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Alcohol and Sexual Health Behavior: "What We Know and How We Know It"

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Alcohol, despite salutary associations with sexuality, has been implicated in sexual health problems. This review examines the relationship between alcohol and outcomes related to sexual health. Methodological considerations limiting causal assertions permissible with nonexperimental data are discussed, as are advantages of experimental methods. Findings from laboratory experiments are reviewed evaluating causal effects of acute alcohol intoxication on a variety of outcomes, including sexual arousal, sexual desire, orgasm, and sexual risk behaviors related to sexually transmitted infections (STIs) and human immunodeficiency virus (HIV). Several variables exerting mediating and moderating influences are identified. It is concluded that acute alcohol intoxication is capable of exerting a causal impact on multiple constituent components of sexual responding related to sexual health. Both alcohol expectancy and alcohol myopia theories have been supported as explanations for these causal effects. Furthermore, for sexual risk behavior, noteworthy recent developments include research highlighting the importance of women's sexual victimization history and men's condom use resistance. Limitations and implications associated with this body of research are also discussed.

Engaging in sexual behavior while under the influence of alcohol is not uncommon. We all know stories about it involving others, if not ourselves. Certainly in the media (e.g., advertising, art, music, movies, television) sex after drinking is depicted as commonplace. Contemporary cultures are replete with illustrative associations and memes: beer goggles, wedding night champagne, wine and candlelight, tropical drinks and bikinis, fraternity keggers—the list goes on. While alcohol has many positive associations with sexuality, it unfortunately has also been implicated in problematic sexual health outcomes, including unintended pregnancy, sexual dysfunction, and sexual risk behaviors related to sexually transmitted infections (STIs) and human immunodeficiency virus (HIV). A collective impact of these stories and associations is the idea not only that sex after drinking is common but also that alcohol plays a causal role. Does it? That is, does drinking alcohol have a causal effect determining whether a person engages in sexual activity and consequently experiences problematic sexual health outcomes?

Alternatively, perhaps alcohol is merely an accomplice, serving as scapegoat to truer causes of sex after drinking, such as diminished parental supervision, youthful adventuresomeness, peer pressure, or permissive bar/party settings. Given the overwhelming abundance of correlational evidence—anecdotal, clinical, and research—and the seemingly ubiquitous claim that alcohol potentiates sexuality, the causality issue stands as the most insistent imperative question for advancing scientific understanding of alcohol's role in sexual health.

The current review addresses the following question: What is known scientifically about alcohol's causal impacts on responding that pertain to sexual health, particularly sexual functioning and STI/HIV-related sexual risk taking? Secondary questions follow: How do we know what is known? And how does it advance theory and understanding? In addressing these questions, I aim to highlight the role of methodology and specifically the role of experiments in evaluating alcohol's causal influence. Also, most reviews of alcohol and sexuality research have concentrated only on sexual risk taking because it has been the focus of

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considerable research attention (and funding)—understandably so, given the persistence of the HIV/AIDS crisis. Therefore, a second aim is to consider alcohol experiments that relate to sexual functioning as well as those on sexual risk taking. Third, findings that bear on the intersection of sexual arousal and risk taking are highlighted. Intuitively, sexual arousal and sexual risk taking seem intertwined. Sexual risk takes place in the heat of the moment, thereby privileging "hot" emotional determinants over "cold" cognitive ones. Yet many alcohol and sexual risk studies have stressed cognitive factors, such as expectancies and attitudes, at the expense of considering arousal and other emotional variables. Fourth, future research and intervention implications are considered. Finally, the current review affords an opportunity to encompass more recent experimental findings, such as those investigating condom use resistance and those addressing the role of sexual victimization history.

How We Know What We Know About Alcohol and Sexual Health

Misconceptions About Alcohol and Sex Experiments

As an experimentalist researching alcohol and sexuality, I find that casual conversations about this research—with scientists and nonscientists alike—often reveal misconceptions that warrant attention. For instance, some contend that experiments about alcohol and sex are unnecessary. The basis of this view is that because of the profusion of anecdotal evidence, we already know everything there is to know. Thus, according to this view, evidence from experiments is superfluous, only reaffirming what is obvious. From this view, the idea that alcohol leads to sex is so intuitive and axiomatic that experiments could not possibly shed further light on a self-evident truth; drinking so commonly precedes sex that something about alcohol must systematically influence and foster something about sexual responding. For example, in final judgment in a civil suit weighing the risks associated with serving alcohol in strip clubs, a judge ridiculed the value of experiments and lamented government expenditures wasted on funding them. The judge cited *The Pardoner's Tale*, circa 1380 (which, among many interpretations, portrays alcohol as leading men to evil), and opined, "What did he (the researcher) and taxpayers learn that Chaucer did not already know 600 years ago?"

In point of fact, we have learned plenty. For example, until 45 years ago, alcohol's acute effects on behavior were understood to be exclusively a product of alcohol's pharmacological properties. Alcohol and sex experiments conducted by G. Terence Wilson and colleagues in the 1970s played a significant role in furthering and solidifying a fundamental shift in our scientific understanding of how alcohol affects people. By combining earlier groundbreaking experimental methods in both alcohol research (e.g., balanced placebo design [BPD]) and sexology research (e.g., vaginal pulse plethysmography), Wilson's team elucidated the understanding that drinking alcohol evokes two conceptually separable components: a psychologically driven expectancy component and a pharmacologically driven bioactive component. Further, they established that those alcohol components can have independent and even opposing effects on conceptually separable components of sexual functioning: subjective arousal versus physiological arousal (Crowe & George, 1989; George & Stoner, 2000). The insights gleaned from those early alcohol and sex experiments have so stood the test of time that it is now passé among scientists to speak of alcohol's effects on human behavior—sexual behavior particularly—as being attributable to strictly biological, nonpsychological processes. Today, journal submissions and grant proposals addressing alcohol and sexuality are reflexively probed for consideration of expectancy effects and placebo designs.

Another common misconception is that sexual behavior in general and intoxicated sexual behavior in particular cannot be meaningfully studied in experiments. The basis for this view is that sexual behavior is so private that it is essentially unavailable for scientific scrutiny. Indeed, most human subject research ethics regulations would prohibit direct observation and measurement of any naturalistic postdrinking sexual behavior. Further, the alternative method of sole reliance on research participants' self-reports can be justifiably deemed suspect, owing to a variety of reporting biases and motives. For example, particpants may exaggerate drinking to deflect responsibility for sexual behavior they later regretted. However, despite genuine constraints on being able to measure sexual behavior directly, researchers have succeeded in developing and establishing an array of laboratory methods for measuring constituent elements of postdrinking sexual behavior, such as sexual arousal (e.g., George et al., 2011, 2006), condom use intention (e.g., George et al., 2014; Woolf-King, Maisto, Carey, & Vanable, 2010), and even sexual aggression (see reviews by Abbey & Wegner, 2015; Davis et al., 2014). Thus, the aforementioned misconceptions—dismissive about the idea of subjecting sex after drinking to scientific scrutiny—are handily countered by theory and method developments evident in the alcohol and sexuality experiments reported to date.

Why Focus on Experiments?

The research literature regarding alcohol and sex health is extensive, and relevant research activity has experienced a marked upsurge since the beginning of the HIV/AIDS crisis (Hendershot & George, 2007). The vast majority of studies linking alcohol with sexual health outcomes have been nonexperimental (Hendershot & George, 2007). This nonexperimental work was vital in establishing the reliability and generalizability of empirical associations between alcohol consumption and sexual behavior and in identifying individual difference variables and contextual factors that bear on those associations (Cooper, 2010). However, nonexperimental methods are, of course, ill-suited for addressing questions of causality. For example, global association studies do not even permit the simple determination that the drinking and sexual behavior occurred on the same day. Use of sophisticated

measurement and statistical approaches can enhance the ability to draw causal inferences in nonexperimental contexts. For example, event-level studies (e.g., Kilwein & Looby, 2018), daily diary assessments (e.g., Wilhite, Mallard, & Fromme, 2018), and ecological momentary assessments (e.g., Simons, Simons, Maisto, Hahn, & Walters, 2018) can offer nuanced assessments of covariation between drinking and behavior across discrete sexual events. The use of prospective designs and advanced statistical modeling approaches can further enhance the ability to infer causal associations through determinative temporal ordering of events across longitudinal time epochs (e.g., Bryan et al., 2016). Still, nonexperimental methods remain beset with inherent design and measurement limitations that are insurmountable for ascertaining true proximal causal linkages and, therefore, for illuminating what transpires in crucial moments culminating in intoxicated sexual behavior.

Scientifically speaking, sex after drinking causality can be truly determined only by using controlled experiments in which alcohol is systematically manipulated. In addition to the obvious need to control extraneous influences (e.g., predrinking food consumption) that might contaminate the findings, the sine qua non of an experiment is the random assignment of research participants to conditions—say, being given an alcoholic versus a nonalcoholic beverage. As a consequence, any differences between the two groups in, for example, sexual behavior can only be caused by alcohol; rival explanations can be ruled out. Experimental paradigms have other distinct advantages over nonexperimental methods. First, experiments permit investigation and evaluation of in-the-moment operation of proximal situational variables and processes that reflect or affect sexual behavior. Second, alcohol variables, such as amount consumed and achieved intoxication level, can be validated unequivocally. Third, experiments permit a definitive determination of the causal ordering of events. The latter advantage, specifying that the drinking precedes sexual risk behavior, is significant because it controls for the reverse-causation possibility. For example, in real life, a person may decide to pursue an opportunity for risky sexual behavior and proceed to drink alcohol in anticipation, creating the artifactual impression that alcohol played a causal role. Finally, because experiments allow simultaneous consideration of distal and proximal determinants, they foster theory refinement and advancement based on identification, isolation, and evaluation of mediating and moderating mechanisms. Given these important advantages, experimental studies provide the best source of evidence for evaluation of causal linkages between alcohol and sexual behavior.

As another essential consideration, it is important to distinguish between chronic versus acute alcohol use. Chronic alcohol consumption refers to the amount of alcohol consumed habitually over time and is operationalized based on research participants' retrospective reporting of drinking frequency, drinking quantity, and drinking consequences. Acute use refers to the amount of alcohol consumed and the level of intoxication attained in a particular drinking episode and the resultant effects in that same episode. Acute use is operationalized in experiments by having research participants enter a controlled laboratory setting and ingest beverages containing predetermined dosages of alcohol, designed to result in precisely targeted blood alcohol concentrations (BACs). Coverage in this review focuses primarily, but not exclusively, on acute use, which has been the predominant focus of alcohol–sexuality experiments.

What We Know About Alcohol and Sexual Functioning

The available findings from experiments indicate that acute alcohol intoxication can in fact exert a short-term causal impact on aspects of sexual functioning (see reviews by George & Gilmore, 2013; George & Stoner, 2000), and much of the ongoing experimentation has been oriented toward explaining the nature of these effects. In this section I review literature on alcohol's effects—primarily acute intoxication effects—on processes and outcomes related to three aspects of sexual functioning, including sexual arousal, sexual desire, and orgasm. The experimentation on arousal constitutes separate bodies of literature examining men and women.

Alcohol and Men's Sexual Arousal

Regarding men's chronic alcohol consumption, it has been fairly well established that chronic high alcohol consumption indicative of alcohol dependence is linked with impaired erectile functioning and associated sexual dysfunctions in men, such as erectile disorder, hypoactive sexual desire, and either premature or delayed ejaculation (e.g., O'Farrell, Choquette, Cutter, & Birchler, 1997; Ponizovsky, 2008). Among samples of men seeking treatment for erectile disorder, moderate to high alcohol use was associated with worse sexual function (e.g., Boddi et al., 2010; Dachille et al., 2008). Perhaps because of such findings obtained with alcoholic and sexually dysfunctional men. and because of findings associating acute alcohol intoxication with attenuated erectile response (e.g., see reviews by Crowe & George, 1989; George & Stoner, 2000), it was presumed that chronic consumption of low and moderate amounts of alcohol also had deleterious effects on erectile functioning for all men. This presumption was in spite of survey findings and ample anecdotal evidence indicating that modest alcohol consumption was associated with enhanced sexual activity (e.g., Christensen, Grønbaek, Pedersen, Graugaard, & Frisch, 2011). However, two review papers evaluating numerous population-based studies examining the effects of chronic alcohol consumption on erectile functioning have indicated that consuming low to moderate amounts of alcohol actually offered some protection against experiencing erectile dysfunction (Cheng, Ng, Chen, & Ko, 2007; Chew, Bremner, Stuckey, Earle, & Jamrozik, 2009). This conclusion suggests that chronic drinking becomes a problem for erectile responding only at high levels of chronic consumption. Overall, such evidence has ushered a shift in our understanding of chronic alcohol consumption and erectile functioning, from appraising all consumption as deleterious to recognizing

nondeleterious effects, and perhaps even protective effects, for low to moderate consumption (e.g., see Furukawa et al., 2016).

Acute Alcohol Attenuation of Erectile Responding. There has been a similar shift in understanding acute alcohol consumption effects on erectile functioning. While it once seemed conclusive that moderate to high doses of alcohol administered acutely attenuated men's erectile responding, recent research has illuminated more qualifying parameters on this so-called attenuation effect.

More than two dozen laboratory experiments have been conducted examining the effects of acute alcohol consumption on erectile responding. Evidence of an attenuation effect, in which alcohol reduces erection magnitude, was initially established based on findings published by three investigative teams, coincidentally in the same year (Briddell & Wilson, 1976; Farkas & Rosen, 1976; Rubin & Henson, 1976). In these pioneering experiments, young men were administered alcoholic or nonalcoholic drinks and presented with explicit sexual stimuli while erectile responding was assessed using a strain gauge to measure erection magnitude. The findings revealed that, except at very low dosages, alcohol attenuated erectile response. Intoxicated men showed less erection magnitude than sober men. Another early study showed that alcohol also increased the amount of time it took to achieve orgasm (Malatesta, Pollack, Wilbanks, & Adams, 1979). After this pioneering work, alcohol-induced attenuation of erectile responding was corroborated in subsequent experiments within the next decade (Wilson, Lawson, & Abrams, 1978; Wilson, Niaura, & Adler, 1985; Wormith, Bradford, Pawlak, Borzecki, & Zohar, 1988) and became axiomatic.

Two aspects of this attenuation effect were noteworthy. First, it cohered with what were understood at the time to be the attenuating effects of chronic alcohol consumption. Thus, the scientific conclusion seemed unambiguous: Both chronic and acute (except for very low dosages) drinking undermined erectile performance. This conclusion, of course, contradicted the lore about aphrodisiac properties and about alcohol disinhibiting men's sexual responding. Second, in light of then-expanding work on alcohol expectancy effects, the attenuation effect seemed to contribute to a narrative that resolved the old Shakespearean paradox about alcohol and sex: "Lechery, sir, it [drink] provokes, and unprovokes: it provokes the desire, but it takes away the performance" (Macbeth, 2.3.32). Specifically, it seemed that alcohol's salutary effects ("it provokes") were attributable to its psychological expectancy properties, whereas its deleterious effects ("it unprovokes") were attributable to its pharmacological properties.

The role of alcohol expectancies on men's postdrinking erectile response was first identified with experiments (e.g., Wilson & Lawson, 1976) using BPD (Marlatt & Rohsenow, 1980), a method for evaluating the independent and interactive influences of alcohol's physiological and psychological or expectancy effects (George, Gilmore, & Stappenbeck, 2012). Two properties of drinking are manipulated independently and systematically in BPD: expected alcohol content (expectancy) and actual alcohol content. Research participants either (1) expect alcohol/receive alcohol, (2) expect alcohol/receive none, (3) expect no alcohol/receive none, or (4) expect no alcohol/receive alcohol. Use of drink "look-/taste-alikes" and limiting dosage to a low level, such as .04%, made the deception necessitated in Groups 2 and 4 possible. Landmark experiments showed that men in the expect-alcohol conditions exhibited greater erectile response compared to men in expect-no-alcohol conditions, and actual alcohol content was inconsequential (Wilson & Lawson, 1976).

This expectancy enhancement effect seemed clear: Men who think they are drinking alcohol report and exhibit more erectile arousal, as measured by penile strain gauge, than men who do not. The effect seemed best attributed to the self-fulfilling prophecy explanation whereby men who believe that alcohol increases sexual arousal are more likely to experience heightened arousal after drinking than men who do not believe this (George, Stoner, Norris, Lopez, & Lehman, 2000). The expectancy enhancement effect was somewhat of a breakthrough in that the effect of "drink" on men's arousal could now be seen as consistent with the general sexual disinhibition outcomes conventionally understood as commonplace. However, because people drink real drinks in the real world outside of the laboratory, there was an explanatory problem: Pharmacologically driven attenuation remained unreconciled with anecdotal lore and empirical trends attesting to postdrinking sexual enhancement. This led to reexamination of the attenuation effect.

Critics noted that the attenuation effect was not universal (e.g., George & Stoner, 2000; Langevin et al., 1985), and it emerged that attenuation effects were occurring in less than half of the relevant experiments. Also, in a sleep study evaluating the highest BAC investigated to date (.15%), alcohol attenuation of erectile arousal was not evident among men during sleep (Morlet et al., 1990). Furthermore, methodological problems that may have contributed to attenuation effects were identified (George, Norris, & Schacht, 2003), including (1) inadequate control for alcohol's biphasic limb effects, whereby differential effects at equivalent BAC levels are discernible based on whether the BAC is ascending or descending; (2) reliance on within-subject designs, in which participants might (2a) intuit the hypothesis and respond accordingly across the alcohol versus no-alcohol conditions and (2b) habituate to repeated exposure to the erotic stimuli.

In subsequent experiments published 30 years after the original attenuation findings, these methodological problems were corrected. In a sample of young community men, George et al. (2006)—using between-subject methods and controlling for biphasic effects and nonrepeating erotic stimuli—evaluated the effects of three BAC levels (.00%, .08%, and .10%) and of instructions to either maximize or suppress arousal while viewing an erotic film. These instructions were adapted from nonalcohol experiments on arousal examining voluntary control of genital arousal (Beck & Baldwin, 1994;

Hatch, 1981) and permitted examination of the possibility that alcohol might, in addition to attenuating arousal, impair men's control over arousal. At the .08% BAC, alcohol did not attenuate genital arousal. However, alcohol interacted with arousal instructions to influence arousal onset. Sober men instructed to maximize arousal became aroused more quickly than did sober men instructed to suppress it. Intoxicated men did not show this effect, suggesting that alcohol lessened their ability to control the onset of their arousal. At the higher BAC, alcohol did have an attenuating effect on men's peak genital arousal but had no effect on other erectile measures, including average genital arousal. Two other studies evaluated the effects of different alcohol dosages during exposure to erotic stimuli, and no alcohol attenuation effects were detected (George et al., 2009; Prause, Staley, & Finn, 2011).

In contrast to earlier findings and the premature conclusions they inspired about attenuating effects of acute alcohol consumption, these later findings indicated that relatively high dosages of alcohol have limited impact on men's erectile responding, with only certain types of arousal attenuation emerging and only at very high doses (George et al., 2009; Prause et al., 2011). These findings posed a challenge for the view that acute heavy drinking diminishes erectile performance. Furthermore, alcohol's attenuating effects on erectile functioning appear to be more context dependent than previously recognized; dependent for example, upon the situational instruction-demand to maximize versus suppress arousal. Generally, it now appears that there is little basis to view alcohol as necessarily interfering with sexual response for sexually functional men. It may be that the attenuation effect holds true and is robust, but only at higher degrees of intoxication—much higher than .10%, which, while relatively rare in laboratory experimentation, may be common in men's real-life encounters.

That said, an additional consideration of importance is the biphasic limb pattern; that is, the difference in responding based on whether the BAC level is ascending or descending. In all of the mentioned studies, except the sleep study, investigators sought to assess alcohol's effects on the ascending limb or peak portions of the BAC curve, effects which involve euphoric mood and stimulation. Less is known about alcohol effects during the descending limb, which is generally characterized by dysphoric mood and fatigue. In the one study dedicated to examining alcohol's genital arousal effects on the descending limb, an attenuating effect was evident at a .08% dosage, but again only in a confined way (George et al., 2008). Men who received alcohol (target peak BAC = .08%) exhibited less genital arousal than no-alcohol controls, but only when instructed to maximize arousal and only on one of three erectile measures. This finding indicated again that attenuation effects may be more limited than previously thought and also that the threshold for attenuation effects may be somewhat lower on the descending than on the ascending limb of the alcohol curve. The latter point is consistent with the idea that the descending limb could be especially prone to potentiate alcohol attenuation effects because of the dysphoria and fatigue associated with descending BAC. A potential implication of this finding is that in circumstances where men are deliberately exerting conscious effort to become aroused, alcohol likely undermines peak erectile response for quite some time after drinking has ended and intoxication subsides. An interesting twist to these findings was that intoxicated men instructed to suppress their arousal exhibited more genital arousal than intoxicated men instructed to maximize their arousal. By implication, in circumstances where men are deliberately trying to resist becoming aroused, being on the descending alcohol limb may hamper their effectiveness. Reduced effectiveness at suppressing arousal in conjunction with alcohol's tendency to heighten risk taking could result in an increased likelihood of pursuing sexual activity in circumstances, such as disease risk situations, where sexual pursuit should be curtailed. Alcohol and STI/HIV sexual risk taking are discussed in further detail later.

A noteworthy limitation in this experimentation is that all but two of the studies reported (George et al., 2008; Morlet et al., 1990) investigated alcohol's effects under conditions of short-term acute administration on the ascending limb of the BAC curve. Thus, while there has been considerable research devoted to understanding alcohol's moderate-to-high dose (.04 to .10) effects within an hour or so after drinking, very little is known about alcohol's effects at greater acute doses over longer periods of time—for example, hours after heavy drinking.

Acute Episodic Erectile Failure. There are clinical and anecdotal reports of men experiencing erectile failure during episodes of excessive drinking. Colloquial phrases (e.g., "whiskey dick," "brewer's droop") reflect this phenomenon. While the causal mechanism would clearly seem to be physiological, there are significant implications for sexual health. First, frequent episodes of alcohol-induced erectile failure may signal the presence or development of alcohol abuse or alcohol dependence disorders and could serve as a prompt for seeking addiction evaluation and treatment. Second, even a single episode can contribute to the development of an erectile disorder. Specifically, if the episode is not accurately attributed to alcohol, but if instead it is misattributed to psychological concerns about virility, sexual potency, partner issues, and so on, then a recurrent pattern of erectile failure can develop and persist. Although initially caused by physiological processes, this pattern can become psychogenic in nature. For instance, subsequent alcohol-free sexual encounters can become characterized by partner conflict and performance anxiety about repeated erectile failure, both of which can drive emotional and cognitive distractibility that, in turn, disrupts normal erectile responding (e.g., Barlow, 1986). Third, whether it occurs sporadically or chronically, episodic erectile failures can have adverse effects on relationship stability, thereby undermining psychological as well as sexual health. Under these circumstances, alcohol moderation or

abstinence and psychological and/or pharmacological treatment for erectile disorder may be advised.

Alcohol and Erectile Dysfunction Medications. A final note about alcohol and erectile functioning concerns medications used to treat erectile disorder. There is growing evidence that medications such as Viagra (sildenafil) and Cialis (tadalafil) are often used in conjunction with alcohol, both legally and illicitly (Bechara, Casabé, De Bonis, Helien, & Bertolino, 2010). Among men diagnosed as having erectile disorder, alcohol dependence, or both, these medications appear to improve sexual functioning. Among sexually functioning nonalcoholic men, these medications are often used recreationally during drinking episodes, a pattern which has been especially observed in research findings with gay men (e.g., Holt, 2009). Little is known empirically about the interactive effects on sexual functioning of consuming alcohol with these drugs, and research is warranted, particularly given the potential for medically problematic interactions (Calabrò et al., 2015).

Alcohol and Women's Sexual Arousal

Survey studies that have examined sexual arousal and alcohol use in women have had mixed findings. When considering alcohol's effects on sexual arousal and sexual functioning among women, the distinction between subjective or self-reported arousal versus physiologically assessed indices of genital arousal is more important than among men (for whom self-reported and physiological indices are more highly correlated than among women [Chivers, Seto, Lalumiere, Laan, & Grimbos, 2010]).

Self-Reported Arousal. In a daily log study, alcohol was found to have no effect on self-reported arousal (Harvey & Beckman, 1986). However, in another diary study, it was found that alcohol use was related to increased sexual arousal during the post- and intermenstrual phases of the menstrual cycle (Lindman, Koskelainen, & Eriksson, 1999). A study classifying women into abstainer, light, and moderate drinking groups reported that drinking was unrelated to genital vascularization and lubrication and that moderate drinking was associated with lower depression and elevated sexual activity and orgasm (Battaglia et al., 2011). Impairment of sexual arousal has been related to alcohol dependency (for a review, see Sobczak, 2009a). However, premenopausal alcoholic women who were abstinent even less than a year have shown improvement in their ability to become sexually aroused (Gavaler et al., 1993).

As of 2012, approximately 16 experiments had examined acute alcohol intoxication effects on self-reported sexual arousal in women. The majority (specifically, 13 out of 16) found that alcohol intoxication increased self-reported arousal in women. The effect of alcohol has been more pronounced with higher dosages, but the effect was present in a variety of doses. In addition, the effect has been shown with a variety of different stimuli, including erotic films, vignettes, and slides. One explanation for alcohol's effect on increasing self-reported sexual arousal in women is alcohol expectancy theory. As noted, this theory suggests a self-fulfilling prophecy whereby individuals who hold the belief that alcohol increases sexual arousal are more likely to perceive that alcohol enhances sexual arousal in alcohol-involved sexual situations than those who do not hold that belief. A second explanation for increased self-reported sexual arousal may be alcohol myopia theory, which asserts that alcohol-induced cognitive impairment constricts the range of cues to which one is capable of attending in a situation (Steele & Josephs, 1990). Alcohol myopia theory would therefore posit that intoxicated individuals may focus more intently on salient sexual cues, such as erotic stimuli, than sober individuals and consequently experience themselves as more aroused. A third possible explanation for the increase in self-reported sexual arousal when intoxicated may be mediated by testosterone, which has been associated with heightened sexual feelings. A study of Finnish women found that alcohol increased testosterone levels, depending on different phases of the menstrual cycle and on oral contraception use (Eriksson, Fukunaga, & Lindman, 1994). For women not using oral contraceptives, the increase was evident during the ovulatory phase; for women using oral contraceptives, it was evident during days 17 through 20 of the cycle. These findings suggest that alcohol's effects on self-reported arousal may be related to testosterone in women. These potential explanations for how low to moderate dosages of alcohol can enhance women's self-reported sexual arousal warrant further research.

Nevertheless, regardless of the explanation, it seems clear that low to moderate dosages of alcohol result in women feeling and reporting more sexual arousal than if they were sober. Paradoxically, it seems that this effect can occur concurrently while alcohol is actually attenuating genital arousal. That is, in some experiments, women reported postdrinking increases in sexual arousal, yet they also exhibited alcohol-induced decreases in physiologically measured genital arousal (e.g., George et al., 2009). This paradox is not surprising given the low agreement generally between women's self-report and genital measures across all sexual arousal studies (not just alcohol studies). In a review of studies examining the correspondence between self-report versus genital arousal, self-report and genital measures had substantially lower correlations for women than for men (Chivers et al., 2010). Therefore, although paradoxical, it is not contradictory empirically that alcohol can have opposite effects on women's self-reported versus genital arousal.

Genital Arousal. Experiments evaluating alcohol's effects on genital arousal typically use a vaginal photoplethysmograph as measurement. Of 11 such experiments that have been reported to date, six (George et al., 2009, 2011, Study 2; Gilmore et al.,

2010; Malatesta, Pollack, Crotty, & Peacock, 1982; Wilson & Lawson, 1976, 1978) found that alcohol attenuated women's genital arousal and five (Bird et al., 2018; George et al., 2011, Study 1; Prause et al., 2011; Schacht et al., 2007, 2010) found no evidence of attenuation. In an illustrative study with women between the ages of 21 and 35 years, the effects of two BACs (.08% and .10%) were evaluated in the context of instructions to control arousal by either maximizing or suppressing it while viewing an erotic film (George et al., 2009). At the lower BAC, which is widely recognized as substantially intoxicating in that it is the legal criterion for drunk driving and it is widely associated with generalized impairment, alcohol did not attenuate genital arousal.

Furthermore, women exhibited greater genital arousal under conditions of being instructed to maximize arousal than to suppress arousal. These findings were interesting in demonstrating that, despite being intoxicated and despite the generically low correspondence between subjective and genital arousal, women were able to exert control over their genital arousal. However, at the .10% BAC, alcohol attenuated women's genital arousal and no arousal control was evident. Thus, it seems clear that alcohol can attenuate women's genital arousal, and this effect is most evident at very high dosages. As noted earlier, the mechanisms responsible for this attenuation effect have not been clearly identified. It has been presumed that the mechanisms are strictly physiological. However, this has not been established, and psychological mechanisms have not been ruled out.

Looking across both the self-report and genital assessment studies, it appears reasonable to contend that until the BAC exceeds .08%, women are likely to exhibit an arousal response pattern characterized by enhanced self-reported sexual arousal, no alcohol-induced diminution in genital arousal, and an effective capability to control their arousal response volitionally. These considerations suggest that women's postdrinking sexual responses are subject to considerable variability and are not robustly dictated by physiological imperatives or limits. However, this variability apparently decreases at higher dosages where it appears that intoxication effects may become less conducive to positive sexual responding and experience. Potentially, physiology related to high alcohol levels may overwhelm the sexuality-related response systems.

Alcohol, Sexual Interest, Desire, and Orgasm

Research on alcohol use and sexual interest and desire is limited, and practically all of it has examined women. The studies that have examined these relationships among women consist predominantly of descriptive surveys. Most studies have found that alcohol is related to increased sexual desire among nonclinical samples (e.g., Beckman, 1979; Harvey & Beckman, 1986; Woods, Mitchell, & Smith-Di Julio, 2010). An illustrative study specifically examined red wine intake in Italian women and found that women who reported moderate daily red wine intake also reported higher levels of sexual desire compared to women who did not drink alcohol (Mondaini et al., 2009). In two experiments investigating alcohol's effects on sexual desire, findings were mixed. In one, alcohol decreased sexual desire (George et al., 2014); in another, alcohol interacted with expectancy, such that alcohol increased sexual desire as a function of women's endorsement of the expectancy that alcohol disinhibits sexuality (Gilmore et al., 2013).

According to a review (Sobczak, 2009a), findings concerning the relationship between alcohol consumption and other aspects of sexual functioning are mixed. In some studies, women reported increased sexual activity after drinking; in other studies, women reported having less sex when drinking. This review also concluded that heavy alcohol use is related to impaired sexual functioning, including lowered sexual interest, desire, and satisfaction, as well as lack of orgasm and lubrication and painful intercourse. Wilsnack, Klassen, Schur, and Wilsnack (1991) found in a longitudinal study that the most consistent predictor of women's problem drinking was sexual dysfunction. On the positive side, Gavaler et al. (1993) found that among premenopausal alcoholic women, being abstinent for less than a year had beneficial effects on sexual desire and ability to achieve orgasm. Among postmenopausal women, Gavaler, Rizzo, Rossaro, and Van Thiel (1994) found that alcoholic women's satisfaction with their sex lives improved after at least a year of abstinence.

Among women in treatment for alcohol or other drug use disorders, a history of child or adult sexual assault has been related to various types of sexual dysfunction (Wilsnack, Plaud, Wilsnack, & Klassen, 1997), including painful intercourse (Sutherland, Fantasia, & McClain, 2013). Evidence suggests that women with a history of sexual trauma often use alcohol or other drugs to disconnect from in-the-moment sexual experiences (Sanjuan, Langenbucher, & Labouvie, 2009; Sobczak, 2009b). In fact, evidence from laboratory experiments using heat-of-the-moment protocols to simulate a sexual encounter with a nonsteady partner have shown that, for women with varying degrees of historical sexual victimization, acute alcohol intoxication increases dissociation during the simulated encounter (Bird et al., 2017; Stappenbeck et al., 2016). Thus, sexual dysfunction and alcohol abuse likely have a reciprocal relationship.

Research on alcohol use and orgasm is also limited. The available studies, although few, examining the relationship between alcohol and orgasm has found that alcohol is related to more inconsistent orgasms or fewer orgasms overall in women (e.g., Johnson, Sweeney, Herrmann, & Johnson, 2016). One experiment examining the effect of acute alcohol on orgasms in women found that latency to orgasm was longer and subjective intensity of orgasm was decreased for women with higher BACs (Malatesta et al., 1982); however, higher BACs were also associated with greater sexual arousal. Another experiment examining the effect of acute alcohol on orgasms in men found increased orgasm latency after alcohol, increasing linearly with BAC level (.03%, .06%, .09%)

(Malatesta et al., 1979). However, the authors noted that this effect among men "may be viewed as a beneficial consequence" because "sexual intercourse would be prolonged for both partners" (p. 106). Much research is needed to better inform scientific understanding of how alcohol intoxication affects sexual desire and orgasm.

What We Know About Alcohol and STI/HIV Related Sexual Risk Taking

STIs, especially HIV/AIDS, remain a grave health threat, and new infections are predominantly attributable to sexual contact, among both gay men and heterosexuals (Centers for Disease Control and Prevention, 2018). The primary behaviors placing an individual at risk of disease include having condomless anal or vaginal intercourse, doing so with multiple partners, and doing so with partners of unknown infection status—especially partners at elevated risk categorically, such as intravenous drug users. Risks are greater with new and casual partners than with established partners. However, established partners may also pose risks if involved in intravenous drug use or extradyadic sexual activity. Therefore, among sexually active individuals, consistent condom use and avoidance of partners of unknown risk status provide the greatest protection against STIs and HIV. Among factors associated with inconsistent condom use and risky sexual partnering, alcohol intoxication is prominent. If alcohol could be shown and understood to play a truly causal role in risk taking, then targeting alcohol use as an avenue of risk reduction would be indicated. Consequently, the association between alcohol and sexual risk taking has been the subject of considerable research using a variety of methodological approaches, including qualitative (e.g., Carey et al., 2018), quantitative (e.g., see review by Wagenaar, Florence, Adams, & Savahl, 2018), and experimental (e.g., see review by Scott-Sheldon et al., 2016) methods.

The key question in this research has been: Does alcohol contribute to sexual risk taking? Global and situational covariation surveys have shown that alcohol is associated with increased risk, in that individuals who drink to intoxication use condoms less frequently overall than those who do not (e.g., Cooper, 2002; Dingle & Oei, 1997; Donovan & McEwan, 1995; Halpern-Felsher, Millstein, & Ellen, 1996; Leigh & Morrison, 1991). However, event-level and daily diary studies (e.g., Leigh et al., 2008; Simons et al., 2018) have demonstrated that this relationship does not exist when individuals report on their intoxication and condom use during specific sexual events. These mixed findings have highlighted the complexity of alcohol's involvement in sexual risk taking, underscoring the importance of moderating contextual factors such as the risk level and relationship status of sexual partners (Cooper, 2002, 2010; Weinhardt & Carey, 2000). Theory-building longitudinal work by Cooper (2010) supports the idea that contextual and relationship factors, in conjunction with individual characteristics, affect the relationship between alcohol and condomless sex. As noted earlier, survey methods—despite their pioneering advances in documenting alcohol and sexual risk associations and their sustained contributions to elucidating cross-sectional and longitudinal influences of various individual difference factors, situational factors, and interpersonal factors—remain limited in significant ways with respect to evaluating the "does alcohol contribute" question and its implications for causation.

Researchers using experimental methods have addressed questions that are arguably more basic than the "does alcohol contribute" question and, at the same time, more complex: Is alcohol even capable of exerting a genuine causal impact on sexual risk taking? If so, how can this causation best be explained? Despite ambiguity in survey data about alcohol's causal role, it is now well established through rigorous experiments that alcohol can and does have a causal impact on sexual risk behaviors (e.g., see reviews by Berry & Johnson, 2018; Rehm, Shield, Joharchi, & Shuper, 2012; Scott-Sheldon et al., 2016).

To date, numerous independent investigative teams (e.g., Abbey, Saenz, & Buck, 2005; Cho & Span, 2010; Fromme, D'Amico, & Katz, 1999; George et al., 2009; Johnson et al., 2016; MacDonald, Zanna, & Fong, 1996; Maisto, Carey, Carey, & Gordon, 2002; Murphy, Monahan, & Miller, 1998; Prause et al., 2011; Zawacki, 2011) have now conducted more than three dozen alcohol and sexual risk experiments. With little exception (e.g., Kruse & Fromme, 2005), these experiments have supported the hypothesis that rising alcohol intoxication increases the likelihood of condomless intercourse with nonsteady partners (see reviews by Berry & Johnson, 2018; George & Stoner, 2000; Hendershot & George, 2007; Rehm et al., 2012; Scott-Sheldon et al., 2016) and highlight several points. First, and an important point given the current zeitgeist of justifiable concern about replicability of findings from psychological experiments, there is independent replication using a variety of protocols showing clear evidence of causal influences. Second, despite the pragmatic and ethical constraints on analogizing and studying sexual risk in the laboratory, investigators have succeeded in developing and implementing protocols that credibly assess risky sex intentions and likelihood, as well as pertinent constructs such as sexual arousal. Third, these researchers have identified in-the-moment processes and reactions that immediately precede sexual decision making and have demonstrated the theoretical importance of these constructs. Fourth, simultaneous consideration of distal and proximal determinants has advanced identification, isolation, and evaluation of mediating and moderating mechanisms. Several variables may function as cofactors in alcohol-involved sexual situations. For instance, sexual sensation seeking (e.g., Maisto et al., 2004a), alcohol expectancies (e.g., Maisto, Carey, Carey, Gordon, & Schum, 2004b), relationship factors (Zawacki, 2011; Zawacki et al., 2009), effortful control (Simons et al., 2018), and sexual victimization history (e.g., Stoner et al., 2008) have been associated with sexual risk-taking intentions in alcohol experiments.

Finally, these findings almost uniformly support the utility of alcohol myopia theory (Steele & Josephs, 1990) as the best framework to understand intoxicated sexual risk taking. This support is best illustrated in experiments showing that (1) alcohol effects increase with dosage (e.g., George et al., 2009), which would not be predicted from an alcohol expectancy explanation; (2) alcohol affects risk indirectly via relevant subjective states, including (a) sexual arousal (Davis et al., 2009; George et al., 2009; Norris et al., 2009a; Prause et al., 2011; Wray, Simons, & Maisto, 2015), (b) perceived sexual potential (Norris et al., 2009b; Purdie et al., 2011), (c) positive mood (George et al., 2014; Gilmore et al., 2016) and sexual desire (George et al., 2014), (d) anticipated partner response to condom requests (e.g., George et al., 2016; Masters et al., 2014), (e) perceived intoxication (Davis et al., 2009), (f) emotional numbing and refusal self-efficacy (Stappenbeck et al., 2016), and (g) implicit approach bias toward erotic stimuli (Simons, Maisto, Wray, & Emery, 2016); and (3) alcohol focuses the drinker cognitively on impelling cues in the situation (Norris et al., 2009b); and a weighted composite of impelling/inhibiting cognitions has been shown to mediate alcohol's effects (Davis, Hendershot, George, Norris, & Heiman, 2007; MacDonald, MacDonald, Fong, Zanna, & Martineau, 2000). In sum, the available science warrants the conclusion that alcohol can and does increase the likelihood of sexual risk taking through attuning intoxicated participants to internal states congruent with being motivated toward sexual gratification. Alcohol's capacity to focus the drinker myopically on impelling cues, despite extant risks in a sexual encounter, fosters unsafe sex.

Sexual Victimization History

Victimization history has proven to be a particularly important cofactor for women (e.g., Schacht et al., 2010; Staples et al., 2015). Heat-of-the-moment experiments utilizing second-person ("This is you") eroticized scenarios with heavy episodic drinking women at elevated risk of HIV/STI infection have been conducted. In these studies, both a history of sexual victimization and acute alcohol intoxication were hypothesized to affect intentions to have condomless sex. Focusing on sexual-emotional mediators, George et al. (2014) found that both sexual victimization history severity and acute alcohol intoxication affected condomless sex intention, and that these effects were mediated by positive mood and sexual desire. Focusing on cognitive mediators, Masters et al. (2014) found that the effects of sexual victimization history severity and acute alcohol intoxication were mediated through (a) perceptions of the partner's intoxication and STI risk, (b) anticipated negative partner reaction to condom insistence, and (c) willingness to abdicate the decision about having condomless sex to the partner. In another similar study, women were subjected to low or high pressure by the simulated male partner to forgo using a condom (George et al., 2016). When acutely intoxicated, high pressure and women's sexual victimization history severity predicted the anticipated negative partner reaction, which in turn predicted abdication (i.e., willingness to let the partner decide about condom use). Specifically, high pressure increased anticipation that the man would respond negatively to the woman's desire to use a condom. The fact that this anticipation, in turn, increased willingness to abdicate the decision about having condomless sex to him can be seen as potentially instrumental in avoiding his negative reactions by instead yielding to his wish for condomless intercourse. Such an avoidance-by-yielding response could be interpreted as disempowering because it is at the expense of women's own desire, agency, and sexual safety. Yet this pathway could also be self-protective, because it mitigates the prospect of being harmed by a male partner, either psychologically or physically.

These experiments examining the intersection of alcohol intoxication and women's sexual victimization history indicate that, once in an alcohol-involved sexual encounter, sexually victimized women do respond differently than nonvictims in ways that lead to riskier choices. Previous research on victimization and sexual risk behavior has largely been restricted to explanations relying on distal associations: Presumably, victimization fosters trauma symptoms and mood disturbances, which, in turn, foster problematic coping styles and relational instability, resulting in greater exposure to risk opportunities. There has been comparatively scant victimization and sexual risk research about what transpires proximally in the moments preceding sexual decisions. These experimental findings strongly suggest that something about prior victimization threads its way into the current sexual situation to influence affect, sexual responding, and risk outcomes. Further research is needed to identify what features of victimization or reactions to it drive the link to differential responding in subsequent sexual encounters. Also, high intoxication achieved through a heavy-episodic-drinking style—replicated in the laboratory—clearly heightens sexual risk taking. Given that victimized women generally drink more and engage in more frequent heavy episodic drinking than their nonvictimized counterparts, such findings demonstrate a clear risk nexus. By virtue of both their drinking quantity and style and their heightened willingness to abdicate condom decision making to a male partner, sexually victimized women are at substantially elevated risk for behavior that can lead to STI/HIV infection.

Condom Use Resistance

Alcohol experiments have also been valuable in deconstructing and identifying features of sexual risk intentions that had previously received little research attention. One such construct is men's willingness to use coercion to get a woman to have sex without a condom (e.g., Abbey, Parkhill, Jacques-Tiura, & Saenz, 2009). Condom use resistance (CUR; Davis et al., 2012) can be defined as behaviors strategically aimed at having sexual intercourse without a condom when the partner wants to use a condom. In

effect, CUR constitutes effort at nonconsensual condomless sex when consensual protected sex is available, and thus can be seen as a unique form of sexually aggressive behavior. Davis et al. (2014) have identified a number of CUR tactics, both coercive and noncoercive, that men endorse, such as seduction (e.g., "getting her so sexually excited that she agrees to have sex without a condom"), risk-level assurance (e.g., "reassuring her that you are 'clean' so that she will have sex without a condom"), deception (e.g., "pretending that you have been tested and do not have any STIs"), condom sabotage (e.g., "agreeing to use a condom but intentionally breaking the condom when putting it on"), relationship and trust (e.g., "telling her that you trust each other so that she will have sex without a condom"), physical threat/force (e.g., "preventing her from getting a condom by staying on top of her"), and so on. Accordingly, women report substantial rates of experiencing CUR at the hands of male partners, including nonconsensual condom removal ("stealthing") and lower but discernable rates of engaging in CUR themselves (Davis, Stappenbeck, Masters, & George, in press).

Not surprisingly, alcohol affects CUR. Davis et al. (2012) have found that childhood sexual abuse and acute intoxication increased CUR intentions among heavy episodic drinking men. These effects were indirect via their sense of sexual entitlement and perceptions of the woman's sexual arousal. In another experiment, Davis et al. (2016b) found that both acute intoxication and a history of being sexual aggressive increased CUR. Alcohol intoxication also increased men's positive attitudes about CUR, perceptions of more supportive social norms about CUR, and self-efficacy about engaging in CUR (Davis et al., 2016a). Alcohol's effects on men's CUR also vary with risk rationale (e.g., whether a woman expresses concern about "getting pregnant" versus "getting an STI"), depressive symptomology (Neilson, Eakins, Davis, Norris, & George, 2017), and hostility toward women (Wegner et al., 2017). Davis (2010) found that men's aggressive condomless sexual intentions were moderated by alcohol-aggression expectancies and mediated by sexual aggression congruent emotions/motivations. Together, these CUR experiments demonstrate another path through which alcohol fosters outcomes that threaten sexual health, especially for women.

Limitations and Summary

Circling back to the opening questions, what do we know about alcohol's causal impacts pertaining to sexual health and how do we know it? Partial answers are indicated. After a lengthy hiatus in alcohol and sexual arousal experimentation, experiments conducted since the mid-2000s corrected for earlier methodological problems and showed that alcohol's attenuating effects on men's genital arousal are more circumscribed than they were understood to be earlier. There is little evidence for robust attenuation effects below a .08% BAC, and effects obtained at .10% BAC are limited. Among women, attenuation effects on genital arousal are inconsistently evident at .08% BAC, and these effects occur concurrently with alcohol-induced enhancement effects on subjective arousal. Experiments also revealed that alcohol effects on sexual arousal are subject to contextual considerations such as motivational set and whether BAC is rising or falling. Thus, until the BAC exceeds .08%, both men and women are likely to exhibit an arousal response pattern characterized by enhanced self-reported sexual arousal, and no alcohol-induced diminution in genital arousal. That said, due to constraints on acute intoxication laboratory protocols, little is known about alcohol's effects at higher dosages, which may be associated with more robust attenuation effects, suggesting that heavy chronic drinking could potentiate sexual arousal disorders.

A noteworthy contribution of alcohol and sexual arousal experimentation was the demonstration that subjective but not genital arousal mediates alcohol's effects on sexual risk-taking intentions. This finding emerged amid a host of studies showing that alcohol's effects on measures indicative of STI/HIV-related sexual risk taking are robust and causal. Multiple review narrative and meta-analytic papers have confirmed this conclusion and have indicated that alcohol myopia theory best accounts for this causal impact (e.g., Hendershot & George, 2007; Scott-Sheldon et al., 2016). Sexual risk experiments have also identified several other mediating and moderating influences on alcohol's effects, some of which (e.g., alcohol expectancies) suggest intervention targets for clinical work aimed at reducing alcohol-related sexual risk behavior.

An important cofactor identified, especially for women, is sexual victimization history. Previously victimized women are at increased risk for engaging in behaviors, such as sexual decision abdication, that heighten their likelihood of condomless intercourse. Because victimized women drink more than nonvictimized counterparts and because alcohol increases risk taking, the risk nexus for women's sexual health posed by sexual victimization history stands as an important focus for future research. Men's CUR is another important focus to highlight going forward. Alcohol increases men's intentions to violate women's consent by trying to perpetrate condomless intercourse when women want to use a condom. Not surprisingly, mediating and moderating factors identified for CUR reflect some of the same factors known to be associated with alcohol-related sexual assault perpetration, such as hostility toward women and situational anger. There is an extensive literature on experiments examining alcohol's effects on men's sexual assault perpetration, which exceeds the scope of the current review, attesting to alcohol's influential role in men's sexually transgressive behavior toward women (Abbey, Wegner, Woerner, Pegram, & Pierce, 2014). Future work is needed to further unravel the roles of women's sexual assault victimization and men's CUR and sexual assault perpetration in understanding alcohol effects on sexual risk taking and sexual health.

Experiments have clearly and definitively answered the question "Is acute alcohol intoxication capable of exerting a causal influence on behaviors related to sexual function and health?" Yet important limitations warrant acknowledgment about whether and under what conditions these causal effects manifest in the real world. While controlled experiments enjoy high internal validity, their external validity demands further research. While emerging external validity data suggest that what research participants exhibit in the laboratory indeed reflects their naturalistic behaviors, much more research is needed. Perhaps naturalistic experiments in the real world (e.g., MacDonald et al., 2000) combined with ecological momentary assessment technology could be useful in this regard. Another limitation is that the vast majority of the experiments conducted to date have utilized heterosexual young adult samples. Age influences erectile functioning, rendering younger men less affected by alcohol attenuation than older men. Thus, more research is needed with men and women of varying ages across the life span. While there is a small but growing number of experiments with men who have sex with men (e.g., see Maisto, Palfai, Vanable, Heath, & Woolf-King, 2012; Maisto & Simons, 2016; Shuper, Joharchi, & Rehm, 2016), it is imperative that more experimentation be done with this sample, which is at uniquely higher risk for HIV/AIDS. Likewise, there has been no experimentation with other lesbian, gay, bisexual, transgender, and queer (LGBTQ) samples. Indeed, individuals who identify as transgender and individuals who are gender nonconforming also appear to be at elevated risk for sexual risk behaviors. Overall, in spite of these limitations, research shows that, in addition to alcohol's effects on sexual functioning, some of which are salutary, it is clear that alcohol constitutes a threat to sexual health through its effects on risk taking.

Implications

Beyond the basic science aim of elucidating alcohol's role in sexual health, the body of alcohol experimentation research reviewed here was often motivated (and sometimes funded) with an eye toward deriving translatable intervention implications, especially with regard to STI/HIV sexual risk taking. An undergirding refrain has been: The capacity to prevent unsafe sex ultimately hinges on scientists' capacity to understand what transpires in those critical moments in which intoxicated sexual decisions are made, enacted, or "just happen." That has been the mission of the experiments reviewed here. With respect to clinical implications, primary prevention strategies can be categorized into those aimed at scaling up or extending existing interventions, modifying existing interventions, and innovating new interventions. For each of these perspectives there exist insights to be gleaned from findings based on alcohol and sexuality experiments.

Many prevention interventions have been shown to be effective in reducing HIV risk-related behaviors (Centers for Disease Control and Prevention, 2019). By demonstrating that alcohol has a causal effect, a clear implication of alcohol experiments is that sexual risk behavior can be reduced indirectly by reducing alcohol consumption; and there has been documented success in this regard (e.g., Walsh, Weinhardt, Kalichman, & Carey, 2016). Some interventions have combined and/or integrated sexual behavior content with alcohol behavior content and shown risk reduction (e.g., Lewis et al., in press). Thus, scaling up and extending the reach of interventions that reduce alcohol consumption is an important implication, especially for impacting sexual health problems for underinvestigated and underserved populations.

For modifying existing sexual health interventions, important implications include edifying and intensifying psychoeducational content about alcohol and about the alcohol-sexuality intersection. Implications grounded in sexual arousal findings suggest the importance of psychoeducational content (a) debunking myths about male arousal as an uncontrollable determinant of sexual outcomes, (b) noting the prepotency of subjective arousal over genital arousal in driving sexual risk outcomes, and (c) emphasizing the independent and synergistic influences of alcohol and sexual arousal in determining risk outcomes.

Theory-driven implications grounded in alcohol expectancy theory suggest psychoeducational content emphasizing that (a) alcohol's sexual effects are not strictly a result of pharmacology—expectancies matter; (b) alcohol expectancies shape decisions to drink and how much to drink; (c) self-fulfilling prophecies about what is expected from alcohol and deviance disavowal expectancies about alcohol as an excuse for out-of-character behavior both shape how one behaves initially after drinking begins; and (d) importantly, alcohol expectancies are malleable and can be targeted for cognitive restructuring interventions. Also, "expectancy challenge" demonstrations, in which individuals consume placebo drinks while engaging in a socialization activity, can be added as a psychoeducational module to illustrate the power of alcohol expectancies and thereby serve as a springboard to expectancy modification.

Theory-driven implications grounded in alcohol myopia theory suggest psychoeducational content noting that alcohol intoxication results in increased attention on impelling "green light" cues for risky behavior and decreased attention on inhibiting "red light" cues. However, when inhibiting cues are accentuated in a risky situation, alcohol can actually reduce sexual risk taking (MacDonald et al., 2000), which supports the potential utility of signage practices such as highlighting condom availability in drinking venues.

The alcohol experiments reviewed can also provide fodder for innovating new intervention mechanisms and procedures. For instance, heat-of-the-moment modules developed and validated in laboratory work could be repurposed to serve as (a) pre- and postintervention assessment tools; (b) a "reflective tool" exemplifying more vividly for clients how sexual risk decisions unfold (e.g., Andrasik et al., 2013); and (c) a protocol for rehearsing safer decision making. Also, experiments indicate clearly that distal past

experiences with victimization and perpetration influence current-day sexual risk behavior, suggesting utility in routinely assessing such histories in sexual health protocols and developing psychoeducational content about this linkage.

Finally, questions and implications for future basic research are evident. Can risk-reduction strategies and skills be acquired and/or implemented during states of acute alcohol intoxication? Do strategies and skills acquired while sober generalize to alcohol-involved sexual encounters? Given demonstrable emotional processes, more work is needed determining how emotion regulation models pertain. How do drinking motives and sex motives function as predispositions and as in-the-moment factors for alcohol-involved sex? Although it has been amply demonstrated that women with sexual victimization histories actually behave differently in sexual encounters than women without such histories, there is scant understanding of how to best explain this pattern. Lastly, given the growing predominance of medically driven prevention strategies, such as pre-exposure and postexposure prophylaxis, research is needed addressing alcohol's role in sexual risk taking via impaired medication compliance (e.g., Hendershot, Stoner, Pantalone, & Simoni, 2009). More research is needed in identifying explanatory mechanisms in all of these lines of inquiry and in recognizing derivative intervention targets.

Conclusions

We know, through its demonstrable effects on sexual arousal, orgasm, and sexual risk behavior, that alcohol affects sexual health. We know this from a variety of research methods. However, from experiments, we know that these alcohol effects reflect causal influences. From these same experiments, we also know that alcohol expectancy theory and alcohol myopia theory afford plausible explanatory accounts and that a host of contextual, emotional, cognitive, and predispositional factors moderate these effects, often in complex ways. Going forward, it is hoped that these findings and insights will fuel intervention applications. Intervention science must remain linked to basic science aimed at evaluating theory-driven mechanisms accounting for alcohol's relation to sexual health. Further research is needed in both domains of research about alcohol and sexual health: Basic science and prevention/intervention science.

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