Some Original Findings: Comments on Fillmore and Weafer

Fillmore and Weafer (2004) devised a modified stop-signal paradigm by adding cues predicting whether a go or no-go target will be presented, and manipulating the correctness of the information conveyed by the cues. They found that incorrect go cues preceding no-go targets resulted in more alcohol-induced failures to inhibit in men, but not in women. This is a new finding that raises many interesting questions about the factors and processes that might account for these effects.

One possible answer to these questions stemmed from the evidence that subjective stimulation during rising blood alcohol concentrations (BACs) was higher in men than in women. This raised the possibility that heightened stimulation may contribute to inhibitory failures with incorrect go cues. A regression of these inhibitory failures on stimulation ratings could assist in evaluating this idea. Since greater sedation is commonly reported when BACs decline, it would also be interesting to determine whether the gender difference in these inhibitory failures abates on the declining limb of the BAC curve.

The use of cues in the present research is similar to the procedure used in learning studies to investigate ‘preparatory set’ by manipulating the temporal regularity or correctness of cues for the response to be made (Mower 1940). Such work has shown that correct cues speed reaction time (RT) whereas incorrect cues slow RT. This accords with the cue effects on RT to go targets obtained in the current experiment. The slowing effect of incorrect cues on RT is thought to reflect the extra processing time needed to switch from one response and select another (Schmidt 1988). This RT switching has been used to test response flexibility under alcohol when drinkers try to inhibit a pre-potent response and make a different one (Easdon & Vogel-Sprott 2000). That research found failures to inhibit were associated with reduced flexibility. If alcohol-impaired flexibility contributes to inhibition failures with incorrect cues for no-go targets, the RT when inhibition fails may be slower following incorrect rather than correct cues. If it contributes to gender differences, this effect should be more pronounced in male than in female social drinkers.

The stop-signal paradigm presents no predictive cues, and studies using the stop-signal task have shown that alcohol reliably reduces inhibitions to stop (no-go) targets (e.g. Mulvihill, Skilling & Vogel-Sprott 1997; de Wit, Crean & Richards 2000). In contrast, the present study showed that alcohol failed to affect inhibitions when cues correctly predicted a no-go target. This is a potentially important finding because it carries safety implications, suggesting that situations providing drinkers with correct cues for appropriate behavior may counteract the disinhibiting behavioral effects of alcohol. In contrast, the adverse effect of alcohol on inhibitory control may be more intense in settings that provide no cues, or incorrect ones.

The cued go/no-go paradigm used by Fillmore and Weafer seems to have more ecological validity than the stop-signal paradigm because drinking situations commonly contain unreliable or ambiguous cues for appropriate behavior. Their experiment provides provocative findings that seem to parallel observations of more pronounced disinhibited, aggressive behavior in men than in women. The study also generates many interesting questions about the underlying mechanisms and factors that might account for these gender differences in inhibitory control. Additional research with the cued go/no-go task promises to add valuable insights on the puzzle of alcohol-impaired control of inhibition.

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GENDER DIFFERENCES IN ALCOHOL’S DISINHIBITING EFFECTS: A COMMENTARY ON FILLMORE AND WEAFER

Fillmore & Weafer (2004) present some very interesting and potentially important results suggesting that men are more susceptible than women to both the disinhibiting and stimulating effects of a moderate dose of alcohol. Research suggests that men are more susceptible than women to the disinhibiting effects of alcohol on aggression (Giancola & Zeichner 1995; Giancola et al. 2002), and, it has been well documented that men drink more and have more alcohol-related problems than women (Williams et al. 1989; Finn & Hall 2004). The present results suggest a potential mechanism that might contribute to these important gender differences. Alcohol may compromise inhibitory control of certain processes in men more than women, resulting in a tendency for men to consume more drinks on a single occasion than women, and a tendency for men to be more aggressive than women when intoxicated. Although the gender differences observed by Fillmore and Weafer make sense given the known gender differences in excessive alcohol use and alcohol-related problems, a number of questions need to be systematically addressed to establish the reliability of these effects.

The first word of caution stems from the small sample size used in this study. Larger sample sizes are necessary to ensure that the observed gender differences are not just an oddity of the small sample. Furthermore, if these are true gender differences then it is important to assess whether they are reliable across time, dose level, blood alcohol concentration limb location, risk-status, and context. In fact, systematic study of the reliability and correlates of gender differences in the disinhibiting effects of alcohol would be helpful in uncovering the mechanisms responsible for this effect, if it is a true effect, and their relevance for broader gender differences in alcohol consumption and problems. If the differences are observed only at specific dosage levels or in specific contexts, then this might clarify some of the mechanisms underlying this effect. The authors discuss this issue to some degree when noting that gender differences in the disinhibiting effects of alcohol have not been observed on some inhibitory control tasks, such as the the stop-signal task (Mulvihill, Skilling & Vogel-Sprott 1997). We also have not observed gender differences in the disinhibiting effect of alcohol on a different go/no-go task that uses monetary incentives and punishments to activate or inhibit behavior (Finn et al. 1999). Gender differences also might vary in relation to risk status. For instance, while moderate drinking women appear to have significantly fewer alcohol problems than moderate drinking men, heavy drinking women appear to have equal or more alcohol-related problems than heavy-drinking men (Wilsnack, Wilsnack & Klassen 1984), suggesting that heavy drinking women may be more disinhibited than heavy-drinking men. Finally, the authors offer an interesting speculation that the gender differences in alcohol’s stimulating effects may contribute to the observed differences in alcohol’s disinhibiting effect. This is intuitively appealing speculation, but the authors can directly test this speculation by assessing whether alcohol-induced decreases in response inhibition are correlated with alcohol-induced increases in subjective stimulation.

In summary, the gender differences in alcohol’s disinhibiting effect observed by Fillmore and Weafer are potentially important and may have relevance for gender differences in alcohol consumption and problems. However, it is important to systematically study these effects in larger samples that vary in risk status while using different tasks, doses, and contexts. Such work will be very valuable to our understanding of broader gender differences in alcohol use and abuse and the mechanisms contributing to these differences.

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INDIVIDUAL AND BACK AGAIN

Students of the ecology of alcohol-related problems are struck by the consistent correlations observed between locations of alcohol outlets, especially bars, and rates of violence in community areas (Stockwell & Gruenewald 2004). These correlations have been demonstrated across many studies, at many units of analysis, reflect both densities of alcohol outlets and alcohol sales, and are independent of broad population and place characteristics. Students of drinkers’ behavior in bars, and especially ‘risky bars’, are struck by the multifaceted nature of social interactions within these premises, the competitive atmosphere of many of these places, and again the amount of violence that may occur (Homel, Mellwain & Carvolth 2001). Certainly, the criminogenic aspects of these places, the ‘cultures’ in which bar patrons live, somehow combined with the use of alcohol, may explain some of these relationships (Graham et al. 1980). But one might wonder if something else, something more simple and interesting, is afoot.

Perhaps bars are places where the sociogenic and alcogenic sources of violence meet. Bars are often places where very many social and physical interactions take place, often among demographic groups more, rather than less, prone to aggression and violence (e.g. young males). Bars are places that feature the use of alcohol as part of normal social activity. If the use of alcohol intrinsically affects judgement, altering the ability of the user to decide on a course of action that leads to less, rather than more, violence, then we should always expect at the population level to see more, rather than less, violence around alcohol outlets. In this case, regardless of ‘culture’, bars (or their social equivalents) should always be associated with these risks.

The excellent work of Fillmore & Wenaer (2004) paints a picture of the effects of alcohol use upon subjective stimulation and impaired inhibitory control that supports these concerns. As the authors suggest, gender differences in alcohol-induced aggression may be related to greater ‘cultural’ acceptability of aggressive behavior among men (Eagly & Steffen 1996) or from a fundamental difference in the degree to which alcohol disrupts basic mechanisms of behavioral control.’ Regardless of the source of these differences, it is argued that alcohol may reduce the ability to inhibit a response to a stimulus once it has been initiated. With suitable controls for blood alcohol levels, they demonstrate that the ability to inhibit such ‘pre-potent’ responses among men is reduced with the use of moderate doses of alcohol. In addition, although men and women were matched on blood alcohol level, their subjective impressions of intoxication differed; men reported stimulation, women reported sedation. This observation begs the question as to whether women who experience doses of alcohol as a stimulative would exhibit disinhibition, a possibility not explored in the study. But, nevertheless, a consistent picture, for males, of an inability, on average, to inhibit ‘pre-potent’ responses when using alcohol is presented. Combined with our current knowledge about the neurocognitive processes that support alcohol myopia (Steele & Josephs 1990), this picture is actually somewhat distressing: is it the case that the use of alcohol in bar settings both reduces young males’ accounting of the long-term costs of current behaviors (alcohol myopia) and their abilities to inhibit ‘pre-potent’ responses to stimuli that may elicit aggression?

Why is an ecologist concerned about this?

Ecologists generally tend to worry about small things in a big way. General frameworks for mathematically modelling ecological systems (e.g. epidemiological models of disease, Brauer & Castillo-Chavez 2001) focus upon the large-scale population implications of rather small events, such as the contact of a susceptible individual with an infected host. At the population level, multitudes of these rather small events are summarized in terms of contact processes (i.e. interactions between people) and have large-scale implications for the spread and control of disease (e.g. the SARS epidemic). The same is likely true for the growth and spread of alcohol-related violence: as we distribute bars around our communities, we are providing contact sites for individuals to interact, support, and produce violence. This is a simple process that may have large-scale implications for communities everywhere, the establishment of core
group that support violence in settings that extend beyond the local bar (e.g. intimate partner violence). Continued work like that of Fillmore & Weaver (2004) will identify those ‘simple’ processes that enable the development of ecological models of alcohol-related problems and establish rational models for the regulation of alcohol outlets.

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DISINHIBITION, IMPULSE CONTROL, AROUSAL AND GENDER: UNDERSTANDING THE MECHANISMS OF ALCOHOL’S EFFECTS ON AGGRESSION

This study (Fillmore & Weaver 2004) tests gender differences in ‘inhibitory control’ as measured by the cued go/no-go task. The authors suggest that the results have implications for understanding the relationship between alcohol and aggression in that ‘a weakened inhibitory mechanism’ is related to ‘extreme or disinhibited behavior’ i.e. alcohol -> reduced inhibitory control -> increase aggression.

The use of terms such as ‘inhibitory control’ and ‘disinhibited behavior’ harks back to the notion of ‘disinhibition’ as a general explanation for alcohol-related aggression (see Room & Collins 1982). ‘Disinhibition’ is a rather vague and often tautological explanation of aggression which has been largely rejected by most reviewers (see Graham 1980; Giancola 2000; Pernanen 1976, 1993); however, aspects relating to disinhibition or inhibitory control are often reflected in more accepted explanations of alcohol-related aggression such as reduced impulse control, poorer cue perception and impaired decision-making (Giancola 2000; Graham et al. 1997, 1998; Hoaken, Giancola & Phl 1998; Pernanen 1976).

So the question is – does this study give a more meaningful and specific definition of ‘disinhibition’ and in so doing increase our understanding of the role of alcohol in aggression? In general, the link with aggression is not well made. I am not against so-called ‘reductionist’ approaches – in my opinion, it is foolhardy to ignore the pharmacological effects of alcohol in the development of explanatory models relating to social behaviors such as aggression. However, I would like to see more about the external validity of the cued go/no-go task as an analogue for inhibitory behavior in the context of social interaction. The task seems rather mechanical and not necessarily relevant to social and emotional responding. The other critical piece that is missing in terms of the causal links described above is any evidence that performance on the cued go/no-go task is actually related to aggression.

Could arousal be the key?

Although the paper focuses on inhibitory control, it includes a potentially more viable or basic explanation of alcohol-related aggression, namely, alcohol-related arousal (Graham & Wells 2001, 2003; Graham et al. 2000; Pihl & Hoaken 2002). Specifically, it is possible that gender differences in inhibitory control can be explained by gender differences in arousal. For example, arousal might mediate the relationship between alcohol and inhibitory control, that is, alcohol -> heightened arousal -> reduced inhibitory control -> aggression. If this were true, then inhibitory control would still be the mechanism linking alcohol to aggression but only for those who experience heightened arousal. Another possibility is that arousal affects both inhibitory control and aggression but that the relationship between reduced inhibitory control and aggression is small or even spurious. Unfortunately, although the authors acknowledge in the discussion that arousal level may be an important factor in their findings, they do not actually test the potential mediating role of arousal in the alcohol-inhibitory control relationship.

Despite these concerns, I think the paper is valuable. There are a number of reasons, both social and biological, why men and women may experience alcohol’s
effects differently, and this study is important because it highlights the potential for exploring gender differences in mechanisms linking alcohol to aggression. For example, gender differences in both arousal and inhibitory control may provide important insight not only in the occurrence of aggression but also relating to gender differences in the escalation of aggression (Martin & Bachman 1997).

The findings from this study also point to a number of areas for further research. First, it would be useful to examine gender differences in the effects of alcohol on other cognitive functions linked to increased risk of aggression such as risk taking, cue perception and problem solving. Second, if arousal is actually a key variable linking alcohol and aggression, it would be important to explore the added impact of high arousal drinking environments (which are found in some public drinking establishments) on the frequency and severity of aggression.

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AN EXTRAORDINARILY INTERESTING ARTICLE: COMMENTS ON FILLMORE AND WEAFER

Two taken-for-granted facts in the criminologist’s universe are that most crime (especially violent crime) is perpetrated by males, and that a great deal of crime is alcohol-related. Put young men and easy access to alcohol together, and one has, by common consent, the ingredients for ‘a hot time in the old town tonight.’ Of course the causal status of alcohol is widely disputed. Many criminologists of a sociological bent argue, for example, that alcohol-related violence by men, especially in disadvantaged or indigenous communities, reflects the specific socially structured ways in which alcohol is used, as well as the dependent or powerless state of the users (Homel, Lincoln & Herd 1999). Aboriginal leaders counter that the alcohol epidemic, whatever its social origins, is a powerful force in its own right perpetuating intolerable levels of violence by men against women (Aboriginal & Torres Strait Islander Women’s Task Force on Violence 2000; Pearson 2001).

These criminological debates illustrate a wider dispute: to what extent is ethanol the substance that influences mood, modifies behaviour, impairs inhibitory controls, and heightens the risk of aggression and violence, and to what extent are social and cultural factors to blame? Should those—like myself—who are interested in prevention concentrate on modifying physical and social environments or on controlling drinking itself? In this respect the article by Fillmore & Weafer (2004) is extraordinarily...
interesting, giving me pause for thought. The challenge to my sociological biases is particularly acute given the avowedly reductionist approach of the authors.

My world is not that of the laboratory and I don’t read the experimental literature much. Perhaps therefore I was more surprised than I should have been when I read that ‘To date, studies based on behavioral control models have not reported gender differences in the degree to which alcohol impairs response inhibition. . . little is known about potential gender differences in the impairing effect of alcohol on inhibitory control.’ (Fillmore & Weafer 2004, p. 4). This, it seems, is because experiments have not been designed to investigate the issue. How could such a fundamental question be overlooked for so long? How could the issue of gender differences, that is so fundamental to the social and behavioural sciences, be so marginalised in this specialised field?

Although it is perhaps 50 years late, I am impressed with the thoroughness of Fillmore and Weafer’s experiment and the way that they rule out competing explanations for their results. It does seem that there are real gender differences in alcohol-induced aggression, at least in situations where ‘response pre-potency’ must be overcome to inhibit responding to a ‘no-go’ target. But I was left wondering what the ‘real world’ parallels are to such experimental manipulations.

Certainly there is no lack of evidence for gender effects in research on violence in licensed environments (Babor et al. 2003). In our own work in nightclubs in Sydney (Homel & Clark 1994) violence instigated by men occurred at least three times more often than female-instigated violence, with non-physical aggression an even more masculine phenomenon. What female-initiated violence occurred tended to be observed mainly in ‘skid-row’ establishments which were qualitatively unlike the mainstream venues that were the main study focus. In a multivariate analysis of risk factors across all venues, male drunkenness and ‘round shouting’ by males emerged as key variables. The importance of male drunkenness (along with a range of non-alcohol factors such as physical comfort and the availability of transport) was confirmed in recent analyses of changed environmental factors related to large reductions in violence and aggression observed before and after the implementation of the safety action program in nightclubs in North Queensland in the mid-1990s (Homel et al. 2004).

What now needs to be done, in the light of Fillmore and Weafer’s experiment, is detailed qualitative analyses of the circumstances surrounding the observed incidents of aggression and violence. The question is how one could observe response pre-potency that must be overcome because of changed circumstances. One could perhaps hypothesise that in many cases intoxicated males—in contrast to females—became aggressive when their intention to act in a particular way was thwarted, perhaps by security or bar staff. If the experimental findings are consistent with observational data, one could then give detailed consideration to the implications for staff training. The more specific the preventive responses, and the more they are based on knowledge of the dynamics of male aggression, the more likely they are to succeed. The refinement of training programs could be one of the main ‘practical products’ of the type of experimental research that is the focus of this commentary.

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ALCOHOL IMPAIRMENT OF BEHAVIOR IN MEN AND WOMEN: A REPLY TO THE COMMENTARIES

We thank the commentators for their insightful remarks. Their diverse backgrounds offer a broad spectrum of perspectives on these findings, ranging from basic pharmacology to sociology and ecology.

Our primary finding in the study was that, compared with women, men experienced greater impairment of
inhibitory control and greater stimulant effects in response to a dose of alcohol. Vogel-Sprott (2004), Finn (2004), and Graham (2004) all raised questions about the role of the increased stimulation effects as a possible mediator for impaired inhibitory control observed in men. The possibility that impaired inhibitory control is a secondary effect that results from a general increase in stimulation or arousal by alcohol, is interesting because it would offer new insights into the circumstances in which alcohol and other psychoactive drugs are likely to result in disinhibited, under-controlled behavior. With regard to alcohol, much is known about the drug’s biphasic effects on arousal, with greater stimulation and arousal associated with the rising phase of the blood alcohol concentration (BAC) curve, and more sedative-like effects as BAC declines. To the extent that impaired inhibitory control results from general stimulation, the time-course of stimulation and sedation suggests that disinhibited effects should be most evident during the ascending limb of the BAC curve, early after drinking has begun. Such possible time-course effects on impaired inhibitory control need systematic study.

Although Graham (2004) argues that increased stimulation is a viable explanation for the observed impairment of inhibitory control under alcohol, such an inference should be cautioned. It is also entirely possible that alcohol-induced impairment of inhibitory control is independent of alcohol effects on arousal. Vogel-Sprott (2004) and Finn (2004) made an excellent suggestion that we test for a correlation between individual differences in inhibition failures and self-reported stimulation to determine if subjects who displayed poorer inhibitory control also reported greater stimulant effects under the drug. We performed correlation tests and found no significant relationship between these measures under alcohol or placebo in either gender group.

Graham (2004), citing early work by Room & Collins (1982) and Graham (1980), states that our use of the term inhibitory control ‘harks back to the notion of ‘disinhibition’ as a general explanation for aggression . . . that is a rather vague and often a tautological explanation of aggression’. We agree that early use of the ‘disinhibition’ term was vague and circular. At that time, impaired inhibitory control was inferred by observations of increased activity which did not necessarily indicate that alcohol actually reduced the ability to inhibit behavior. However, over the past two decades, cognitive neuroscience has made considerable advancement with respect to measuring the specific, covert act of inhibiting a behavioral response (Logan 1994; Fillmore 2003). In particular, stop-signal and go/no-go tasks have been developed that model behavioral control as the net effect of competitive activating and inhibiting processes. The models can better identify the source of impaired control by measuring the independent ability to activate and to inhibit a response. Most importantly, these models measure the covert act of response inhibition. To date, studies of alcohol effects based on these models have provided consistent evidence that moderate doses of the drug reduce behavioral control by impairing response inhibition while leaving the ability to activate behavior unaffected (Fillmore 2003). Thus alcohol’s apparent disinhibiting effects appear to be mediated primarily by acute impairment of normal inhibitory mechanisms. Thus, these process-model approaches, which our study represents, have advanced the field beyond the early, vague notions of disinhibition as simply ‘overactive’ and ‘aggressive’ behavior.

Homel (2004) and Gruenewald (2004) provide some interesting sociological and ecological perspective on our findings. Both commentators found that evidence for alcohol-impairment of a simple laboratory response to be intriguing, particularly given the absence of any obvious social or cultural factors traditionally associated with the display of alcohol-related impulsive and under-controlled behavior (e.g. a ‘risky bar’ setting or competitive atmosphere). Although social and cultural factors play a huge role in mediating behavioral control under alcohol (e.g. MacAndrew & Edgerton 1969), there is growing evidence for a direct pharmacological effect of alcohol on behavioral control. For example, neural systems associated with inhibitory and activational mechanisms of control also respond to the reinforcing properties of drugs via their effects on presynaptic dopaminergic nerve terminals that stimulate dopaminergic pathways of the mesoaccumbens (Braver, Barch & Cohen 1999; Cohen, Braver & Brown 2002). Both commentators also speculate as to how our finding of impaired inhibition of pre-potent responses under alcohol might relate to observations of alcohol-related violence. We found that impairment was restricted to the condition in which invalid go cues made responses pre-potent. Thus, our findings highlight the importance of response pre-potency (i.e. instigation) in the display of alcohol-induced impairment of inhibitory control. This condition may parallel a more complex social situation in which a drinker’s motivated, goal-directed behavior is somehow obstructed, prompting a violent response. Controlled laboratory studies of alcohol effects on complex social interaction could address these types of hypotheses.

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