The effects of exposure to appetitive cues on inhibitory control: A meta-analytic investigation

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ABSTRACT

Inhibitory control refers to the ability to stop, change or delay a response, and is often used in order to protect higher order goals. Theoretical models suggest that appetitive cues such as pictures of alcoholic drinks or food evoke strong automatic appetitive responses which lead to transient impairments in inhibitory control, and that these effects of cues may be related to individual differences (e.g. in body mass index, or alcohol consumption).

In order to investigate these claims we conducted a random effects meta-analysis of 66 effect sizes (35 alcohol, 31 food) from 37 articles that tested the effect of exposure to appetitive (alcohol/food) cues on indices of inhibitory control. The overall effect of cue exposure was small, but robust (SMD = −0.12 [95% CI −0.23, −0.02]; \(Z = 2.34, p = .02, \text{i}^2 = 84\%\)). Exposure to alcohol-related cues significantly impaired inhibitory control (SMD = −0.21 [95% CI −0.32, −0.11]; \(Z = 4.17, p < .001\), however exposure to food-related cues did not lead to impairments (SMD = −0.03 [95% CI −0.21, 0.15]; \(Z = 0.36, p = .720\)). There was no evidence that drinking or weight status significantly moderated the effects of cues on inhibitory control. Similarly, cue modality (words, pictures, or smells) did not significantly moderate the effects. Trim and Fill analysis suggested bias in the literature, which when corrected, made the overall effect of cues non-significant. Overall, these findings provide some tentative support for theoretical claims that exposure to appetitive cues prompts transient impairments in inhibitory control. Further research is required to determine the clinical significance of these observations. However, care should be taken when drawing conclusions from a potentially biased evidence base.

1. Introduction

Inhibitory control refers to the ability to stop, change or delay a response that is inappropriate given current environment demands (Logan, Cowan, & Davis, 1984; Logan, Schachar, & Tannock, 1997). This (in)ability is a key component of impulsivity and executive functioning (Bickel, Jailmolowicz, Mueller, Gatchalian, & McClure, 2012), and it overlaps considerably with broader constructs such as self-control and disinhibition (Baumeister, 2014; Tarter, Kirisci, Reynolds, & Ridderinkhof, 2013) and maintain their higher order goals such as abstaining from alcohol or weight-loss, even when tempted by environmental cues such as the sight or smell of appetising foods or alcoholic drinks (Jones, Hardman, Lawrence, & Field, 2017; Verbruggen, Best, Bowditch, Stevens, & McLaren, 2014).

Theoretical models of both addiction and obesity posit that deficits in inhibitory control are an important contributor to the development and maintenance of these conditions (Goldstein & Volkow, 2011; Volkow, Wang, Tomasi, & Baler, 2013). Laboratory research using established measures of inhibitory control such as the Stop Signal and Go/No-Go tasks (Verbruggen & Logan, 2008) generally supports the predictions made by these models. For example, there are robust cross-sectional associations between impairments in inhibitory control and hazardous drinking (Christiansen, Cole, Goudie, & Field, 2012; Smith, Mattick, Jamadar, & Iredale, 2014), and also Body Mass Index (BMI) and indicators of unhealthy eating (Nederkoorn, Smulders, Havermans, Roefs, & Jansen, 2006; Vainik, Dagher, Dubé, & Fellows, 2013). Longitudinal studies demonstrate that deficits in inhibitory control predict subsequent alcohol use (Fernie et al., 2013), transition to dependence (Rubio et al., 2008) and treatment outcomes (Petit et al., 2014). Similar studies also show poorer inhibitory control predicts weight gain over
one year (Nederkoorn, Houben, Hofmann, Roefs, & Jansen, 2010). Findings such as these are generally interpreted as indicating that inhibitory control is a stable trait characteristic that differs between individuals but remains fairly constant within individuals, which is why it reliably predicts between-subject variability in behaviour months or years later. However, more recent theoretical models have suggested that inhibitory control functions as a transient state which can fluctuate in response to environmental or internal ‘events’ (De Wit, 2009), and these short term impairments in inhibitory control may increase the immediate risk of temptation and subsequent (re) lapse. In a recent narrative review we (Jones, Christiansen, Nederkoorn, Houben, & Field, 2013a) suggested that individuals have a general stable capacity for inhibitory control, however this capacity can fluctuate (both improve and worsen) in response to environmental and internal events. Exposure to appetitive cues is one potential environmental event that may negatively influence inhibitory control, because those cues evoke automatic appetitive tendencies (Brockmeyer, Hahn, Reetz, Schmidt, & Friederich, 2015; Field, Kiernan, Eastwood, & Child, 2008; Kemps & Tiggemann, 2015; Kemps, Tiggemann, Martin, & Elliott, 2013), and these responses should conflict with inhibition of behaviour. Indeed, exposure to both alcohol (Czapla et al., 2015a,b) and food-related cues (Phelan et al., 2011) result in short-lived impairments in inhibitory control. Furthermore, these ‘cue-specific’ deficits in inhibition may predict greater variance in individual differences in health-related behaviours than general inhibition deficits (Houben, Nederkoorn, & Jansen, 2013; Petit, Kornreich, Noël, Verbanck, & Campanella, 2012), including ad-libitum food and alcohol consumption in the laboratory (Field & Jones, 2017; Price, Lee, & Higgs, 2016). However, as with many research questions that are studied intensively, there are some null or equivocal findings in the literature, in which cue-exposure has not impaired inhibitory control (Mainz et al., 2012; Nederkoorn, Baltus, Guerrieri, & Wiers, 2009).

The aim of the present meta-analytic investigation was to quantify the extent to which exposure to appetitive-cues (alcohol and food-related pictures) causes transient impairments in the ability to inhibit behaviour, and to identify procedural variables or participant characteristics that may moderate this effect. Following initial scoping searches, we limited our investigation to alcohol and food-related cues because the vast majority of studies in the field were limited to these domains (however, the disinhibiting effects of smoking: (Luijten, Littel, & Franken, 2011) and drug-related cues (Pike, Stoops, Fillmore, & Rush, 2013), have been investigated). We sought to identify potential moderators of the effects of appetitive cues on inhibitory control, including: drinking/weight status, the modality of cue-exposure (pictorial, lexical, olfactory), type of task used to measure inhibitory control, and to determine if cue-specific inhibitory deficits are associated with unhealthy behaviours or outcomes, such as alcohol consumption or BMI. We hypothesised that inhibitory control would be worse during or after exposure to appetitive cues compared to neutral cues, or the absence of cues. For our moderator analyses we predicted that this effect would be larger in heavier drinkers, individuals with alcohol use disorder and individuals with overweight/obesity because, theoretically, automatic appetitive responses to appetitive cues should be stronger in people who consume them more frequently (Volkow et al., 2013; Wiers et al., 2007). We had no a priori predictions regarding cue-modality, but we investigated this on the basis of findings from some individual studies which demonstrated differential effects of cues presented in different modalities on both inhibitory control and subjective craving (Boswell & Kober, 2016; Monk, Sunley, Qureshi, & Heim, 2016). Similarly, we examined the effects of different inhibitory control tasks as each have differing inhibitory pressures and may measure a different type of inhibitory control, e.g. action cancellation versus action inhibition (Eagle, Bari, & Robbins, 2008). However, we made no specific hypothesis as to which may be most affected by cue-exposure.

2. Methods

2.1. Information sources and search strategy

We conducted scoping searches using three commonly used electronic databases (Scopus, PubMed and PsycInfo) in November and December 2015. We pre-registered our protocol and analysis strategy on Open Science Framework (https://osf.io/c9j8/). Full searches were carried out in October 2017. Our literature search was guided by the Preferred Reporting Items for Systematic Review (PRISMA) guidelines. See supplementary materials for full search strategy and terms. Following identification of full text articles we conducted manual searches on reference lists, and identified further articles based on authors’ knowledge. In total we identified 35 effect sizes for alcohol and 31 for food.

2.2. Eligibility criteria

All studies had to meet the following criteria in order to be included in the meta-analysis; (i) include human participants aged 18+, (ii) include alcohol or food-related (appetitive) cue exposure, i.e. olfactory or visual cues, prior to or during an inhibitory control task, (iii) a control comparison, for example exposure to neutral cues during, or the absence of cue exposure (baseline) prior to or during, an inhibitory control task. Cue exposure involved food/alcohol and neutral images/words that were embedded into an inhibitory control task (Houben et al., 2013; Jones et al., 2015), or the holding and sniffing of food/alcohol prior to completing an inhibitory control task (Gauggel et al., 2010; Lattimore & Mead, 2015).

2.3. Outcome measure(s)

Studies were required to have an outcome measure of inhibitory control during/following appetitive cue exposure, and either a measure of inhibitory control at baseline (prior to cue-exposure) or during exposure to non-appetitive (neutral) cues. Proposed measures of inhibitory control were cross-checked against previous literature and review papers to ensure that they were validated measures (e.g. Diamond, 2013). All authors agreed on the tasks for inclusion.

2.4. Data extraction and coding

Three independent coders (JD, IK, NC) performed the searches and identified the relevant articles. After removal of duplicates, 4151 unique articles were identified. These articles were screened via title and abstract, which resulted in exclusion of 3819 articles with agreement from all coders. Data were extracted by the coders and cross-checked by the first author. In cases where insufficient data was available the authors were contacted to provide this data. If the authors did not respond to the data request and it was possible, we used Web Plot Digitizer (Version 3.10, Rohatgi, 2016) to estimate means and variances from figures presented in publications, as recommended (Jelicic Kadic, Vucic, Dosenovic, Sapunar, & Puljak, 2016; Vucic, Jelicic Kadic, & Puljak, 2015).

To code moderator variables such as drinking and weight status we first examined if any participants were described in the article in a specific way (e.g. alcohol dependent, overweight). If no explicit claims were made we made group level inferences on alcohol use based on established cut-offs for ‘heavy’ or ‘hazardous’ drinking via scores on the AUDIT (score > 8 indicative of hazardous drinking (see Saunders, Asland, Babor, De La Fuente, & Grant, 1993) or estimates of units of alcohol consumed per week (> 14 units per week indicative of heavy drinking)). For weight status we examined if group mean BMI > 25 kg/
m² (for overweight/obese). Three studies compared alcohol-dependent participants to a control group (Noel et al., 2005; Noel et al., 2007; Sion, Jurado-Barba, Alonso, & Rubio-Valladares, 2017), but provided no information as to whether the control group drank any alcohol. Therefore, the control groups in these studies were not included in any analysis.

2.5. Variables of interest

The indices of inhibition used for each task are stated in Table 1. The most common tasks were the Stop Signal, Go/No-Go and Go/No-Go shifting tasks. The Stop Signal and Go/No-Go tasks require motor inhibition of a pre-potent response following a visual or auditory ‘stop signal’ or ‘No-Go cue’. In the Stop Signal task this cue is presented following a variable delay after initial stimulus onset and therefore motor behaviour has to be cancelled, whereas in the No-Go task the No-Go cue is presented concurrently with the target stimulus, and therefore behaviour must be restrained rather than cancelled (Eagle et al., 2008).

In the shifting version of the task the cues for ‘Go’ and ‘No-Go’ are switched on a block-by-block basis (Meule, 2017). In the anti-saccade task participants have to inhibit an involuntary oculomotor response (saccade) to a visual stimulus that appears in the periphery of a visual display (Hallett, 1978). In the Stroop task (Stroop, 1935) participants have to name the colour of target words whilst ignoring the semantic content of the word (e.g., the word ‘red’ printed in blue ink). Finally, in the flanker task participants have to categorise a target stimulus whilst ignoring distractor stimuli that appear alongside it (Eriksen & Eriksen, 1974). Stop Signal Reaction Time and Commission errors were the most common outcomes from these tasks. We also extracted and coded a number of variables for our main and supplementary analyses, including: type of task used, modality of cue exposure, drinking and weight status, and any correlations with BMI, typical alcohol use or AUDIT scores (see Table 1). We selected these variables as they were the most commonly measured across all studies.

2.6. Statistical analyses

Our main statistical analyses were carried out using Review Manager 5.3 (Cochrane Informatics & Knowledge Management Department, UK, 2014), with supplementary analyses conducted using JASP (JASP team, Version 0.8.4). All outcomes were continuous, therefore we computed the Standardised Mean Difference (SMD) effect size using the equation $SMD = (M_a - M_r)/S_p$, where $M_a$ is the mean inhibitory control measure following exposure to appetitive cues, $M_r$ is the mean following neutral cues (or no cues) and $S_p$ is the pooled standard deviation. We also computed the Standard Error of the SMD; calculated as $d = \frac{d}{\sqrt{(Q - df/Q)} \times 100\%}$, where $Q$ is the chi-squared statistic and df is the accompanying degrees of freedom. We used random-effects models due to substantial heterogeneity between studies (Riley, Higgins, & Deeks, 2011). In the case of substantial heterogeneity we provided estimates of subgroup effects to aid interpretation of the data. Finally, to remove outlying effect sizes we calculated z-scores and identified any effect size which was an extreme value at .001 alpha level (i.e. $Z > 3.30$). As a result one effect size (Petit et al., 2012; Light drinkers: SMD > 6) was excluded from all subsequent analyses. This decision was made a-priori, as evidenced in our pre-registration.

2.7. Characteristics of studies

The majority of studies employed within-subject (repeated measures) designs, in which participants either (a) completed similar inhibitory control tasks during or after exposure to appetitive cues and neutral cues, or (b) completed one inhibitory control task with embedded appetitive and neutral cues that permitted separate indices of inhibitory control to be computed for each type of cue (e.g. (Jones & Field, 2015; Nederkoorn et al., 2009; Petit et al., 2012)). We also identified some studies that employed between-subject designs in which participants were randomized to exposure to either appetitive or neutral cues (e.g. (Jones, Rose, Cole, & Field, 2013b; Lattimore & Maid, 2015; Muraven & Shmueli, 2006). Field and Jones (2017) employed a mixed design in which inhibitory control was measured at baseline in all participants, before a between-subjects cue exposure manipulation (appetitive, neutral), followed by a second measure of inhibitory control. In this case we took the difference between the two groups after cue exposure.

Some studies also contrasted the effects of appetitive cues on inhibitory control in different groups of participants using mixed designs. For example, heavy vs. light drinkers (Nederkoorn et al., 2009), people with alcohol dependence vs. controls (Czapla et al., 2015a,b), obese/overweight vs. normal weight (Loeber et al., 2012). In these studies we computed within-subject comparisons based on these groups where possible to allow for individual comparisons to be included in different moderator analyses (see Table 1). Finally, some studies used multiple inhibitory control tasks or parameters (e.g., Adams, Ataya, Attwood, & Munafò, 2013) and in these cases we adjusted the sample sizes in the control conditions (N/Control/number of tasks) accordingly to ensure each comparison could be included in our pooled analyses, as recommended (Higgins & Green, 2011).

3. Results

The article selection process and flow is shown in Fig. 1. Following exclusion of irrelevant articles by title and abstract scanning we identified 37 full-text articles. See Table 1 for full details.

3.1. Pre-registered analyses

3.1.1. Primary hypothesis: the overall effect of appetitive cues on inhibitory control

Our main analysis consisted 66 effect sizes. We included a subgroup of appetitive cue-type: alcohol-related ($k = 35$) or food-related ($k = 31$). The overall effect of appetitive cues was small but statistically significant ($SMD = -0.12$ [95% CI $-0.23$, $-0.02$]; $Z = 2.34$, $p = .02$, $I^2 = 84\%$). Exposure to alcohol-related cues significantly impaired inhibitory control ($SMD = -0.21$ [95% CI $-0.32$, $-0.11$]; $Z = 4.17$, $p < .001$). There was no evidence that food-related cues led to observable deficits in inhibitory control ($SMD = -0.03$ [95% CI $-0.21$, 0.15]; $Z = 0.36$, $p = .720$). There was weak statistical evidence for subgroup differences ($X^2 (1) = 2.97$, $p = .090$). These results suggest that exposure to appetitive cues impairs inhibitory control compared to neutral cue exposure/no cue exposure, and overall the impairing effects of alcohol-related cues are more robust (see Supplementary Fig. 1).

(footnote continued)

Department of Health Guidelines for low risk drinking.
# Table 1
Details of studies included in the meta-analysis.

| Authors and Study | Participants and Design | Cue exposure and Control | Outcome | Notes | Included subgroup analyses |
|-------------------|-------------------------|--------------------------|---------|-------|---------------------------|
| Adams et al. (2013) | N = 96 Mean age: 21.0 Inclusion criteria: Social drinkers Exclusion criteria: None stated | Pictorial and Lexical cue exposure: Alcohol-related cues and words were embedded into the task Control: Soft-drink images and musical instrument words were embedded into the task | Commission errors to alcohol cues (pictorial) and words (lexical) | Participants completed the task under placebo and alcohol intoxication sessions. Data from the placebo session only was analysed. Participants were also split into heavy and light drinkers based on units consumed. | Drinking status: Heavy drinkers, Light drinkers Cue modality: Pictorial, Lexical Task: Go/No-Go Switching |
| (Czapla et al., 2015a,b) | N = 16 Mean age: 47.0 Inclusion criteria: Alcohol dependent patients or health matched controls Exclusion criteria: Current drug abuse or dependence (except alcohol/nicotine); severe somatic, neurological or psychiatric diseases; pregnancy | Pictorial cue exposure: Alcohol-related cues were embedded into the inhibition task Control: Neutral (geometric shapes) were embedded into the task. | Commission errors | We considered the control group to be light drinkers based on their drinking characteristics (number of drinking days and cumulative alcohol consumption) reported in the article. | Drinking status: Alcoholics, Light drinkers Cue modality: Pictorial Task: Go/No-Go |
| Field & Jones (2017) | N = 81 Mean age: 23.8 Inclusion criteria: Heavy drinking Exclusion criteria: No history of substance use disorder or ADHD | Pictorial and olfactory cue exposure: Participants sniffed beer Control: Participants sniffed water | Stop Signal Reaction time | | |
| Gauggel et al. (2010) | N = 20 Mean age: 44.9 Inclusion criteria: Detoxified alcohol dependent patients Exclusion criteria: None stated | Olfactory cue exposure: Participants sniffed beer prior to the inhibition task Control: Participants sniffed water prior to the inhibition task | Stop Signal Reaction Time | Drinking Status: Alcoholics Cue exposure type: Olfactory Task: Stop Signal | |
| Jones et al. (2013a,b) | N = 60 Mean age: 21.1 years Inclusion criteria: Heavy drinking Exclusion criteria: No history of alcohol-related problems | Olfactory cue exposure: Participants sniffed water prior to the inhibition task Control: Participants sniffed water prior to the inhibition task | Stop Signal Reaction Time | Drinking Status: Heavy drinkers Cue modality: Olfactory Task: Stop Signal | |
| Jones and Field (2015) study 1 | N = 64 Mean age: 22.34 years Inclusion criteria: Social drinking Exclusion criteria: No history of alcohol-related problems or ADHD | Pictorial cue exposure: Alcohol-related cues were embedded into the inhibition task Control: Neutral (scenery) cues were embedded into the task. | Stop Signal Reaction Time | Alcohol vs Neutral cues comparison was used. Positively and negatively valenced cues were also included in the Stop Signal task. | Drinking status: Heavy drinkers, Light drinkers Cue exposure type: Pictorial Task: Stop Signal |
| Jones and Field (2015) study 2 | N = 117 Mean age: 24.8 Inclusion criteria: Social drinking Exclusion criteria: No history of alcohol-related problems or ADHD | Pictorial cue exposure: Alcohol-related cues were embedded into the task Control: Neutral (scenery) cues were embedded into the task. | Proportion of correct responses | Alcohol vs Neutral cues comparison was used. Positively and negatively valenced cues were also included in the Stop Signal task. | Drinking status: Heavy drinkers, Light drinkers Cue modality: Pictorial Task: Stop Signal |
| Kreusch, Vienne, and Quertemont (2013) study 1 | N = 71 Mean age: 21.1 Inclusion criteria: Social drinker Exclusion criteria: Drug consumption; psychiatric symptoms | Pictorial cue exposure: Alcohol related cues were embedded into the task Control: Neutral (object images) were embedded into the task. | Proportion of commission errors | Sample was split by problem and non-problematic drinkers based on AUDIT scores. | Drinking status: Heavy drinkers and Light drinkers Cue modality: Pictorial Task: Go/No-Go switching |

(continued on next page)
| Authors and Study | Participants and Design | Cue exposure and Control | Outcome | Notes |
|-------------------|------------------------|-------------------------|---------|-------|
| Kreusch et al. (2013) | study 2 | Alcohol-cue exposure articles | groups created based on awareness of the study aims. | Included subgroup analyses |
| Kreusch, Billieux, and Quertemont (2017) | | Pictorial cue exposure | Inclusion criteria: Social drinking; Exclusion criteria: Drug consumption; psychiatric symptoms | |
| Kreusch, Billieux, and Quertemont (2017) | | Lexical cue exposure | Inclusion criteria: No history of other substance dependence or schizoaffective disorders | |
| Monk et al. (2016) | | Olfactory and pictorial cue exposure | Inclusion criteria: None stated | |
| Muraven & Schmeuli (2006) | | Olfactory cue | Participants sniffed beer prior to the inhibition task | |
| Nederkoorn et al. (2009) | | Pictorial cue exposure | Inclusion criteria: Social drinking; Exclusion criteria: None stated | |
| Nikolaou, Field, and Duka (2013) | | Olfactory cue | Inclusion criteria: None stated | |
| Noel et al. (2007) | | Lexical cue exposure | Inclusion criteria: None stated | |

### Table 1 (continued)

| Authors and Study | Participants and Design | Cue exposure and Control | Outcome | Notes |
|-------------------|------------------------|-------------------------|---------|-------|
| A. Jones et al. Appetite 128 (2018) 271–282 | | | | |
### Alcohol-cue exposure articles

| Authors and Study | Participants and Design | Cue exposure and Control | Outcome | Notes | Included subgroup analyses |
|-------------------|-------------------------|--------------------------|---------|-------|----------------------------|
| Noel et al. (2009) | N = 40<br>Mean age: 44.1<br>Inclusion criteria: Alcoholics had to meet DSM criteria; controls aged matched<br>Exclusion criteria: Current DSM-IV Axis I diagnoses, a history of significant medical illness, head injury; use of other psychotropic drugs or substances that influence cognition; overt cognitive dysfunction. | Lexical cue exposure<br>Appetitive: Alcohol-related words were embedded into the task<br>Control: Neutral-related words were embedded into the task | Decision bias (C) | No information was given for control group alcohol consumption. Therefore we were unable to include this group in our analyses. | Drinking status: Alcoholics, no information on controls<br>Cue modality: Lexical<br>Task: Go/No-Go Switching |
| Petit et al. (2012) | N = 35<br>Mean age: 21.3<br>Inclusion criteria: Regular alcohol consumption<br>Exclusion criteria: Alcohol abstainers; major medical problems; history of alcohol-related problems. | Pictorial cue exposure<br>Appetitive: Alcohol-related cues were embedded into the task<br>Control: Neutral (non-alcohol related) cues were embedded into the task | Commission errors | Alcohol vs Non-alcohol context comparison was used. Black screen context was also included into the Go/No-Go task. Participants were split into heavy and light drinkers based on AUDIT scores. | Drinking status: Heavy<br>Cue modality: Pictorial<br>Task: Go/No-Go task |
| Petit et al. (2014) | N = 54<br>Mean age: 45.0<br>Inclusion criteria: Patients with alcoholism undergoing treatment or healthy age/sex matched controls<br>Exclusion criteria: Diagnosis of axis I disorders (DSM-IV) or significant CNS or visual impairment | Pictorial cue exposure<br>Appetitive: Alcohol-related cues were embedded into the task<br>Control: Neutral (non-alcohol related) cues were embedded into the task | Commission errors | Alcohol vs Non-alcohol context comparison was used. Black screen context was also included into the Go/No-Go task. Control group drank < 14 standard (7 for woman) drinks per week, to ensure low risk for alcohol-related problems. As such we classed the control group as light drinkers for our subgroup analyses. | Drinking status: Alcoholics, Light drinkers<br>Cue modality: Pictorial<br>Task: Go/No-Go task |
| Sion et al. (2017) | N = 85<br>Mean age: Not reported<br>Inclusion criteria: Alcohol dependent patients attending detoxification and recovery<br>Exclusion criteria: Psychiatric morbidities | Lexical cue exposure<br>Appetitive: Alcohol-related words were embedded into the task<br>Control: Neutral (non-alcohol related) words were embedded into the task | Stop Signal Reaction time | Alcohol vs Non-alcohol word comparisons were used. Non-word comparisons were also used. Control group were not included as no information about drinking status was available. | Drinking status: Alcoholics<br>Cue modality: Lexical<br>Task: Stop Signal |
| Weafer and Fillmore (2012) | N = 50<br>Mean age: 23.9<br>Inclusion criteria: Adult beer drinkers<br>Exclusion criteria: Head trauma, psychiatric disorder, or substance abuse disorder | Pictorial cue exposure<br>Appetitive: Alcohol-related cues (Beer only) were embedded into the task<br>Control: Neutral (non-alcohol related) cues were embedded into the task | Proportion of Inhibition errors |  | Drinking status: Heavy<br>Cue modality: Pictorial<br>Task: Go/No-Go task |
| Weafer and Fillmore (2014) | N = 40<br>Mean age: 23.3<br>Inclusion criteria: Adult beer drinkers<br>Exclusion criteria: Head trauma, psychiatric disorder, or substance abuse disorder | Pictorial cue exposure<br>Appetitive: Alcohol-related cues (Beer only) were embedded into the task<br>Control: Neutral (non-alcohol related) cues were embedded into the task | Proportion of Inhibition errors |  | Drinking status: Heavy<br>Cue modality: Pictorial<br>Task: Go/No-Go task |

### Food-cue exposure articles

| Authors and Study | Participants and Design | Cue exposure and Control | Outcome | Notes | Included subgroup analyses |
|-------------------|-------------------------|--------------------------|---------|-------|----------------------------|
| He et al. (2014)  | N = 30<br>Mean age: 19.7<br>Inclusion criteria: None stated<br>Exclusion criteria: Individuals with neuropsychiatric disorders, medication or health issues which interfered with neuroimaging data Between Subjects | Pictorial cue exposure<br>Appetitive: High calorie food-related cues were embedded into the task<br>Control: Low calorie food-related cues were embedded into the task | Commission errors |  | Weight Status: Normal weight<br>Cue modality: Pictorial<br>Task: Go/No-Go Switching |
| Authors and Study | Alcohol cue exposure articles | Participants and Design | Inclusion criteria | Exclusion criteria | Outcome | Notes |
|------------------|-------------------------------|-------------------------|-------------------|-------------------|---------|-------|
| Hobson et al. (2013) | N = 87 | Participants were split into normal weight, overweight and obese based on their BMI | Mean age: 36.3 | Male: 22/22; Female: 35/35 | Weight Status: Normal weight, Overweight/Obese | Included subgroup analyses |
| Kroff et al. (2015) | N = 51 | Participants were split into restrained or unrestrained eaters based on the Restraint Scale | Mean age: 26.2 | | Weight Status: Normal weight, Overweight/Obese | |
| Houben et al. (2013) | N = 81 | Participants were split into normal weight, overweight and obese based on their BMI | Mean age: 25.9 | | Weight Status: Normal weight, Overweight/Obese | |
| Hume, Howells, Rauch, Kraft, and Lambert (2013) | N = 91 | Participants were split into normal weight, overweight and obese based on their BMI | Mean age: 48.9 | | Weight Status: Normal weight, Overweight/Obese | |
| Lattimore and Mead (2015) | N = 51 | Participants were split into restrained or unrestrained eaters based on the Restraint Scale | Mean age: 25.0 | | Weight Status: Normal weight, Overweight/Obese | |
| Luebber et al. (2012) | N = 40 | Participants were split into high and low food addiction groups based on the Yale Food Addiction Scale | Mean age: 46.4 | | Weight Status: Normal weight, Overweight/Obese | |
| Houben et al. (2013) | N = 87 | Participants were split into normal weight, overweight and obese based on their BMI | Mean age: 36.3 | | Weight Status: Normal weight, Overweight/Obese | |
| Hume, Howells, Rauch, Kraft, and Lambert (2013) | N = 91 | Participants were split into normal weight, overweight and obese based on their BMI | Mean age: 48.9 | | Weight Status: Normal weight, Overweight/Obese | |
| Lattimore and Mead (2015) | N = 51 | Participants were split into restrained or unrestrained eaters based on the Restraint Scale | Mean age: 25.0 | | Weight Status: Normal weight, Overweight/Obese | |
| Luebber et al. (2012) | N = 40 | Participants were split into high and low food addiction groups based on the Yale Food Addiction Scale | Mean age: 46.4 | | Weight Status: Normal weight, Overweight/Obese | |
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| Lattimore and Mead (2015) | N = 51 | Participants were split into restrained or unrestrained eaters based on the Restraint Scale | Mean age: 25.0 | | Weight Status: Normal weight, Overweight/Obese | |
| Luebber et al. (2012) | N = 40 | Participants were split into high and low food addiction groups based on the Yale Food Addiction Scale | Mean age: 46.4 | | Weight Status: Normal weight, Overweight/Obese | |
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| Hume, Howells, Rauch, Kraft, and Lambert (2013) | N = 91 | Participants were split into normal weight, overweight and obese based on their BMI | Mean age: 48.9 | | Weight Status: Normal weight, Overweight/Obese | |
| Lattimore and Mead (2015) | N = 51 | Participants were split into restrained or unrestrained eaters based on the Restraint Scale | Mean age: 25.0 | | Weight Status: Normal weight, Overweight/Obese | |
| Luebber et al. (2012) | N = 40 | Participants were split into high and low food addiction groups based on the Yale Food Addiction Scale | Mean age: 46.4 | | Weight Status: Normal weight, Overweight/Obese | |
| Authors and Study | Participants and Design | Cue exposure and Control | Outcome | Notes | Included subgroup analyses |
|-------------------|-------------------------|--------------------------|---------|-------|---------------------------|
| Meule, Lutz, Vögele, and Kübler (2014a) study 1 | N = 50 Mean age: 22.3 Inclusion criteria: Female Exclusion criteria: Mental disorders, psychoactive medication, under- or overweight; aged > 40 | Pictorial cue exposure  Appetitive: High calorie food-related cues were embedded into the task Control: Neutral (office equipment) cues were embedded into the task | Commission errors | | Weight Status: Normal weight  Cue modality: Pictorial |
| Meule et al. (2014a) study 2 | N = 102 Mean age: 22.8 Inclusion criteria: Female Exclusion criteria: Mental disorders, psychoactive medication, aged > 40 | Pictorial cue exposure  Appetitive: High calorie food-related cues were embedded into the task Control: Neutral (office equipment) cues were embedded into the task | Commission errors | Participants were split into hungry or satiated experimental groups. The satiated group were given a yogurt to consume prior to the task | Weight Status: Normal weight  Cue modality: Pictorial  Task: Go/No-Go Switching |
| Meule and Kübler (2014) | N = 55 Mean age: 24.4 Inclusion criteria: Female Exclusion criteria: None stated | Pictorial cue exposure  Appetitive: High calorie food-related cues were embedded into the task Control: Low calorie food-related were embedded into the task | Commission errors | | Weight Status: Normal weight  Cue modality: Pictorial  Task: Go/No-Go Switching |
| Meule et al. (2014b) | N = 50 Mean age: 22.3 Inclusion criteria: Female Exclusion criteria: Mental disorders, psychoactive medication, aged > 40 | Pictorial cue exposure  Appetitive: High calorie food-related cues were embedded into the task Control: Neutral (household items) cues were embedded into the task | Commission errors | | Weight Status: Normal weight  Cue modality: Pictorial  Task: Stop Signal |
| Phelan et al. (2011) | N = 68 Mean age: 46.8 Inclusion criteria: None stated Exclusion criteria: Binge eating, food allergies, and vegetarianism | Lexical cue exposure  Appetitive: High calorie food-related words were embedded into the task Control: Neutral words were embedded into the task | Number of valid words | Participants were split into experimental groups based on current weight and dieting status (Normal Weight, Obese, Weight loss maintainer). | Weight Status: Normal weight, Overweight/Obese  Cue modality: Lexical  Task: Stroop |
| Schag et al. (2013) | N = 76 Mean age: 39.67 Inclusion criteria: Females Exclusion criteria: Impaired vision; somatic diseases; medications; pregnancy or lactation; psychosis or bipolar disorder | Pictorial cue exposure  Appetitive: Food-related cues were embedded into the task Control: Neutral (non-food) related cues were presented prior to the task | Anti-saccade errors | Participants were split into experimental groups based on current weight and the presence of binge eating disorder (Binge eating disorder +, binge eating disorder -, Normal weight controls) | Weight Status: Normal weight, Overweight/Obese  Cue modality: Pictorial  Task: Anti-saccade |
| Yeomans and Brace (2015) | N = 36 Mean age: 21.4 Inclusion criteria: None stated Exclusion criteria: None stated | Pictorial cue exposure  Appetitive: Food-related cues were embedded into the task Control: Neutral related cues were presented prior to the task | Commission errors | Participants were randomly allocated to food cue-exposure or neutral exposure. | Weight Status: Normal weight  Cue modality: Pictorial  Task: Go/No-Go task |
3.1.2. Moderation by drinking status

We identified \( k = 9 \) effect sizes for light drinkers, \( k = 15 \) effect sizes for heavy drinkers and \( k = 8 \) effect sizes for alcohol dependent patients\(^2\). There was no significant effect of alcohol-related cues on inhibitory control in light drinkers (SMD = \(-0.15\) [95% CI = \(-0.33, 0.03\)]; \( Z = 1.59, p = .110; I^2 = 53\% \)). There was a significant effect of alcohol-related cues on inhibitory control in heavy drinkers (SMD = \(-0.26\) [95% CI = \(-0.46, -0.06\)]; \( Z = 2.59, p < .010; I^2 = 80\% \)) and alcohol-dependent patients (SMD = \(-0.22\) [95% CI = \(-0.41, -0.04\)]; \( Z = 2.35, p = .020; I^2 = 58\% \)). However, there was no significant subgroup effect (\( X^2 (2) = 0.79, p = .67 \)) suggesting that drinking status did not reliably moderate the effects of alcohol-related cues on inhibitory control.

3.1.3. Moderation by weight status

We identified \( k = 10 \) effect sizes for overweight/obese participants and \( k = 21 \) effect sizes for normal weight or underweight participants. There was no significant effect of food-related cues on inhibitory control in overweight/obese individuals (SMD = \(-0.31\) [95% CI = \(-0.73, 0.12\)]; \( Z = 1.42, p = .16; I^2 = 91\% \)) or normal weight individuals (SMD = 0.09 [95% CI = \(-0.10, 0.28\)]; \( Z = 0.95, p = .34; I^2 = 86\% \)). There was weak evidence for a subgroup effect (\( X^2 (2) = 2.83, p = .09 \)).

3.1.4. Moderation by modality of cue-exposure

Across both food and alcohol cues we identified \( K = 42 \) effect sizes from studies that employed pictorial cues, \( k = 16 \) effect sizes from studies that employed lexical (word) cues and \( k = 10 \) effect sizes from studies that employed olfactory/in vivo cues. Note that two studies\(^3\) used combined cue-exposure paradigms. Therefore they contributed to more than one group in these analyses, but removal of effect sizes from these studies did not significantly alter the results. Exposure to olfactory/in vivo cues led to significant impairments in inhibitory control (SMD = \(-0.24\) [95% CI = \(-0.41, -0.07\)]; \( Z = 2.83, p < .001, I^2 = 65\% \)). Whereas, pictorial (SMD = \(-0.07\) [95% CI = \(-0.19, 0.05\)]; \( Z = 1.07, p = .280, I^2 = 83\% \)) and lexical (SMD = \(-0.26\) [95% CI = \(-0.57, 0.06\)]; \( Z = 1.62, p = .110, I^2 = 90\% \)) cues did not significantly impair inhibitory control. However, the test for subgroup differences was not statistically significant (\( X^2 (2) = 3.47, p = .180 \)).

Due to the differences between alcohol-related and food-related cues we analysed the effects of cue-exposure modality separately. For alcohol, we identified \( k = 23 \) effect sizes from studies that employed pictorial cues, \( k = 6 \) effect sizes were from studies that employed lexical cues and \( k = 8 \) effect sizes were from studies that employed olfactory/in vivo cues. Pictorial alcohol-related cues (SMD = \(-0.22\) [95% CI = \(-0.35, -0.08\)]; \( Z = 3.11, p = .002; I^2 = 75\% \)) and olfactory alcohol-related cues (SMD = \(-0.27\) [95% CI = \(-0.46, -0.08\)]; \( Z = 2.76, p < .001; I^2 = 69\% \)) significantly impaired inhibitory control, however lexical cues did not have a significant effect (SMD = \(-0.13\) [95% CI = \(-0.44, 0.18\)]; \( Z = 1.18, p = .237, I^2 = 81\% \)).

\(^2\) We were unable to categorise individuals in Kreusch et al. (2013, study 2), or Muraven and Schmeuli (2006) as there was no information provided on participants’ alcohol consumption.

\(^3\) Field and Jones (2017) combined pictorial and olfactory cues, whereas Kreusch et al. (2017) combined lexical and olfactory cues.
CL = 0.27, 0.02); Z = 1.74, p = .080; I² = 0%). The test for subgroup differences was not significant (χ² (2) = 1.51, p = .470).

For food, we identified k = 19 effect sizes from studies that employed pictorial cues, k = 10 effect sizes from studies that employed from lexical cues and k = 2 effect sizes from studies that employed olfactory cues/in vivo cues. Only lexical cues impaired inhibitory control (SMD = −0.30 [95% CI = −0.83, −0.24]; Z = 1.09, p = .28, I² = 94%); pictorial (SMD = 0.10 [95% CI = −0.06, 0.27]; Z = 1.20, p = .23, I² = 82%) and olfactory cues (SMD = −0.12 [95% CI = −0.53, 0.295]; Z = 0.58, p = .56, I² = 54%) did not. However, the test for subgroup differences was not statistically significant (χ² (2) = 2.69, p = .260).

3.2. Exploratory analyses

3.2.1. Effect of inhibitory control task

We conducted exploratory analyses on the type of task used to operationalize inhibitory control (see Table 1). Following appetite cue exposure, inhibitory control was impaired on the Stop Signal (k = 18; SMD = −0.15 [95% CI = −0.25, −0.05]; Z = 3.00, p < .001, I² = 47%), Stroop (k = 6; SMD = −0.66 [95% CI = −1.28, −0.03]; Z = 2.04, p = .040, I² = 92%), and Anti-saccade tasks (k = 5; SMD = −0.19 [95% CI = −0.33, −0.04]; Z = 2.57, p = .010, I² = 0%), but there was no reliable effect on Go/No-Go tasks (k = 16; SMD = −0.15 [95% CI = −0.35, 0.05]; Z = 1.46, p = .14, I² = 84%) or Go/No-Go shifting tasks (k = 20; SMD = 0.10 [95% CI = −0.15, 0.35]; Z = 0.80, p = .430, I² = 89%). However, the subgroup effect was not statistically significant (χ² (4) = 6.64, p = .160).

Due to methodological considerations regarding the Go/No-Go shifting task (see Meule, 2017 and discussion) we repeated our primary analysis after excluding studies that used this task. In this case both alcohol-related cues (k = 26; SMD = −0.23 [95% CI = −0.35, −0.11]; Z = 3.85, p < .001, I² = 73%) and food-related cues (k = 20; SMD = −0.19 [95% CI = −0.37, −0.01]; Z = 2.03, p = .04; I² = 82%) impaired inhibitory control. We note that this was not an a-priori analysis. We present all analyses with exclusion of effect sizes from the Go/No-Go shifting task in online supplementary materials.

3.2.2. Examination of bias

Visual inspection of the funnel plot (see Supplementary Fig. 2) for all studies suggested asymmetry, and Trim and Fill analyses suggested 17 effect sizes would need to be added to achieve symmetry (see Supplementary Fig. 3). Adding these effect sizes made the overall point estimate non-significant (SMD = 0.05 [95% CI = −0.08, 0.18]). This suggests some degree of bias was evident across the effect sizes included. We also conducted Egger’s test to formally examine asymmetry by regressing the effect size against the precision, however the test was not statistically significant (Z = −0.56, p = .574).

4. Discussion

The results of this meta-analytic investigation demonstrate that exposure to alcohol-related cues prompts robust, albeit small impairments in inhibitory control, although the evidence for comparable effects of food-related cues was not reliable. We observed substantial heterogeneity across effect sizes, which remained high despite several subgroup analyses that attempted to identify moderating variables. There was limited evidence to suggest that drinking status moderated the effect of alcohol-related cues, or that weight status moderated the effect of food-related cues on inhibitory control. Similarly, the modality of cue exposure did not significantly moderate our findings. Statistical correction for bias made the main effect of appetitive cues on inhibitory control no longer statistically significant.

Our primary hypothesis – exposure to appetitive cues would prompt a deficit in inhibitory control – was not fully supported. Overall, appetitive cues impaired inhibitory control, however this was driven by a significant effect for alcohol-related cues. These findings provide partial support for theoretical models which suggest inhibitory control is a transient process that is sensitive to environmental and internal events (Jones, Christiansen, Nederkoorn, Houben, & Field, 2013a; Verbruggen et al., 2014), and the transient nature of inhibitory control cues may be one psychological mechanism that underlies the influence of alcohol cues on drinking behaviour (De Wit, 2009; Field & Jones, 2017). We found no evidence that transient impairments in inhibitory control were associated with individual differences in alcohol or food intake over the longer term, such as (self-reported) quantity of alcohol consumed per week, hazardous drinking scores or BMI. Future studies should attempt to clarify the associations between the disinhibiting effects of appetitive cues and food or alcohol intake that is measured immediately afterwards, such as ad-libitum intake (Field & Jones, 2017; Jones et al., 2015) or operant choice (Veling, Aarts, & Stoebbe, 2013).

Consideration of publication bias suggests that even these small effects of appetitive cues may be inflated, and when we accounted for ‘missing’ (small and non-significant; k = 17) effect sizes using Trim and Fill analyses (Duval & Tweedie, 2000), the overall effect size was no longer statistically significant. This suggests that this literature is characterised by ‘small study effects’, often of poor methodological quality, and reporting biases that can substantially influence pooled-estimates (Schwarzer, Carpenter, & Rücker, 2015). Future research should conduct well powered studies and aim to publish all data to mitigate these biases and improve our confidence in pooled estimates of effect.

As hypothesised, impairments in inhibitory control following exposure to alcohol-related cues were comparable heavy drinkers and people with alcohol dependence, but these effects were absent in light drinkers. However, the test for subgroup differences was not statistically significant. Therefore, these subgroup differences are merely suggestive, and they should be interpreted with caution because many subgroups were poorly defined and created post-hoc using median split techniques (Jones & Field, 2015; Nederkoorn et al., 2009). Future studies might use established criteria to define heavy drinking or alcohol dependence (Babor, Higgins-Biddle, Saunders, & Monteiro, 2001; Edwards, 1996), in order to determine if these subgroup differences are indeed robust.

The absence of a robust effect of food-related cues on inhibitory control was surprising but is difficult to interpret given methodological features of the original studies. Eleven (35%) of studies used the Go/No-Go switching task for food-related cues. In this task, contingencies between making a motor response and inhibiting to high caloric vs. control cues are regularly switched on a block-by-block basis (Loeb et al., 2012). The repeated shifting of task contingencies between blocks mean that this task is likely to capture the effect of cues on inhibition and shifting, two distinct subcomponents of executive functions, and therefore this task provides an impure measure of inhibitory control (Miyake et al., 2010). Furthermore, variations on this task have also used low-calorific food cues (rather than non-food items) which may still be appetitive (He et al., 2014). Notably, when we removed effect sizes generated from Go-No/Go shifting tasks from our analysis the effect of food cues on inhibitory control was robust.

The modality in which appetitive cues were presented (e.g. visual vs. olfactory) did not moderate the effect of appetitive cues on inhibitory control. The absence of a moderation effect here should be interpreted cautiously until further direct comparisons across modalities are attempted (cf. Boswell & Kober, 2016; Monk et al., 2016). Overall, our moderator analyses suggest that we were unable to reduce the substantial heterogeneity by identifying variables that might moderate the influence of appetitive cues on inhibitory control. It is possible that other variables may influence this relationship, but we did not identify enough studies to examine this. For example, heterogeneity may have been caused by considerable variability in food-related and control images (see Table 1) and individual differences in reactivity to these cues, or differing levels of motivation to restrict unhealthy
behaviours across the samples. Future studies should investigate these potential moderators in more detail.

Finally, the effect of alcohol-related cues on inhibitory control supports the recent development of Inhibitory Control Training (ICT) as a behavioural intervention, to mitigate against cue-specific inhibition deficits. ICT creates an associative link between appetitive cues and inhibition of behaviour, which is thought to extinguish the associative link between appetitive cues and approach behaviour (Stice, Lawrence, Kemps, & Veling, 2016; Verbruggen et al., 2014). Promising effects of ICT have been demonstrated for both ad-libitum food and alcohol consumption in the laboratory (Allom, Mullan, & Hagger, 2015; Jones et al., 2016).

Some limitations of our analyses should be taken into consideration. First, we only included studies that examined cue reactivity to alcohol or food-related cues. There is evidence in other domains for cue-specific impairments of inhibitory control, including smoking (Luijten et al., 2011) and illicit drug use (Pike et al., 2013; Verdejo-Garcia et al., 2012) which we did not integrate into our current analysis due to the limited number of available studies. Similarly, although we limited our findings to adult participants (18 + in United Kingdom, the legal drinking age) research also demonstrates inhibitory deficits following alcohol cues and food cues in younger people (Ames et al., 2014; Korucuoglu, Gladwin, & Wiers, 2015). Second, we were unable to directly measure associations between subjective and physiological cue-reactivity (e.g. craving and arousal) and inhibitory control as few studies measured this consistently. Finally, it is unknown whether these deficits in inhibitory control are a capacity deficit, or a motivational deficit (i.e. participants do not evoke effortful inhibition; Fujita, 2011). Future research should aim to overcome these limitations but also identify the mechanisms through which appetitive cues impair inhibitory control, for example through competition with attentional processes (Pessoa, Padmala, Kenzer, & Bauer, 2012), reductions in limited self-regulatory resources (Muraven & Shmueli, 2006) or a reduction in their motivations (Pessoa, 2009). Furthermore, the influence of appetitive cues on inhibitory control in real-world settings (outside of the laboratory) should be investigated in order to elucidate the significance of these deficits for health-related behaviour. An interesting way to do this would be to examine real-time cue-exposure and inhibitory control using Ecological Momentary Assessment techniques (Shiffman, 2009). Given that cue-reactivity demonstrates substantial within- and between-subject variability (Serre, Fatou, Swendsen, & Auriacombe, 2015) the real-world effects of repeated (LaR owe, Saladin, Carpenter, & Upadhyaya, 2007), cumulative and personalised cues (MacKillop et al., 2010) may identify more robust effects of exposure to appetitive cues on inhibitory control.

To conclude, the results from this meta-analytic investigation demonstrate that inhibitory control is sensitive to the presence of alcohol-related cues. The effect of food-related cues was less robust and may be confounded by methodological features of the tasks used. Overall, these findings provide some tentative support for theoretical predictions that inhibitory control is sensitive to exposure to appetitive cues. However whether these effects are robust, and if they play an important role in health-related behaviour, are important questions for future research.

Appendix A. Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.appet.2018.06.024.

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