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Indirect Effects of Acute Alcohol Intoxication on Sexual Risk-Taking: The Roles of Subjective and Physiological Sexual Arousal

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Abstract

Three experiments supported the idea that alcohol fosters sexual risk-taking in men and women, in part, through its effects on sexual arousal. In Experiment 1, increasing alcohol dosage (target blood alcohol levels of .00, .04, .08%) heightened men's and women's risk-taking intentions. Alcohol's effect was indirect via increased subjective sexual arousal; also, men exhibited greater risk-taking than women. In Experiment 2, an extended dosage range (target blood alcohol levels of .00, .06, .08, .10%) heightened men's risk-taking intentions. Alcohol's effect again was indirect via subjective arousal. Physiological sexual arousal, which was unaffected by alcohol, increased risk-taking via increased subjective arousal. In Experiment 3, alcohol increased women's risk-taking indirectly via subjective arousal, but alcohol-attenuated physiological arousal had no effect on risk-taking. Implications for alcohol myopia theory and prevention interventions are discussed.

Keywords

Alcohol; Sexual arousal; HIV/AIDS; Sexual risk

INTRODUCTION

HIV/AIDS remains a grave health threat and new infections are increasingly attributable to heterosexual contact with a person who has, or is at high risk for, HIV infection (e.g., injection drug users, men who have unprotected sex with men, individuals with multiple sex partners, etc.). In the U.S., the proportion of AIDS cases attributed to heterosexual contact increased from 3% in 1985 to 32% in 2005 (CDC, 2005). At present, approximately 80% of new HIV cases in U.S. women and 15% of new cases in U.S. men are attributable to heterosexual transmission (CDC, 2007). In sub-Saharan Africa, heterosexual transmission is understood to be the primary mode of HIV infection (e.g., Mills, Singh, Nelson, & Nachega, 2006). As such, our capacity to curtail HIV incidence now hinges greatly on our ability to

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understand heterosexual risk behaviorally, which necessitates a comprehensive account of the processes unfolding during the crucial moments leading up to decisions about unsafe sex. Conventional wisdom and emerging research (e.g., Ariely & Loewenstein, 2006; Bancroft et al., 2004) suggest that choosing whether or not to have unsafe sex occurs often in the “heat of the moment,” when rational decision making can be overwhelmed by immediate motivational states, especially being sexually aroused (Gerrard, Gibbons, & Bushman, 1996) and intoxicated by alcohol (MacDonald, Zanna, & Fong, 1996).

Scant research has examined these joint influences and none has considered whether alcohol's effects on arousal, in turn, influence risk-taking. The resultant knowledge gap limits our understanding of this fundamental in-the-moment progression. This deficiency is further exacerbated by an apparent paradox. On the one hand, alcohol is widely associated with unsafe sex (Hendershot & George, 2007), as well as other sexually disinhibited outcomes (George & Stoner, 2000). On the other hand, high intoxication is known to attenuate sexual arousal—especially physiological arousal (Crowe & George, 1989), which has inherent relevance for sexual risk behavior. The present work investigates sexual risk in the context of alcohol's contradictory sexual effects, examining whether alcohol's impact on sexual decisions is differentially mediated by subjective versus physiological sexual arousal. We first examined effects of low to moderate dosages (target blood alcohol levels (BALs) of .00, .04, .08%) and biological sex (male, female), evaluating subjective sexual arousal as a determinant of intoxicated risk-taking (Experiment 1). We then examined effects of a higher dosage range (target BALs of .00, .06, .08, .10%) and compared the influences of subjective versus physiological sexual arousal in men's (Experiment 2) and women's (Experiment 3) sexual risk-taking under variable arousal conditions.

SEXUAL AROUSAL AND HIV-RELATED RISK-TAKING

Sexual arousal is a potent motivational state that has not received much attention in research on HIV-related risk-taking although it is understood intuitively to play an influential role in risky sexual encounters. Empirical evidence of this influence is beginning to accrue. In a survey of adolescent sexual encounters, Boldero, Moore, and Rosenthal (1992) found that pre-encounter sexual arousal, which was assessed *post hoc*, was associated with lower condom use during the encounter. Blanton and Gerrard (1997) found that college men reduced their perception of STD risk after sexual motivation was stimulated by exposure to photographs of sexually appealing women. Bancroft et al. (2003) found that being dispositionally low in inhibition of sexual arousal predicted unprotected anal intercourse and high risk oral sex among gay men. Among heterosexual men, being dispositionally low in inhibition of sexual arousal predicted having more partners with whom participants had not used condoms (Bancroft et al., 2004). Ariely and Loewenstein (2006) asked heterosexual men, in a sexually aroused state and an unaroused state, whether they would use condoms in risky sexual encounters and found that being aroused reduced the reported likelihood of condom use. Two other experiments revealed that self-reported sexual arousal during a hypothetical sexual encounter predicted sexually riskier outcomes, in conjunction with alcohol intoxication among men (MacDonald, MacDonald, Zanna, & Fong, 2000) and independent of alcohol intoxication in a mixed sex sample (Abbey, Saenz, & Buck, 2005). Thus, growing empirical evidence shows that sexual arousal increases risk-taking.

ALCOHOL, SEXUAL AROUSAL, AND RISK-TAKING

Until recently, alcohol and sexual risk-taking research had been conducted predominantly using surveys (Hendershot & George, 2007). Critical incident studies and diary studies have yielded conflicting evidence about the reliability and causality of alcohol's role (e.g., Gillmore et al., 2002). However, data from nonexperimental methods are limited for

clarifying what transpires in real time during the decisive moments preceding sex. By contrast, experimental methods allow researchers to capture in-the-moment states. Such methods also permit stronger causal statements because they permit random assignment to intoxicated versus sober conditions, specify precise intoxication levels, ascertain that intoxication precedes sexual outcomes, and do not rely on participants' potentially faulty retrospections. Given these important advantages, experiments are appearing with greater frequency and are fast becoming essential to deepening our understanding of the alcohol-risky sex linkage (Hendershot & George, 2007).

Five independent investigative teams have conducted more than 20 alcohol and sexual risk experiments (e.g., Abbey et al., 2005; Fromme, D'Amico, & Katz, 1999; Maisto, Carey, Carey, & Gordon, 2002; MacDonald, Zanna, & Fong, 1996; Murphy, Monahan, & Miller, 1998). With little exception (e.g., Kruse & Fromme, 2005), these experiments have supported the hypothesis that alcohol intoxication fosters riskier sexual outcomes (see reviews by George & Stoner, 2000; Hendershot & George, 2007) and have collectively substantiated the importance of dissecting in-the-moment states and processes that directly precede sexual decisions. However, thus far, only two experiments have investigated the joint influence of being both aroused and intoxicated (Abbey et al., 2005; MacDonald et al., 2000). Although these initial experiments provided preliminary evidence that both arousal and alcohol heighten risk-taking, they were limited in their capacity to address essential questions concerning the range and interplay of these effects. Several key questions stand out as important and remain largely unaddressed.

First, how does sexual arousal affect intoxicated individuals when the level of arousal is high, as would be the case in naturalistic sexual encounters? Investigating the "heat of the moment" necessitates creating an analogue of a highly sexually charged ambiance. Previous experiments examining the alcohol-arousal-risk relationships utilized analogue stimuli consisting of a written vignette designed to be "moderately sexually arousing" (Abbey et al., 2005, p. 85) or a videotape vignette that evoked an overall modest level of arousal (median of 3 on a 9-point scale; MacDonald et al., 2000). Relative to real-life interchanges preceding risky sexual decisions, previous stimuli were not—by design—intended to simulate a highly sexually charged encounter. Arousal levels nevertheless proved influential in both of these earlier studies. Abbey et al. (2005) found that men's and women's sexual arousal to written vignettes predicted their risky sex intentions and did so over and above alcohol's effects. MacDonald et al. (2000) distinguished men reporting above and below the median level of arousal to a videotape vignette and found an interaction; alcohol increased risk indices only for men above the median level of arousal. An aim of the current work was to intensify the experimental heat-of-the-moment analogue by incorporating an explicit erotic film to prime sexual arousal, as a prelude to an eroticized written vignette measuring risk while aroused. Together, the film and vignette allow a more sexually provocative context in which to evaluate the impact of sexual arousal on risk behavior.

Second, what roles do alcohol's effects on sexual arousal play in risky sexual encounters? This question springs from well-established research indicating that alcohol has demonstrable effects on sexual arousal itself (Crowe & George, 1989). Therefore, in naturalistic alcohol-involved sexual encounters, alcohol can be construed hypothetically as having direct effects on sexual risk-taking, as well as potentially indirect effects via its impact on sexual arousal. These potential influences have not been jointly evaluated before. As we have reviewed elsewhere (Crowe & George, 1989; George & Norris, 1991; George & Stoner, 2000), alcohol's effects on sexual arousal are complex and depend on several factors, including whether arousal is operationalized subjectively or physiologically. Crowe and George (1989) concluded (1) that alcohol increases or disinhibits subjective sexual arousal, (2) that it conversely decreases physiological genital arousal, and (3) that these

subjective and physiological effects can occur separately or jointly. Therefore, it seems that only alcohol's effects on subjective sexual arousal appear to be congruent with its effects on sexual risk-taking. It is unclear how alcohol's effects on physiological sexual arousal might relate to sexual risk-taking.

Third, are there sex differences in how alcohol and sexual arousal affect sexual risk-taking? As Abbey et al. (2005) note, while many alcohol and sexual risk experiments have examined only one sex, there are important reasons to expect differences. Compared to women, men are generally less concerned about the dangers of unprotected sex (Amaro, 1995) and more interested in casual sex (Oliver & Hyde, 1993). Also, alcohol's effects on sexual arousal and responses are more pronounced in men than in women (George & Stoner, 2000). Consequently, indirect effects of alcohol on sexual risk-taking via sexual arousal may vary by biological sex. Indeed, Abbey et al. (2005) found that while men and women achieved equal levels of sexual arousal during an experimental risk-taking protocol, men expressed greater risk-taking intentions than women; however, biological sex did not interact with alcohol to predict risk.

PRESENT EXPERIMENTS

Across three experiments, we studied the joint influences of alcohol and sexual arousal on sexual risk behavior, including a first-time evaluation of whether and how alcohol's effects on arousal, in turn, determine risk-taking. With little exception (Maisto, Carey, Carey, Gordon, & Shum, 2004), all previous experiments have used a single alcohol dosage. Given that naturalistic drinking experiences vary widely in dosage, we considered two multiple dosage ranges. In Experiment 1, we investigated low to moderate dosages (target BALs of .00, .04, .08%) and sex differences on subjective sexual arousal and the likelihood of having unprotected sex. In Experiments 2 (men) and 3 (women), we investigated a higher range of dosages (target BALs of .00, .06, .08, .10%) and the effects of sexual arousal on sexual risk behavior; instructional sets were used to vary arousal systematically (e.g., Beck & Baldwin, 1994). In an advance over previous work in which sexual arousal was measured by self-report only, Experiments 2 and 3 included a first-time consideration of how physiological genital arousal relates to risk-taking. Overarching hypotheses were (1) that both alcohol and subjective sexual arousal would independently increase sexual risk-taking intentions and (2) that alcohol's effects would be indirect, via subjective sexual arousal. Overarching questions across the latter two experiments concerning the intoxication → arousal → risk pathway included (3) whether subjective and physiological sexual arousal would function differently under conditions of high (maximized) versus low (suppressed) arousal and (4) whether these functions would vary based on participant sex and alcohol dosage.

Experiment 1

METHOD

Participants—Participants were recruited through campus newspaper advertisements and flyers for a study on “alcohol and social perception.” Callers were informed that to participate, they must be at least occasional drinkers, between 21 and 35 years of age,¹ heterosexual (self-defined), and native English speakers. Exclusion criteria—based on self-report—were current problem drinking² and/or taking medications or having a health

¹The main rationale for the age range is to bracket ages at which men and women are highly likely to engage in risky sexual behaviors. The range represents a balance among four concerns: minimum drinking age, age of appreciable risk, enhanced external validity, and reduced threat to internal validity. Therefore, although the selected range constrains generalizability; it captures an age of risk; it enables greater generalizability than is usual in experiments with college samples; and yet it does not allow age to become a potential source of excessive error variance.

condition contraindicated with alcohol consumption. The final sample ($N = 115$; 51% women) had a mean age of 24.9 years ($SD = 3.6$); consumed an average of 6.1 ($SD = 5.5$) drinks per week and was predominantly unmarried (91%), and educated (92% with some college). The self-identified racial/ethnic makeup of the sample was 77% European American, 8% Asian/Pacific-Islander, 3% African-American, and 12% other (including multiracial). Participants received \$8/hr for their participation in the study.

Procedure

Pre-experimental instructions and initial procedures: After screening and scheduling, each participant was instructed to bring photo identification, not to drive to the lab, not to eat or consume caloric drinks for the preceding three hours, and not to drink alcohol or use recreational or over-the-counter drugs for the preceding 24 hours. Upon arrival, participants were greeted by a same-sex experimenter (E), escorted to a private room, and provided information about the study procedures, risks, and benefits. All participants signed an informed consent form and were conducted through the session individually. Participants were screened to verify their age, to ensure that they had followed the pre-experimental instructions, and to ensure that they were free of any medical conditions or prescription drug usage that would contraindicate drinking alcohol. Women were required to complete a urinalysis to ensure that they were not pregnant; none resulted in a positive or ambiguous result. Participants were then administered a breath test (breathalyzer) to ensure that their baseline blood alcohol level (BAL) was zero. After E left the room, participants completed background questionnaires via computer, including measures of social desirability, sexual attitudes, and alcohol expectancies, which are beyond the scope of the present paper.

Beverage administration: Participants were randomly assigned to one of three beverage conditions: low alcohol dose (target peak BAL = .04, $n = 27$), moderate-high alcohol dose (target peak BAL = .08, $n = 29$), or control (no alcohol, $n = 59$). To control for individual differences in speed of alcohol absorption—and, consequently, elapsed time and number of breath samples to criterion—each participant in an alcohol condition was yoked to a control participant who was required to provide the same number of breath samples over the same number of minutes before beginning the experimental vignette (George et al., 2004; Giancola & Zeichner, 1997). Alcoholic beverages consisted of four parts orange juice to one part 100 proof vodka. Control beverages consisted of five parts of pure orange juice. Dosage was determined by dosage condition, body weight, and biological sex (Gussler-Burkhardt & Giancola, 2005). In the high dose condition, women received .69 g ethanol/kg body weight whereas men received .82 g ethanol/kg body weight. In the low dose condition, women received .35 g/kg body weight whereas men received .41 g/kg body weight³. Beverages were divided into three equal portions, and participants consumed each portion over a period of 3 minutes for a total of 9 minutes. BALs were measured at regular intervals, and participants were given accurate BAL feedback. Participants were required to reach a criterion BAL in order to proceed to the experimental vignette to ensure that participants within conditions began the vignette at comparable BALs and to control for blood alcohol curve limb effects by having participants complete the vignette while their BALs were ascending. Criterion BALs were determined by extensive piloting (George et al., 2004) and were set at .030 (en route to .04) and .065 (en route to .08) for low dose and high dose participants, respectively.

²Potential participants were excluded if they reported having been told by a professional that they had a problem with alcohol, ever seriously concerned about their own drinking, or treated or advised to seek treatment for drinking. For callers screened across studies, approximately 2% of women and 3% of men were excluded on the basis of these three problem-drinking items.

³The total amount of beverage consumed was based on sex, bodyweight, and beverage condition. For example, based on our formula for the high dose condition, a 150 lb man would receive 704 ml of total beverage and a 150 lb woman would receive 589 ml of total beverage.

Sexual arousal induction: After participants reached their criterion BALs, they were presented with a film clip viewing task, which was designed to induce subjective sexual arousal. Participants were shown 16 10-second sexual film clips depicting a variety of consensual sexual activities including caressing, kissing, masturbation, and heterosexual vaginal, oral, and anal sex. Participants then rated their subjective sexual arousal. The task took approximately 10 minutes.

Sexual risk-taking vignette: After the sexual arousal induction procedure, participants were presented with an eroticized vignette (~ 1000 words) describing a first date between a man and a woman that was written in the second person to facilitate participants' ability to project themselves into the storyline as the protagonist. Instructions for the vignette were as follows: "You are now going to read a brief scenario and answer some questions. Imagine that you are the person being described in the scenario and try and put yourself in the situation. When the scenario involves drinking, imagine that you have had a similar amount to drink as you have had today in the lab." For participants in alcohol conditions, the protagonist in the vignette was drinking alcohol while for those in the control condition, the protagonist was drinking soft drinks. For female participants, their dating companion in the story was named "Dan," and the companion for male participants was named "Ellen." Regardless of the protagonist's drinking, the companion was drinking beer to control for the potential confounding influence of companion's drinking. In addition, the female character was consistently portrayed as being on oral contraceptives to minimize the potential confounding influence of concerns about pregnancy. In the vignette, the couple was introduced at a party by a mutual friend. There was a mutual attraction over the course of the evening, and the companion invited the protagonist back to his/her place. Eventually, the couple progressed from kissing to petting under clothes and then realized that neither person had a condom. During the vignette, the storyline was interrupted to assess participants' intention to engage in various sexual activities.

After reading the vignette and responding to questions regarding their intention to engage in sexual activities, participants rated to what extent the vignette depicted a realistic situation that might happen to a normal 21-35 year old man, a normal 21-35 year old woman, and to the participant himself/herself on a Likert-type scale ranging from 1 "very unrealistic" to 5 "very realistic." Mean ratings for these questions were 4.3 ($SD = 1.0$), 4.1 ($SD = 1.1$), and 4.0 ($SD = 1.2$), respectively. Participants also rated the extent to which they found the vignette to be sexually arousing on a Likert-type scale ranging from 1 "not arousing at all" to 5 "very arousing." The mean rating was 4.0 ($SD = 1.0$).

After completing the vignette, participants were debriefed and paid \$8 per hour for their participation. Those who received alcohol were required to remain in the laboratory until they had detoxified to a blood alcohol concentration of 0.03 or below.

Measures

Subjective sexual arousal: After the sexual arousal induction, prior to the vignette, participants were administered a mood questionnaire including four items intended to assess participants' sexual arousal. Participants were asked to rate to what extent they felt each of 25 emotions in the preceding moments on a 5-point Likert scale, ranging from 1 = "very slightly or not at all" to 5 = "extremely". Sexual arousal was measured by computing a mean of the following items: sexually aroused, excited, interested, and horny. The scale alpha was .92. The overall mean was 3.20 ($SD = 1.02$). Example filler items are as follows: happy, angry, proud, ashamed, jittery, lonely, guilty, and alert.

Likelihood of unprotected sex: Participants answered four items concerning the likelihood that they would engage in various sexual activities as protagonist of the vignette. The items were as follows: “At this point, how likely are you to have sex with Dan (Ellen) even if he (she) does not have a condom?” “At this point, how likely are you to perform oral sex on Ellen/Dan?” “At this point, how likely are you to rub your clitoris against Dan’s penis (your penis against Ellen’s clitoris)?” and “At this point, how likely are you to allow Dan to put his penis inside of you (to put your penis inside of Ellen)?” Each item was rated on a Likert scale ranging from 1 “not at all” to 5 “very much.” To reflect sexual risk-taking intentions, a likelihood of unprotected sex scale score was calculated by taking a mean of these items; the scale alpha was .82. The overall mean was 2.85 ($SD = 1.19$).

RESULTS

Path Analysis—Following procedures recommended by Pedhazur (1997), path analysis using multiple regression was used to examine whether subjective sexual arousal mediated effects of biological sex and alcohol dose on likelihood of unprotected sex. First, likelihood of unprotected sex was regressed on subjective sexual arousal, alcohol condition (.00%, .04%, .08%), and biological sex. Next, subjective sexual arousal was regressed on alcohol dose (coded 0, 1, 2) and biological sex. Figure 1 shows the path model. All paths that are drawn were tested; only those in bold were significant. The interaction between biological sex and alcohol was examined but omitted from the model because it was not significant. Table 1 presents the bivariate correlations.

Self-reported sexual arousal after the arousal induction: For self-reported sexual arousal, the regression equation accounted for approximately 5% of the variance, $R^2 = .05$, $p = .054$. As hypothesized and shown in Figure 1, beverage condition significantly predicted self-reported sexual arousal, with intoxicated participants reporting greater sexual arousal than sober participants, $\beta = .19$, $p < .05$. Post hoc tests indicated that high dose (.08%) participants ($M = 3.56$; $SD = 0.94$) reported greater sexual arousal than control participants ($M = 3.06$; $SD = 1.08$). Low dose (.04%) participants ($M = 3.16$; $SD = 0.89$) did not significantly differ from either group. There was no significant effect for biological sex.

Likelihood of unprotected sex: For estimates of likelihood of unprotected sex, the regression equation accounted for approximately 19% of the variance, $R^2 = .19$, $p < .001$. Figure 1 shows that, as hypothesized, self-reported sexual arousal significantly predicted sexual risk likelihood, $\beta = .23$, $p < .05$, with estimated likelihood increasing as self-reported sexual arousal increased. There was also a significant effect of biological sex, $\beta = -.30$, $p < .01$. Women ($M = 2.48$; $SD = 1.05$) reported a lower likelihood of engaging in unprotected sex than men did ($M = 3.24$; $SD = 1.22$). There were no other significant effects. See Fig. 1 for the path model.

DISCUSSION

As hypothesized, alcohol intoxication, when considered alone, increased sexual risk-taking. Consistent with the Maisto et al. (2004) finding, only the higher dosage increased risk-taking beyond the control condition. When intoxication and self-reported sexual arousal were examined simultaneously, alcohol had no direct impact on risky sexual behavior likelihood. This pattern marks two noteworthy points. First, consistent with Abbey et al.’s (2005) findings, sexual arousal had predictive value over and above alcohol’s effects. Sexual arousal is a potent motivational state and experiencing oneself as being aroused exerts a strong proximal and direct influence on sexual decisions and does so even against the backdrop of another salient phenomenological state, being acutely intoxicated. Second, the states of being intoxicated and aroused were not independent. Consistent with our

hypothesis, alcohol's risk-taking effects were indirect via postdrinking increases in subjective sexual arousal.

This is the first demonstration of alcohol affecting risk-taking via arousal and it portrays subjective arousal as a potentially important mechanism in understanding alcohol-involved sexual risk. Moderate to high levels of intoxication may set the stage for experiencing a comparably heightened sense of subjective sexual arousal, which functions as a proximal and direct motivational state steering the individual toward sexually gratifying outcomes in spite of manifest health risks.

Support for this arousal-as-mechanism claim in our data, however, was limited in two ways. First, because sexual arousal was allowed to vary freely, its link to risk taking was correlational. Without arousal being manipulated systematically, whether it has a causal impact on risk-taking cannot be ascertained. Second, only subjective arousal was considered. A more complete evaluation of this mechanism argument necessitates the use of physiological, as well as subjective, indicators of sexual arousal. This is especially so given long established evidence that increasing intoxication attenuates physiological sexual arousal (Wilson, 1977). An intermediary mechanistic role for sexual arousal may be limited to the subjective dimension, which would suggest that physiological sexual arousal functions differently in intoxicated risk-taking. To advance a fuller understanding of how sexual arousal influences intoxicated risk-taking, arousal should be varied systematically and physiological arousal should be evaluated concurrently.

Finally, our biological sex findings were perfectly consistent with those observed in the other experiments (Abbey et al., 2005; Abbey, Saenz, Buck, Parkhill, & Hayman, 2006): Men reported higher risk-taking than women, and biological sex did not interact with alcohol. Compared to women, men's greater willingness to engage in risky sex has generally been attributed to their more liberal attitudes about casual sex (Oliver & Hyde, 1993). Also, use of explicit erotica in the present paradigm may have contributed to sex differences because men tend to respond more favorably than women to explicit erotica (e.g., Lopez & George, 1995).

Experiment 2

Four key objectives were addressed in follow-up experiments, based on the findings, questions, and limitations characterizing Experiment 1. First, to evaluate the role of sexual arousal more fully, we examined it bi-dimensionally, including both physiological genital response and subjective self-report. Because genital response assessment protocols and metrics differ across the sexes (Laan & Everaerd, 1995) and because the correspondence between subjective and physiological indices of sexual responding is also understood to differ across the sexes (Rellini, McCall, Randall, & Meston, 2005), meaningful statistical comparisons were precluded, and we therefore examined men (Experiment 2) and women (Experiment 3) separately.

Second, we expanded the range of alcohol dosages examined (target BALs = .00, .06, .08, .10). This range permitted us to include the most risk-inducing dosage from Experiment 1 while evaluating two additional dosages, one above and one below it. The top dosage (target BAL = .10) constitutes one of the highest tested in alcohol risky sex experiments to date. Nonetheless, it is likely that, in many real-life encounters of alcohol-involved sexual risk-taking, individuals achieve higher levels of intoxication than typically studied in lab experiments.

Third, to evaluate more directly the causal importance of sexual arousal, which was measured but not manipulated in Experiment 1, we varied arousal systematically by

instructing participants either to maximize or to suppress their arousal. Research unrelated to alcohol has shown that sexually functional men (e.g., Hatch, 1981) and women (e.g., Beck & Baldwin, 1994) can control their genital arousal such that they show higher arousal levels under instructions to maximize arousal than under instructions to suppress it (Barlow, 1986). Consistent with Experiment 1 findings, the MacDonald et al. (2000) findings, and our earlier reasoning about arousal's proximal motivational impact, we hypothesized that men experiencing higher arousal by virtue of instructions to maximize arousal would exhibit greater risk than would men experiencing lower arousal by virtue of instructions to suppress it.

Fourth, we hypothesized that subjective sexual arousal would again function as a mechanism through which alcohol would exert indirect effects, such that intoxication would enhance sexual arousal which would, in turn, enhance risk-taking. Less clear is the role of physiological sexual arousal. There has been no research linking physiological genital arousal to sexual risk-taking, and research linking intoxication to genital arousal has generated inconsistent findings. Initial studies found that high intoxication attenuated men's penile tumescence (e.g., Rubin & Henson, 1976), but later studies found null effects (e.g., Morlet et al., 1990). Recently, we found that high alcohol dosages (target BALs of .08%, .10%) had negligible attenuating effects on genital arousal to erotic films (George et al., 2006). In light of these recent data and methodological confounds raised about some initial findings (see George & Stoner, 2000), we did not anticipate that alcohol would attenuate genital arousal. Therefore, we expected that physiological arousal—unlike subjective arousal—would not in itself influence alcohol's effects on risk. However, because men show high correspondence between physiological and subjective indices of sexual arousal (Nobre et al., 2004), we anticipated that genital arousal would be indirectly associated with risk-taking through its association with subjective arousal.

METHOD

Participants—Participants ($n = 165$ men) were recruited for a study on “social drinking and decision-making” by ads in campus and community newspapers and flyers posted in urban zip codes associated with high HIV prevalence. Callers interested in participating were told that procedures included the use of sexually explicit films and physiological measures of sexual arousal and were screened to determine eligibility. Callers were informed that to participate, they must be between 21 and 35 years of age; interested in dating opposite-sex partners; not currently in a committed dating relationship; and a social drinker (drinks at least five drinks per week). Exclusion criteria were self-reported current problem drinking or a history of problem drinking and/or currently taking medications or having a health condition that contraindicated alcohol consumption. The final sample had a mean age of 25.0 years ($SD = 3.8$), consumed a mean of 15.7 drinks/week ($SD = 11.5$), and was predominantly European-American (76%); 4% were African-American, 5% were Asian, 2% were Native American, 5% were Latino, and 6% were multi-racial. Most (59%) were employed; 44% were students, 67% reported an income of less than \$31,000/yr. They received \$15/hr for study participation. Table 2 lists descriptive data for all three samples.

Measures

Stimulus films: Participants viewed a sexually neutral 2.5 min long documentary about birds (BBC-TV) followed by two three min erotic films (New Era Productions and VCA Productions). Pilot testing established that the erotic films induced equivalent self-reported increases in sexual arousal. Both erotic films explicitly depicted normative, consensual sexual activities (kissing, oral sex, and vaginal intercourse) between a man and a woman.

Physiological sexual arousal: Sexual arousal was measured using penile plethysmography (BioPac Systems, Inc., Santa Barbara, CA; model MP 150) and a mercury-in-rubber strain gauge (D. M. Davis Inc. Hackensack, NJ) positioned mid-shaft on the penis (E. Janssen, personal communication, July, 2002). Gauges were disinfected following each use with a glutaraldehyde solution (Cidex OPA, Advanced Sterilization Products, Irvine, CA). Using Acqknowledge software (version 3.7.2), data were collected at a rate of 62.5 samples per sec then reduced to 25 samples per sec. Movement artifacts, defined as clear spikes of more than 5 mm in an otherwise smooth curve, were deleted based on visual inspection of raw data. Data were then digitally transformed and exported to statistical software for analysis (Hoffmann, Janssen, & Turner, 2004). Fig. 2 illustrates overall means for our measure of male participants' physiological sexual arousal to the neutral stimulus and the risky sex vignette stimulus, which are plotted in millimeters and correspond to the left Y-axis (note the solid line).

Self-reported ratings of sexual arousal: Participants rated their perceived level of arousal on a 4-item seven-point Likert scale (e.g. 1 = "no sexual arousal at all"; 7 = "extremely sexually aroused"). Items were: (1) "Overall, how much sexual arousal did you feel during the story?" (Heiman, 1977); (2) "To what extent did you feel sensation in your genitals during the story?" (Heiman & Rowland, 1983); (3) "How much sexual warmth (in your genitals, breasts, and body) did you feel during the story?" (Meston & Heiman, 1998); (4) "To what extent did you feel sexually absorbed in the sensory components of the story?" (Koukounas & McCabe, 2001). Ratings on these four items were averaged to form a sexual arousal scale with good inter-item reliability ($\alpha = .94$). Figure 2 presents overall means for male participants' subjective sexual arousal to the neutral and risky sex vignette stimuli, corresponding to the center Y-axis (note the grey columns).

Sexual risk-taking vignette and likelihood of unprotected sex: The same vignette paradigm and 5-point Likert-type likelihood of unprotected sex dependent measures used in Experiment 1 were used to assess hypothetical sexual risk-taking and yielded acceptable reliability ($\alpha = .79$).

Procedure

Beverage administration: Except for differences described below, the pre-experimental instructions, initial procedures, and beverage administration were the same as in Experiment 1. Each participant was weighed to determine the amount of alcohol mixed with fruit juice needed to achieve target BAL (g ethanol/kg body weight for .06% target BAL = .612; .08% target BAL dosage = .816 for men; .10% target BAL dosage = 1.02 for men). BALs were tested every three minutes until they reached criterion (BAL .03% for .06% target, .045% for .08% target, and .06% for .10% target) after which participants began the experimental procedures. Mean pre-assessment BAL was .048% ($SD = .012$) for the .06% target group, .060% ($SD = .010$) for the .08% target group, and .070% ($SD = .011$) for the .10% target BAL group.

Sexual arousal instructional set: Participants were randomly assigned to a maximize-arousal or suppress-arousal instructional set condition. Experimenters were blind to participants' arousal instruction condition. Before the story, participants received audio instructions informing them of their arousal instruction and directing them to open an envelope containing a card reiterating this instruction. In the maximize condition, the instructions were to "try as much as possible to relax and maximize your arousal during the remainder of the experiment. We would like you to try and become as aroused as possible." In the suppress condition, the instructions were to "try as much as possible to suppress your

sexual arousal during the remainder of the experiment. In other words, please keep from becoming sexually aroused.”

Sexual risk-taking assessment: After reaching criterion BAL, participants placed the gauge. Participants watched the neutral and erotic films, rated their sexual arousal, and read and responded to the sexual risk-taking vignette. They were then instructed by intercom to remove the gauge. Afterwards, participants were debriefed and paid, following a detoxification period when necessary.

RESULTS

Path Analysis—Table 3 presents the bivariate correlations among the Study 2 measures (above the diagonal). To test our hypotheses, path analysis was conducted using structural equation modeling and maximum likelihood estimation with robust standard errors (MLR estimator, Mplus statistical software version 4; Muthén & Muthén, 2007). Figure 3 illustrates the path model. All paths that are drawn were tested, $\chi^2(1) = .016$, $p = .844$, CFI = 1.000, TLI = 1.103, RMSEA = 0.000, SRMR = 0.002; only those in bold were significant.

Genital response during the sexual risk-taking vignette was computed by subtracting each participant's lowest achieved mm circumference during the neutral stimulus from his highest achieved mm circumference during the vignette and dividing the difference by his lowest achieved mm circumference during the neutral stimulus to yield his percentage of circumference change from baseline.

Genital response during the sexual risk-taking vignette: For genital response during the sexual risk-taking vignette, the model predicted 6% of the variance. As hypothesized, arousal instructional set condition predicted physiological sexual arousal, $\beta = .24$, $p < .01$; participants instructed to maximize their arousal achieved a greater percentage circumference change from baseline than those instructed to suppress their arousal. There were no other significant effects.

Self-reported sexual arousal during the sexual risk-taking vignette: For self-reported sexual arousal, the model predicted 5% of the variance. As hypothesized, genital response during the sexual risk-taking vignette significantly predicted self-reported sexual arousal, $\beta = .47$, $p < .001$. Participants who experienced a greater percentage circumference change from baseline reported greater sexual arousal than those who experienced less of a circumference change from baseline. Additionally, alcohol condition significantly predicted self-reported sexual arousal; the more intoxicated participants were, the more sexual arousal they reported, $\beta = .18$, $p < .05$. Post hoc tests indicated that high dose (.10%) participants ($M = 4.63$; $SD = 1.48$) reported greater sexual arousal than moderate-high dose (.08%) participants ($M = 3.82$; $SD = 1.60$) and control participants ($M = 3.71$; $SD = 1.55$). Moderate dose (.06%) participants ($M = 4.28$; $SD = 1.48$) did not differ from any other group. There were no other significant effects.

Likelihood of unprotected sex: For estimates of likelihood of unprotected sex, the model predicted 23% of the variance. As hypothesized, self-reported sexual arousal during the risk-taking vignette significantly predicted unprotected sex likelihood, $\beta = .47$, $p < .001$, with estimated likelihood increasing as self-reported sexual arousal increased. In addition to direct effects, we tested the significance of indirect effects, as recommended by Bryan et al. (2007). Results showed that alcohol condition had a significant indirect effect on likelihood of unprotected sex via subjective sexual arousal, $\beta = .09$, $p < .05$.

DISCUSSION

Alcohol-induced increases in subjective sexual arousal, but not physiological sexual arousal, led to increased sexual risk taking. The sexual arousal instruction manipulation produced differences in physiological genital arousal. This self-controlled heightening and suppressing of genital arousal was directly related to subjective sexual arousal and indirectly related to risk-taking. As hypothesized, compared to the lower arousal exhibited by men in the suppress condition, the higher arousal experienced by men in the maximize condition resulted in greater subjective sexual arousal, which in turn led to greater likelihood of sexual risk-taking. Thus, regardless of whether subjective sexual arousal was free to vary, as in Experiment 1, or whether it was manipulated via instructional set, increased subjective sexual arousal led to increased sexual risk-taking. Also, as hypothesized, subjective sexual arousal mediated the effects of alcohol on risk-taking; alcohol had no direct effects on risk-taking. In keeping with recently published guidelines for mediational analyses in HIV/AIDS research (Bryan et al., 2007), we used structural equation modeling to evaluate our mediational hypotheses. This approach allowed us to simultaneously consider multiple mediational paths, ruling out paths dependent on genital arousal and bolstered confidence in the mediational effects observed. Taken together, these findings provide strong support for the idea that subjective sexual arousal functions causally as an important mechanism in determining alcohol's effects on men's sexual risk-taking. The well-established correspondence between men's genital and subjective arousal (Nobre et al., 2004) was evident in our findings and suggests that what mattered most in men's risk-taking was their perceptions of how aroused they felt.

Experiment 3

Based on Experiment 1 findings, we anticipated that alcohol and subjective arousal would increase women's self-reported likelihood of sexual risk-taking and that alcohol's effects would be indirect via subjective sexual arousal. However, scant evidence exists for formulating hypotheses about the role of women's physiological sexual arousal. Despite evidence linking alcohol to women's sexual risk-taking as well as other sexual experiences (Norris, Masters, & Zawacki, 2004), basic research delineating alcohol's effects on women's sexual physiological responding remains scarce. Only two studies have evaluated the effects of alcohol on women's vaginal arousal (Wilson & Lawson, 1976, 1978). Both reported that alcohol had an attenuating effect. Consequently, while hypothesizing that alcohol would increase subjective sexual arousal, we anticipated opposite effects on physiological arousal. These opposing predictions are consistent with conclusions advanced by Crowe and George (1989) and with evidence for low correspondence between indices of subjective and physiological sexual responding among women (Laan & Everaerd, 1995). No hypotheses were proffered about the role of physiological responding on women's intoxicated risk-taking.

We anticipated that instructional set would affect women's subjective and physiological arousal. Several studies not examining alcohol have shown that sexually functional women can control their genital arousal such that they show higher arousal levels under instructions to maximize arousal than to suppress arousal (e.g., Beck & Baldwin, 1994). Therefore, as with men, we hypothesized that women experiencing higher arousal due to instructions to maximize arousal would exhibit greater risk taking than would women experiencing lower arousal due to instructions to suppress.

METHOD

Participants—Participants ($n = 173$ women) had a mean age of 25.3 years ($SD = 3.8$), consumed a mean of 10.6 drinks/week ($SD = 7.5$), and were predominately European-

American (75%); 6% were African-American, 6% were Asian, 2% were Native American, 6% were Latina, and 7% were multi-racial. Most (71%) were employed, 41% were students, and 75% reported an income of less than \$31,000/year.

Procedure and Measures—The sampling and methods were identical to Experiment 2 except for the following. Female sexual arousal was measured using vaginal photoplethysmography (Geer, Morokoff, & Greenwood, 1974; BioPac Systems, Inc., Santa Barbara, CA; model MP 150) by means of a tampon-sized Plexiglas vaginal probe inserted into the vagina (Behavioral Technology, Inc., Salt Lake City, UT). Probes were disinfected following each use with a glutaraldehyde solution (Cidex OPA, Advanced Sterilization Products, Irvine, CA). The probe, which contains a light-emitting diode and a photo cell, measures changes in the amount of light backscattered from the vaginal wall as it becomes engorged with blood, termed vaginal pulse amplitude (VPA). Using Acqknowledge software (version 3.7.2, BioPac Systems, Inc.), VPA was continuously sampled at a rate of 62.5 samples per second, reduced to 25 samples per second, and transformed into digital data for analysis. Movement artifacts revealed through visual inspection were removed from waveform data, which were then reduced to 30-second base-to-peak millivolt means. Following digital transformation, remaining movement artifacts, defined as a 100% increase or decrease in VPA relative to the adjacent 30-second intervals, were smoothed by averaging the values of the adjacent intervals (Schacht et al., 2007). Self-reported sexual arousal ($\alpha = .95$) and risk taking ($\alpha = .75$) were measured reliably with Study 2 scales using the same 7-point and 5-point Likert-type scales (respectively). Fig. 2 shows overall means for our measure of female participants' physiological sexual arousal, which are plotted in millivolts and correspond to the right Y-axis (note the dashed line). Means for female participants' subjective arousal are plotted on the center Y-axis (note the white columns).

Before alcohol administration, women were given a pregnancy test (Osom hCG-Urine Test, Genzyme General Diagnostics, San Diego, CA) to ascertain that they were not pregnant. Weight- and sex-adjusted alcohol dosing resulted in women receiving .514, .686, or .857 g ethanol/kg body weight, respectively, for the .06%, .08%, and .10% target BALs.

RESULTS

Path Analysis—Table 3 presents the bivariate correlations among the Study 3 measures (below the diagonal). Figure 4 depicts the path model for Study 3. Identical analytic strategies were used in Experiments 2 and 3 with one exception. Genital response during the sexual risk-taking vignette was computed by subtracting women's lowest achieved VPA during the neutral stimulus from their highest achieved VPA during the risk-taking vignette to yield a VPA difference score (Rellini & Meston, 2006; Schacht et al., 2007). All paths that are drawn were tested, $\chi^2(1) = .039$, $p = .844$, CFI = 1.000, TLI = 1.299, RMSEA = 0.000, SRMR = 0.004; only those in bold were significant.

Genital response during the sexual risk-taking vignette: For women's genital response during the sexual risk-taking vignette, the model predicted 3% of the variance. Contrary to our hypothesis, there was no effect of arousal instructional set on physiological sexual arousal. However, alcohol condition significantly predicted women's genital response during the risk-taking vignette, $\beta = -.16$, $p < .05$. Post hoc tests indicated that control participants ($M = .036$; $SD = .030$) had a significantly larger change in VPA from neutral video than high dose (.10%) participants ($M = .024$; $SD = .019$). Moderate dose (.06%) participants ($M = .028$; $SD = .022$) and moderate-high dose (.08%) participants ($M = .029$; $SD = .021$) did not differ from any of the other groups. There were no other significant effects.

Self-reported sexual arousal during the sexual risk-taking vignette: For women's self-reported sexual arousal, the model accounted for 11% of the variance. As hypothesized, the more intoxicated participants were, the more sexual arousal they reported, $\beta = .31$, $p < .001$. Post hoc tests indicated that, compared to control participants ($M = 4.16$; $SD = 1.52$), moderate dose (.06%) participants ($M = 4.82$; $SD = 1.37$), moderate-high dose (.08%) participants ($M = 4.82$; $SD = 1.48$), and high dose (.10%) participants ($M = 5.42$; $SD = 1.22$) reported significantly higher sexual arousal. Contrary to our hypothesis, there was no effect of instructional set on self-reported sexual arousal.

Likelihood of unprotected sex: For women's estimates of their likelihood of unprotected sex, the model accounted for 8% of the variance. As hypothesized, self-reported sexual arousal during the risk-taking vignette significantly predicted self-reported likelihood of unprotected sex, $\beta = .28$, $p = .001$, with estimated likelihood increasing as self-reported sexual arousal increased. There were no other significant direct effects. Again following procedures recommended by Bryan et al. (2007), we tested the significance of indirect effects. Results showed that alcohol condition had a significant indirect effect on likelihood of unprotected sex via subjective sexual arousal, $\beta = .09$, $p < .01$.

DISCUSSION

The basic alcohol→subjective arousal→risk pathway identified in Experiment 1 and replicated among men in Experiment 2 was essentially the same for women in Experiment 3. Alcohol increased subjective sexual arousal, which in turn increased self-reported likelihood of risk-taking. This finding suggests that no sex difference exists in this core dynamic of alcohol-involved risky sexual encounters. In both men and women, alcohol increased their subjective sense of being sexually aroused, and this sense contributed to greater willingness to engage in unprotected intercourse. Also, among women as with men, alcohol had no direct effects on risk-taking; its impact was exerted only via subjective arousal.

Apart from this core similarity, women exhibited important differences relative to men. First, arousal instructions surprisingly had no effect on women's responding. This finding diverges from earlier evidence that women can increase or decrease their physiological and subjective arousal in accordance with instructional set (Beck & Baldwin, 1994). Methodological differences between previous studies and the current work may have contributed to this discrepancy, such as inclusion of beverage administration procedures and use of computerized vignettes. Because arousal control is thought to be accomplished primarily through cognitive strategies (Beck & Baldwin, 1994), it is possible that these and other unspecified methodological differences interrupted women's optimal use of such strategies. Second, alcohol attenuated women's physiological genital arousal. This finding concurs with two previous experiments (Wilson & Lawson, 1976, 1978). Therefore, among now three studies reporting alcohol effects on vaginal responding, all have reported attenuation effects. Thus, although alcohol has inconsistent and context specific effects on men's genital arousal, it appears to attenuate women's genital arousal more generally. Third, alcohol increased subjective responding while decreasing physiological responding. This suggests genital arousal operates differently in men and women. In sum, subjective sexual arousal was as pivotal for women's intoxicated risk-taking as men's. However, given several paradoxical patterns for women and paucity of alcohol and sexual studies, it is clear that more experimentation is needed to elucidate alcohol's sexual effects in women.

GENERAL DISCUSSION

Most risky first-time sexual encounters involve being sexually aroused and many involve being inebriated. Yet, little scientific information has been available about the intersection of these critical in-the-moment states. Our experiments showed that these states and associated

sexual decisions can be credibly analogized experimentally and that these states operate jointly to heighten risk-taking for both men and women. Alcohol intoxication led to increased subjective sexual arousal. Also, escalating subjective sexual arousal—whether or not it occurred in the context of self-regulating instructions—led to increased risk-taking.

Subjective sexual arousal proved such a crucial factor that all of alcohol's impact was a product of its effect on arousal. Indeed, alcohol surprisingly had no direct effects on risk-taking when subjective arousal was considered. This finding, obtained across all three experiments, spotlighted subjective sexual arousal as a pivotal mechanism in understanding alcohol's effects on risk-taking. Use of structural equation modeling to evaluate multiple mediational paths in the Experiment 2 and 3 data supported subjective arousal's mediational role in post-drinking risk. Indeed, these are the first findings to indicate that alcohol's effects on sexual arousal can determine how alcohol increases sexual risk-taking. Strikingly, concurrent physiological genital arousal was not helpful in these studies for understanding alcohol's impact on risk-taking. Men's genital arousal, which was highly correlated with their subjective arousal, was not significantly affected by alcohol. For women, genital arousal was attenuated by alcohol but unrelated to sexual risk-taking.

Alcohol Myopia Theory (AMT) remains the leading theory for explaining intoxicated risk-taking (MacDonald et al., 1996, 2000). It posits a pharmacologically-determined tunnel vision that focuses the drinker on salient factors that impel risk-taking and diverts attention from less salient considerations that might otherwise inhibit risk-taking when sober. Our findings emphasize that a prominent focal point for an intoxicated person is the phenomenological state of perceiving oneself as sexually aroused. From an AMT framework, alcohol seemed to focus the person myopically and fixedly on this internal sense of feeling sexual, thereby intensifying the impact of this feeling state and its capacity to motivate the person toward sexual gratification despite prevailing risks. Alongside evidence that alcohol focuses the drinker cognitively on impelling cues in sexually risky situations and that a weighted composite of impelling/inhibiting cognitions can mediate alcohol's effect (Davis, Hendershot, George, Norris, & Heiman, 2007; MacDonald, Fong, Zanna, & Martineau, 2000), the current findings stress the importance of the alcohol-induced focus on one's affective or sensorial state of feeling sexual. This suggests that AMT explanations of sexual risk-taking should expand beyond primarily cognitive characterizations to incorporate sexual and affective variables that are motivationally congruent with risky outcomes.

It should be noted that, traditionally, AMT has been applied primarily to explain post-drinking *behavioral* outcomes, rather than subjective states. While AMT has proven useful to explain alcohol enhanced subjective sexual arousal, the explanation essentially breaks new ground and should thus be considered speculative. It is noteworthy that the enhancement effects on subjective sexual arousal observed here are, at first blush, inconsistent with recently reported null effects at the same alcohol dosages (George et al., 2006). On closer scrutiny, however, these discrepant findings are in keeping with the Inhibition Conflict Model of AMT (Steele & Josephs, 1990; Steele & Southwick, 1985), which may reasonably be applied to post-drinking subjective sexual arousal. This more specific model postulates that alcohol facilitates disinhibitory behavioral outcomes only under high conflict conditions where impelling and inhibiting pressures/cues are both strong. Because the vignette used here contained both impelling (willing partner) and inhibiting (risks associated with unprotected intercourse) cues, it could be construed as engendering high inhibition conflict. In the context of our vignette, alcohol could "disinhibit" subjective sexual arousal by reducing attention to inhibitory cues and thereby resulting in an intensified focus on cues impelling an erotic response. In contrast, the stimulus materials utilized in the George et al. (2006) study were largely devoid of inhibitory cues. Under such low-conflict conditions, the Inhibition Conflict Model of AMT would predict null effects for alcohol.

Apart from AMT considerations, how else might one explain alcohol enhancement effects on arousal? One possibility is to invoke alcohol's anxiolytic effects. Sayette (1993) posited that alcohol dampens anxiety by impairing cognitive processes required for accurately appraising and evaluating anxiety inducing stimuli. Therefore, intoxicated men may have experienced less anxiety and anxiety related distractions – such as spectating – than their sober counterparts. On the other hand, in light of the null (men) and negative (women) alcohol effects on physiological findings, it is worth cautioning that alcohol may not have enhanced subjective sexual arousal *per se*, but only increased the willingness either to acknowledge arousal to oneself or to report it to others. More research is needed to evaluate the merit of these speculations about explanatory mechanisms.

Potential applied implications of these findings for prevention programs are that there may be merit in providing educational content that distinguishes between genital versus subjective sexual arousal, emphasizes the decisional impact of the latter over the former, and indicates that alcohol works to increase risk in part by enhancing subjective arousal. Also, because subjective arousal was so pivotal, our findings suggest that arousal-reduction strategies could prove a useful adjunct to prevention content. For instance, in situations that involve risky partners or condom unavailability, efforts to suppress subjective arousal may aid in mitigating risk. However, because the particular arousal-reduction strategies we used were only effective for men, further research is needed identifying effective strategies for women and their potential for mitigating risk. Alternatively, indications of heightened subjective sexual arousal can come to be construed as prompts for seeking condoms or pursuing an early exit from an evolving sexual encounter.

The current set of studies has both strengths and limitations. Alcohol factors, including dosage, BAL limb (insuring that participants were on the ascending limb of alcohol metabolism rather than on the descending limb⁴), and absorption times were tightly controlled through rigorous procedures involving consistent BAL monitoring, specific BAL criterion starting points, and yoked controls. To better approximate real-world risky sexual situations, an arousal induction protocol, utilizing erotic films as priming stimuli and an eroticization of the experimental vignette, was used to create a highly charged sexual ambiance during the sexual decision-making process. Although lab analogues of sexual situations can never fully capture all elements—including the timing of sexual stimuli and alcohol intake—involved in real sexual situations, participants reported that the vignette was very realistic and arousing. Such vignettes have effectively analogized real-life encounters for both sexual risk-taking (Stoner, George, Norris, & Peters, 2007) and sexual assault (Davis, George, & Norris, 2004; Norris et al., 2006). Another strength was present in our sampling of both college students and urban community residents, including higher risk individuals recruited from neighborhoods with elevated HIV prevalence rates, bolstering the generalizability of our findings.

Limitations include the lack of an alcohol expectancy (placebo) condition, arbitrary ordering of physiological and subjective arousal assessments, sample characteristics, and the prospect of volunteer bias. First, previous work has shown that expectancy set (the belief that one has been drinking) enhances men's self-reported and physiological arousal independent of low-dosage intoxication (Wilson & Lawson, 1976). Because of the difficulty of convincing placebo participants that they have received a high dose of alcohol, and because our specific interest was in high dosage effects, we did not manipulate alcohol expectancy set. At high

⁴Research has shown that alcohol's effects on many dependent measures vary based on BAL limb (Pohorecky, 1977). Alcohol effects assessed at a particular BAL on the ascending portion—or “limb”—of the alcohol metabolism curve when the BAL is rising are distinguishable from effects assessed at an equivalent BAL on the descending limb when BAL is falling. Consequently, failure to control for BAL limb in alcohol experiments introduces unnecessary error variance.

dosages, physiological effects were expected to override any expectancy effects. Likewise, physiological effects were expected to override any effects due to “vignette-drinking,” which was varied to match actual drinking (alcohol subjects were told that they consumed alcohol in the vignette, while control subjects were told that they consumed soda). Given the wide range of blood alcohol levels examined and the direction of the effects, however, expectancy effects and vignette-drinking effects cannot be ruled out as having influenced the findings.

Second, regarding the sexual arousal protocol, we assessed physiological sexual arousal before subjective sexual arousal and evaluated whether the former predicted the latter. This sequential order was somewhat arbitrary and does not imply that physiological arousal necessarily precedes or determines subjective arousal. Third, despite utilizing a more sexually charged protocol than typically utilized in sexual risk experiments, the levels of arousal experienced in the lab likely under-represent the maximum levels experienced in real life and the generalizability of our findings should be seen in this context. Fourth, although there is evidence and theory indicating that sexually functional and sexually dysfunctional individuals respond differently to sexual arousal protocols (e.g., Barlow, 1986), we were unable to discriminate sexually functional and dysfunctional participants in this study.⁵ Future research should consider the interesting possibility that alcohol’s effects on sexual risk taking via sexual arousal may vary systematically based on sexual functioning.

Finally, sample characteristics, such as exclusion of heavy problem drinkers, limit the generalizability of our findings. For example, because age influences erectile functioning, younger men may be less affected by alcohol than older men (Blanchard & Barbaree, 2005). In addition, volunteers for sexual psychophysiological studies tend to have more sexual experience and liberal sexual attitudes than non-volunteers (Strassberg & Lowe, 1995). Our findings should be interpreted accordingly.

This research analogized heat-of-the-moment processes preceding sex and revealed important new evidence about sexual arousal as a mechanism underlying alcohol’s role in fostering unsafe sex related to HIV and other sexually transmitted infections. Acute intoxication heightened willingness to engage in unprotected intercourse with a new casual partner. Subjective, but not physiological, sexual arousal also heightened unsafe sex and it indirectly accounted for alcohol’s effect on unsafe sex. Thus, in highly sexually charged situations, moderately high levels of intoxication foster risky sex by facilitating the drinker’s subjective sense of being aroused. These findings encourage continued basic research further delineating the role of subjective sexual arousal, particularly in the context of AMT. These findings also suggest applied research exploring the utility of deliberately targeting subjective arousal in prevention interventions as a way of mitigating alcohol’s effects on risky sex.

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⁵We administered an instrument designed to assess sexual functioning. Unfortunately, however, these data proved uninterpretable because many items assumed regularized access to a sexual partner yet participants were recruited in part on the basis of being “not currently in a committed dating relationship.” Consequently, we were unable to effectively discriminate sexually functional from dysfunctional participants in this sample.

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Figure 1.

Path model from Experiment 1. All paths that are shown were tested; only those in bold were significant. $^* p < .05$, $^{**} p < .01$.

Figure 2.

Men (Experiment 2) and women's (Experiment 3) physiological and subjective arousal to the neutral stimulus and to the risky sex vignette stimulus. Subjective arousal (columns) is plotted on the center Y-axis. Men's physiological arousal (solid line) is plotted on the left Y-axis. Women's physiological arousal (dashed line) is plotted on the right Y-axis.

Figure 3.

Path model from Experiment 2. All paths that are shown were tested; only those in bold were significant. * $p < .05$, ** $p < .01$, *** $p < .001$.

Figure 4.

Path model from Experiment 3. . All paths that are shown were tested; only those in bold were significant. * $p < .05$, ** $p < .01$, *** $p < .001$

Table 1

Bivariate Correlations among Variables in Study 1

Variable	1	2	3	4
1. Biological Sex	--	-.03	-.13	-.32***
2. Alcohol Dosage		--	.20*	.21*
3. Self-reported Sexual Arousal			--	..30***
4. Likelihood of Unprotected Sex				--

* Note. $p < .05$.

** $p < .01$.

*** $p < .001$.

Table 2

	Experiment 1 (N = 115)			Experiment 2 (N = 165)			Experiment 3 (N = 173)		
	<i>M</i>	<i>SD</i>	%	<i>M</i>	<i>SD</i>	%	<i>M</i>	<i>SD</i>	%
Women	---	---	51	---	---	0	---	---	100
Age	24.9	3.6	---	25.0	3.8	---	25.3	3.8	---
Age first consensual intercourse	17.0	2.0	---	16.8	2.4	---	17.0	2.6	---
Drinks per week	6.1	5.5	---	15.7	11.5	---	10.6	7.5	---
Currently a student	---	---	N/A	---	---	44	---	---	41
Currently employed	---	---	N/A	---	---	59	---	---	71
Annual income < \$31,000	---	---	N/A	---	---	67	---	---	75
Ethnicity									
European American/White	---	---	77	---	---	76	---	---	75
African American/Black	---	---	3	---	---	4	---	---	6
Asian American	---	---	8	---	---	5	---	---	6
Native American	---	---	N/A	---	---	2	---	---	2
Latino/Hispanic	---	---	N/A	---	---	5	---	---	6
Other/Multiracial	---	---	12	---	---	6	---	---	7

Note. N/A means not assessed.

Table 3**Bivariate Correlations among Variables in Studies 2 and 3**

Variable	1	2	3	4	5
1. Alcohol Dosage	--	-.01	.00	.18 [*]	.16 [*]
2. Arousal Instructional Set	-.01	--	.23 ^{**}	.12	.05
3. Genital Response	-.18 [*]	.07	--	.47 ^{***}	.17 [*]
4. Self-reported Sexual Arousal	.31 ^{***}	.10	.04	--	.47 ^{***}
5. Likelihood of Unprotected Sex	.08	.00	.01	.28 ^{***}	--

Note. Correlations for Study 2 (men) are presented above the diagonal, and correlations for Study 3 (women) are below the diagonal.

^{*}
 $p < .05$.

^{**}
 $p < .01$.

^{***}
 $p < .001$.