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Effects of Acute Alcohol Consumption on the Processing of Emotion in Faces:

Implications for Understanding Alcohol-Related Aggression

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Abstract

The negative consequences of chronic alcohol abuse are well known, but heavy episodic consumption ("binge drinking") is also associated with significant personal and societal harms. Aggressive tendencies are increased after alcohol but the mechanisms underlying these changes are not fully understood. While effects on behavioural control are likely to be important, other effects may be involved given the widespread action of alcohol. Altered processing of social signals is associated with changes in social behaviours, including aggression, but until recently there has been little research investigating the effects of acute alcohol consumption on these outcomes.

Recent work investigating the effects of acute alcohol on emotional face processing has suggested reduced sensitivity to submissive signals (sad faces) and increased perceptual bias towards provocative signals (angry faces) after alcohol consumption, which may play a role in alcohol-related aggression.

Here we discuss a putative mechanism that may explain how alcohol consumption influences emotional processing and subsequent aggressive responding, via disruption of OFC-amygdala connectivity. While the importance of emotional processing on social behaviours is well established, research into acute alcohol consumption and emotional processing is still in its infancy. Further research is needed and we outline a research agenda to address gaps in the literature.

Effects of Acute Alcohol Consumption and Processing of Emotion in Faces:
Implications for Understanding Alcohol-Related Aggression

1. Introduction
2. Alcohol and aggression
3. Emotional facial processing and behaviour
4. Alcohol and emotional face processing
5. A neurological framework for the effects of alcohol within the emotional brain
6. Individual differences
7. Conclusions

1. Introduction

While a link between aggression and acute alcohol consumption is well established [Bushman *et al.*, 1990, Chermack *et al.*, 1997, Hoaken *et al.*, 2003], the nature of this relationship is not fully understood. Alcohol may increase aggression directly, but a direct pharmacological mechanism is somewhat undermined by the fact that not every drinking episode results in heightened aggression or violence. Instead, alcohol's pharmacological action may be indirect, via disruption of a cognitively-mediated system that, in turn, increases the likelihood of an aggressive response [Bushman, 1997]. At doses consistent with normal consumption, alcohol causes disruption of frontal areas of the brain that control executive functions, and these frontal effects may mediate the alcohol-aggression relationship [Giancola, 2000; 2004]. However, areas within the frontal cortex, such as the orbitofrontal cortex (OFC), are also involved in the perception and regulation of emotion. Effective emotional processing is vital for social

functioning, and dysregulation of emotional circuitry has been identified as a factor underlying impulsive aggression [Davidson *et al.*, 2000]. Therefore, disruption of these processes may represent an additional mechanism by which acute alcohol consumption increases aggression. For example, in normal functioning there is a tendency to attend to emotionally-valenced stimuli, particularly if the stimuli convey threat (e.g., angry faces) [Holmes *et al.*, 2009]. This is adaptive as it facilitates early identification of potentially hazardous encounters, but can result in inappropriate behavioural responses when impaired [Davidson *et al.*, 2000]. Furthermore, deficits in emotional processing have been reported in those with aggressive traits [Kimonis *et al.*, 2006, Hoaken *et al.*, 2007, Lee *et al.*, 2009], and similar deficits have been identified following chronic alcohol abuse [Oscar-Berman *et al.*, 1990, Townshend *et al.*, 2003, Foisy *et al.*, 2007].

Here we review the literature on the effects of alcohol on the processing of emotionally salient cues, and argue that dysregulated emotional processing following acute alcohol consumption mediates alcohol-related aggression. We suggest that disrupted processing of emotionally-salient cues after alcohol instigates aberrant social and emotional responses, which are further exacerbated by the concomitant disruption of behavioural control and evaluation of behavioural consequences.

2. Alcohol and Aggression

There is an extensive literature examining the association between both chronic and acute alcohol consumption and various types of aggressive behaviour [Pihl *et al.*, 2009], including interpersonal violence between strangers [Cogan *et al.*, 2006] intimate partner aggression [Leonard *et al.*, 1992, Leonard *et al.*, 1996, Foran *et al.*, 2008], sexual aggression [Testa, 2002], violent crime [Murdoch *et al.*, 1990, Zhang *et al.*, 1997, McClelland *et al.*, 2001, Franklin *et al.*,

2010] and homicide [Darke, 2010]. Forensic data have identified general alcohol consumption as a risk factor for violent crime [Murdoch *et al.*, 1990, Lipsey *et al.*, 1997, Lundholm *et al.*, 2012], and this may be stronger in societies where drinking to intoxication is common [Graham *et al.*, 2011]. Studies in the general population show that lifetime / habitual levels of drinking correlate positively with the perpetration of aggressive acts [Scott *et al.*, 1999, Wells *et al.*, 2000, Bye, 2007], as well as the likelihood of being the victim of violence [Scott *et al.*, 1999, Shepherd *et al.*, 2006]. Furthermore, greater alcohol outlet density and longer opening hours are associated with greater reports of violent crimes and injury resulting from violence [Ray *et al.*, 2008, Schofield *et al.*, 2013], particularly in low income and high population density areas [Resko *et al.*, 2010, Liang *et al.*, 2011, Livingston, 2011, Mair *et al.*, 2013].

The effect of alcohol on aggression may be greater for physical displays of aggression, rather than verbal aggression [Wells *et al.*, 2000], and when heaviness of drinking or intoxication, rather than frequency, are considered [Wells *et al.*, 2000, Richardson *et al.*, 2003, Wells *et al.*, 2003]. Many studies have compared lifetime or general drinking rates with general aggression. However, event level studies (which analyse whether alcohol was consumed at the time of an aggressive incident), indicate that a high proportion of aggressive episodes occur after one or more of the individuals involved in the incident have recently consumed alcohol. A Canadian study of the general population reported that individuals had consumed alcohol in 38% of arguments, 57% of threats and 68% of physical assaults [Wells *et al.*, 2000], while a study of US college freshmen reported that alcohol consumption occurred in 9% of cases of general aggression, and 28% of cases of sexual aggression [Stappenbeck *et al.*, 2010].

These findings are compelling but, historically, much of the data have been drawn from clinical and criminal cohort studies that often lack appropriate comparison groups, and only

include cases that result in police involvement or hospitalisation. More recently, there has been an increase in the number of population-based surveys, utilising existing national statistics or telephone / postal survey methods, but response biases and memory recall inaccuracies are problematic when using self-report retrospective accounts. In particular, the correlational nature of the data precludes causal inferences, while the focus on general patterns of use does not address the specific effects of acute consumption. Experimental studies that directly manipulate alcohol consumption address the question of causality and can examine other important features of the relationship, such as individual differences. Findings from animal studies support a causal link between acute alcohol consumption and aggression, and have demonstrated positive effects of alcohol on relevant variables, including greater risk of victimisation [Miczek *et al.*, 1977, Miczek *et al.*, 1984], lengthening of aggressive episodes [Miczek *et al.*, 1992], disinhibition of suppressed behaviours (including suppressed aggression in unfamiliar environments) [Miczek *et al.*, 1980, Miczek *et al.*, 1998] and reduced responsivity to submissive signals [Miczek *et al.*, 1997]. There is substantial individual variation, with only a minority of animals consistently displaying aggressive behaviours after alcohol [Miczek *et al.*, 1998, Fish *et al.*, 2002]. Biological and behavioural explanations of this variation include differing levels of testosterone [Debold *et al.*, 1985], serotonin receptor mRNA expression in the pre-frontal cortex [Chiavegatto *et al.*, 2010], and baseline/trait levels of aggression [Blanchard *et al.*, 1987].

Most laboratory experiments of human aggressive behaviour have utilised variants of the Buss teacher-learner [Buss, 1961] or Taylor reaction time [Taylor, 1967] tasks. In both, the participant is required to deliver an electric shock when a fictitious opponent (an experimental confederate) makes an error (Buss) or is slower to respond than the confederate in competitive trials (Taylor); the dependent variable is typically the average level of shock set by the

participant, although alternatives such as number of extreme shocks have been used [McCloskey *et al.*, 2009]. Despite some methodological differences across studies, the widespread use of these tasks enables reasonable comparability, and these support a causal relationship between alcohol and heightened aggression. In a meta-analysis of 30 studies (in males), Bushman and Cooper [1990] considered four comparisons: alcohol vs. placebo (i.e., non-alcoholic drink disguised as alcohol), alcohol vs. control (i.e. a non-alcoholic drink described as non-alcoholic and not disguised as alcohol), anti-placebo (i.e., an alcoholic beverage described as non-alcoholic) vs. control, and placebo vs. control. This enabled the independent analysis of alcohol and expectancy effects of alcohol, and indicated an effect of alcohol vs. placebo ($d = 0.61$, $CI = 0.51-0.70$). However, there was no evidence of an effect of anti-placebo vs. control (which best models a pure pharmacological comparison), leading the authors to conclude that the relationship is not due to a direct pharmacological effect of alcohol. However, Chermack and Taylor [1995] found effects of alcohol on aggression regardless of participants' expectancies. They suggest that the expectancy that alcohol increases aggression may facilitate intense levels of aggression under high provocation. In a narrative summary of research using the Taylor task, Taylor and Chermack [1993] suggested that aggression proceeds through sequential stages, identified as pre-escalation, escalation and post-escalation, and that differences can be observed between intoxicated and sober individuals throughout these stages. For example, intoxicated individuals are more likely to set higher shocks earlier (i.e., during the pre-escalation stage), and are slower to reduce levels of shock if their opponent does so. However, intoxicated individuals rarely set the highest level of shock, indicating that they retain a level of control, and are sensitive to social influence.

The alcohol-related aggression literature is large and diverse, with important limitations including differences in the populations studied (e.g., adolescents, college students, convicted criminals and clinical populations), and contradictory results possibly due to failure to identify and control for factors mediating or moderating the relationship. Despite this, the overall consensus indicates a positive causal relationship between acute alcohol consumption and aggression. Nevertheless, alcohol consumption clearly does not *always* result in aggression across individuals and situations. Therefore, alcohol consumption *per se* is not sufficient to explain heightened aggression, and is likely to operate via cognitive mechanisms – exposure to alcohol cues also increases in aggression and aggressive cognitions, implying a role for learned associations [Subra *et al.*, 2010]. The specific mechanisms underlying the alcohol-aggression relationship are therefore complex, with alcohol affecting multiple systems and processes that make aggression more likely, but not inevitable [Graham *et al.*, 2000].

Several explanations of how alcohol consumption may increase the *probability* of aggression have been proposed. One of the most prominent is disinhibition of behaviour. It is now well established that alcohol consumption leads to impairments in cognitive functioning [Bartholow *et al.*, 2003, Casbon *et al.*, 2003], in particular executive function and behavioural control [Giancola, 2000]. These processes are controlled by prefrontal regions of the brain, which are particularly vulnerable to disruption and damage following acute and chronic alcohol consumption [Volkow *et al.*, 1995, Harper, 1998, Oscar-Berman *et al.*, 2007, Harris *et al.*, 2008, Makris *et al.*, 2008]. Therefore, socially unacceptable or risky behaviours may occur following alcohol consumption because frontal regulatory systems that usually inhibit impulsive responding are disrupted. There is evidence of individual sensitivity to these effects; for example, individuals high in sensation seeking show greater impairment in inhibitory control

after alcohol than those low in sensation seeking [Fillmore *et al.*, 2009]. If impairments in behavioural control mediate the alcohol-aggression relationship, we might expect more alcohol-related aggression in these susceptible individuals, for which there is some support [Cheong *et al.*, 1999].

Alcohol may also promote aggression indirectly via its anxiolytic properties. Sayette [1993] developed an appraisal-disruption model to account for alcohol's attenuation of the stress response. According to this model, stress-dampening effects occur because alcohol disrupts appraisal of stressful information, resulting in lower negative affect, autonomic arousal, and anxiety in relation to personal harms or social sanctions. Alcohol may therefore facilitate aggression by reducing anxiety and increasing approach behaviours in threatening or hostile situations, and/or by reducing tendencies to relent when threatened [Vogel-Sprott, 1967, Lindman, 1983].

According to a two-factor model of aggression [Geen, 1990], these "background" variables of behavioural disinhibition and anxiety reduction interact with a second environmental factor, related to frustrating or provocative cues, to induce arousal and anger. While these may be related to genuine threat, alcohol can also distort perception of social cues, such that intoxicated individuals are more likely to erroneously *perceive* threat and provocation [Zeichner *et al.*, 1979; 1980, Steele *et al.*, 1985, Parnanen, 1991].

Alcohol myopia theory (AMT) [Steele *et al.*, 1990] posits that alcohol disinhibits behaviour by narrowing attentional focus to the most salient environmental cues [Steele *et al.*, 1990, Giancola *et al.*, 2011]. This occurs due to alcohol's negative impact on cognitive processing capacity. AMT can be used to explain a wide range of disinhibited behaviours after alcohol, including risky sexual behaviours and drink driving [Giancola *et al.*, 2010]. In the case

of alcohol-related aggression, attention will be preferentially allocated to attention-grabbing provocative cues that signal (real or perceived) danger or attack, over non-provocative cues that may otherwise attenuate aggression. This implies that alcohol would decrease aggression when the salience of inhibitory cues is increased, which has been supported empirically [Giancola *et al.*, 2007].

Given the contexts in which alcohol is commonly consumed, we propose that social cues are of particular importance. More specifically, this myopic effect may extend to emotionally salient cues including facial expressions of emotion. In addition to the attentional changes implicated by AMT, we propose that alcohol alters *perception* of emotional cues, thereby increasing the likelihood of (erroneously) perceiving social cues as threatening. In line with this model, Nelson and Trainor [2007] identify impaired recognition of social cues and impulsivity as important components underlying uncontrolled aggression. We suggest that this, coupled with behavioural disinhibition and attentional myopia, creates a volatile cognitive state that significantly increases the likelihood of aggression following acute alcohol consumption.

3. Emotional facial processing and behaviour

The processing of emotional cues has been shown to impact on behaviour in the non-intoxicated state. Facial expressions are of particular importance as they are a rich source of emotional information, enabling the viewer to infer the thoughts, feelings, mood and intentions of others. Six primary emotional expressions (fear, sadness, disgust, anger, surprise and happiness) are recognised cross-culturally, suggesting that an innate neurobiological system exists to recognise these basic emotions. This is supported by work in infants showing that the use of social cues, including emotional faces, develops early [Mineka *et al.*, 1984, Klinnert *et al.*, 1987]. In infancy there is a preference for viewing positive versus negative faces, but as the child

develops negative faces are attended to for longer and are used increasingly to guide behaviour [Klannert, 1983]. The ability to recognise facial expressions in childhood is also predictive of later social and academic competence [Izard *et al.*, 2001], and impaired emotional face processing is observed in children with high levels of aggression [Kimonis *et al.*, 2006]. In adults, emotional expressions guide social interactions and behaviour; for example, facial displays of sadness and fear are distress cues that can inhibit aggression and promote prosociality in others [Miller *et al.*, 1988, Eisenberg *et al.*, 1989, Marsh *et al.*, 2007], while displays of anger can curtail unwanted or socially unacceptable behaviour [Blair *et al.*, 1999]. However, in some circumstances where there is perceived threat to the self, social status or resources, the primary adaptive response can be approach behaviour towards the source of threat in order to overcome it (if the threat is perceived as surmountable), rather than lose social position or resources [Wilkowski *et al.*, 2010].

The ability to quickly identify and respond to danger in the environment conveys substantial survival advantage, and threat-related stimuli therefore enjoy a processing advantage [Vuilleumier *et al.*, 2001, Calvo *et al.*, 2005]. This includes threat-related facial expressions [Holmes *et al.*, 2009, Feldmann-Wustefeld *et al.*, 2011, Marinetti *et al.*, 2012], even when presented at low levels of awareness [Mogg *et al.*, 1999]. However, this bias may lead to socially undesirable behaviour if dysregulated. In an integrated model of emotion and cognition in psychopathy, Blair [2005] proposes that a failure to efficiently process distress cues (e.g., sad or fearful faces) can result in inappropriate aggressive responses, due to failure to engage the normal inhibitory responses. Consistent with this model, impairments in processing of facial emotions is seen in aggressive or aggression-prone populations, including schizophrenic patients that present violent or psychopathic traits [Marwick *et al.*, 2008], violent offenders [Hoaken *et*

al., 2007], sex offenders [Gery *et al.*, 2009], intermittent explosive disorder patients [Best *et al.*, 2002], individuals with attention deficit/hyperactivity disorder symptoms [Sinzig *et al.*, 2008] and individuals with psychotic [Blair *et al.*, 2001] or anti-social [Marsh *et al.*, 2008, Pfabigan *et al.*, 2012] traits or disorders. In addition, children and adolescents identified as having disruptive or anti-social behaviour problems show deficits in facial emotion recognition [Wright *et al.*, 2000], and altered neural, physiological and behavioural responses to emotionally expressive faces and situations [Dougherty *et al.*, 1999, Aleman *et al.*, 2005] and negatively valenced and distressing stimuli [Sterzer *et al.*, 2005, Kimonis *et al.*, 2006].

These findings have been echoed in non-clinical and non-forensic samples. For example, Hall [2006] report that self-reported aggressive tendencies positively correlate with misidentification of anger in a facial recognition task. Yet arguably the strongest evidence for a causal effect of emotional recognition on behaviour comes from research showing behavioural changes following emotional recognition manipulation. Using an adapted psychophysical computer-based task, Penton-Voak *et al.* [Penton-Voak *et al.*, 2013] modified biases in emotion recognition by promoting recognition of happiness (over anger) in a group of healthy volunteers and a group of adolescents with problem behaviours (either criminal offenders or deemed “at risk” of criminal offending). Training resulted in a shift in bias to perceiving happiness in both cohorts relative to controls (“placebo” training). Furthermore training led to decreases in self-reported anger and aggression in both groups, and decreases in independently-rated aggression in the juvenile sample.

4. Alcohol and emotional face processing

Sterzer and colleagues [2005] suggest that the propensity towards aggressive behaviour in conduct disorder may be due to a combination of impairment in recognition of emotional stimuli

and the ability to control emotional behavioural responses. We argue that a similar profile of impairment may exist following acute alcohol consumption. A specific tendency to perceive provocation signalled by angry expressions, for example, paired with reduced behavioural control, may enhance the overall likelihood of aggressive behaviour when intoxicated. There are several key findings which support this. First, different emotional expressions seem to be predominantly processed by different brain regions [Adolphs, 2002]. It is therefore plausible that site-specific disruption induced by a pharmacological agent, such as alcohol, could cause selective impairment in the processing of specific emotional expressions, such as those signalling threat. Second, disrupted facial processing is present in several population subtypes associated with heightened levels of aggression. While a causal relationship needs to be established empirically, this link supports the hypotheses that alcohol-related disruption to facial processing could promote some incidence of aggression. Third, emotional processing impairments have been observed following chronic alcohol abuse, indicating that alcohol disrupts processes that underlie emotional processing at least when taken repeatedly.

Alcohol-dependent participants show deficits in the recognition of emotional facial expressions when compared to healthy control groups and patients with a range of neurological disorders [Hulshoff Pol *et al.*, 2001]. These deficits include slower identification times [Foisy *et al.*, 2007], reduced identification accuracy [Schneider *et al.*, 1998], overestimations of emotional intensity [Philippot *et al.*, 1999] and hostile attribution bias (i.e., a tendency to erroneously label expressions such as sadness or disgust as anger) [Philippot *et al.*, 1999, Frigerio *et al.*, 2002]. Altered emotional face processing may be of particular importance as it appears to be related to social and interpersonal problems that are common in alcoholism [Kornreich *et al.*, 2001].

Although the underlying nature of these deficits are not known, there are several plausible explanations, including problems in visuospatial processing that may precede alcohol dependency [Philippot *et al.*, 1999], altered amygdala sensitivity, dysregulation of pre-frontal regions [Pfefferbaum *et al.*, 1998, Chanraud *et al.*, 2007, Makris *et al.*, 2008], and disruption of cortico-limbic circuitry [Marinkovic *et al.*, 2009]. Reduced or altered responses (e.g., amygdala hypoactivity) to emotionally-salient stimuli, including facial emotional expressions, have been observed in individuals with a familial history of alcoholism [Miranda *et al.*, 2002, Glahn *et al.*, 2007], suggesting that at least part of this deficit is genetically-influenced and may precede drinking onset.

In contrast, there is a lack of research investigating the acute effects of alcohol on processing of facial emotional expressions in social, non-dependent, drinkers. While data from chronic populations is informative, the effects of acute alcohol and the processes underlying these effects may differ markedly from chronic alcohol use. Tucker and Vuchinich [1983] used a balanced-placebo design to assess the effects of acute alcohol consumption and expectancy of consuming alcohol on emotion recognition. Male and female social drinkers were randomised to either receive 0.50 g/kg alcohol or placebo, and instructed that they had received alcohol or placebo, in a crossover design. The study found reduced overall emotion recognition accuracy after alcohol consumption, which was greater in those who had been told that they had received alcohol, indicating an additive effect of expectancy. As analyses were not stratified by emotion type, it is unclear whether alcohol had emotion-specific effects. More recently, Kano and colleagues [2003] reported that 0.14 g/kg alcohol speeded discrimination of happy-neutral morph faces compared with 0.56 g/kg alcohol, but not placebo. There were no effects on the discrimination of negative facial expressions (anger, surprise, sadness). The interpretation of

these results is complicated by the use of reaction time as the dependent variable, which provides little information on the mechanism by which performance is altered. Furthermore, the task lacked sensitivity as the facial stimuli comprised only four levels of emotional intensity for each emotion (0%, 33%, 66% and 100%).

More sensitive measures of emotional processing have been developed by modifying tasks typically used to measure perceptual sensitivity in visual psychophysics. This allows a standard facial stimulus (i.e., neutral face) to be presented alongside a second facial stimulus (of the same individual) that varies in the degree of emotional expression. By using a larger range of stimuli, varying in intensity between the neutral and emotional expression exemplars across a number of emotional expressions, and asking participants to identify the stimulus containing the relevant emotion over repeated trials, a measure of perceptual threshold can be obtained. Scores on this task are an index of how much emotional information is required in a face in order for it to be reliably identified (i.e., recognition *sensitivity*). Using this task, we [Craig *et al.*, 2009] investigated the effect of an acute dose of 0.4 g/kg alcohol on the processing of angry, happy and sad emotional expressions in social alcohol drinkers. Alcohol impaired the ability to identify sadness compared to placebo (i.e., reduced sensitivity), but there was no difference in the identification of anger or happiness. A selective effect of alcohol on identification of sadness was partially replicated in follow-up studies. First, a within-subject study using three doses of alcohol (0.0, 0.2, 0.4 g/kg) found a three-way interaction between alcohol dose, participant sex and emotion, with male participants showing a reduced sensitivity to sadness compared to females after the highest dose of alcohol [Attwood *et al.*, 2009]. More recently, a study using dynamic facial morphing sequences using three doses of alcohol (0.0, 0.4 and 0.8 g/kg) found a higher response bias to neutral expressions after the low dose [Kamboj *et al.*, 2012]. Further analysis

indicated a tendency to misidentify sadness as neutral, with no effects on other emotions. This relative inability to identify sadness in faces has implications for alcohol-related aggression, as sadness may be seen as a sign of submission [Hart, 2011], which may curtail aggressive behaviour [Miczek *et al.*, 1977].

The lack of increased sensitivity to anger after alcohol is unsurprising considering that alcohol is generally associated with impaired performance. Rather than increase sensitivity to anger, alcohol may induce a *bias* towards perceiving it, particularly when the emotional content is ambiguous. To address this, we developed an alternative task to assess the *categorisation* of ambiguous emotional facial stimuli (i.e., a single stimulus comprising varying degrees of two morphed expressions, such as angry-happy or angry-disgusted) [Attwood *et al.*, 2009]. After consuming 0.4 g/kg alcohol, participants were more likely to categorise an ambiguous negative (anger-disgust morph) emotional face as angry, compared to placebo, but only if the target face was male. These data suggest that in social situations where alcohol is consumed, social drinkers are more likely to perceive a negative male facial cue as angry. It is plausible that these effects may be specific to disgusted/angry morphs as anger and disgust recognition involve neural regions in close proximity to each other in the orbitofrontal cortex, which may make these morphs particularly prone to disruption [Townshend *et al.*, 2003]. Future research should examine the extent to which anger is preferentially perceived when morphed with other negative emotions.

Preliminary findings therefore suggest a reduced ability to recognise distress cues (i.e., sad expressions), and a bias towards perceiving threat (i.e., angry expressions), after acute alcohol consumption. The neural mechanisms by which alcohol alters processing of emotional cues are yet to be established. Data from imaging, lesion and pharmacological challenge studies

indicate that dissociable neural regions are involved in the processing of different facial emotions [Kelly *et al.*, 1988, Kirsch, 2009, Vasquez *et al.*, 2012], with anger being linked to frontal regions of the brain. We next need to turn to findings that provide insight on the emotional circuitry of the brain.

5. A neurological framework for the effects of alcohol within the emotional brain

The emotional circuitry of the brain comprises a complex series of interconnections between numerous neural regions at all levels of the neuraxis [Adolphs, 2002]. Higher order association areas are required for the experience of emotion through attributing emotional significance to events, while lower level autonomic areas are critical in expressing emotion [Adolphs, 2002]. Prominent regions of the emotional brain are the OFC, amygdala and anterior cingulate cortex, which are highly interconnected. Damage to these areas has independently shown to lead to changes in emotional behaviour and the experience of emotion [Davidson *et al.*, 2000, Barbas *et al.*, 2003].

The amygdala is known for its role in emotional processing and responding, and is particularly responsive to stimuli that suggest environmental threat [Ohman *et al.*, 2001, Ohman, 2005]. Although small, it is heterogeneous, consisting of nucleic sub-regions, each with its own range of functions and profile of wider connectivity (for specialised reviews, see: [Aggleton, 1993, Davidson *et al.*, 2000, Ledoux, 2000]). The amygdala is particularly important in the processing of, and response to, fearful stimuli [Romanski *et al.*, 1992]; these automatically activate the amygdala in functional imaging studies [Breiter *et al.*, 1996, Morris *et al.*, 1996], and bilateral damage to the amygdala results in disproportionate deficits in the processing of fearful facial expressions [Adolphs *et al.*, 1999].

The amygdala has also been implicated in the processing of sad facial expressions [Whalen *et al.*, 1998, Wang *et al.*, 2005]. However, amygdala damage has not been consistently associated with impairments in the recognition of sadness, which raises a potentially important distinction between responses to emotional facial stimuli and the process of explicitly recognising them. As Blair and colleagues [1999] note, their finding of amygdala activation to sad facial expressions is discrepant with neuropsychological data showing normal recognition of sadness in these patients, but discrepancies between neuropsychological, neuroimaging and behavioural outcomes are not uncommon. It is plausible that the amygdala is not essential for recognising sad facial expressions, but plays other functional roles, such as in autonomic responses to sad stimuli [Blair *et al.*, 1999]. In support, emotional empathy, which is characterised by the ability to share the emotional experience of others through affective reactions, is impaired following bilateral amygdala damage, while cognitive empathy (i.e., theory of mind-like ability to understand another's perspective) remains unaffected [Hurlemann *et al.*, 2010] (for further discussion of emotional and cognitive empathy see reviews [Moya-Albiol *et al.*, 2010, Shamay-Tsoory, 2011, Thoma *et al.*, 2013]). Adolphs [2002] suggests that trying to link the processing of specific emotions to the amygdala may be misguided. Instead, the amygdala may contribute to an unspecified basic biological process important in emotional processing more generally, but which does not directly translate clearly to categories of emotion. Less is known about the neurobiology underlying other emotional expressions such as anger, although the amygdala would be expected to be involved to some degree. Artificial stimulation of the amygdala produces aggressive vocalisations in animals [Blanchard *et al.*, 1972] and reduced amygdala volume is associated with aggressive tendencies [Matthies *et al.*, 2012]. Amygdala activation occurs in response to angry faces [Derntl *et al.*, 2009], and impairments in

the ability to recognise anger have been reported in patients with amygdala lesions [Phillips *et al.*, 1997]. However these findings are not consistently replicated and, when observed, the effects are often not specific to anger.

Experiencing anger and expressing aggression engages higher-level cortical areas, particularly the pre-frontal cortex (PFC) [Rolls *et al.*, 2006], and sub-regions of the PFC have been identified as sites that may preferentially process facial displays of anger. In particular, the OFC appears to be directly involved in face processing. Neurons that selectively respond to facial stimuli have been identified in the primate OFC [Coccaro *et al.*, 2007] and damage to the OFC induces deficits in the recognition of emotions when presented in the face and in the voice [Hornak *et al.*, 1996]. Moreover, Blair and colleagues [1999] showed selective activation in the right OFC in response to passive viewing of angry faces, compared to other negative emotions, and this positively correlated with the emotional intensity of the stimuli

Patients with OFC damage often display aberrant emotional responses characterised by heightened reactivity to emotionally valenced stimuli [Rule *et al.*, 2002], supporting a role of the OFC in top-down modulation of neural responses to emotional stimuli in the healthy brain (as described in the dynamic filtering model of OFC function). Its role in assigning reward and punishment value to stimuli and behavioural reactions to stimuli extends to the social domain. Patients with fronto-temporal dementia, who have significant OFC atrophy, demonstrate insensitivity to the reward or punishment value of social cues, and often engage in inappropriate social behaviours, and show deficits in empathy and the processing of facial and vocal displays of emotion [Rolls, 2004]. It is also well established that the frontal cortex, including the OFC, is particularly sensitive to the effects of alcohol [Abernathy *et al.*, 2010]. Therefore, the effects of

alcohol on emotional processing may be mediated by disruption to these areas and related circuitry, including the connectivity between the OFC and amygdala.

Disruption of the OFC, amygdala and their connectivity has been identified as underlying emotional expression and recognition deficits in many disorders with emotional dysregulation and aggression as prominent features [Davidson *et al.*, 2000, Hulshoff Pol *et al.*, 2001]. However, more direct support regarding OFC-amygdala coupling comes from a study investigating neural reactivity to emotional cues in individuals with impulsive aggressive problems. Compared to healthy controls, patients with a diagnosis of intermittent explosive disorder show altered OFC-amygdala connectivity, and exaggerated amygdala activity in conjunction with reduced OFC activity, in response to angry facial expressions [Coccaro *et al.*, 2007], with amygdala activation to angry faces positively associated with prior aggressive behaviour. Recent fMRI data shows that acute alcohol consumption reduces functional connectivity between the amygdala and OFC during emotional face processing [Gorka *et al.*, 2013], further substantiating this system as one underlying alcohol's effects on social behaviour and, in particular, as a possible mechanism underlying alcohol-related aggression.

Davidson and colleagues [2000] suggest that the activity of the OFC in response to angry facial expressions is part of an emotion-behaviour regulation system. OFC inhibitory neurons project and terminate in portions of the amygdala which act as relay centres, innervating regions of the hypothalamus and brain stem that control autonomic activity. Thus, it appears that, in the healthy brain, the OFC mediates aggressive behaviour by suppressing limbic activation, and that impulsive aggression occurs when failures in this system occur [McDonald, 1991]. This is supported by animal data showing amygdala hyperactivity and orbitofrontal hypoactivity in aggressive rats [Marquez *et al.*, 2013]. Thus, alcohol-induced disruption of these neurons leads

to a disinhibition of amygdala, which results in a state of *emotional disinhibition* [Rule *et al.*, 2002] and subsequent vulnerability to aggression.

Taken together, these observations suggest a model of OFC functioning that may increase aggression through a series of cognitive and behavioural adaptations. First, attenuated OFC regulation of parts of the limbic system (including the amygdala) results in exaggerated or aberrant processing of emotional stimuli. Second, reduced OFC-related behavioural control increases the likelihood of an inappropriate behavioural response (e.g., aggression) to these stimuli.

6. Individual Differences

Only a small proportion of alcohol consumers reliably display alcohol-related aggression.

Similar variation has been observed in several animal species, with only a subset of animals displaying aggressive behaviours after alcohol consumption [Miczek *et al.*, 1998].

Understanding these individual differences is important in order to elucidate underlying mechanisms and to develop interventions [Parrott *et al.*, 2012]. This section briefly discusses some factors that may contribute to this individual variation. While they are presented in isolation, many factors interact to heighten alcohol-aggression risk.

Personality

Aggressive personality traits predict aggressive behaviours in a sober state, and interactions between trait aggression/anger and alcohol have been reported in studies of alcohol-related aggression [Giancola, 2002, Tremblay *et al.*, 2007, Tremblay *et al.*, 2008]. As alcohol is a potent disinhibitor of behaviour, part of its action may be to amplify natural behavioural tendencies or cognitions, making trait aggressive individuals particularly susceptible to these effects. Although trait aggression is a relatively consistent predictor of alcohol-related aggression, there are also

interesting interactions with other personality and situational variables, such as anger control [Parrott *et al.*, 2004], provocation [Bettencourt *et al.*, 2006] and physical context. For example, Tremblay, Graham and Wells [2008] found that trait aggression predicted real-world aggression severity in a student population within all environments examined (i.e., bar/pub/nightclub, home, party/social event), but an interaction between alcohol and [physical] trait aggression was only evident in bar/pub/nightclub locations, which may reflect moderating effects of social norms.

Individuals high in self-reported aggression are also more likely to misidentify anger in facial cues [Hall, 2006]. Therefore, individuals high in trait anger/aggression may have a sensitivity or bias towards recognising/categorising threatening emotional stimuli during alcohol intoxication, although this needs to be tested empirically.

Cognition and executive function

In a study investigating the relationship between executive function and alcohol-related aggression, Giancola [2004] reported that alcohol selectively increased aggression in men with lower executive function. However, executive function comprises numerous independent processes, including working memory, planning, reasoning and problem solving. Unsurprisingly, behavioural regulation indices appears to be one of the best predictors of alcohol-related aggression [Giancola *et al.*, 2012], but it is still unclear if, and to what extent, other processes are also involved.

Little research has directly assessed the effect of executive function on emotional face processing. Circelli, Clark and Golomb [2012] found changes in emotional recognition ability and visual scanning patterns of emotional faces in a group of older adults (mean age 69 years). The latter correlated with executive function, which implicated age-related changes in frontal

lobe function as underlying the differences in emotion recognition. Executive function has also been associated with emotion recognition deficits in adult ADHD [Ibanez *et al.*, 2011] and schizophrenic/schizoaffective [Premkumar *et al.*, 2008] patients, although a causal effect has yet to be established.

Genetics

Several genetic polymorphisms have been implicated in regulating aggressive behaviours following voluntary alcohol consumption, including those that encode oxytocin [Johansson *et al.*, 2012, Johansson *et al.*, 2012], GABA and 5HT receptors. The GABA_A subunit of the GABA receptor complex appears to be particularly important in the expression of aggression, and genes that encode the alpha and gamma units of this receptor may be markers of individuals who are sensitive to alcohol-related aggression [Miczek *et al.*, 2004].

Serotonin has long been associated with negative emotionality and aggression [Takahashi *et al.*, 2011, Kulikov *et al.*, 2012, Montoya *et al.*, 2012]. Both 5-HT_{1A} and 5-HT_{1B} agonists dose-dependently decrease alcohol-induced aggression in rodents [Miczek *et al.*, 2001, Miczek *et al.*, 2002], and exaggerated aggression can be reduced to species typical levels with chronic administration of citalopram, a selective serotonin reuptake inhibitor [Caldwell *et al.*, 2008]. Thus, genes controlling serotonin function and serotonin receptor expression (e.g., serotonin transporter, monoamine oxidase A tryptophan hydroxylase-2 genes) may underlie some of the individual variation in aggression, including aggressive responses after alcohol [Popova, 2008, Takahashi *et al.*, 2011]. Sander *et al.* [1998] found partial support for higher frequency of the short allele of the *SLC6A4* gene in alcohol dependent patients with concomitant personality disorder, and reduced levels of endogenous serotonin have been reported in Type II alcoholics

(i.e., a subgroup of alcohol-dependent individuals characterised by early onset of alcohol-related problems, more severe dependence, alcohol-related aggression, high sensation seeking and anti-social behaviour) [Virkkunen *et al.*, 1993].

Dysregulated serotonergic function is a feature of many psychiatric disorders comprising deficits in emotional processing, such as depression and anxiety, and some of the pharmacotherapies used to treat these disorders target the serotonergic system. It has been proposed that these drugs exert their clinical effect through positive changes in emotional processing [Harmer *et al.*, 2009]. It is therefore plausible that individual differences in aggression based on serotonergic function may be mediated, at least in part, by differences in emotional processing. Studies that manipulate central serotonin using acute tryptophan depletion show altered processing of emotional facial expressions with lowered serotonin (although the precise nature of this effects differs across studies) [Van Der Veen *et al.*, 2007, Williams *et al.*, 2007, Daly *et al.*, 2010]. Of particular interest, lower serotonin is associated with reductions in functional connectivity between the amygdala and frontal regions in response to angry (compared to sad and neutral) faces [Passamonti *et al.*, 2012].

Gender

Alcohol-related aggression and violence is more commonly associated with men [Archer, 2004, Quinn *et al.*, 2013]. Although there is inconsistency in findings and suggestion that differences are diminishing over time [Archer, 2004, Graves, 2007], experimental studies which administer alcohol acutely have found effects of alcohol on aggression are stronger in males than females [Giancola *et al.*, 2009], and this difference may in part be mediated by sex hormones [Archer, 2006, Oyegbile *et al.*, 2006, Soma, 2006]. For example, testosterone is associated with selective

attention [Van Honk *et al.*, 1999] and increased cardiovascular reactivity to angry faces [Van Honk *et al.*, 2001], increased approach to/reduced aversion of threat-related stimuli [Wirth *et al.*, 2007], and is positively correlated with amygdala activity to angry and fearful faces [Derntl *et al.*, 2009]. One mechanism by which some of these effects may occur is through a functional uncoupling of the amygdala and prefrontal areas that would normally regulate amygdala activity [Van Wingen *et al.*, 2010].

Social norms and *expectations* about behaviour may also be important in explaining sex differences in aggression. Gender roles are among the most highly established and socialised in modern society. The male gender role is associated with dominance and aggression compared to the antithesis traits of nurturance and empathy typical of the female gender role. However, in modern society, adherence to traditional gender roles is weakening. It is becoming more acceptable for women to act in masculine ways and binge drinking among women is on the rise [Newberry *et al.*, 2013]. Alongside this, there has been a move away from the traditional male-dominated public house with bar room environments being designed to be more "female friendly". These societal changes may reduce the gender gap in alcohol consumption and alcohol-related aggression from previous decades.

Therefore, although aggression, including alcohol-related aggression, is greater in males, it is not solely a male phenomenon. In an experimental paradigm, Giancola [2002] found that alcohol increased aggression (i.e., mean shock intensity in Buss-Taylor paradigm) in males and females reporting high levels of trait anger, and although perpetrators of bar room violence are more often male, incidents of female violence are not uncommon [Forsyth *et al.*, 2010].

Expectancies

Alcohol-related aggression is stronger in drinkers who expect alcohol to increase aggression [Dermen *et al.*, 1989]. These expectations could develop from direct experience, observation or from popular social or cultural belief. Many individuals believe that alcohol leads to aggression, although this belief appears to be stronger when considering the behaviour of others rather than one's own behaviour [Paglia *et al.*, 2006]. The belief that alcohol induces aggressive behaviour is related to having had experience of alcohol-related aggression [Quigley *et al.*, 2002], and in experimental studies, people who believe they have consumed alcohol act more aggressively even in placebo conditions [Bushman, 1997].

Several expectancy theories have been offered as alternatives to pharmacological explanations for alcohol-related aggression. A core tenet of many of these is that alcohol consumption activates the expectancy of aggression, and this expectancy induces heightened aggression, implying that both alcohol consumption *and* expectancy are required for aggression to occur. In addition to offering an explanation for individual differences, these theories imply that an individual's propensity to display alcohol-related aggression could change over time, if beliefs are altered in response to new experience or learning. For example experiencing alcohol-related aggression, either as victim or perpetrator, may increase risk of displaying aggression if the incident strengthens the belief that alcohol and aggression are related. From a clinical perspective, interventions that challenge these beliefs may be efficacious for individuals with a history of problem behaviours after alcohol consumption.

Cultural norms and environmental factors

Behavioural outcomes of alcohol consumption are often consistent with pervading cultural and social expectations [Neff, 1991, Lindman *et al.*, 1994]. These beliefs provide a framework for

behaviour and the consequences of behaviour in social contexts. Thus, alcohol-related aggression is greater in cultures where there is a belief that alcohol is associated with aggression and where binge drinking is the norm, and these differences further undermine a pure pharmacological aggressive action of alcohol.

There are also numerous environmental factors that could promote alcohol consumption, aggression or both. For example early life stress is associated with higher risk of alcohol abuse, early onset drinking and alcohol-related problems [Anda *et al.*, 2002, Enoch, 2011, Sartor *et al.*, 2013]. Acute stress is also identified as a motivator of alcohol use [Pohorecky, 1991]. Alcohol can dampen the response to stress which has become one indirect explanation for alcohol-related aggression [Sayette, 1999]. However, anxiolytic motives of alcohol consumption are inconsistent in clinical samples [Chutuape *et al.*, 1995, Battista *et al.*, 2010]. Stress is associated with relapse in alcohol dependency [Brady *et al.*, 1999], but chronic use is associated with elevations in stress in animals [Becker *et al.*, 2011]. As well as increasing the likelihood to consume alcohol and/or aggress, these environmental factors can interact with other environmental or biological factors that put the individual at higher risk of alcohol abuse or aggression [Heinz *et al.*, 2011].

7. Conclusions

We have hypothesised a two-phase model of alcohol-related aggression, based on studies indicating disrupted processing of emotional information following acute alcohol consumption. We also speculate on possible neural mechanisms for these effects based on recent neurological and clinical data. While many of the ideas presented here are speculative, there are strong theoretical and empirical grounds to suggest that these ideas are worthy of further investigation. Chronic alcohol abuse has previously been associated with altered processing of facial emotional expressions, and recent studies of acute alcohol consumption in healthy social drinkers indicate

altered processing, indicative of reduced sensitivity to sadness and increased bias towards perceiving anger in ambiguous facial morphs. These findings imply that there may be a greater likelihood of erroneously perceiving provocation when intoxicated in social situations and a reduced likelihood of identifying submissive signals that would otherwise curtail aggressive behaviours. This altered processing occurs alongside other effects of alcohol (i.e., behavioural inhibition, anxiolysis) which together increase the likelihood of aggressive behaviours in response to these social cues.

Few studies have examined the effect of acute doses of alcohol on the processing of emotional facial expressions, but the current findings are informative and largely consistent with what is already known regarding emotion processing and the effects of alcohol on the brain. Evidence that alcohol disrupts general processing is not particularly useful when attempting to interpret these effects. It is therefore important to investigate the effects of alcohol on *specific* emotions, rather than on emotion recognition *in general*, and tasks should be sensitive enough to identify relatively small effects in healthy social drinkers. Kano and colleagues [2003], for example, report faster responding after a low dose of alcohol but no effects of a larger dose or on accuracy. The lack of disruption by alcohol at higher doses may be due to ceiling effects since only four levels of emotional intensity were used. Subsequent research used a wider range of emotional intensity and found effects of alcohol that differed depending on the nature of the task indicating that alcohol may differentially affect sensitivity versus bias towards certain emotions. This distinction is critical; for example, social anxiety is associated with an increased *sensitivity* to fear, while depression is associated with a *bias* towards negativity such as sadness. Preliminary findings imply reduced sensitivity towards sadness after alcohol, but no effects of alcohol on the processing of anger [Craig *et al.*, 2009, Kamboj *et al.*, 2012]. In contrast, when

anger was morphed with another emotion to create an ambiguous negative facial morph, there was a *bias* towards identifying anger in a two-alternative forced-choice paradigm [Attwood *et al.*, 2009]. These findings need to be replicated and extended; for example, the bias towards identifying anger was only observed when target images were male. This may be due to expectations of greater aggression in males, but this study only used one male and one female facial image and therefore cannot rule out sex-specific effects as being due to other idiosyncratic characteristics of the facial stimuli. However, one difficulty with these studies is the length of the tasks, given the number of trials required to ensure reliability. This limits the number of variables that can be assessed in a single study. Due to the biphasic effects of alcohol, there is a pharmacological window in which a task needs to be completed, otherwise outcomes will be difficult to interpret, as previous studies have shown different behavioural outcomes on the ascending and descending limbs of the blood alcohol curve [Giancola *et al.*, 1997]. Nevertheless, these findings indicate that alcohol alters facial emotional processing in ways that have implications for aggressive behaviour and provide a methodological framework to build on.

There are a number of studies reporting positive effects of alcohol on the processing of social stimuli that should be considered. First, acute (intravenous) alcohol administration is associated with attenuated neural response to fearful faces in social alcohol drinkers [Gilman *et al.*, 2012]. Behavioural data suggest that acute alcohol consumption leads to reductions in sensitivity to sad facial expressions [Attwood *et al.*, 2009], and a similar insensitivity may exist for fearful faces. This would need to be tested empirically, as we can only speculate what effect a dampened neural response would have behaviourally. If this is indicative of a processing deficit of fear that is similar to sadness, this may have similar implications for alcohol-related aggression (i.e., fear may be an inhibitory cue that curtails aggression when processed

effectively). In social phobic samples, alcohol consumption reportedly decreases perceived rejection and attention bias of angry faces [Stevens *et al.*, 2008, Stevens *et al.*, 2009]. There are distinctions between these studies and those reported in this review. For example, these studies do not measure emotion recognition, which limits comparison. Where patient samples have been used, the findings may not generalise to healthy social drinkers (e.g., the attentional bias effect was not observed in sober controls). In addition, some effects were only seen for full emotional exemplar images and not for images that were emotionally ambiguous [Stevens *et al.*, 2008].

Nevertheless, it is well established that alcohol has positive effects on mood, sociability and interactions with peers [Sayette *et al.*, 2012], which raises the question of why we should see negative effects of alcohol on the processing of emotional faces. We suggest that this is due to a general detrimental effect of cognitive processing ability that is well reported, and which we show extends to processing of negative facial emotional expressions. These effects would only be expected to affect behaviour in social situations that are perceived to be threatening or in which negative facial displays are perceived. As suggested previously, alcohol may amplify current states or enduring traits, such that positive mood and sociability are potentiated in pleasant social situations but negative mood and aggression are enhanced in situations of low mood, perceived threat etc.

An important question that remains unanswered is the effect of repeated dosing, as most studies administer a single dose of alcohol. To achieve high blood alcohol concentrations, doses in region of 1.0 g/kg have been used, but this does not produce the same pharmacological effect as repeated doses over an extended period of time. Therefore, the extent to which multiple dosing changes emotional processing over time, and if there is a critical period when an individual is particularly at risk of alcohol-related aggression, has yet to be established.

Despite the fact that the empirical data on acute alcohol consumption and emotional face processing are limited, in particular linking these effects directly to alcohol-related aggression, there are relevant findings that potentially support a link. First, the results to date are consistent with the known pharmacology of alcohol, and the role of the OFC (i.e., in behavioural control and emotional processing). Second, the importance of emotional processing, including processing of emotion in faces, on behavioural expression is also well reported. Third, there reports of aberrant emotional processing in individuals with disorders associated with violence and in violent offenders. Taken together, this suggests that integration of these themes represents an important avenue for future research.

This review has implicated a role of aberrant emotional processing in alcohol-related aggression. We stress that we do not introduce this as alternative explanation of alcohol-related aggression, but rather as an additional mechanism that has been relatively unexplored to-date. Alcohol undoubtedly increases aggression likelihood via multiple mechanisms, many of which have not been covered in this review. Due to the cue-induced nature of our argument, we have focussed predominately on “reactive” aggression, but alcohol may also alter motivational processes involved in social interaction that could enhance other types of instrumental or “appetitive” aggression. The ideas presented in this paper integrate with the alcohol myopia theory (AMT), which states that, due to limited processing capacity, intoxicated individuals will focus attention on the most salient cues. For adaptive reasons, threat cues are particularly salient and draw attention, and therefore AMT has most frequently been used to explain alcohol-related aggression. We suggest that as well as honing attentional focus (in line with AMT), acute alcohol consumption has the additional effect of altering perception of emotional expressions. This, in turn, will affect which cues are perceived as salient, and focussed on. Finally, it is important to

consider individual variation in response to alcohol. It is clear that not everybody who drinks alcohol displays aggression. Instead alcohol may "amplify" pre-existing behavioural tendencies, making those high in trait aggression the most likely to display aggression when intoxicated. There is also some evidence of inter-individual variation in emotional face processing, but the variability of alcohol-related disruption to this processing is less well studied, and warrants further investigation. Alcohol may disrupt emotional processing in most individuals, increasing *susceptibility* to perceiving social cues as threatening or provocative, but the subsequent *expression* of aggression may be limited to those who have aggressive tendencies, or are particularly sensitive to failures of behavioural inhibition.

In summary, it is well established that alcohol induces behavioural disinhibition, most likely via direct pharmacological action on the frontal regions of the brain. Aggressive behaviour is not an inevitable consequence of this, but will be mediated by numerous cognitive and environmental factors. The social environment comprises a wealth of information regarding the intentions of others and threats to the self, and the processing of this information may be altered by alcohol. Both chronic and acute alcohol consumption appear to disrupt the processing of emotional stimuli, such that the individual is more likely to perceive provocation, and less likely to perceive submission, in social environments. Therefore, alcohol's effects on emotional processing may be a factor that increases the likelihood that outward expressions of disinhibited behaviour are aggressive in nature, although this itself may also be subject to individual variation. Recent advances in computer-based interventions to improve emotional face processing in psychiatric disorders [Browning *et al.*, 2012, Penton-Voak *et al.*, 2013] mean that if a causal link between alcohol-induced emotion processing disruption and aggressive behaviour

can be established, there is scope to develop novel interventions to tackle this problem in individuals prone to alcohol-related aggression.

References

- Abernathy, K., Chandler, L.J., and Woodward, J.J. (2010) Alcohol and the Prefrontal Cortex. *Int Rev Neurobiol* 91: 289-320.
- Adolphs, R. (2002) Neural Systems for Recognizing Emotion. *Curr Opin Neurobiol* 12: 169-177.
- Adolphs, R. (2002) Recognizing Emotion from Facial Expressions: Psychological and Neurological Mechanisms. *Behav Cogn Neurosci Rev* 1: 21-62.
- Adolphs, R., Tranel, D., Hamann, S., Young, A.W., Calder, A.J., Phelps, E.A. *et al.* (1999) Recognition of Facial Emotion in Nine Individuals with Bilateral Amygdala Damage. *Neuropsychologia* 37: 1111-1117.
- Aggleton, J.P. (1993) The Contribution of the Amygdala to Normal and Abnormal Emotional States. *Trends Neurosci* 16: 328-333.
- Aleman, A. and Kahn, R.S. (2005) Strange Feelings: Do Amygdala Abnormalities Dysregulate the Emotional Brain in Schizophrenia? *Prog Neurobiol* 77: 283-298.
- Anda, R.F., Whitfield, C.L., Felitti, V.J., Chapman, D., Edwards, V.J., Dube, S.R. *et al.* (2002) Adverse Childhood Experiences, Alcoholic Parents, and Later Risk of Alcoholism and Depression. *Psychiatr Serv* 53: 1001-1009.
- Archer, J. (2004) Sex Differences in Aggression in Real-World Settings: A Meta-Analytical Review. *Review of General Psychology* 8:
- Archer, J. (2006) Testosterone and Human Aggression: An Evaluation of the Challenge Hypothesis. *Neurosci Biobehav Rev* 30: 319-345.
- Attwood, A.S., Ataya, A.F., Benton, C.P., Penton-Voak, I.S., and Munafo, M.R. (2009) Effects of Alcohol Consumption and Alcohol Expectancy on the Categorisation of Perceptual Cues of Emotional Expression. *Psychopharmacology (Berl)* 204: 327-334.

Attwood, A.S., Ohlson, C., Benton, C.P., Penton-Voak, I.S., and Munafo, M.R. (2009) Effects of Acute Alcohol Consumption on Processing of Perceptual Cues of Emotional Expression. *J Psychopharmacol* 23: 23-30.

Barbas, H., Saha, S., Rempel-Clower, N., and Ghashghaei, T. (2003) Serial Pathways from Primate Prefrontal Cortex to Autonomic Areas May Influence Emotional Expression. *BMC Neurosci* 4: 25.

Bartholow, B.D., Pearson, M., Sher, K.J., Wieman, L.C., Fabiani, M., and Gratton, G. (2003) Effects of Alcohol Consumption and Alcohol Susceptibility on Cognition: A Psychophysiological Examination. *Biol Psychol* 64: 167-190.

Battista, S.R., Stewart, S.H., and Ham, L.S. (2010) A Critical Review of Laboratory-Based Studies Examining the Relationships of Social Anxiety and Alcohol Intake. *Curr Drug Abuse Rev* 3: 3-22.

Becker, H.C., Lopez, M.F., and Doremus-Fitzwater, T.L. (2011) Effects of Stress on Alcohol Drinking: A Review of Animal Studies. *Psychopharmacology (Berl)* 218: 131-156.

Best, M., Williams, J.M., and Coccaro, E.F. (2002) Evidence for a Dysfunctional Prefrontal Circuit in Patients with an Impulsive Aggressive Disorder. *Proc Natl Acad Sci U S A* 99: 8448-8453.

Bettencourt, B.A., Talley, A., Benjamin, A.J., and Valentine, J. (2006) Personality and Aggressive Behavior under Provoking and Neutral Conditions: A Meta-Analytic Review. *Psychol Bull* 132: 751-777.

Blair, R.J. (2005) Applying a Cognitive Neuroscience Perspective to the Disorder of Psychopathy. *Dev Psychopathol* 17: 865-891.

Blair, R.J., Colledge, E., Murray, L., and Mitchell, D.G. (2001) A Selective Impairment in the Processing of Sad and Fearful Expressions in Children with Psychopathic Tendencies. *J Abnorm Child Psychol* 29: 491-498.

Blair, R.J., Morris, J.S., Frith, C.D., Perrett, D.I., and Dolan, R.J. (1999) Dissociable Neural Responses to Facial Expressions of Sadness and Anger. *Brain* 122 (Pt 5): 883-893.

Blanchard and Blanchard, R.J. (1972) Innate and Conditioned Reactions to Threat in Rats with Amygdaloid Lesions. *J Comp Physiol Psychol* 81: 281-290.

Blanchard, R.J., Hori, K., Blanchard, D.C., and Hall, J. (1987) Ethanol Effects on Aggression on Rats Selected for Different Levels of Aggressiveness. *Pharmacol Biochem Behav* 27: 641-644.

Brady, K.T. and Sonne, S.C. (1999) The Role of Stress in Alcohol Use, Alcoholism Treatment, and Relapse. *Alcohol Res Health* 23: 263-271.

- Breiter, H.C., Etcoff, N.L., Whalen, P.J., Kennedy, W.A., Rauch, S.L., Buckner, R.L. *et al.* (1996) Response and Habituation of the Human Amygdala During Visual Processing of Facial Expression. *Neuron* 17: 875-887.
- Browning, M., Holmes, E.A., Charles, M., Cowen, P.J., and Harmer, C.J. (2012) Using Attentional Bias Modification as a Cognitive Vaccine against Depression. *Biol Psychiatry* 72: 572-579.
- Bushman, B.J. (1997) Effects of Alcohol on Human Aggression. Validity of Proposed Explanations. *Recent Dev Alcohol* 13: 227-243.
- Bushman, B.J. and Cooper, H.M. (1990) Effects of Alcohol on Human Aggression: An Integrative Research Review. *Psychol Bull* 107: 341-354.
- Buss, A.H. (1961) *The Psychology of Aggression*. Wiley: New York.
- Bye, E.K. (2007) Alcohol and Violence: Use of Possible Confounders in a Time-Series Analysis. *Addiction* 102: 369-376.
- Caldwell, E.E. and Miczek, K.A. (2008) Long-Term Citalopram Maintenance in Mice: Selective Reduction of Alcohol-Heightened Aggression. *Psychopharmacology (Berl)* 196: 407-416.
- Calvo, M.G. and Castillo, M.D. (2005) Processing of Threat-Related Information Outside the Focus of Visual Attention. *Span J Psychol* 8: 3-11.
- Casbon, T.S., Curtin, J.J., Lang, A.R., and Patrick, C.J. (2003) Deleterious Effects of Alcohol Intoxication: Diminished Cognitive Control and Its Behavioral Consequences. *J Abnorm Psychol* 112: 476-487.
- Chanraud, S., Martelli, C., Delain, F., Kostogianni, N., Douaud, G., Aubin, H.J. *et al.* (2007) Brain Morphometry and Cognitive Performance in Detoxified Alcohol-Dependents with Preserved Psychosocial Functioning. *Neuropsychopharmacology* 32: 429-438.
- Cheong, J. and Nagoshi, C.T. (1999) Effects of Sensation Seeking, Instruction Set, and Alcohol/Placebo Administration on Aggressive Behavior. *Alcohol* 17: 81-86.
- Chermack, S.T. and Giancola, P.R. (1997) The Relation between Alcohol and Aggression: An Integrated Biopsychosocial Conceptualization. *Clin Psychol Rev* 17: 621-649.

Chermack, S.T. and Taylor, S.P. (1995) Alcohol and Human Physical Aggression: Pharmacological Versus Expectancy Effects. *J Stud Alcohol* 56: 449-456.

Chiavegatto, S., Quadros, I.M., Ambar, G., and Miczek, K.A. (2010) Individual Vulnerability to Escalated Aggressive Behavior by a Low Dose of Alcohol: Decreased Serotonin Receptor Mrna in the Prefrontal Cortex of Male Mice. *Genes Brain Behav* 9: 110-119.

Chutuape, M.A. and De Wit, H. (1995) Preferences for Ethanol and Diazepam in Anxious Individuals: An Evaluation of the Self-Medication Hypothesis. *Psychopharmacology (Berl)* 121: 91-103.

Circelli, K.S., Clark, U.S., and Cronin-Golomb, A. (2012) Visual Scanning Patterns and Executive Function in Relation to Facial Emotion Recognition in Aging. *Neuropsychol Dev Cogn B Aging Neuropsychol Cogn*:

Coccaro, E.F., Mccloskey, M.S., Fitzgerald, D.A., and Phan, K.L. (2007) Amygdala and Orbitofrontal Reactivity to Social Threat in Individuals with Impulsive Aggression. *Biol Psychiatry* 62: 168-178.

Cogan, R. and Ballinger, B.C., 3rd. (2006) Alcohol Problems and the Differentiation of Partner, Stranger, and General Violence. *J Interpers Violence* 21: 924-935.

Craig, L.C., Attwood, A.S., Benton, C.P., Penton-Voak, I.S., and Munafo, M.R. (2009) Effects of Acute Alcohol Consumption and Alcohol Expectancy on Processing of Perceptual Cues of Emotional Expression. *J Psychopharmacol* 23: 258-265.

Daly, E., Deeley, Q., Hallahan, B., Craig, M., Brammer, M., Lamar, M. *et al.* (2010) Effects of Acute Tryptophan Depletion on Neural Processing of Facial Expressions of Emotion in Humans. *Psychopharmacology (Berl)* 210: 499-510.

Darke, S. (2010) The Toxicology of Homicide Offenders and Victims: A Review. *Drug Alcohol Rev* 29: 202-215.

Davidson, R.J., Jackson, D.C., and Kalin, N.H. (2000) Emotion, Plasticity, Context, and Regulation: Perspectives from Affective Neuroscience. *Psychol Bull* 126: 890-909.

Davidson, R.J., Putnam, K.M., and Larson, C.L. (2000) Dysfunction in the Neural Circuitry of Emotion Regulation - a Possible Prelude to Violence. *Science* 289: 591-594.

Debold, J.F. and Miczek, K.A. (1985) Testosterone Modulates the Effects of Ethanol on Male Mouse Aggression. *Psychopharmacology (Berl)* 86: 286-290.

Dermen, K.H. and George, W.H. (1989) Alcohol Expectancy and the Relationship between Drinking and Physical Aggression. *J Psychol* 123: 153-161.

Derntl, B., Windischberger, C., Robinson, S., Kryspin-Exner, I., Gur, R.C., Moser, E. *et al.* (2009) Amygdala Activity to Fear and Anger in Healthy Young Males Is Associated with Testosterone. *Psychoneuroendocrinology* 34: 687-693.

Dougherty, D.D., Shin, L.M., Alpert, N.M., Pitman, R.K., Orr, S.P., Lasko, M. *et al.* (1999) Anger in Healthy Men: A Pet Study Using Script-Driven Imagery. *Biol Psychiatry* 46: 466-472.

Eisenberg, N., Fabes, R.A., Miller, P.A., Fultz, J., Shell, R., Mathy, R.M. *et al.* (1989) Relation of Sympathy and Personal Distress to Prosocial Behavior: A Multimethod Study. *J Pers Soc Psychol* 57: 55-66.

Enoch, M.A. (2011) The Role of Early Life Stress as a Predictor for Alcohol and Drug Dependence. *Psychopharmacology (Berl)* 214: 17-31.

Feldmann-Wustefeld, T., Schmidt-Daffy, M., and Schubo, A. (2011) Neural Evidence for the Threat Detection Advantage: Differential Attention Allocation to Angry and Happy Faces. *Psychophysiology* 48: 697-707.

Fillmore, M.T., Ostling, E.W., Martin, C.A., and Kelly, T.H. (2009) Acute Effects of Alcohol on Inhibitory Control and Information Processing in High and Low Sensation-Seekers. *Drug Alcohol Depend* 100: 91-99.

Fish, E.W., Debold, J.F., and Miczek, K.A. (2002) Repeated Alcohol: Behavioral Sensitization and Alcohol-Heightened Aggression in Mice. *Psychopharmacology (Berl)* 160: 39-48.

Foisy, M.L., Kornreich, C., Fobe, A., D'hondt, L., Pelc, I., Hanak, C. *et al.* (2007) Impaired Emotional Facial Expression Recognition in Alcohol Dependence: Do These Deficits Persist with Midterm Abstinence? *Alcohol Clin Exp Res* 31: 404-410.

Foran, H.M. and O'leary, K.D. (2008) Alcohol and Intimate Partner Violence: A Meta-Analytic Review. *Clin Psychol Rev* 28: 1222-1234.

Forsyth, A.J.M. and Lennox, J.C. (2010) Gender Differences in the Choreography of Alcohol-Related Violence: An Observational Study of Aggression within Licensed Premises. *Journal of Substance Use* 15: 75-88.

Franklin, F.A., Laveist, T.A., Webster, D.W., and Pan, W.K. (2010) Alcohol Outlets and Violent Crime in Washington D.C. *West J Emerg Med* 11: 283-290.

Frigerio, E., Burt, D.M., Montagne, B., Murray, L.K., and Perrett, D.I. (2002) Facial Affect Perception in Alcoholics. *Psychiatry Res* 113: 161-171.

Geen, R. (1990) *Human Aggression*. Open University Press: Milton Keynes.

Gery, I., Miljkovitch, R., Berthoz, S., and Soussignan, R. (2009) Empathy and Recognition of Facial Expressions of Emotion in Sex Offenders, Non-Sex Offenders and Normal Controls. *Psychiatry Res* 165: 252-262.

Giancola, P.R. (2000) Executive Functioning: A Conceptual Framework for Alcohol-Related Aggression. *Exp Clin Psychopharmacol* 8: 576-597.

Giancola, P.R. (2002) The Influence of Trait Anger on the Alcohol-Aggression Relation in Men and Women. *Alcohol Clin Exp Res* 26: 1350-1358.

Giancola, P.R. (2004) Executive Functioning and Alcohol-Related Aggression. *J Abnorm Psychol* 113: 541-555.

Giancola, P.R. and Corman, M.D. (2007) Alcohol and Aggression: A Test of the Attention-Allocation Model. *Psychol Sci* 18: 649-655.

Giancola, P.R., Duke, A.A., and Ritz, K.Z. (2011) Alcohol, Violence, and the Alcohol Myopia Model: Preliminary Findings and Implications for Prevention. *Addict Behav* 36: 1019-1022.

Giancola, P.R., Godlaski, A.J., and Roth, R.M. (2012) Identifying Component-Processes of Executive Functioning That Serve as Risk Factors for the Alcohol-Aggression Relation. *Psychol Addict Behav* 26: 201-211.

Giancola, P.R., Josephs, R.A., Parrott, D.J., and Duke, A.A. (2010) Alcohol Myopia Revisited: Clarifying Aggression and Other Acts of Disinhibition through a Distorted Lens. *Perspectives on Psychological Science* 5: 265-278.

Giancola, P.R., Levinson, C.A., Corman, M.D., Godlaski, A.J., Morris, D.H., Phillips, J.P. *et al.* (2009) Men and Women, Alcohol and Aggression. *Exp Clin Psychopharmacol* 17: 154-164.

Giancola, P.R. and Zeichner, A. (1997) The Biphasic Effects of Alcohol on Human Physical Aggression. *J Abnorm Psychol* 106: 598-607.

Gilman, J.M., Smith, A.R., Ramchandani, V.A., Momenan, R., and Hommer, D.W. (2012) The Effect of Intravenous Alcohol on the Neural Correlates of Risky Decision Making in Healthy Social Drinkers. *Addict Biol* 17: 465-478.

Glahn, D.C., Lovallo, W.R., and Fox, P.T. (2007) Reduced Amygdala Activation in Young Adults at High Risk of Alcoholism: Studies from the Oklahoma Family Health Patterns Project. *Biol Psychiatry* 61: 1306-1309.

Gorka, S.M., Fitzgerald, D.A., King, A.C., and Phan, K.L. (2013) Alcohol Attenuates Amygdala-Frontal Connectivity During Processing Social Signals in Heavy Social Drinkers : A Preliminary Pharmacofmri Study. *Psychopharmacology (Berl)* 229: 141-154.

Graham, K. and Livingston, M. (2011) The Relationship between Alcohol and Violence: Population, Contextual and Individual Research Approaches. *Drug Alcohol Rev* 30: 453-457.

Graham, K., West, P., and Wells, S. (2000) Evaluating Theories of Alcohol-Related Aggression Using Observations of Young Adults in Bars. *Addiction* 95: 847-863.

Graves, K.N. (2007) Expanding Theoretical and Functional Explanations of Why Females Aggress. *Aggression and Violent Behavior* 12: 131-140.

Hall, C.W. (2006) Self-Reported Aggression and the Perception of Anger in Facial Expression Photos. *J Psychol* 140: 255-267.

Harmer, C.J., Goodwin, G.M., and Cowen, P.J. (2009) Why Do Antidepressants Take So Long to Work? A Cognitive Neuropsychological Model of Antidepressant Drug Action. *Br J Psychiatry* 195: 102-108.

Harper, C. (1998) The Neuropathology of Alcohol-Specific Brain Damage, or Does Alcohol Damage the Brain? *J Neuropathol Exp Neurol* 57: 101-110.

Harris, G.J., Jaffin, S.K., Hodge, S.M., Kennedy, D., Caviness, V.S., Marinkovic, K. *et al.* (2008) Frontal White Matter and Cingulum Diffusion Tensor Imaging Deficits in Alcoholism. *Alcohol Clin Exp Res* 32: 1001-1013.

Hart, S. (2011) *The Impact of Attachment*. W. W. Norton & Co.: New York.

Heinz, A.J., Beck, A., Meyer-Lindenberg, A., Sterzer, P., and Heinz, A. (2011) Cognitive and Neurobiological Mechanisms of Alcohol-Related Aggression. *Nat Rev Neurosci* 12: 400-413.

Hoaken, P.N., Allaby, D.B., and Earle, J. (2007) Executive Cognitive Functioning and the Recognition of Facial Expressions of Emotion in Incarcerated Violent Offenders, Non-Violent Offenders, and Controls. *Aggress Behav* 33: 412-421.

- Hoaken, P.N. and Stewart, S.H. (2003) Drugs of Abuse and the Elicitation of Human Aggressive Behavior. *Addict Behav* 28: 1533-1554.
- Holmes, A., Bradley, B.P., Kragh Nielsen, M., and Mogg, K. (2009) Attentional Selectivity for Emotional Faces: Evidence from Human Electrophysiology. *Psychophysiology* 46: 62-68.
- Hornak, J., Rolls, E.T., and Wade, D. (1996) Face and Voice Expression Identification in Patients with Emotional and Behavioural Changes Following Ventral Frontal Lobe Damage. *Neuropsychologia* 34: 247-261.
- Hulshoff Pol, H.E., Schnack, H.G., Mandl, R.C., Van Haren, N.E., Koning, H., Collins, D.L. *et al.* (2001) Focal Gray Matter Density Changes in Schizophrenia. *Arch Gen Psychiatry* 58: 1118-1125.
- Hurlemann, R., Patin, A., Onur, O.A., Cohen, M.X., Baumgartner, T., Metzler, S. *et al.* (2010) Oxytocin Enhances Amygdala-Dependent, Socially Reinforced Learning and Emotional Empathy in Humans. *J Neurosci* 30: 4999-5007.
- Ibanez, A., Petroni, A., Urquina, H., Torrente, F., Torralva, T., Hurtado, E. *et al.* (2011) Cortical Deficits of Emotional Face Processing in Adults with Adhd: Its Relation to Social Cognition and Executive Function. *Soc Neurosci* 6: 464-481.
- Izard, C., Fine, S., Schultz, D., Mostow, A., Ackerman, B., and Youngstrom, E. (2001) Emotion Knowledge as a Predictor of Social Behavior and Academic Competence in Children at Risk. *Psychol Sci* 12: 18-23.
- Johansson, A., Bergman, H., Corander, J., Waldman, I.D., Karrani, N., Salo, B. *et al.* (2012) Alcohol and Aggressive Behavior in Men--Moderating Effects of Oxytocin Receptor Gene (Oxtr) Polymorphisms. *Genes Brain Behav* 11: 214-221.
- Johansson, A., Westberg, L., Sandnabba, K., Jern, P., Salo, B., and Santtila, P. (2012) Associations between Oxytocin Receptor Gene (Oxtr) Polymorphisms and Self-Reported Aggressive Behavior and Anger: Interactions with Alcohol Consumption. *Psychoneuroendocrinology* 37: 1546-1556.
- Kamboj, S.K., Joye, A., Bisby, J.A., Das, R.K., Platt, B., and Curran, H.V. (2012) Processing of Facial Affect in Social Drinkers: A Dose-Response Study of Alcohol Using Dynamic Emotion Expressions. *Psychopharmacology (Berl)*:
- Kano, M., Gyoba, J., Kamachi, M., Mochizuki, H., Hongo, M., and Yanai, K. (2003) Low Doses of Alcohol Have a Selective Effect on the Recognition of Happy Facial Expressions. *Hum Psychopharmacol* 18: 131-139.

Kelly, T.H., Cherek, D.R., Steinberg, J.L., and Robinson, D. (1988) Effects of Provocation and Alcohol on Human Aggressive Behavior. *Drug Alcohol Depend* 21: 105-112.

Kimonis, E.R., Frick, P.J., Fazekas, H., and Loney, B.R. (2006) Psychopathy, Aggression, and the Processing of Emotional Stimuli in Non-Referred Girls and Boys. *Behav Sci Law* 24: 21-37.

Kirsch, S.J., Mounts, J. R. W., Olczak, P. V. (2009) Violent Media Consumption and the Recognition of Dynamic Facial Expressions. *Journal of Interpersonal Violence* 21: 571-584.

Klinnert. (1983) Social Referencing: Emotional Expressions as Behavior Regulators. In: Plutchik, R. and Kellerman, H., (eds), *Emotion: Theory, Research, and Experience*, Vol. 2. Academic Press: Orlando, Florida.

Klinnert, Emde, R.N., Butterfield, P., and Campos, J.J. (1987) Social Referencing: The Infant's Use of Emotional Signals from a Friendly Adult with Mother Present. *Annual Progress in Child Psychiatry and Child Development* 22: 427-432.

Kornreich, C., Blairy, S., Philippot, P., Hess, U., Noel, X., Streel, E. *et al.* (2001) Deficits in Recognition of Emotional Facial Expression Are Still Present in Alcoholics after Mid- to Long-Term Abstinence. *J Stud Alcohol* 62: 533-542.

Kulikov, A.V., Osipova, D.V., Naumenko, V.S., Terenina, E., Mormede, P., and Popova, N.K. (2012) A Pharmacological Evidence of Positive Association between Mouse Intermale Aggression and Brain Serotonin Metabolism. *Behav Brain Res* 233: 113-119.

Ledoux, J.E. (2000) Emotion Circuits in the Brain. *Annu Rev Neurosci* 23: 155-184.

Lee, T.M., Chan, S.C., and Raine, A. (2009) Hyperresponsivity to Threat Stimuli in Domestic Violence Offenders: A Functional Magnetic Resonance Imaging Study. *J Clin Psychiatry* 70: 36-45.

Leonard, K.E. and Blane, H.T. (1992) Alcohol and Marital Aggression in a National Sample of Young Men. *Journal of Interpersonal Violence* 7: 19-30.

Leonard, K.E. and Senchak, M. (1996) Prospective Prediction of Husband Marital Aggression within Newlywed Couples. *J Abnorm Psychol* 105: 369-380.

Liang, W. and Chikritzhs, T. (2011) Revealing the Link between Licensed Outlets and Violence: Counting Venues Versus Measuring Alcohol Availability. *Drug Alcohol Rev* 30: 524-535.

Lindman, R. (1983) Alcohol and the Reduction of Human Fear. In: Pohorecky, L. and Brick, J., (eds), *Stress and Alcohol Use*. Elsevier: Amsterdam.

Lindman, R.E. and Lang, A.R. (1994) The Alcohol-Aggression Stereotype: A Cross-Cultural Comparison of Beliefs. *Int J Addict* 29: 1-13.

Lipsey, M.W., Wilson, D.B., Cohen, M.A., and Derzon, J.H. (1997) Is There a Causal Relationship between Alcohol Use and Violence? A Synthesis of Evidence. *Recent Dev Alcohol* 13: 245-282.

Livingston, M. (2011) A Longitudinal Analysis of Alcohol Outlet Density and Domestic Violence. *Addiction* 106: 919-925.

Lundholm, L., Haggard, U., Moller, J., Hallqvist, J., and Thiblin, I. (2012) The Triggering Effect of Alcohol and Illicit Drugs on Violent Crime in a Remand Prison Population: A Case Crossover Study. *Drug Alcohol Depend*:

Mair, C., Gruenewald, P.J., Ponicki, W.R., and Remer, L. (2013) Varying Impacts of Alcohol Outlet Densities on Violent Assaults: Explaining Differences across Neighborhoods. *J Stud Alcohol Drugs* 74: 50-58.

Makris, N., Oscar-Berman, M., Jaffin, S.K., Hodge, S.M., Kennedy, D.N., Caviness, V.S. *et al.* (2008) Decreased Volume of the Brain Reward System in Alcoholism. *Biol Psychiatry* 64: 192-202.

Marinetti, C., Mesquita, B., Yik, M., Cragwall, C., and Gallagher, A.H. (2012) Threat Advantage: Perception of Angry and Happy Dynamic Faces across Cultures. *Cogn Emot* 26: 1326-1334.

Marinkovic, K., Oscar-Berman, M., Urban, T., O'reilly, C.E., Howard, J.A., Sawyer, K. *et al.* (2009) Alcoholism and Dampened Temporal Limbic Activation to Emotional Faces. *Alcohol Clin Exp Res* 33: 1880-1892.

Marquez, C., Poirier, G.L., Cordero, M.I., Larsen, M.H., Groner, A., Marquis, J. *et al.* (2013) Peripuberty Stress Leads to Abnormal Aggression, Altered Amygdala and Orbitofrontal Reactivity and Increased Prefrontal Maa Gene Expression. *Transl Psychiatry* 3: e216.

Marsh, A.A. and Blair, R.J. (2008) Deficits in Facial Affect Recognition among Antisocial Populations: A Meta-Analysis. *Neurosci Biobehav Rev* 32: 454-465.

Marsh, A.A., Kozak, M.N., and Ambady, N. (2007) Accurate Identification of Fear Facial Expressions Predicts Prosocial Behavior. *Emotion* 7: 239-251.

Marwick, K. and Hall, J. (2008) Social Cognition in Schizophrenia: A Review of Face Processing. *Br Med Bull* 88: 43-58.

Matthies, S., Rusch, N., Weber, M., Lieb, K., Philipsen, A., Tuescher, O. *et al.* (2012) Small Amygdala-High Aggression? The Role of the Amygdala in Modulating Aggression in Healthy Subjects. *World J Biol Psychiatry* 13: 75-81.

Mcclelland, G.M. and Teplin, L.A. (2001) Alcohol Intoxication and Violent Crime: Implications for Public Health Policy. *Am J Addict* 10 Suppl: 70-85.

Mccloskey, M.S., Berman, M.E., Echevarria, D.J., and Coccaro, E.F. (2009) Effects of Acute Alcohol Intoxication and Paroxetine on Aggression in Men. *Alcohol Clin Exp Res* 33: 581-590.

Mcdonald, A.J. (1991) Organization of Amygdaloid Projections to the Prefrontal Cortex and Associated Striatum in the Rat. *Neuroscience* 44: 1-14.

Miczek, K.A., Barros, H.M., Sakoda, L., and Weerts, E.M. (1998) Alcohol and Heightened Aggression in Individual Mice. *Alcohol Clin Exp Res* 22: 1698-1705.

Miczek, K.A. and Barry, H., 3rd. (1977) Effects of Alcohol on Attack and Defensive-Submissive Reactions in Rats. *Psychopharmacology (Berl)* 52: 231-237.

Miczek, K.A. and De Almeida, R.M. (2001) Oral Drug Self-Administration in the Home Cage of Mice: Alcohol-Heightened Aggression and Inhibition by the 5-Ht1b Agonist Anpirtoline. *Psychopharmacology (Berl)* 157: 421-429.

Miczek, K.A., Debold, J.F., Van Erp, A.M., and Tornatzky, W. (1997) Alcohol, Gabaa-Benzodiazepine Receptor Complex, and Aggression. *Recent Dev Alcohol* 13: 139-171.

Miczek, K.A., Fish, E.W., De Almeida, R.M., Faccidomo, S., and Debold, J.F. (2004) Role of Alcohol Consumption in Escalation to Violence. *Ann N Y Acad Sci* 1036: 278-289.

Miczek, K.A., Fish, E.W., De Bold, J.F., and De Almeida, R.M. (2002) Social and Neural Determinants of Aggressive Behavior: Pharmacotherapeutic Targets at Serotonin, Dopamine and Gamma-Aminobutyric Acid Systems. *Psychopharmacology (Berl)* 163: 434-458.

Miczek, K.A. and O'donnell, J.M. (1980) Alcohol and Chlordiazepoxide Increase Suppressed Aggression in Mice. *Psychopharmacology (Berl)* 69: 39-44.

Miczek, K.A., Weerts, E.M., Tornatzky, W., Debold, J.F., and Vatne, T.M. (1992) Alcohol and "Bursts" of Aggressive Behavior: Ethological Analysis of Individual Differences in Rats. *Psychopharmacology (Berl)* 107: 551-563.

- Miczek, K.A., Winslow, J.T., and Debold, J.F. (1984) Heightened Aggressive Behavior by Animals Interacting with Alcohol-Treated Conspecifics: Studies with Mice, Rats and Squirrel Monkeys. *Pharmacol Biochem Behav* 20: 349-353.
- Miller, P.A. and Eisenberg, N. (1988) The Relation of Empathy to Aggressive and Externalizing/Antisocial Behavior. *Psychol Bull* 103: 324-344.
- Mineka, S., Davidson, M., Cook, M., and Keir, R. (1984) Observational Conditioning of Snake Fear in Rhesus Monkeys. *J Abnorm Psychol* 93: 355-372.
- Miranda, R., Jr., Meyerson, L.A., Buchanan, T.W., and Lovallo, W.R. (2002) Altered Emotion-Modulated Startle in Young Adults with a Family History of Alcoholism. *Alcohol Clin Exp Res* 26: 441-448.
- Mogg, K. and Bradley, B.P. (1999) Orienting of Attention to Threatening Facial Expression Presented under Conditions of Restricted Awareness. *Cognition and Emotion* 13: 713-740.
- Montoya, E.R., Terburg, D., Bos, P.A., and Van Honk, J. (2012) Testosterone, Cortisol, and Serotonin as Key Regulators of Social Aggression: A Review and Theoretical Perspective. *Motiv Emot* 36: 65-73.
- Morris, J.S., Frith, C.D., Perrett, D.I., Rowland, D., Young, A.W., Calder, A.J. *et al.* (1996) A Differential Neural Response in the Human Amygdala to Fearful and Happy Facial Expressions. *Nature* 383: 812-815.
- Moya-Albiol, L., Herrero, N., and Bernal, M.C. (2010) The Neural Bases of Empathy. *Rev Neurol* 50: 89-100.
- Murdoch, D., Pihl, R.O., and Ross, D. (1990) Alcohol and Crimes of Violence: Present Issues. *Int J Addict* 25: 1065-1081.
- Neff, J.A. (1991) Race, Ethnicity and Drinking Patterns: The Role of Demographic Factors, Drinking Motives and Expectancies. In: Pittman, D.J. and White, H.R., (eds), *Society, Culture and Drinking Patterns Re-Examined*. Rutgers Centre for Alcohol Studies: Piscataway, NJ, USA.
- Nelson, R.J. and Trainor, B.C. (2007) Neural Mechanisms of Aggression. *Nat Rev Neurosci* 8: 536-546.
- Newberry, M., Williams, N., and Caulfield, L. (2013) Female Alcohol Consumption, Motivations for Aggression and Aggressive Incidents in Licensed Premises. *Addict Behav* 38: 1844-1851.
- Ohman, A. (2005) The Role of the Amygdala in Human Fear: Automatic Detection of Threat. *Psychoneuroendocrinology* 30: 953-958.

- Ohman, A. and Mineka, S. (2001) Fears, Phobias, and Preparedness: Toward an Evolved Module of Fear and Fear Learning. *Psychol Rev* 108: 483-522.
- Oscar-Berman, M., Hancock, M., Mildworf, B., Hutner, N., and Weber, D.A. (1990) Emotional Perception and Memory in Alcoholism and Aging. *Alcohol Clin Exp Res* 14: 383-393.
- Oscar-Berman, M. and Marinkovic, K. (2007) Alcohol: Effects on Neurobehavioral Functions and the Brain. *Neuropsychol Rev* 17: 239-257.
- Oyegbile, T.O. and Marler, C.A. (2006) Weak Winner Effect in a Less Aggressive Mammal: Correlations with Corticosterone but Not Testosterone. *Physiol Behav* 89: 171-179.
- Paglia, A. and Room, R. (2006) Expectancies About the Effects of Alcohol on the Self and on Others as Determinants of Alcohol Policy Attitudes. *Journal of Applied Social Psychology* 29: 2632-2651.
- Parrott, D.J., Gallagher, K.E., and Zeichner, A. (2012) Liquid Courage or Liquid Fear: Alcohol Intoxication and Anxiety Facilitate Physical Aggression. *Subst Use Misuse* 47: 774-786.
- Parrott, D.J. and Giancola, P.R. (2004) A Further Examination of the Relation between Trait Anger and Alcohol-Related Aggression: The Role of Anger Control. *Alcohol Clin Exp Res* 28: 855-864.
- Passamonti, L., Crockett, M.J., Apergis-Schoute, A.M., Clark, L., Rowe, J.B., Calder, A.J. *et al.* (2012) Effects of Acute Tryptophan Depletion on Prefrontal-Amygdala Connectivity While Viewing Facial Signals of Aggression. *Biol Psychiatry* 71: 36-43.
- Penton-Voak, I.S., Thomas, J., Gage, S.H., McMurrin, M., McDonald, S., and Munafò, M.R. (2013) Increasing Recognition of Happiness in Ambiguous Facial Expressions Reduces Anger and Aggressive Behavior. *Psychol Sci* 24: 688-697.
- Pernanen, K. (1991) Alcohol in Human Violence. Guildford Press: New York.
- Pfabigan, D.M., Alexopoulos, J., and Sailer, U. (2012) Exploring the Effects of Antisocial Personality Traits on Brain Potentials During Face Processing. *PLoS One* 7: e50283.
- Pfefferbaum, A., Sullivan, E.V., Rosenbloom, M.J., Mathalon, D.H., and Lim, K.O. (1998) A Controlled Study of Cortical Gray Matter and Ventricular Changes in Alcoholic Men over a 5-Year Interval. *Arch Gen Psychiatry* 55: 905-912.

- Philippot, P., Kornreich, C., Blairy, S., Baert, I., Den Dulk, A., Le Bon, O. *et al.* (1999) Alcoholics' Deficits in the Decoding of Emotional Facial Expression. *Alcohol Clin Exp Res* 23: 1031-1038.
- Phillips, M.L., Young, A.W., Senior, C., Brammer, M., Andrew, C., Calder, A.J. *et al.* (1997) A Specific Neural Substrate for Perceiving Facial Expressions of Disgust. *Nature* 389: 495-498.
- Pihl, R.O. and Sutton, R. (2009) Drugs and Aggression Readily Mix; So What Now? *Subst Use Misuse* 44: 1188-1203.
- Pohorecky, L.A. (1991) Stress and Alcohol Interaction: An Update of Human Research. *Alcohol Clin Exp Res* 15: 438-459.
- Popova, N.K. (2008) From Gene to Aggressive Behavior: The Role of Brain Serotonin. *Neurosci Behav Physiol* 38: 471-475.
- Premkumar, P., Cooke, M.A., Fannon, D., Peters, E., Michel, T.M., Aasen, I. *et al.* (2008) Misattribution Bias of Threat-Related Facial Expressions Is Related to a Longer Duration of Illness and Poor Executive Function in Schizophrenia and Schizoaffective Disorder. *Eur Psychiatry* 23: 14-19.
- Quigley, B.M., Corbett, A.B., and Tedeschi, J.T. (2002) Desired Image of Power, Alcohol Expectancies, and Alcohol-Related Aggression. *Psychol Addict Behav* 16: 318-324.
- Quinn, P.D., Stappenbeck, C.A., and Fromme, K. (2013) An Event-Level Examination of Sex Differences and Subjective Intoxication in Alcohol-Related Aggression. *Exp Clin Psychopharmacol* 21: 93-102.
- Ray, J.G., Moineddin, R., Bell, C.M., Thiruchelvam, D., Creatore, M.I., Gozdyra, P. *et al.* (2008) Alcohol Sales and Risk of Serious Assault. *PLoS Med* 5: e104.
- Resko, S.M., Walton, M.A., Bingham, C.R., Shope, J.T., Zimmerman, M., Chermack, S.T. *et al.* (2010) Alcohol Availability and Violence among Inner-City Adolescents: A Multi-Level Analysis of the Role of Alcohol Outlet Density. *Am J Community Psychol* 46: 253-262.
- Richardson, A. and Budd, T. (2003) Young Adults, Alcohol, Crime and Disorder. *Crim Behav Ment Health* 13: 5-16.
- Rolls, E.T. (2004) Convergence of Sensory Systems in the Orbitofrontal Cortex in Primates and Brain Design for Emotion. *Anat Rec A Discov Mol Cell Evol Biol* 281: 1212-1225.
- Rolls, E.T., Critchley, H.D., Browning, A.S., and Inoue, K. (2006) Face-Selective and Auditory Neurons in the Primate Orbitofrontal Cortex. *Exp Brain Res* 170: 74-87.

Romanski, L.M. and Ledoux, J.E. (1992) Equipotentiality of Thalamo-Amygdala and Thalamo-Cortico-Amygdala Circuits in Auditory Fear Conditioning. *J Neurosci* 12: 4501-4509.

Rule, R.R., Shimamura, A.P., and Knight, R.T. (2002) Orbitofrontal Cortex and Dynamic Filtering of Emotional Stimuli. *Cogn Affect Behav Neurosci* 2: 264-270.

Sander, T., Harms, H., Dufeu, P., Kuhn, S., Hoehe, M., Lesch, K.P. *et al.* (1998) Serotonin Transporter Gene Variants in Alcohol-Dependent Subjects with Dissocial Personality Disorder. *Biol Psychiatry* 43: 908-912.

Sartor, C.E., Waldron, M., Duncan, A.E., Grant, J.D., Mccutcheon, V.V., Nelson, E.C. *et al.* (2013) Childhood Sexual Abuse and Early Substance Use in Adolescent Girls: The Role of Familial Influences. *Addiction* 108: 993-1000.

Sayette, M.A. (1993) An Appraisal-Disruption Model of Alcohol's Effects on Stress Responses in Social Drinkers. *Psychol Bull* 114: 459-476.

Sayette, M.A. (1999) Does Drinking Reduce Stress? *Alcohol Res Health* 23: 250-255.

Sayette, M.A., Creswell, K.G., Dimoff, J.D., Fairbairn, C.E., Cohn, J.F., Heckman, B.W. *et al.* (2012) Alcohol and Group Formation: A Multimodal Investigation of the Effects of Alcohol on Emotion and Social Bonding. *Psychol Sci* 23: 869-878.

Schneider, F., Weiss, U., Kessler, C., Salloum, J.B., Posse, S., Grodd, W. *et al.* (1998) Differential Amygdala Activation in Schizophrenia During Sadness. *Schizophr Res* 34: 133-142.

Schofield, T.P. and Denson, T.F. (2013) Alcohol Outlet Business Hours and Violent Crime in New York State. *Alcohol Alcohol*:

Scott, Schafer, J., and Greenfield, T.K. (1999) The Role of Alcohol in Physical Assault Perpetration and Victimization. *J Stud Alcohol* 60: 528-536.

Shamay-Tsoory, S.G. (2011) The Neural Bases for Empathy. *Neuroscientist* 17: 18-24.

Shepherd, J.P., Sutherland, I., and Newcombe, R.G. (2006) Relations between Alcohol, Violence and Victimization in Adolescence. *J Adolesc* 29: 539-553.

Sinzig, J., Morsch, D., and Lehmkuhl, G. (2008) Do Hyperactivity, Impulsivity and Inattention Have an Impact on the Ability of Facial Affect Recognition in Children with Autism and Adhd? *Eur Child Adolesc Psychiatry* 17: 63-72.

Soma, K.K. (2006) Testosterone and Aggression: Berthold, Birds and Beyond. *J Neuroendocrinol* 18: 543-551.

Stappenbeck, C.A. and Fromme, K. (2010) Alcohol Use and Perceived Social and Emotional Consequences among Perpetrators of General and Sexual Aggression. *J Interpers Violence* 25: 699-715.

Steele, C.M. and Josephs, R.A. (1990) Alcohol Myopia: Its Prized and Dangerous Effects. *American Psychologist* 45: 921-933.

Steele, C.M. and Southwick, L. (1985) Alcohol and Social Behavior I: The Psychology of Drunken Excess. *Journal of Personality and Social Psychology* 48: 18-34.

Sterzer, P., Stadler, C., Krebs, A., Kleinschmidt, A., and Poustka, F. (2005) Abnormal Neural Responses to Emotional Visual Stimuli in Adolescents with Conduct Disorder. *Biol Psychiatry* 57: 7-15.

Stevens, S., Gerlach, A.L., and Rist, F. (2008) Effects of Alcohol on Ratings of Emotional Facial Expressions in Social Phobics. *J Anxiety Disord* 22: 940-948.

Stevens, S., Rist, F., and Gerlach, A.L. (2009) Influence of Alcohol on the Processing of Emotional Facial Expressions in Individuals with Social Phobia. *Br J Clin Psychol* 48: 125-140.

Subra, B., Muller, D., Begue, L., Bushman, B.J., and Delmas, F. (2010) Automatic Effects of Alcohol and Aggressive Cues on Aggressive Thoughts and Behaviors. *Pers Soc Psychol Bull* 36: 1052-1057.

Takahashi, A., Quadros, I.M., De Almeida, R.M., and Miczek, K.A. (2011) Brain Serotonin Receptors and Transporters: Initiation Vs. Termination of Escalated Aggression. *Psychopharmacology (Berl)* 213: 183-212.

Taylor. (1967) Aggressive Behavior and Physiological Arousal as a Function of Provocation and a Tendency to Inhibit Aggression. *Journal of Personality* 35: 297-310.

Taylor, S.P. and Chermack, S.T. (1993) Alcohol, Drugs and Human Physical Aggression. *J Stud Alcohol Suppl* 11: 78-88.

Testa, M. (2002) The Impact of Men's Alcohol Consumption on Perpetration of Sexual Aggression. *Clin Psychol Rev* 22: 1239-1263.

Thoma, P., Friedmann, C., and Suchan, B. (2013) Empathy and Social Problem Solving in Alcohol Dependence, Mood Disorders and Selected Personality Disorders. *Neurosci Biobehav Rev* 37: 448-470.

Townshend, J.M. and Duka, T. (2003) Mixed Emotions: Alcoholics' Impairments in the Recognition of Specific Emotional Facial Expressions. *Neuropsychologia* 41: 773-782.

Tremblay, P.F., Graham, K., and Wells, S. (2008) Severity of Physical Aggression Reported by University Students: A Test of the Interaction between Trait Aggression and Alcohol Consumption *Personality and Individual Differences* 45: 3-9.

Tremblay, P.F., Mihic, L., Graham, K., and Jelley, J. (2007) Role of Motivation to Respond to Provocation, the Social Environment, and Trait Aggression in Alcohol-Related Aggression. *Aggress Behav* 33: 389-411.

Tucker, J.A. and Vuchinich, R.E. (1983) An Information Processing Analysis of the Effects of Alcohol on Perceptions of Facial Emotions. *Psychopharmacology (Berl)* 79: 215-219.

Van Der Veen, F.M., Evers, E.A., Deutz, N.E., and Schmitt, J.A. (2007) Effects of Acute Tryptophan Depletion on Mood and Facial Emotion Perception Related Brain Activation and Performance in Healthy Women with and without a Family History of Depression. *Neuropsychopharmacology* 32: 216-224.

Van Honk, J., Tuiten, A., Hermans, E., Putman, P., Koppeschaar, H., Thijssen, J. *et al.* (2001) A Single Administration of Testosterone Induces Cardiac Accelerative Responses to Angry Faces in Healthy Young Women. *Behav Neurosci* 115: 238-242.

Van Honk, J., Tuiten, A., Verbaten, R., Van Den Hout, M., Koppeschaar, H., Thijssen, J. *et al.* (1999) Correlations among Salivary Testosterone, Mood, and Selective Attention to Threat in Humans. *Horm Behav* 36: 17-24.

Van Wingen, G., Mattern, C., Verkes, R.J., Buitelaar, J., and Fernandez, G. (2010) Testosterone Reduces Amygdala-Orbitofrontal Cortex Coupling. *Psychoneuroendocrinology* 35: 105-113.

Vasquez, E.A., Pedersen, W.C., Bushman, B.J., Kelley, N.J., Demeestere, P., and Miller, N. (2012) Lashing out after Stewing over Public Insults: The Effects of Public Provocation, Provocation Intensity, and Rumination on Triggered Displaced Aggression. *Aggress Behav*:

Virkkunen, M. and Linnoila, M. (1993) Brain Serotonin, Type II Alcoholism and Impulsive Violence. *J Stud Alcohol Suppl* 11: 163-169.

Vogel-Sprott, M. (1967) Alcohol Effects on Human Behaviour under Reward and Punishment. *Psychopharmacologia* 11: 337-344.

Volkow, N.D., Wang, G.J., and Doria, J.J. (1995) Monitoring the Brain's Response to Alcohol with Positron Emission Tomography. *Alcohol Health Research World* 19: 296-299.

Vuilleumier, P. and Schwartz, S. (2001) Beware and Be Aware: Capture of Spatial Attention by Fear-Related Stimuli in Neglect. *Neuroreport* 12: 1119-1122.

Wang, L., McCarthy, G., Song, A.W., and Labar, K.S. (2005) Amygdala Activation to Sad Pictures During High-Field (4 Tesla) Functional Magnetic Resonance Imaging. *Emotion* 5: 12-22.

Wells, S. and Graham, K. (2003) Aggression Involving Alcohol: Relationship to Drinking Patterns and Social Context. *Addiction* 98: 33-42.

Wells, S., Graham, K., and West, P. (2000) Alcohol-Related Aggression in the General Population. *J Stud Alcohol* 61: 626-632.

Whalen, P.J., Rauch, S.L., Etkoff, N.L., Mcinerney, S.C., Lee, M.B., and Jenike, M.A. (1998) Masked Presentations of Emotional Facial Expressions Modulate Amygdala Activity without Explicit Knowledge. *J Neurosci* 18: 411-418.

Wilkowski, B.M. and Meier, B.P. (2010) Bring It On: Angry Facial Expressions Potentiate Approach-Motivated Motor Behavior. *J Pers Soc Psychol* 98: 201-210.

Williams, J.H., Perrett, D.I., Waiter, G.D., and Pechey, S. (2007) Differential Effects of Tryptophan Depletion on Emotion Processing According to Face Direction. *Soc Cogn Affect Neurosci* 2: 264-273.

Wirth, M.M. and Schultheiss, O.C. (2007) Basal Testosterone Moderates Responses to Anger Faces in Humans. *Physiol Behav* 90: 496-505.

Wright, I.C., Rabe-Hesketh, S., Woodruff, P.W., David, A.S., Murray, R.M., and Bullmore, E.T. (2000) Meta-Analysis of Regional Brain Volumes in Schizophrenia. *Am J Psychiatry* 157: 16-25.

Zeichner, A. and Pihl, R.O. (1979) Effects of Alcohol and Behaviour Contingencies on Human Aggression. *Journal of Abnormal Psychology* 88: 153-160.

Zeichner, A. and Pihl, R.O. (1980) Effects of Alcohol and Instigator Intent on Human Aggression. *Journal of Studies on Alcohol* 41: 265-276.

Zhang, L., Wieczorek, W.F., and Welte, J.W. (1997) The Nexus between Alcohol and Violent Crime. *Alcohol Clin Exp Res* 21: 1264-1271.