 1988). This expectancy effect also was observed for at-risk college drinkers trained to reduce consumption by demonstrating that the students experienced enhanced mood and conviviality when they were induced to think they were consuming alcohol but were not (Fromme, Marlatt, Baer, & Kivlahan, 1994). As greater positive expectancies have been associated with binge drinking, expectancy differences appear to be a strong influence on alcohol's individual effects (Blume et al., 2003).

Tolerance to Alcohol

Individual responsivity or “tolerance” to alcohol also is important and has been assessed by the BAC curve changes with consumption (Fillmore & Vogel-Sprott, 1998). The *rising limb theory* supposes that heavy drinkers are more sensitive than light drinkers to the subjective positive euphorigenic effects during the early portion of the BAC curve but less sensitive to the sedative-like effects during both the rising and declining phases (Holdstock, King, & de

Wit, 2000). Young adult heavy binge drinkers (≥5/4 drinks on one occasion at least once a week) were found to produce this biphasic response on a battery of behavioral scales. An initial pattern of positive reinforcement and absence of negative effects was obtained for the binge compared with nonbinge drinkers (<5/4 drinks per occasion), who did not show a biphasic alcohol response and reported heightened sedation throughout both limbs of the BAC curve (King, Houle, de Wit, Holdstock, & Schuster, 2002). Although the biphasic response may have been produced by the binge pattern of consumption, the authors speculated that the differential sensitivity between binge and nonbinge drinkers may have contributed to the enhanced risk for the development of alcohol-use disorders and the acquisition of binge-drinking patterns.

Social Issues

Drinking in a group leads to the experience of greater euphoria than drinking the same quantity alone (Pliner & Cappell, 1974), and drinking in a social setting facilitates more consumption than solitary drinking (Storm & Cutler, 1981). A survey of 409 college students found that a drinking event with many people intoxicated and having school friends present were factors predictive of binge drinking with five or more drinks (Clapp & Shillington, 2001). Students often seek out environments that facilitate binge drinking (Clapp et al., 2003; Lange & Voas, 2000b). Indeed, peer relationships can be a risk factor for increased alcohol consumption, as collegiate living arrangements—especially fraternities and sororities—are a significant correlate of binge drinking. Other factors include living with a roommate, stressing the importance of parties, and having five or more close student friends (Wechsler, Dowdall, Davenport, & Castillo, 1995).

Binge drinking can affect quality of life in terms of general health. After adjustment for age, frequent binge drinkers (≥5 drinks on one occasion > 3 times in last 30 days) compared with infrequent binge drinkers (≥5 drinks on one occasion < 3 times in the last 30 days) were more likely than nonbinge drinkers to report fair or poor health and experience more sick days. These findings appear to reflect the generally negative consequences of alcohol abuse but at an earlier stage in poor health development (Okoro et al., 2004).

In contrast, the benefits of light and moderate alcohol consumption have been well documented for stress reduction, mood enhancement, reduced depression symptoms, improved functioning in the elderly (Baum-Baicker, 1985; Pernanen, 1991), as well as for protection against coronary artery disease (Sacco et al., 1999). These issues often are reported as reasons for consuming alcohol. Only when the perceived drinking effects are detached from personal experience are harmful effects of drinking cited as “objective” assessments (Peele & Brodsky, 2000). The term “moderate” drinking, therefore, should not be confused with “binge drinking,” as the latter implies irregular intake and withdrawal from large quantities of alcohol and often leads to different outcomes than the positive ones associated with moderate drinking.

Binge Drinking

The current binge-drinking literature varies widely on the nature of the individual studies and definitions used to categorize alcohol consumption. Interpreting the results of these studies, therefore, requires a perspective that includes comparative awareness of sample characteristics, binge-drinking definition, and the control/nonbinge-drinking group inclusion criteria. Important too is to maintain the distinction between human and animal studies, as the former are typically much less specific than the later with respect to the neurophysiological underpinnings of binge-drinking effects. However, an overview of the general findings helps provide a fundamental grounding in what is known about binge-drinking outcomes at different levels of effect.

Cognitive Effects

Binge-drinking studies that measure cognitive function have found frontal lobe and working memory deficits, although an empirical definition of binging has not been used consistently. Heavy social drinkers, defined to include those who engaged in binge-drinking episodes, demonstrated delayed auditory and verbal memory deficits that were related to task difficulty. These deficits were not found for the light social drinkers. The findings implied that "frequent intake of large amounts of alcohol in any one sitting (i.e., 'binge' drinking) may place individuals at an increased risk for suffering alcohol-related cognitive impairment" (Nichols & Martin, 1997, p. 455). However, the conflation of participant drinking levels with descriptive labels colors statements about binge-drinking effects, thereby making comparisons unclear.

In Table 2, we summarize neurocognitive studies of binge-drinking studies using standard neuropsychological tests. The Binge-Drinking Score method was employed in several of these to define research participant drinking groups (Townshend & Duka, 2005). Binge drinkers compared with nonalcohol drinkers evinced cognitive impairments in the Paced Auditory Serial Addition Test, executive planning function, and episodic memory tasks—findings similar to frontal function deficits found in Korsakoff alcoholics (Hartley et al., 2004). Another report found that binge drinkers relative to nonbinging drinkers produced errors in a spatial working memory and pattern recognition tasks (Weissenborn & Duka, 2003). Furthermore, female compared with male binge drinkers were more impaired on these paradigms and unable to inhibit their response to an alerting stimulus in a vigilance task. Thus, binge drinking may be associated with deficits in frontal inhibitory control (Townshend & Duka, 2005).

It is important in this context to distinguish binge drinking from alcohol dependence. For example, alcohol dependent individuals who did binge drink—that is, regularly consumed more than 10 successive drinks—were compared with an alcohol dependent group who did not binge drink. No differences in performance were found for visuo-motor speed, visuo-spatial organization/planning, learning, proactive/retroactive interference, and item retrieval efficiency (Kokavec & Crowe, 1999). Comparable executive functioning results were obtained for both groups, and binge drinkers performed better than nonbinge drinkers on memory tasks. Although binge drinking was associated with impaired performance on immediate and delayed recall of verbal and visual information (Wechsler Memory Scale–Revised), retrieval ability was similar so that semantic organizational ability may be superior in binge compared with nonbinge drinkers. The pattern of binge versus nonbinge findings is likely affected by the inclusion of alcohol dependence criteria and the disproportionate number of drinks required in the binge definition.

Physiological Factors

The consensus from animal model studies is that "binge" effects require a long-term (multiple days) exposure to alcohol (e.g., Greiffenstein, Mathis, Stouwe, & Molina, 2007; Moore et al., 2007; Wezeman, Juknelis, Himes, & Callaci, 2007)—a viewpoint similar to the clinical alcoholic binge but quite different from the most common interpretations of binge drinking discussed above. Moreover, animal studies of alcohol binge exposure have led to the conclusion that such ethanol intake can lead to neurodegeneration in corticolimbic areas linked to learning and spatial memory (Aggleton, Hunt, & Rawlins, 1986; Haberly, 1998; Jarrard, 1993), such as the olfactory bulb, piriform cortex, perirhinal cortex, entorhinal cortex, and the hippocampal dentate gyrus (Collins, Corso, & Neafsey, 1996; Collins, Zou, & Neafsey, 1998; Corso, Mostafa, Collins, & Neafsey, 1998; Crews, Braun, Switzer, & Knapp, 2000; Zou, Martinez, Neafsey, & Collins, 1996). Researchers have found extensive neurodegeneration of the entorhinal cortex in rats after 2 days of "binge" alcohol exposure using stomach catheters that produced learning deficits (Obernier, White, Swartzwelder, & Crews, 2002). The vulnerability of this region after a single "binge" episode (i.e., 2 days of alcohol exposure) implies that long-

term ethanol exposure may not produce the neurotoxicity commonly associated with heavy alcohol use. However, the duration of alcohol exposure time that leads to neurotoxicity is still unknown.

The Iowa Gambling Task (IGT) has been used to measure decision making skills in a sample of human binge (≥5 drinks on one occasion, more than one time in the past 30 days) and nonbinge alcohol drinkers. Diminished IGT performance was found in chronic high-binge drinkers (binge drinking 2 or more times a week 95% of the time) compared with low-binge drinkers (binge drinking 2 or more times a week 3% of the time). Heavy drinkers and possible alcohol dependent/abusers were included, and it was acknowledged that the findings did not permit differentiation of whether the quantity/frequency of drinking or the pattern of drinking was the cause of the diminished IGT performance (Goudriaan, Grekin, & Sher, 2007).

Magnetic resonance imaging measures of regional white and gray matter regional volumes were used to quantify N-acetylaspartate (NAA) concentrations—a metabolite biomarker of neural integrity. For bingers (> 100/80 alcohol drinks/month on <21 days in the past 3 years) compared with nonbingers, decreased NAA concentrations were associated with increased metabolism and frontal white matter loss, with higher parietal gray matter NAA. Consumption amount for heavy drinkers (> 100/80 drinks per month over past 3 years, which included binge drinkers) was correlated with lower executive functioning and working memory test scores. In addition, their relative frontal NAA loss was associated with impaired executive functioning and processing speed. Taken together, the results imply that these bingers have less parietal neuron damage than continual heavy drinkers (Meyerhoff et al., 2004), and that binge drinking may result in relatively specific neural deficits that differ from those associated with continual drinking levels.

Withdrawal Effects

A related issue is whether binge drinking causes permanent cognitive deficits. Previous studies of alcohol dependent adolescents suggest that frequent heavy drinking produces long-term memory deficits (Tapert et al., 2001). A study of nondependent binge drinkers examined hangover effects from binge drinking (≥5 drinks on a single occasion), which were assessed with memory tasks to determine whether cognitive deficits were related to the hangover episode or long-term neural damage. Encoding and consolidation processes were impaired, but delayed recall was intact, suggesting that retrieval processes were affected only during the hangover (Verster, van Duin, Volkerts, Schreuder, & Verbaten, 2003). The implications of these findings may be best described by the Federal Aviation Administration's Pilot Safety Guidelines on alcohol and flying: “eight hours from bottle to throttle” (Salazar & Antuñano, 2008, p. 3). Moreover, hours from last drink appear unrelated to cognitive performance (Townshend & Duka, 2005), and neuropsychological impairment from heavy social drinking over 6 months has not been observed (Alterman & Hall, 1989). Thus, the relationship between heavy alcohol consumption and subsequent cognitive capability is unclear.

Another interpretation suggests that increased binging causes a greater number of withdrawals, which produce the long-term deficits (Glenn, Parsons, Sinha, & Stevens, 1988; Parsons & Stevens, 1986; Stephens et al., 2005). The number of alcohol withdrawals has been linked to impairments of long-term nonverbal memory in adolescents and to poor memory in adult alcoholics (Glenn et al., 1988). Alcoholic patients with two or more medically supervised alcohol detoxifications demonstrated more frontal lobe cognitive dysfunction than patients with a single or no previous detoxification (Duka, Townshend, Collier, & Stephens, 2003).

Neural “kindling” has been proposed as the mechanism by which alcohol ingestion and subsequent withdrawal produce cognitive damage (Ballenger & Post, 1978). Repeated withdrawals are thought to generate an accumulative adaptive process that underlies the

"advancing pathogenesis associated with the development of alcoholism [such that] continued alcohol abuse could be related to an avoidance of distress from worsening acute withdrawal symptoms induced by a kindling process that advances the course of alcoholism" (Breese, Overstreet, & Knapp, 2005, pp. 371–372). This view is consistent with an increased risk for brain damage from binge drinking and subsequent withdrawal (Hunt, 1993; Wechsler et al., 1994).

The occurrence of "blackouts" in which complex activities are performed with no recollection of the behavior available may be a related phenomenon and perhaps a biomarker for the mechanism of neurotoxicity observed in binge drinkers. Blackouts occur often in binge drinkers and could originate from reduced activity of N-methyl-D-aspartate (NMDA) receptors in the hippocampus, which would impair long-term potentiation (Izumi, Nagashima, Murayama, & Zorumski, 2005; for a review, see Allgaier, 2002). Excessive glucocorticoid release induced by the withdrawal stress could intensify the responses of already overactive NMDA receptors, thereby initiating blackouts (Hunt, 1993). Periods of binging followed by abstinence then trigger a neural cycle that leads to increased neurotoxicity of structures involved in learning and memory.

Alcoholism, Alcohol Dependence, and Other Determinants

Table 3 summarizes the definitions of alcohol abuse and dependence from the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; American Psychiatric Association, 1994). Inclusion of frank alcoholism in binge samples may result in biased drinking correlates stemming from the negative consequences of alcoholism as well as binging. Alcohol dependence also can alter binge-drinking outcomes. College students who are frequent heavy episodic drinkers (5/4 or more drinks on three or more occasions in the past 2 weeks) had 19 times greater odds of being classified with alcohol dependence and 13 times greater odds of being classified with alcohol abuse compared with nonheavy episodic drinkers. The occasional episodic drinkers (heavy drinking on one or two occasions during the past 2 weeks) were found to have 4 times greater odds of dependence or abuse compared with the nonheavy episodic drinkers (Knight et al., 2002). However, earlier reports suggest that the comorbidity of binge drinking (periodic heavy drinking followed by a period of abstinence), with alcohol addiction or dependence, is not clinically supported (Levy, 1988;Levy & Kunitz, 1974).

Parental history for alcoholism and binge drinking (≥5 drinks per occasion) in a sample of alcohol dependent individuals both have been found to influence short-term outcome of alcohol dependence (Hasin, Paykin, & Endicott, 2001). An additional factor is gender, because as many as 81% of all binge-drinking episodes are attributed to men (Naimi et al., 2003), but men also demonstrate increased frequency of alcohol dependence (Robin, Long, Rasmussen, Albaugh, & Goldman, 1998). These data suggest that the relationships among binge-drinking definitions, epidemiological findings, and alcohol-related diagnostic categories need additional refinement.

Family History

Presence of alcoholism in the family covaries with behavioral and neuroimaging measures of binge drinking (Ehlers et al., 2007; Kokavec & Crowe, 1999). Alcohol expectancies have been shown to be a genetically influenced characteristic having a heritability between 0.4 and 0.6 (Heath et al., 1999; Schuckit et al., 2001), with greater alcohol consumption in high-risk than in low-risk control families (Newlin & Thomson, 1990). After the consumption of the lower or higher ethanol dose (approximately three or five drinks, respectively), men with high risk for alcoholism reported significantly less intense feelings of intoxication compared with low-risk men (Ehlers & Schuckit, 1988; O'Malley & Maisto, 1988; Schuckit, 1980, 1984, 1988). As outlined above, individuals who are homozygous for the ALDH2 gene are less likely to

binge drink (Luczak, Wall, Shea, Byun, & Carr, 2001), which needs to be considered in such studies.

These associations have spurred the search for a binge-drinking gene. College students with the short version of the serotonin transporter gene (5-HTT) consumed more alcohol per occasion, more often drank expressly to become inebriated, and were more likely to engage in binge drinking than college students without the 5-HTT variant (Herman, Philbeck, Vasilopoulos, & Depetrillo, 2003). The 5-HTT gene is thought to be involved in serotonin reuptake, and the students who were homozygotic for the short version of 5-HTT were more likely to report troublesome drinking patterns. Students with at least one copy of the 5-HTT long variant gene consume fewer alcoholic drinks per episode but are equal in the number of episodes. Individuals who are homozygous for the short version are also at risk for higher levels of anxiety and depression and may use alcohol to reduce tension (Mazzanti et al., 1998).

Event-Related Potentials (ERPs)

ERPs are sensitive to the neural effects of alcohol intake (Porjesz & Begleiter, 1996). Several studies have reported decreases in ERP component (N1, MMN, P300) amplitudes with ethanol doses ranging from 0.50 g/kg to 0.85 g/kg (Campbell & Lowick, 1987; Grillon, Sinha, & O'Malley, 1995; Jääskeläinen et al., 1995, 1998; Rohrbaugh et al., 1987; Sommer, Leuthold, & Hermanutz, 1993). The P300 component reflects attention and memory operations engaged when stimulus change occurs (Polich, 2007). P300 variation with ethanol ingestion has been interpreted as demonstrating adverse effects on perceptual processing resources, a measure of central nervous system disinhibition, or frontal executive dysfunction (Begleiter & Porjesz, 1999; George, Potts, Kothman, Martin, & Mukundan, 2004; Kim, Kim, & Kwon, 2001).

ERPs also have been used to assess familial history as a neural signature or “marker” of alcoholism (Begleiter, Porjesz, Bihari, & Kissin, 1984; Hill et al., 1998; Hill & Steinhauer, 1993; O'Connor, Hesslebrock, Tasman, & DePalma, 1987; Porjesz & Begleiter, 1990). A meta-analysis of the early studies found that these effects were variable (Polich, Pollock, & Bloom, 1994), and that difficult visual discrimination tasks produced the strongest family history effects (e.g., Carlson, Iacono, & McGue, 2002; Iacono, Carlson, Malone, & McGue, 2002; Reese & Polich, 2003). These findings suggest that the P300 component in particular can index the effects of alcohol intake and may reflect the genetic background of alcoholism.

ERPs are just beginning to be used to assay binge drinking. A facial discrimination task yielded P300 amplitudes that were smaller for adolescents exposed to alcohol (i.e., ≥5 drinks per occasion), with a positive family history for alcohol dependence acting as a significant covariate. Further, P300 latency was decreased for alcohol and drug-exposed young adults in the absence of an alcohol challenge relative to control participants (Ehlers et al., 2007). Recent ERP studies suggest that high-binge compared with low-binge college student groups can be differentiated with tasks requiring strong visual stimulus processing: P300 amplitude tends to be smaller for the high- compared with the low-binge groups, although the quantity and frequency of alcohol intake that produces these effects are still unclear (Courtney & Polich, 2008).

Discussion

The present review highlights issues that contribute to the definition of binge drinking, with the main variables centering on the quantity consumed and the time-frame of consumption. However, alcohol consumption effects are modulated by individual variation with respect to expectancy, how expectations influence the perception of inebriation, tolerance to alcohol ingestion, and the social environment. These factors contribute to the characterization of binge drinking in relation to its cognitive, physiological, and withdrawal effects. Moreover, the

relevant findings empirically differentiate binge drinking from clinical alcoholism by defining how these variables influence alcohol effects. Thus, the interactive milieu of alcohol's internal determinants is complex and surprisingly subtle, so that binging to some is not necessarily binging to others.

An Operational Definition

Epidemiological reports of binge drinking vary in definitional consistency, but for young adults they indicate a large prevalence and imply a clear burden of suffering. The individual and social costs associated with binge drinking—such as drunken driving, induced violence, and personal injury—are profound. The cognitive damage that may be inflicted by binge drinking appears to involve alteration in critical neural mechanisms. However, experimental binge-drinking studies vary in their definitional approaches so that the what, where, and when of the neurocognitive insult is uncertain. Functional magnetic resonance imaging and ERP methods are beginning to assay such outcomes, but these approaches require sustained definitional rigor to inform public health policies.

The current NIAAA (2004) definition has provided a structure for binge drinking, but scientific and clinical assessments would benefit from the formation of a definition that facilitates comparison among studies. Given the findings outlined above, this definition should encompass three factors: alcohol quantity consumed, time-frame of consumption, and time period of past binging episodes. A definition of binge drinking that integrates these issues is as follows: *A pattern of drinking alcohol that brings BAC to 0.08 gram percent or above (≥5/4 for men/women in 2 hr) on more than one occasion within the past 6 months*. This definition (1) is operational in structure, (2) delimits consumption amount and time-frame (taking into account gender), and (3) specifies a time period that encompasses individual variation.

Future Directions

The intriguing hints provided by initial genetic studies may ultimately identify the neural origins of propensity to binge drink, which likely reflect fundamental individual differences to alcohol intake and interact with the wider context of personality or psychiatric variables. Searching for the primary reasons why some young adults binge would foster genetic links between binge drinking and subsequent alcohol dependence. Characterizing the association between binge-drinking mechanisms and the development of alcoholism could reveal a means to pursue and evaluate treatment interventions before the addictive disease is fully developed.

Neurophysiological and neurocognitive assessments of binge drinking are demonstrating promise in specifying biological differences between bingers and controls. The biphasic alcohol response exhibited by young binge drinkers and the associated neuropsychological impairments found for frontal lobe processing provide clues to the origins of binge drinking. Preliminary findings suggest working memory deficits in binge drinkers, but whether these are long-term or abate after withdrawal is unknown. Although difficult to execute, longitudinal studies of adolescent binge drinking could establish whether and how future alcohol dependence and abuse originates from this pattern of alcohol consumption while controlling for family history. Addressing these issues with a quantifiable and consistent binge-drinking definition would encourage comparisons among studies and increase their societal impact.

Conclusion

Scientific understanding of how alcohol produces reactions that vary across individuals from pleasurable to deadly requires clear observation of the phenomena and definitional agreement about what is observed. The public health concerns about young adult binge drinking have helped to motivate refinement of its definition. The implications of the empirical framework outlined here can be used to evaluate the proposed quantities, time-frame, and consumption

frequencies as factors that may contribute to subsequent alcohol-related problems. The proposed binge-drinking definition should therefore help provide the operational utility that will facilitate inferences across studies.

Acknowledgments

This work was supported by National Institute on Alcohol Abuse and Alcoholism Grant AG10604. This article is 19458-MIND (Molecular and Integrative Neuroscience Department) from The Scripps Research Institute. We thank Shirley Y. Hill and Brian Lopez for very helpful comments on earlier versions of this article.

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Table 1

Summary of Binge Drinking Studies

Author (year)	Binge definition	Population	*N*	Age (years)	Method	Conclusion
Wechsler et al. (1994)	5/4+ drinks past 2 weeks	U.S. college students	F = 10,203 M = 7,389	N/A	Survey (CAS)	44% binge drinkers, 19% frequent binge drinkers, 47% frequent binge drinkers who had 5+ drinking-related problems.
Wechsler, Dowdall, Davenport, & Rimm (1995)	Developed the 5/4 measure	U.S. college students	*N* = 12,243	N/A	Survey (CAS)	Women who drink 4 drinks in a row have same likelihood of experiencing drinking-related problems as men who drink 5 drinks in a row.
Wechsler, Dowdall, Davenport, & Castillo (1995)	5/4+ drinks past 2 weeks	U.S. college students	F= 10,203 M = 7,389	N/A	Survey (CAS)	Prior high school binging; gender and race significant correlates; predictors = fraternity or sorority, party-centered life, and risky behaviors.
Kokavec & Crowe (1999)	10+ drinks <2 days/ week, irregular basis	Referred from alcohol-related brain injury support groups	F= 18 M = 82	25–68	Neuropsychological tests[a]	Semantic organizational ability poorer in constant drinkers versus bingers; executive performances on tasks are similar for both groups.
Turrisi & Wiersma (1999)	Being “drunk or very high from alcohol” in past 90 days	U.S. high schools	F= 159 M = 111	16–18	Scenarios describing drinking situations, questionnaires	**1** Family history positive for alcohol abuse (FH +) compared with family history negative for alcohol abuse (FH−) individuals were more likely to make judgmental errors and underestimate their drunkenness; errors in judgment were more pronounced with moderate to

Author (year)	Binge definition	Population	*N*	Age (years)	Method	Conclusion
						heavy alcohol consumption.
						2 The more FH + individuals tended to underestimate their drunkenness, the more likely they were to binge drink and drive after drinking.
Vik et al. (2000)	5/4 at one time	U.S. college students	F = 72 M = 40	17–43	Questionnaire	38.6% of those that binged during the past 3 months would not have been identified as bingers using a 2-week time period; age, church attendance, alcohol-related consequences, and age of first intoxication were the same for bingers identified from both time periods.
Hasin et al. (2001)	5+ drinks/occasion (all subjects met *DSM–IV* diagnostic criteria for alcohol dependence)	General community	F = 99 M = 70	M = 30.1	Interviews—random telephone	Positive relationship between family history and chronicity (stable relationship between familial/genetic background and alcohol dependence).
Lange & Voas (2000a)	0.08 + BAC	U.S. drinkers in Tijuana	F = 512 M = 547	18–33	Survey	5/4 measure too low for 0.08 BAC; should use 6/5 measure.
Wechsler & Nelson (2001)	5/4+ drinks past 2 weeks	N/A	N/A	N/A	Discussion of binge definition	5/4 measure extensively used and chosen because it provides a simple way to assess a drinking style that threatens the public's health.
King et al. (2002)	5/4+ drinks per occasion	General community	F = 8 M = 26	24–38	Questionnaires: alcohol/drug experimental: BAC, heart rate, Cortisol levels	Young adult binge drinkers show a biphasic BAC alcohol response compared with light drinkers.
Knight et al. (2002)	Binge/episodic: 5/4+ drinks per occasion	U.S. college students (CAS)	F = 8,610 M = 5,505	N/A (50.2% <21)	Survey	31% of students endorsed criteria for an alcohol

Author (year)	Binge definition	Population	*N*	Age (years)	Method	Conclusion
	Heavy episodic drinker = within past 2 weeks (occasional = 1/2 times, within 2 weeks; frequent = 3+ times within 2 weeks)					abuse diagnosis and 6% for a dependence diagnosis in past 12 months. Heavy episodic drinkers more likely to have an alcohol disorder. Frequent heavy episodic drinkers had 13 times greater odds for abuse and 19 times greater odds for dependence.
Weissenborn & Duka (2003)	Binge Drinking Score: Drinks per hour, times drunk within last 6 months, percentage of being drunk when drinking (4 × Item − 10 + 1 × Item − 11 + 0.2 × Item − 12 = drinking pattern)[b]	University of Sussex students	F = 49 M = 47	18–34	Neuropsychological tests[a]	Acute alcohol consumption impairs executive-type cognitive functions, and binge drinking may be associated with impaired cognitive functioning in a working memory and pattern recognition task.
Townshend & Duka (2002)	Binge drinking score	U.S. college students	F = 41 M = 14	18–28	Alcohol Use Questionnaire (AUQ), diary	High drinkers tended to underestimate their drinking behavior, whereas lower drinkers tended to overestimate. Found that relationship between binge scores, beverage specificity, and alcohol consumption supports idea that binge drinking criteria should be based on patterns of drinking rather than alcohol consumption.
Blume et al. (2003)	5/4 per drinking episode	General community	F = 32 M = 44	18–32	Questionnaires	Lower self-efficacy and greater positive alcohol expectancies predicted greater numbers of follow-up binge episodes. Greater positive alcohol expectancies predicted greater follow-up alcohol consumption.
Naimi et al. (2003)	5+ drinks on 1 occasion	General community	*N* = 724,479 F = 19% of bingers M = 81% of bingers	18+	Survey (BRFSS—random telephone)	Per capita binge-drinking episodes have increased since 1995; men account for 81% of binge drinking episodes, and 69% of binge-drinking episodes occurred among those 26 years of age or older.

Author (year)	Binge definition	Population	*N*	Age (years)	Method	Conclusion
Herman et al. (2003)	5/4+ drinks in a row	Caucasian undergraduate college students	F = 147 M = 57	M = 19	Survey, genetic analysis	Students who were homozygous for the 5-HTTLPR short allele were more likely to engage in binge drinking behavior, drank more alcohol per occasion, and reported drinking to get drunk more often, as compared with homozygous or heterozygous for the long allele.
Weitzman et al. (2003)	5/4+ consecutive drinks per occasion 1 + times in past 2 weeks	First-year college students	F = 1,263 M = 631	≤19, first-year	Survey (CAS data)	Students who reported that they were exposed to "wet" environments (prevalent and cheap alcohol availability) were more likely to engage in binge drinking than peers without exposure.
Verster et al. (2003)	5+ drinks on 1 occasion	General community (Dutch)	F = 24 M = 24	M =21.9	Neuropsychological tests[a]	Memory retrieval processes significantly impaired during an alcohol hangover after binge drinking.
Clapp et al. (2003)	5+ drinks in a row "Heavy episodic drinking"	U.S. college students	F = 885 M = 724	18–61	Survey, interviews (telephone)	Having many people, available illicit drugs, "BYOB" events, playing of drinking games predicts heavy episodic drinking; students actively seek out environments with characteristics that facilitate heavy drinking.
Hartley et al. (2004)	Binge Drinking Score	King's College students	F = 12 M = 15	18–23	Neuropsychological tests[a]	Binge drinkers compared with nondrinkers have lower trait anxiety and depression, as well as poorer performance for sustained attention, episodic memory, and planning ability.
NIAAA (2004)	0.08 + BAC (approximately = 5/4 + drinks in 2 hr)	N/A	N/A	N/A	Definition	Drink = a half ounce of alcohol.
Oei & Morawska (2004)	6/4+ drinks per drinking period in past 2 weeks	N/A	N/A	N/A	Theoretical article	Proposes a cognitive model for binge drinking that uses alcohol expectancies and drinking refusal self-efficacy to explain the acquisition

Author (year)	Binge definition	Population	*N*	Age (years)	Method	Conclusion
						and maintenance of binge drinking. Suggests that the combinations of alcohol and drinking refusal self-efficacy can explain the four drinking styles (social, binge, heavy, and alcoholic).
Meyerhoff et al. (2004)	100/80+ drinks per month (<21 days) in past 3 years (about = 5/4+ drinks in a row)	General community	F = 28 M = 70	M = 41	MRI and MRSI, neurocognitive tests	Binge drinking modulates brain metabolite abnormalities.
Okoro et al. (2004)	5+ drinks on 1 occasion in past month (frequent = 3+ days, infrequent = 1–2 days)	General community	F = 44,995 M = 54,788	Frequent bingers: M = 34.6; infreq bingers: M = 35.6; nonbingers: M = 45.7	Survey (BRFSS—random telephone), HRQOL	In 2001, 11% of current drinkers were frequent bingers (3+ in past month), and 14% were infrequent (1–2 in past month). Frequent bingers more likely than nonbingers to have ≥ 14 unhealthy days in past month (unhealthy = stress depression and emotional problems).
Zeigler et al. (2005)	5+ drinks on 1 occasion 1 + times in past 30 days (NHSDA definition)	Adolescents and college students	N/A	N/A	Review	Underage alcohol use is associated with brain damage and neurocognitive deficits.
Boyd et al. (2005)	N/A	College students	N/A	N/A	Review	Provides a review on the biology, identity, cognition, affiliation, and achievement of college student alcohol use.
Townshend & Duka (2005) [original authors of Binge Drinking Score]	Binge Drinking Score	General community (England)	F = 47 M = 50	18–30	Neuropsychological tests[a]	Cognitive performance and mood differences between binge and nonbinge drinkers: Movement time on 4 and 8 patterns, and MTS choice time on 8 pattern condition faster in bingers; female bingers worse on SWM and vigilance task than female nonbingers.
Miller et al. (2007)	5+ drinks in a row on 1 + of the 30 days preceding survey	U.S. high schools	F = 6,889 M =7,028	Grades 9–12	Survey (Youth Risk Behavior Surveillance. 2005)	Episodic heavy drinking higher among male (27.5%) than female (23.5%) students.

Author (year)	Binge definition	Population	*N*	Age (years)	Method	Conclusion
SAMHSA (2005)	5+ drinks on 1 occasion 1 + times in past 30 days	General community	*N* = 68,308	12 +	Survey (National Household Survey on Drug Abuse)	Other researchers derive actual findings from survey outcomes.
Cranford et al. (2006)	5/4+ drinks in past 12 months	U.S. college students	F = 2,304 M = 2,276	M = 19.9	Web survey	Past month time-frame lead to higher prevalence estimate than 2-week time-frame and was positively associated with negative consequences.
Wechsler & Nelson (2006)	5/4+ drinks in a row past 2 weeks	U.S. college students	N/A	N/A	Review binge cutoff points	5/4 measure valuable for assessing alcohol related harms in a college population.
Ehlers et al. (2007)	5+ drinks per occasion	Southwest California Indians	F = 73 M = 52	M = 19.9	ERP (facial discrimination task)	Adolescent exposure (alcohol and drugs) associated with latency decrease in P350 and amplitude decrease in P450.
Goudriaan et al. (2007)	5+ on one occasion 2 + times in the past 30 days	U.S. college students	F = 25 M = 25	M = 20	Iowa Gambling Task (IGT) questionnaires	Stable high binge drinking group made less advantageous choices on the IGT than the low binge drinking group. Impulsivity was not related to decision-making performance.
LaBrie et al. (2007)	5/4+ drinks in a row	U.S. college students	F = 182 M = 355	M = 18.6	Questionnaires	Nearly 1/3 of those classified as nonbinge drinkers in the second 2 weeks of the month were classified as either binge drinkers or frequent binge drinkers in the first 2 weeks of the month.
Read et al. (2008)	Standard binge = 5/4+ drinks per occasion; Heavy binge = 7/6+ drinks per occasion	U.S. college students	F = 184 M = 172	M = 19	Questionnaires	Binge drinkers differed from heavy binge drinkers on actual drunkenness (measured by estimated blood alcohol levels); only the heavy binge drinkers differed from the nonbinge drinkers on measures of total alcohol consequences and drinking frequency.

Note. F = female; M = male; N/A = not applicable; CAS = College Alcohol Study; BAC = blood alcohol concentration; BRFSS = Behavioral Risk Factor Surveillance System; BYOB = “bring your own booze”; NIAAA = National Institute on Alcohol Abuse and Alcoholism; MRI = magnetic resonance imaging; MRSI = magnetic resonance spectroscopic imaging; HRQOL = health-related quality of

life; NHSDA = National Household Survey on Drug Abuse; MTS = Matching to Sample Visual Search task; SWM = Spatial Working Memory task; SAMHSA = Substance Abuse and Mental Health Services Administration; ERP = event-related potential.

[a] See Table 2 for specific tests.

[b] Items refer to responses on the Alcohol Use Questionnaire (Mehrabian & Russell, 1978).

Table 2

Neuropsychological Studies of Binge Drinking

Study (year)	Study characteristics	Sample size F	Sample size M	Tasks	Outcome[a] (C = Control, B = Binge)
Kokavec & Crowe (1999)	Binge: Consume >10 drinks irregularly (<2 days per week)	12	38	WMS-R Logical Memory	C > B
	Nonbinge: Consume >10 alcoholic drinks/day	6	44	WMS-R Visual Reproduction	C = B
	Conditions: All subjects diagnosed alcohol dependent, detoxified, gender/family history matched, subjects abstained from alcohol >2 weeks prior, no smoking data on subjects			Rey Complex Figure	C = B
				Rey Auditory Verbal Learning Test	
				Trail Making Test	C = B
					C = B
Weissenborn & Duka (2003)	Binge: 0.8 g/kg of 90% volume/volume alcohol in 30 min	24	24	CANTAB–Tower of London	C > B
				CANTAB–Spatial Recognition Task	C > B
	Nonbinge: placebo group	25	22	CANTAB–Spatial Working Memory Task	C = B
	Conditions: Alcohol challenge, no alcohol abuse, subjects abstained from alcohol for >12 hr prior, smoking data unreported, gender taken into account			CANTAB–Pattern Recognition Task	C = B
Verster et al. (2003)	Binge: Moderate alcohol drinkers 8–9 standard drinks (1.4 g/kg body weight) in 30 min	Not reported		Word Learning Test–Learning	C > B
				Word Learning Test–Delayed Recall	C > B
	Nonbinge: Moderate alcohol drinkers in placebo			Word Learning Test–Immediate Recall	C = B
				Word Learning Test–Recognition Score	C = B
	Conditions: Alcohol challenge, no alcohol dependency, gender taken into account, <10 cigarettes per day allowed, no alcohol recency data			Word Learning Test–Recognition Time	C = B
				Mackworth Clock Test	C = B
Hartley et al. (2004)	Binge: Binge Drinking Score > cutoff, 10 units (8 g alcohol) per occasion, ≥5 M (≥4 F) per occasion	5	9	Hospital Anxiety and Depression Scale	C > B
				Line Drawing Recall Test	C > B
	Nonbinge: Nondrinkers	7	6	Paced Auditory Serial Addition Test	C > B
	Conditions: Some alcohol dependent subjects included, gender taken into account, alcohol intake in past week			CANTAB–Stockings of Cambridge Test	C > B

Study (year)	Study characteristics	Sample size F	Sample size M	Tasks	Outcome[a] (C = Control, B = Binge)
	measured, only minimal smoking in subjects (<5 per day)				
				CANTAB–IDED Test	C = B
				Bond and Lader Mood Rating Scale	C = B
				Spatial Working Memory Test	C = B
				Spatial Recognition Memory Test	C = B
				Pattern Recognition Memory Test	C = B
				Word List Recall Test	C = B
Townshend & Duka (2005)	Binge: Binge Drinking Score > cutoff	15	23	CANTAB–Matching-to-Sample Visual	C > B
	Nonbinge: Binge Drinking Score < cutoff	21	13	Search task: Choice Time (bingers shorter some conditions)	
	Conditions: No subjects with alcohol dependence, gender taken into account, subjects abstained from alcohol for >12 hr prior, smoking not controlled			CANTAB–Matching to Sample Visual	C > B
				Search task: Movement Time (binger shorter)	
				CANTAB-Spatial Working Memory task (F > M errors than nonbinger group)	C > B
				CANTAB-Matching to Sample Visual Search task: Errors Made	C = B
				Gordon Diagnostic System-Vigilance task	C = B
Goudriaan et al. (2007)	Binge: ≥5 drinks on one occasion, ≥2 times past 30 days	Not reported		Iowa Gambling Task–Effects of Punishment Frequency	C > B (heavy bingers only)
	Nonbinge: ≥5 drinks on one occasion, <2 times past 30 days				
	Conditions: Groups matched on age, gender, ethnicity, smoking				

Note. C = B: No significant difference between control and binge group. F = female; M = male; WMS-R = Wechsler Memory Scale-Revised; CANTAB = Cambridge Neuropsychological Test Automated Battery; IDED = Intra-Extra Dimensional Set Shift.

[a] C > B: Controls scored significantly higher than binge group.

Table 3
Definitions of Alcohol Abuse, Dependence, and Binge Drinking

Binge drinking	Alcohol abuse[a]	Dependence[a]
NIAAA (2004): Pattern of drinking alcohol that brings BAC to 0.08 gram % and above (approximately equivalent to the consumption of 5 drinks for men, 4 for women, in 2 hr)	A maladaptive pattern of substance use leading to clinically significant impairment or distress, as manifested by one (or more) of the following, occurring within a 12-month period:	A maladaptive pattern of substance use, leading to clinically significant impairment or distress, as manifested by three (or more) of the following, occurring at any time in the same 12-month period:
Proposed: Pattern of drinking alcohol that brings BAC to 0.08 gram % and above (approximately equivalent to the consumption of 5 drinks for males, 4 for females, in 2 hours), occurring more than once within a 6-month period.	• Recurrent substance use resulting in a failure to fulfill major role obligations at work, school, home (e.g., repeated absences or poor work performance related to substance use; substance-related absences, suspensions, or expulsions from school; neglect of children or household) • Recurrent substance use in situations in which it is physically hazardous (e.g., driving an automobile or operating a machine when impaired by substance use) • Recurrent substance-related legal problems (e.g., arrests for substance-related disorderly conduct) • Continued substance use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the substance (e.g., arguments with spouse about consequences of intoxication, physical fights) • The symptoms have never met the criteria for Substance Dependence for this class of substances.	• Tolerance, as defined by either of the following: – A need for markedly increased amounts of the substance to achieve intoxication or desired effect – Markedly diminished effect with continued use of the same amount of substance • Withdrawal, as manifested by either of the following: – The characteristic withdrawal syndrome for the substance – The same (or a closely related) substance is taken to relieve or avoid withdrawal symptoms • The substance is often taken in larger amounts or over a longer period than was intended • There is a persistent desire or unsuccessful efforts to cut down or control substance use • A great deal of time is spent in activities to obtain the substance, use the substance, or recover from its effects • Important social, occupational or recreational activities are given up or reduced because of substance use • The substance use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance (e.g., continued drinking despite recognition that an ulcer was made worse by alcohol consumption)

Note. NIAA = National Institute on Alcohol Abuse and Alcoholism; BAC = blood alcohol concentration.

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