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A systematic review of the relationship between eating, weight and inhibitory control using the stop signal task.

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HIGHLIGHTS

- Being able/unable to inhibit behaviour has implications for eating and weight
- Inhibitory control is multifaceted: its individual components should be considered
- Restrained eaters were most consistently reported to have poor inhibitory control
- There are few studies on eating disorders, and samples were small and heterogeneous
- Obese people may show poor inhibitory control in specific contexts (e.g., to food)

ABSTRACT:

Altered inhibitory control (response inhibition, reward-based inhibition, cognitive inhibition, reversal learning) has been implicated in eating disorders (EDs) and obesity. It is unclear, however, how different types of inhibitory control contribute to eating and weight-control behaviours. This review evaluates the relationship between one aspect of inhibitory control (a reactive component of motor response inhibition measured by the stop signal task) and eating/weight in clinical and non-clinical populations. Sixty-two studies from 58 journal articles were included. Restrained eaters had diminished reactive inhibitory control compared to unrestrained eaters, and showed greatest benefit to their eating behaviour from manipulations of inhibitory control. Obese individuals may show less reactive inhibitory control but only in the context of food-specific inhibition or after executive resources are depleted. Of the limited studies in EDs, the majority found no impairment in reactive inhibitory control, although findings are inconsistent. Thus, altered reactive inhibitory control is related to some maladaptive eating behaviours, and hence may provide a therapeutic target for behavioural manipulations and/or neuromodulation. However, other types of inhibitory control may also contribute. Methodological and theoretical considerations are discussed.

Keywords:

Eating disorders; obesity; eating; weight; inhibitory control; stop signal task

1. INTRODUCTION:

1.1. Inhibitory control, EDs and obesity

We live in an obesogenic environment. Highly palatable and often calorific foods are readily available/ easily obtained. To maintain a healthy lifestyle, a degree of self-control is needed to overcome temptation towards these easy and unhealthy options. Overcoming temptation or urges requires the ability to withhold inappropriate or unwanted behaviour, a broad concept referred to as inhibitory control. Inefficient inhibitory control may therefore play a role in the development and/or maintenance of obesity. Aberrant inhibitory control has also been implicated in the pathology of eating disorders (EDs): anorexia nervosa (AN), bulimia nervosa (BN) and binge eating disorder (BED) (Brooks et al., 2012; Dawe and Loxton, 2004). For example, poor inhibitory control may contribute to the inability to control urges to binge eat or to purge. In contrast, restrictive-type AN has been proposed to be underscored by excessive inhibitory control (Brooks et al., 2012). Alternatively, it may be that exercising behavioural inhibition is a means of re-establishing the feeling of control during a more general experience of loss of control. In the general context of inhibitory control, it is also of note that EDs (particularly BN and BED) and obesity are often highly comorbid with impulse-control disorders characterised by poor inhibitory control, including ADHD, substance abuse and pathological gambling (e.g., Biederman et al., 2007; Fernández-Aranda et al., 2008; Hudson et al., 2007; Nazar et al., 2014; Nazar et al., 2008), suggesting that deficits in inhibitory control and/or its underlying neural correlates are a vulnerability factor for a range of disordered behaviours. It is unclear, however, whether these deficits are general or specific to particular contexts or behaviours, whether inhibitory control is similarly affected across different conditions, and which types of inhibitory control are related to different clinical and nonclinical eating behaviours.

A number of narrative and systematic reviews have recently been conducted, most of which concluded that there is some evidence that obesity, overweight and binge-related EDs are associated with poor inhibitory control (Fischer et al., 2008; Guerrieri et al., 2008a; Liang et al., 2014; Reinert et al., 2013; Schag et al., 2013; Thamotharan et al., 2013; Vainik et al., 2013; Waxman, 2009; Wierenga et al., 2014b; Wonderlich et al., 2004; Wu et al., 2013b). Moreover, a systematic review and meta-analysis of inhibitory control in the context of general and disorder-specific stimuli in bulimic-type EDs (anorexia nervosa binge-purge subtype (AN-BP), BN, BED) reported a general inhibitory control deficit with a small effect size in individuals with bulimic-type EDs compared to healthy controls (HCs), with an exaggerated deficit in the context of disorder-relevant stimuli in BN, although disorder-relevant stimuli were only assessed in BN participants (Wu et al., 2013b). These findings were consistent across different tasks assessing response inhibition and tasks assessing control over cognitive interference. In contrast, one review of neurocognition in bulimic-type EDs (BN, eating disorders not otherwise specified (EDNOS-BN type) and BED) found no clear indication of impaired inhibitory control across disorders, and attributed this in part to the heterogeneity of the methods and outcome measures used across studies (Van den Eynde et al., 2011b). However, these reviews have assessed findings from several neuropsychological tasks and questionnaires that may measure distinct components of inhibitory control, and refer to poor performance on these tasks (poor inhibitory control) as indicative of "impulsivity" or impulsive action. Inhibitory control (particularly with respect to motor response inhibition) can be assessed with relative ease using simple laboratory tasks, and poor performance on these tasks has been proposed to serve as an endophenotype for impulsivity (Bari and Robbins, 2013).

However, impulsivity and inhibitory control are terms that should not be used synonymously. Impulsivity refers to the tendency to act prematurely, without sufficient evidence, foresight, or consideration of the consequences, or in a way that is poorly conceived, risky or inappropriate (Dalley et al., 2011). In addition to impaired inhibitory control, it requires the co-occurrence of a

strong desire, urge or habit to initiate response. In contrast, inhibitory control refers to the general ability to withhold or inhibit inappropriate behaviour. Furthermore, impaired motor response inhibition is linked to impulsivity as well as compulsivity. Thus, it is important to recognise that impulsivity and inhibitory control are distinct concepts with some overlap.

1.2. Elements of inhibitory control

Inhibitory control encapsulates several components underscored by overlapping but distinct neurobiological mechanisms (for rev., see van Velzen et al., 2014). It includes, but is not limited to, (a) response inhibition (i.e., the ability to withhold an inappropriate motor response, assessed by tasks such as the stop signal task [SST] or the go/no-go task [GNG]), (b) reward-based or motivational inhibition (e.g., the ability to delay reward or gratification, assessed by tasks such as temporal discounting tasks), (c) reversal learning (e.g., tasks assessing cognitive flexibility such as set-shifting tasks), and (d) cognitive inhibition (e.g., overcoming interference from distracting information, such as the Stroop task) (Bari and Robbins, 2013).

Response inhibition can be further subcategorised into proactive and reactive inhibition. Proactive inhibition includes strategic adjustment of response speed (e.g. post-error slowing, or target-frequency effects) and also a general suppression of response tendencies in the context of uncertainty (e.g., Aron, 2011; Bissett et al., 2009; Boulinguez et al., 2009; Criaud et al., 2012; Jaffard et al., 2008). Reactive inhibition is when a response is withheld in reaction to a signal indicating response inhibition is required, i.e., a "stop signal". Reactive inhibition can take the form of action restraint (as in the GNG task), or action cancellation (as in the SST, in which the stop signal appears shortly after a target).

Several brain areas have been implicated across these tasks of response inhibition, i.e., they constitute a 'core stopping network': these include the inferior frontal gyrus (rIFG), middle frontal

gyrus (MFG), pre-supplementary motor area (pre-SMA), anterior insula (aIns) and subthalamic nucleus (STN). However, additional regions are differentially activated by these tasks, supporting the notion of separate components (Aron, 2007; Frank, 2006; Hampshire and Sharp, 2015; Robbins, 2007; Sebastian et al., 2013; Swick et al., 2011; Verbruggen and Logan, 2008). This is further supported by weak correlations between self-report and behavioural (task-based) measures of inhibitory control, as well as between inhibitory control tasks (e.g., Cyders and Coskunpinar, 2011; Enticott et al., 2006; Wingrove and Bond, 1997). Such tasks may therefore provide insight into the specific neural and behavioural manifestations of failed inhibitory control, and contribute to the understanding of the mechanisms underlying the development and/or maintenance of psychiatric disorders and symptom experience.

At present, it is unclear which types of inhibitory control may be aberrant in disordered eating or in obesity, i.e., whether only particular types of inhibitory control are affected or if there is a more general/overarching problem, and whether it is consistent across different symptoms or conditions. There is a lack of systematic exploration of evidence investigating specific inhibitory control capacities assessed by a single neuropsychological paradigm, and the relation between task performance and eating or weight status. Such assessments will provide an indication as to whether specific components of inhibitory control are related to, and are consistent across, different eating behaviours or weight categories, and may help to identify components of inhibitory control that are potential targets for treatment (e.g., modifying eating behaviours), or identifying individuals more likely to respond to treatment. As a result of reports of a relationship between unhealthy eating behaviours and impulsivity, neuropsychological interventions have been developed that aim to reduce such behaviours by altering neurocognitive processes involved in executive decision making. In these, established behavioural measures of inhibitory control are modified to allow the *training* of inhibitory control to evaluate the ability of these paradigms to facilitate weight loss and reduce unhealthy food intake in obese and overweight individuals (for rev., see Juarascio et al., 2015; Spierer et al., 2013). It is unclear, however, which types of inhibitory control should be

targeted to elicit the greatest benefit in terms of eating behaviour or weight control. Thus, systematic examination of individual components of inhibitory control in relation to eating and weight-related behaviours will help to identify (a) which components of inhibitory control (i.e., reactive motor inhibition, cognitive inhibition, etc.) contribute to various behaviours (e.g., binge eating, dietary restraint); and consequently (b) which behaviours might benefit from behavioural interventions aimed at training specific types of inhibitory control.

1.3. Present review

This review examines studies exploring the relationship between eating/weight and one measure of inhibitory control, namely the ability to withhold an already initiated response, measured by the stop signal task (SST; Logan, 1994; Logan and Cowan, 1984). This review does not answer the broader questions of whether *only* reactive inhibition is affected, if there is a more general/overarching problem, or whether other components of inhibitory control are involved. Rather, it aims to address whether reactive inhibitory control, specifically, is involved in a range of clinical and non-clinical eating and weight behaviours. This will help to inform whether difficulties in reactive inhibition are consistent across different ages, behaviours and conditions, and identify which behaviours may benefit from training or manipulation of reactive inhibitory control.

The SST was chosen as it is commonly used to assess motor control, with cognitive underpinnings that are clearly established and may have relevance to eating and weight-control behaviours (e.g., cognitive control exercised when resisting urges to eat). A single task was assessed to improve homogeneity in the specific inhibitory control-related construct under review. The review seeks to provide a better understanding of the specific involvement of reactive inhibitory control in a spectrum of healthy and disordered eating/weight-control behaviours. It is hoped that this is useful in identifying components of inhibitory control that are potential targets for treatment or identifying individuals likely to respond to treatment. It expands on previous reviews by including non-clinical groups exhibiting specific eating behaviours (e.g., dieting) and groups characterised by

weight status, and also by including studies that have looked at the impact of inhibitory control on eating/weight in addition to those that explore the influence of eating behaviour on inhibitory control. Specifically, it aims to:

(1) Characterise the relationship between reactive inhibitory control and both clinical and non-clinical eating behaviours;

(2) Explore whether reactive inhibitory control varies as a function of weight status (obese, overweight, normal weight, underweight) or body mass index (BMI);

(3) Summarise the evidence for weight loss interventions that train inhibitory control.

2. METHODS:

2.1. Search strategy

Four electronic databases were searched (PsycInfo, Medline and Embase (all via OvidSP) and PubMed), with no restrictions on publication date. Additionally, a manual search on Google Scholar and a review of the reference lists of related systematic reviews and meta-analyses were conducted to search for articles that may have been missed in the initial database search. The database and manual searches were completed by 30th September 2015. After initial screening of the title and abstract, the full text of relevant articles and articles for which the abstract did not provide sufficient information was retrieved and evaluated. Reference lists and articles citing (using Google Scholar) potentially relevant full-text articles were reviewed. Articles were retrieved from citation and reference lists until 14th October 2015.

The search was conducted independently by two authors (SB, BD). For every abstract identified as potentially relevant by at least one of the two authors, the full-text was evaluated by both

independently. Discrepancies were resolved through discussion between the two authors conducting the search. All papers that did not meet the inclusion criteria were excluded, with reasons provided in Figure 1. The search was based on the following keyword terms:

A. Search and explode the following terms to look for studies containing information regarding eating and/or weight: [eating disorder* OR eating* (map to subject headings: eating attitudes, eating behaviour, purging (eating disorders), rumination (eating) feeding and eating disorders of childhood, eating habit) OR anorexi* OR bulimi* OR binge (map to subject headings: binge eating OR binge eating disorder OR binge-eating disorder) OR obesity (map to subject headings: obesity morbid) OR overweight OR underweight (map to subject headings: thinness) OR body weight (map to subject headings: weight control, weight gain, weight loss, weight reduction, body weight disorder, weight) OR eating behaviour OR diet (map to subject heading: diets, diet reducing, diet therapy, diet restriction) OR fasting (map to subject heading: food deprivation, fasting) OR energy intake OR calori* intake OR food intake OR loss of control eat* OR LOC eat*]
B. Search and explode the following terms to look for studies using the stop signal task: [(stop signal* OR stop signal task OR stop task OR stop go task OR SSRT)]
C. Limit the search to human samples and written in English language.

These three search terms were then combined using the AND function (A AND B AND C), to limit the search to studies meeting all three criteria.

2.2. Data extraction

The data was extracted from all included studies into an electronic summary table (SB) and was checked by another author (BLD). Information collected related to sample size and characteristics, study design, SST outcomes assessed, eating/weight-related outcomes assessed, and relevant findings. A narrative synthesis of the data was performed. The large methodological diversity of the studies and the heterogeneity of samples in the included studies precluded meta-analyses.

2.3. Eligibility criteria

Studies reported in English that assessed the relationship between at least one measure of eating or weight status and performance on any variant of the SST in humans were included in the analysis. Samples included males and females of any age with a DSM or ICD diagnosis of an ED (including AN, BN, BED, purging disorder, EDs not otherwise specified (EDNOS), feeding disorder, loss of control (LOC) eating), individuals with obesity, individuals characterised by weight status (i.e., underweight, normal weight, overweight, obese), individuals on a weight loss programme, individuals characterised by eating behaviour (e.g., emotional eating, eating restraint, diet), and studies that categorised participants according to impulsivity but included an analysis of the relationship between SST performance and eating/weight status.

Studies were excluded if the sample did not meet the above criteria, was comprised of animals, was comprised of human participants with a psychiatric disorder without an assessment of eating/weight status, participants with a neurological disorder or illness due to brain damage, participants with anorexia/cachexia due to other medical illness (e.g., cancer), participants characterised by drinking behaviour without concurrent comparison of eating behaviour (e.g., alcohol intake: social drinkers, binge drinkers, alcoholics), participants with a genetic disorder (e.g., phenylketonuria, PKU), studies assessing relatives of individuals with an ED or obesity with no reference to the relationship between inhibitory control and the participants' eating/weight, or studies only assessing exercise without additional reporting of eating behaviours or weight. Meta-analyses and systematic reviews were not included in the narrative synthesis, though studies referenced within the reviews were assessed for eligibility. Theses and conference abstracts were excluded.

2.3.1. The stop signal task (SST)

The SST is a behavioural measure of reactive inhibition that assesses an individual's ability to inhibit an already initiated response (Logan, 1994; Logan and Cowan, 1984). Computational

models of the SST, based on a horse-race model of two competitive and independent go and stop mechanisms (Logan and Cowan, 1984), have been used to estimate the speed of the putative inhibitory process (Robbins, 2007). The SST involves two concurrent tasks that are assumed to be underscored by two independent 'going' and 'stopping' processes: a go task, typically choice reaction time (RT) task, in which participants are asked to respond to a target (e.g., location of a stimulus), and a stop task, in which stop signals (e.g., an auditory tone or red dot) are presented at a variable delay after the onset of the go target to indicate that the response should be withheld. In most studies, stop signals are present on approximately 25% of the go trials to encourage rapid responding. In the SST, the delay between stop and go signals is adjusted in a stepwise manner to directly influence the difficulty of response inhibition, and to ascertain the delay that enables successful inhibition on approximately 50% of the stop trials, referred to as the stop signal delay (SSD). For example, the delay will increase in 50ms increments to increase the difficulty of withholding a response, and will decrease after unsuccessful stops to facilitate inhibition.

This review only included studies using a variant of the SST described above. This was to specifically assess *action cancellation*, i.e., cancellation of an already initiated response, compared to *action restraint* as measured by tasks such as the GNG in which either a go or no-go stimulus is presented on each trial (Lawrence et al., 2015b). The classification of the task as an eligible version of the SST was based on the following criteria:

1) Stop signals that occur on a 'go' trial of a simple reaction time (RT) task (e.g., an auditory tone presented at a variable delay after the go target).
2) The stop signal was presented at a variable delay after the go target.

Studies may report a range of outcome measures from this task, the most common being the SSD, the probability of failed inhibition, the latency of correct responses (usually measured by mean or median RT on go trials), and the stop signal reaction time (SSRT). The SSRT is calculated by

subtracting the SSD (the delay between target and stop signal onset that is dynamically adjusted to enable successful inhibition on approximately 50% of the stop trials) from the mean reaction time (MRT) on go trials, although the median reaction time has also been used in place of the MRT. The SSRT is thought to be an index of impulsivity, with longer SSRTs indicative of greater impulsivity, and therefore poorer inhibitory control.

3. RESULTS:

A total of 321 studies were identified through database searching. After screening titles and abstracts, the full manuscript of 148 articles were assessed for eligibility. Of these, 44 studies were identified as meeting eligibility criteria. Hand-search and review of articles citing the eligible studies and relevant titles identified in the reference lists yielded an additional 14 articles deemed eligible for inclusion, resulting in a total of 58 articles included in the final narrative synthesis (see Figure 1).

Records were classified into two outcome categories: a) studies that assessed the relationship between eating/weight and inhibitory control as measured by the SST and b) studies that manipulated the SST procedure to influence inhibitory control ability.

[Insert Figure 1]

3.1. Study characteristics

A total of 62 studies from 58 journal articles were included. Of these, 51 studies assessed the relationship between eating or weight-related behaviours and performance on the SST. Of these, 8 studies included a sample of individuals with an ED (1 in youths), 19 studies included a sample categorised by weight status (10 in youths), 7 studies assessed defined non-clinical eating behaviours including LOC eating, dieting status and restrained eating (1 in youths), and 17 studies

explored this relationship in healthy individuals not characterised by a particular eating or weight-related behaviour (1 in youths). Data extracted from these studies are presented in table 1. A further 11 studies from 8 articles involved a manipulation of the SST to assess the possibility of altering inhibitory control abilities. Of these, 1 study was conducted in obese youths as part of a residential treatment for obesity, 2 studies were conducted in adults categorised by non-clinical eating behaviours, and 9 studies were conducted in healthy adults not otherwise categorised by eating or weight behaviour. Data from these studies are presented in table 2.

3.2. Main findings: the relationship between eating/weight and SST performance

3.2.1. Eating disorders

Eight studies compared SST performance between people with an ED and healthy individuals without an ED. Five reported no differences in behavioural SST measures such as the SSRT, MRT and accuracy between adult ED and HC groups: between AN-R, AN-BP, BN and HC (Claes et al., 2006), individuals recovered from AN (AN-rec) and HC (Oberndorfer et al., 2011), BED and matched HC (Mole et al., 2015; Wu et al., 2013a), a mixed ED sample (BN and EDNOS) compared to HC (Boisseau et al., 2012) and one study reported no differences in adolescents with AN compared to HC (Wierenga et al., 2014a). In contrast, higher SSRT have been reported in individuals with both AN subtypes (Galimberti et al., 2012), BN (Wu et al., 2013a) and BED (Svaldi et al., 2014) compared to HC. No study reported differences in mean reaction time, suggesting any deficits in inhibitory control (as indicated by the SSRT) are not due to impairments in motor response. In one study, reduced performance accuracy on go trials has been observed in BN (Galimberti et al., 2012). Two neuroimaging studies revealed reduced recruitment of medial frontal regions on difficult trials of the SSRT in small samples of adult women recovered from AN (Oberndorfer et al., 2011) and adolescents with current AN (Wierenga et al., 2014a), with no neural differences on easy trials nor any behavioural differences in accuracy or MRT. Finally, SSRT was observed to correlate with eating pathology (Svaldi et al., 2014; Wu et al., 2013a) and BMI ((trend)

Mole et al., 2015) for individuals with BED, whereas no correlations between eating/weight and SSRT were observed in individuals with AN or BN (Galimberti et al., 2012; Wu et al., 2013a).

3.2.2. Weight status

Nineteen studies (10 in child/adolescent samples) explored the relationship between SST performance and BMI/weight status. In both adults and adolescents, the findings were largely inconsistent. Three studies reported greater SSRTs in obese adults (Chamberlain et al., 2015; Mole et al., 2015) and overweight adults (Houben et al., 2014) compared to HC. In contrast, six studies found no overall differences in SSRT between normal weight HC and overweight (Chamberlain et al., 2015; Nederkoorn, 2014) or obese adults (Grant et al., 2015; Hendrick et al., 2012; Lawyer et al., 2015; Nederkoorn et al., 2006c). Similarly, while some studies reported greater SSRT in obese (Kulendran et al., 2014; Nederkoorn et al., 2006a) and overweight ((trend) Nederkoorn et al., 2012; Verbeken et al., 2009) youth, this was not consistent (Fields et al., 2013; Guerrieri et al., 2008b; Lokken et al., 2009). While one study revealed obese adults who do not binge eat to show greater SSRTs compared to individuals with BED (Mole et al., 2015), such findings did not translate to children, with no difference observed between obese individuals who do or do not binge eat (Nederkoorn et al., 2006a). Furthermore, two studies revealed that SSRTs were higher in overweight children (Nederkoorn et al., 2012) and adults (Houben et al., 2014) compared to HC only on food-specific trials of the SST or in later but not earlier blocks of the SST (Guerrieri et al., 2008b; Nederkoorn et al., 2006c). This suggests that deficient inhibitory control emerges in overweight individuals once cognitive resources begin to deplete, or that any inhibitory control deficits are specific to food-related contexts. No differences in MRT were reported in the majority of studies except for one, which revealed longer reaction times in obese compared to normal weight adults (Grant et al., 2015), suggesting that on the whole, poorer inhibitory control was not due to differences in general motor responding.

Correlational and regression analyses revealed SSRT to be relatively consistently related to and predictive of weight/BMI status in overweight/obese adults (Chamberlain et al., 2015; Mole et al., 2015 (trend)), and children (Kulendran et al., 2014; Levitan et al., 2015), however this was not observed in all studies (Lawyer et al., 2015; Lokken et al., 2009). Moreover, Levitan et al. (2015) found that this may only be the case for females of a pre-school age rather than for males, and two studies did not observe an association between SSRT and BMI or weight status (Allom and Mullan, 2014; Lawyer et al., 2015). A further three studies have revealed that SSRT correlated with or predicted weight loss during treatment (Kulendran et al., 2014; Nederkoorn et al., 2006a; Nederkoorn et al., 2006b). In an influential study, Nederkoorn et al. (2006b) found that impulsivity was related to weight change, defined as the change in percentage overweight, in overweight children enrolled in an 8-week behavioural weight-loss intervention: children with the highest percentage overweight were the most impulsive and lost the least percentage overweight, and change in percentage overweight was predicted by SSRT.

With regards to food/eating-related behaviours, SSRT had no main effect or interaction with weight status in adults on attention bias towards food in adults (Bongers et al., 2015), or on food intake (Guerrieri et al., 2008b) or BMI (Fliers et al., 2013) in children. However, in adults, SSRT did have a main effect on number of calories purchased from a virtual supermarket (Nederkoorn, 2014), and interacted with weight status: better inhibitory control was associated with greater purchasing of snack calories in the context of promotional advertising in overweight but not healthy weight individuals (Nederkoorn, 2014). Moreover, SSRT has been reported by two studies to predict food consumption: specifically, SSRT predicted saturated fat intake but not fruit or vegetable consumption in adults (Allom and Mullan, 2014), and predicted sugar and carbohydrate intake but not total calorie, protein or fat intake in pre-school children (Levitan et al., 2015).

One study used neuroimaging to compare neural recruitment during the SST between obese and normal weight adults (Hendrick et al., 2012). It reported that in the absence of differences in

behavioural performance, obese individuals showed reduced recruitment of regions that were more active during stop compared to go trials (including the cuneus, insula, SMA and IPC bilaterally) than HC women. Moreover, recruitment of regions more active during stop than go trials inversely correlated with BMI across obese and normal weight females (Hendrick et al., 2012), suggesting less efficient inhibitory control with increasing BMI.

3.2.3. Other eating behaviours

SST was assessed in relation to a number of non-clinical eating behaviours in a total of 7 studies. These included eating restraint, eating disinhibition, dieting status and emotional eating in adults, and LOC eating in children.

Greater SSRTs were observed for restrained eaters (RE) compared to unrestrained eaters (URE) (Dong et al., 2014; Nederkoorn et al., 2004). Additionally, one study reported lower SSDs in RE compared to URE, and this finding was not affected by self-reported eating disinhibition (Leitch et al., 2013). Moreover, Dong et al. (2014) revealed that SSRT correlated with regional homogeneity (ReHo), an index of resting state brain activity, in RE only, observing a negative correlation with the left DLPFC and a positive correlation with the left insula. No differences in SSRTs were found between dieters and non-dieters (Meule et al., 2014) or between individuals with high or low emotional eating (van Strien et al., 2014). SSRT was not found to correlate with emotional eating (van Strien et al., 2014). Restraint and emotional eating were both found to interact with SSRT to influence food intake but in the opposite directions: food intake was affected by emotional eating only in individuals with high inhibitory control (van Strien et al., 2014), whereas caloric intake correlated with dietary restraint only in individuals low in inhibitory control (i.e., high impulsive participants, Jansen et al., 2009). No differences in mean RT were reported in any of the studies.

One study explored the relationship between LOC eating and SST performance (Hartmann et al., 2013). It found no behavioural differences between adolescents who reported LOC eating

compared to those who did not; however, greater go reaction time variability (GRTV, i.e., the range of reaction times over all trials) after mood induction was observed only in the LOC eating group, suggesting a reduced ability to maintain control over behaviour in the context of negative mood in individuals exhibiting LOC eating.

3.2.4. Healthy individuals

Seventeen studies assessed the relationship between SST performance and a range of eating/weight behaviours in healthy individuals, one of which was conducted in child samples.

Healthy individuals showed greater inhibitory control (i.e., lower SSRT) if they initiated opportunistic snacking compared to non-initiators (Fay et al., 2015). Moreover, food exposure (via a food-specific SST) impaired inhibitory control in unsuccessful but not successful weight regulators (Houben et al., 2012).

The majority of studies found no main effect of SSRT on food intake (Guerrieri et al., 2007a; Guerrieri et al., 2007b; Hall et al., 2015; Lattimore and Mead, 2015), number of calories purchased in a virtual supermarket (Giesen et al., 2012), acquisition or extinction of a conditioned craving or liking response to chocolate (Papachristou et al., 2013) in healthy adults. This lack of effect was not influenced by manipulation of eating restraint, either through eating instructions (Hall et al., 2015) or food intake by a confederate (Hermans et al., 2013). Similarly, no association was found between SSRT and weight change (Nederkoorn et al., 2010) or BMI (Haynes et al., 2015; Nederkoorn et al., 2009; Wang et al., 2013) in university students, or with BMI in children (Nederkoorn et al., 2015). Only one study found BMI to be correlated with accurate inhibition in adults (Lowe et al., 2014).

In contrast, inhibitory control was found to moderate the effect of evaluative conditioning training on food intake (Haynes et al., 2015) and the impact of automatic affective reactions on candy

consumption (Hofmann et al., 2009). For example, snack intake was reduced after pairing unhealthy food words with negative compared to positive images in individuals with low but not high inhibitory control (Haynes et al., 2015). SSRT also interacted with food exposure, as food exposure impaired inhibitory control in high impulsive but not low impulsive individuals (Lattimore and Mead, 2015). Similarly, SSRT interacted with sales promotion, with pricing strategies having a greater impact on food purchase behaviour in individuals with poor inhibitory control, particularly for high energy dense foods (Giesen et al., 2012). This suggests that while SSRT may not have a main effect on non-clinical eating behaviour in healthy adults, it may affect the way in which these eating behaviours are influenced by external factors: individuals with poorer inhibitory control may be more vulnerable to cue-triggered unhealthy eating behaviours.

Some studies observed SSRT to be positively correlated with food intake (Guerrieri et al., 2007b; Nederkoorn et al., 2009 (snack calories)) and predicted food intake (Guerrieri et al., 2007b; Houben et al., 2012), however this was not consistent (Haynes et al., 2015; Hermans et al., 2013; Lowe et al., 2014). Furthermore, this may only be the case for the food-specific but not general SST (Houben et al., 2012), suggesting poor inhibitory control may be context-specific.

Lastly, hunger interacted with inhibitory control to influence food intake and food purchase in healthy adults (Nederkoorn et al., 2009, studies 1 and 2), but not food intake in children. In healthy children, SSRT influenced intake of HED foods: individuals with poor inhibitory control consumed more high energy dense foods than individuals with better inhibitory control regardless of time of testing (before/after lunch), with no difference observed for intake of MED or LED foods (Nederkoorn et al., 2015). This suggests that for children, greater inhibitory control is required to overcome the temptation to eat HED food, regardless of hunger, whereas this may be more dependent on hunger in adults.

[Insert Table 1]

3.3. Manipulating inhibitory control

3.3.1. Weight status

Verbeken et al. (2013) compared weight loss and neurocognitive changes in children towards the end of an inpatient stay after receiving either care as usual (CAU) or CAU plus executive function training (CAU-EF), including inhibitory control training using the SST. They did not find any improvement in SST performance in the CAU-EF group, although the protocol for the SST was adjusted by requiring participants to respond within a certain response time range. However, despite not observing any changes in SST performance, children who received the CAU-EF treatment showed improved maintenance of weight loss at 8-weeks post-treatment than children in the CAU condition. While this suggests that executive function training can promote healthy eating behaviours, the degree to which training in inhibition contributes to this result is unclear.

3.3.2. Other eating behaviours

Two studies manipulated state impulsivity in RE and both reported an interaction between dietary restraint and inhibitory control in the absence of any main effects of either inhibition or restraint. Houben and Jansen (2014) found that rewarding accurate performance impaired impulsivity and increased food craving and consumption in RE, while reducing food intake and craving for URE. Guerrieri et al. (2009) manipulated SST instructions to prioritise either the go or stop task (to promote impulsivity or inhibitory control, respectively). They found that high and low restrained non-dieters had greater food intake in the impulsivity compared to inhibition condition, whereas the opposite was true for current dieters. However, they did not include a control manipulation, and therefore it is unclear whether this difference in food intake was due to greater impulsivity in the impulsivity condition or greater inhibitory control in the inhibition condition.

3.3.3. Healthy individuals

Nine studies assessed the relationship between eating and impulsivity in young healthy adults by manipulating inhibitory control (either in general or towards particular food stimuli) using the SST and have reported mixed findings. Six observed reduced food intake behaviours after the SST was manipulated to promote inhibition (Allom and Mullan, 2015 (study 1); Guerrieri et al., 2012; Houben, 2011; Lawrence et al., 2015b (studies 1 and 2); Sellitto and di Pellegrino, 2014). For example, Guerrieri et al. (2012) manipulated impulsivity and inhibition through SST instructions, revealing significantly greater food intake by individuals trained in impulsivity compared to the neutral and control (reading and summarising text) manipulations, with no difference in hunger or BMI. Moreover, food that was more strongly associated with stopping on the SST were associated with fewer impulsive choices in a hypothetical food choice task (Sellitto and di Pellegrino, 2014), and were consumed less compared to control foods (equally associated with stopping and responding) in individuals with poor inhibitory control (Houben, 2011), whereas individuals with better inhibitory control ate more of the food associated with go trials compared to control foods (Houben, 2011).

However this finding was inconsistent (Allom and Mullan, 2015 (study 2); Lawrence et al., 2015b (study 3)), with some studies suggesting that such a training effect is specific to certain individuals and contexts. Allom and Mullan (2015) conducted two studies assessing the impact of a 10 day food-specific (stop trials paired only with unhealthy food images) and general (stop trials randomly paired with healthy/unhealthy food images) inhibition training on BMI and fat intake, revealing a decrease in BMI after the food-specific inhibition intervention in one study, but no effect of intervention in the other. Finally, in a series of three studies, Lawrence et al. (2015b) compared the effect of signal response, in which participants were either instructed to withhold their response, provide two key presses, or respond to the go target as usual on signal trials, and assessed the generalisability of inhibitory control training by varying the association between food/neutral stimuli and signal trials. Again, results were mixed: less calories were consumed by individuals in

the stop group compared to the double response group (study 1), with no interaction with dietary restraint. This was subsequently found to be specific for foods strongly associated with the signal for individuals high in dietary restraint (study 2), and this training effect was absent on a general SST with no food stimuli, regardless of dietary restraint (study 3). Moreover, stop accuracy on food trials was not associated with food intake (studies 1 and 2).

[Insert Table 2]

4. DISCUSSION

This systematic review is the first to synthesise the findings of studies that have assessed the relationship between eating behaviour, weight and inhibitory control assessed by the SST. It has the advantage of including a broad range of clinical and non-clinical groups, SST paradigm designs (with respect to study aims [i.e., assessment/manipulation], trial type, stimuli [food/general], etc.) and SST outcome measures (e.g., MRT, SSRT, neuroimaging/behavioural, etc.); this was done in an attempt to reduce bias in the studies that were included. However, this heterogeneity in the sample populations, task protocols and outcome measures precludes definitive conclusions on the relationship between reactive inhibitory control and eating/weight. Thus, although a single neurocognitive construct was assessed, the synthesis of the findings should be interpreted with caution as differences may be due to methodological differences between studies. Future research should aim to replicate or standardise the methodology and outcome measures.

4.1. Inhibitory control and eating disorders

The literature on the relationship between eating, weight and inhibitory control was largely inconsistent. In EDs, there were insufficient studies to permit definitive conclusions. In accord with previous reviews implicating poorer inhibitory control in bulimic-type EDs (Waxman, 2009;

Wierenga et al., 2014b; Wu et al., 2013b), higher SSRTs (reflecting poorer inhibitory control) in people who binge eat and/or purge were reported in three studies, although these individuals belonged to different ED diagnostic categories. It appears, therefore, that poor reactive inhibition is related to more impulsive symptoms (e.g., bingeing) in adults, characterised by strong urges and by an experience of loss of control. However, in line with previous reviews (Van den Eynde et al., 2011b), the majority of studies reported no behavioural differences in reactive inhibition in people with an ED compared to HCs. Interestingly, however, while the meta-analysis by Wu et al. (2013b) revealed a general and disorder-specific inhibitory control deficit in bulimic-type disorders and BN, respectively, the authors note that the majority of the individual studies did not observe impaired inhibitory control in their ED sample (17/25 studies exploring general stimuli and 14/20 studies exploring disorder-specific stimuli). Meta-analyses of SST performance in relation to each ED will therefore be of interest in the future, after more studies employing the SST in ED samples have surfaced. However, given the inconsistency across the findings in EDs, it seems likely that reactive inhibition is not the only component of inhibitory control that contributes to symptom presentation or development.

Neuroimaging studies in acutely ill and recovered AN samples revealed reduced medial prefrontal activity during harder trials of inhibition in the absence of behavioural differences (Oberndorfer et al., 2011; Wierenga et al., 2014a). This suggests that individuals with AN have a higher tonic state of inhibitory control and/or may recruit higher cortical regions involved in cognitive control to a lesser degree. This is consistent with the broad hypothesis that AN is maintained by an excessive capacity for self-regulation (Brooks et al., 2012). Alternatively, these findings may reflect more efficient control-related prefrontal activity or alterations in the neurocircuitry involved in inhibitory control, e.g., greater reliance on other brain regions. Whole-brain functional connectivity analyses are required to elucidate what drives these neural differences. However, as this was found across ages and across illness states, altered prefrontal activity during the SST may reflect a persistent trait or endophenotype. It will be of interest to establish whether such differences are present

before disorder onset, and also to assess the potential of inhibitory-control related prefrontal activity as a biomarker of future illness (Bartholdy et al., 2015b; Bartholdy et al., 2015c).

4.2. Inhibitory control and weight status

The data on inhibitory control were inconsistent in relation to weight status. There were no obvious differences between different weight categories in terms of SST performance, however a few studies reported that poor inhibitory control emerged in later blocks of the SST. This suggests that obese individuals have difficulty in maintaining inhibitory control, rather than a general impairment. Moreover, obese and overweight participants have been reported to have poorer inhibitory control on food-specific versions of the task (Guerrieri et al., 2008b; Nederkoorn et al., 2006c), indicating difficulties in inhibitory control but only in the context of food. This is in accord with studies assessing other forms of inhibitory control, e.g., food compared to monetary discounting tasks (e.g., Hendrickson and Rasmussen, 2013; Rasmussen et al., 2010), and may be related to saliency, relevance of stimuli or motivation/reward. Moreover, this is consistent with a observations that the interaction between food motivation and executive function had a greater impact on eating behaviour than either factor alone (for rev., see Vainik et al., 2013).

In contrast to the above, inhibitory control was more consistently found to predict BMI/weight status and weight change. Thus, inhibitory control (as assessed by the SST) may be an indicator of individuals both at risk of developing unhealthy behaviours and those most likely to be unresponsive to weight loss interventions. In addition, BMI negatively correlated with recruitment of regions during stop trials requiring response inhibition (Hendrick et al., 2012), although no behavioural differences were observed. Together, these findings suggest that there is a cause and effect issue between inhibitory control and BMI. While there was some evidence for a relationship between SSRT and food intake in normal weight to obese participants, this may be specific to certain types of food and to the specificity of the SST (e.g., food-specific vs. general).

4.3. Inhibitory control in healthy individuals

In healthy adults, inhibitory control was not associated with food intake. However, it did appear to moderate the extent to which food intake and food purchasing were influenced by external factors such as promotional advertising and food exposure. This suggests that individuals with poorer inhibitory control may be more susceptible to engaging in unhealthy eating behaviours in the face of triggers of temptation or desire (e.g., advertisements).

With regard to non-clinical eating behaviours, restrained eating was most consistently associated with poor inhibitory control, and this appears to be related to altered resting activity in brain regions implicated in inhibitory control. Thus, poorer inhibitory control (high SSRT) correlated with reduced resting activity in the left DLPFC and greater resting activity in the insula (Dong et al., 2014). Both regions have been identified as part of a core network of regions important to many aspects of inhibitory control, although the DLPFC may be more related to the implementation of task rules rather than the ability to stop outright (Aron et al., 2004; Aron et al., 2014; Cai et al., 2014; Hare et al., 2009; Ridderinkhof et al., 2004). These areas have also been implicated in appetitive regulation and EDs (Kaye, 2008; Kaye et al., 2013; Uher et al., 2004). These findings therefore suggest reduced recruitment of higher-level control centres in restrained eaters compared to unrestrained eaters. Moreover, the effects of inhibitory control training are most pronounced in individuals exercising eating restraint. For example, rewarding accurate performance is reported to lead to increased food consumption in restrained eaters (Houben et al., 2014). Moreover, Pavlovian conditioning (i.e., associating food with a stop signal; Lawrence et al., 2015b, study 2) and prioritising stopping (Guerrieri et al., 2009) is reported to lead to reduced food intake in restrained eaters. However, these studies did not distinguish between individuals who were successful at exercising restraint compared to unsuccessful restrained eaters. This may be important as success may be underscored by inhibitory control ability. Restrained eaters frequently report unsuccessful dieting and their efforts at restraint are often accompanied by periods of overeating, particularly when they are disinhibited (Hofmann et al., 2014; Mann et al., 2007). This

may be due to by poor inhibitory control in general, or temporary depletion of inhibitory resources after sustained attempts at exercising dietary restraint (Lawrence et al., 2015a). In restrained eaters, resources needed to exert self-control may be depleted during attempts at exercising restraint (Muraven and Baumeister, 2000), and providing additional instructions or conditions (e.g., reward) may overwhelm their remaining resources, leading to failed inhibition (Vohs and Heatherton, 2000; Ward and Mann, 2000; however, see Inzlicht et al., 2014; and Stroebe et al., 2008). Thus, restrained eaters may benefit from SST manipulations that promote inhibition implicitly through conditioned associations or simple instructions, rather than through explicit demands and performance feedback that may increase stress or pressure experienced during the training.

In addition to restrained eaters, manipulating inhibitory control using the SST appears to influence eating and weight behaviours in non-clinical adult groups but this does not appear to be due to general inhibitory control ability, as the main effects of inhibitory control were not often observed (Allom and Mullan, 2015 (studies 1 and 2); Guerrieri et al., 2009; Houben, 2011; Lawrence et al., 2015b (studies 2 and 3)). These findings are in accord with inhibitory control training using food-specific variations of the go/no-go tasks, that have reported success in reducing appetitive behaviour towards foods frequently paired with stop signals in individuals with non-clinical eating behaviours (Houben and Jansen, 2011, 2015; Lawrence et al., 2015a; van Koningsbruggen et al., 2014; Veling et al., 2011; Veling et al., 2013; Veling et al., 2014). However, the generalisability of these collective inhibitory control training interventions to non-conditioned foods or to other eating behaviours (e.g., clinical eating behaviours and modification of weight status) is not clear.

4.4. Clinical implications

Neurocognitive training may have potential in treating obesity and EDs, particularly those involving binge eating episodes. One published study has assessed inhibitory control training using the SST in obesity (Verbeken et al., 2013), with independent research ongoing (e.g., Halberstadt et al., 2013); however, no studies have attempted to train inhibitory control using the SST in EDs.

Nonetheless, several other techniques for altering impulsivity are currently under investigation in both EDs and obesity. These include non-invasive computerised paradigms, such as attentional bias modification protocols (Boutelle et al., 2014; Kemps et al., 2014; Renwick et al., 2013), that experimentally manipulate changes in attentional processes to manipulate eating behaviour. In addition, the potential of brain stimulation techniques that can alter the activity of specified brain regions is under investigation (Bartholdy et al., 2015a; Bartholdy et al., 2013; Bou Khalil and El Hachem, 2014; Schmidt and Campbell, 2013; Truong et al., 2013; Val-Laillet et al., 2015). These neuromodulation techniques show promise in treating both symptoms and food craving (Kekic et al., 2014), EDs (Khedr et al., 2014; Lipsman et al., 2013; McClelland et al., 2013a; McClelland et al., 2013b; McClelland et al., 2015; Pires Baczynski et al., 2014; Van den Eynde et al., 2011a; Van den Eynde et al., 2010; Van den Eynde et al., 2013) and overweight or obesity (Frank et al., 2012; Gluck et al., 2015; Montenegro et al., 2012). In addition, medications are being explored as potential neurochemical modulators of inhibitory control (e.g., for review see Chamberlain et al., 2011). Selective monoamine reuptake inhibitors such as atomoxetine (Chamberlain et al., 2009; Kehagia et al., 2014) and citalopram (Ye et al., 2014; Ye et al., 2016), psychostimulants such as methylphenidate (Aron et al., 2003; Nandam et al., 2011; Pauls et al., 2012) and wakefulness-promoting medication such as modafinil (Turner et al., 2004; Turner et al., 2003) have shown early evidence of improving inhibitory control, demonstrated by SST performance, in healthy volunteers, and individuals with attention deficit/hyperactivity disorder (ADHD) or Parkinson's disease, although null effects of these medications on SST performance have also been reported (Costa et al., 2013; Nandam et al., 2011; Winder-Rhodes et al., 2009). Future studies may wish to assess whether such neuromodulatory techniques and psychotropic medication can alter inhibitory control to improve or regulate eating behaviour, and the possible additive effect of such interventions with behavioural training paradigms.

4.5. Theoretical considerations

Two concepts appear to be closely related to inhibitory control: impulsivity and compulsivity. There is, however, often confusion between these terms, their definition and how they relate. Inhibitory control is the ability to withhold inappropriate responses. Impulsivity is a broad, multi-faceted term that refers to the tendency to act prematurely without foresight (Dalley et al., 2011). Compulsivity, on the other hand, refers to the persistence of inappropriate behaviours that often result in negative consequences (Dalley et al., 2011; Robbins et al., 2012). Although traditionally impulsivity and compulsivity were considered antonyms, both are considered to result in part from failed inhibitory control, and more recently have been viewed as orthogonal/overlapping concepts (Sohn et al., 2014), reflecting different stages of behavioural control: impulsivity relating to action initiation, and compulsivity relating to action termination (Robbins et al., 2012). When developing transdiagnostic frameworks, attempts have been made to examine and discuss these three concepts together. Poor inhibitory control has been implicated in a range of impulsive and compulsive psychiatric disorders that have high comorbidity with EDs and obesity, including obsessive compulsive disorder (OCD), schizophrenia, ADHD and substance abuse (e.g., for rev., see Chamberlain et al., 2005; Dawe and Loxton, 2004; Lipszyc and Schachar, 2010; Verbruggen and Logan, 2008). Thus, poor inhibitory control may act as a vulnerability factor for a range of psychiatric conditions, and may present a useful concept for transdiagnostic investigations. However, further investigation is needed to determine the precise mechanisms that are involved in each of these discrete conditions and their symptoms, and how these are specifically related to impulsivity and compulsivity. EDs and obesity are characterised by both impulsive and compulsive features (e.g., spontaneous or planned bingeing and purging episodes, inability to control urges, LOC eating, compulsive overeating, compulsive exercise, body/calorie checking, ruminative thinking and difficulties in coping with thoughts/decisions) (e.g., Claes et al., 2002; Dawe and Loxton, 2004; Engel et al., 2005; Robbins et al., 2012). This is not a new observation: 20 years ago, EDs were proposed to be included in a spectrum of disorders characterised by obsessive-compulsive and impulsive traits, jointly termed obsessive compulsive spectrum disorders (McElroy

et al., 1994). However, it will be of interest to determine how and which elements of inhibitory control relate to the individual impulsive and compulsive features of EDs and obesity.

The SST assesses the capacity for action cancellation; one element of reactive motor inhibitory control. Performance on this task is often interpreted in the context of impulsivity, with poor inhibitory control thought to reflect impulsive action (Dalley et al., 2011). As discussed above, such an interpretation is controversial as, in addition to impaired inhibitory control, impulsivity requires the co-occurrence of a strong desire, urge or habit to initiate a response. However, it can be argued that as the typical SST paradigm is predominantly comprised of 'go' trials requiring a response, the task does elicit some urge to respond, though whether this can be considered sufficient for the definition of impulsivity to be used is still a matter of debate.

Inhibitory control in the SST has also been proposed to reflect compulsivity, on the basis that the task measures the ability to inhibit an already initiated response (Robbins et al., 2012). Although the paradigm is designed so that the participant's stop accuracy converges at 50%, the inability to achieve this rate of inhibition may reflect compulsivity: non-convergence either through persistent inhibition (i.e., waiting for the stop signal on each trial) or persistent reaction (i.e., inability to withhold the response). However, it is arguable that in the context that the task was completed correctly (i.e., the participant achieved ~50% correct inhibition), the task may not sufficiently capture drive or habit for persistent inappropriate responding that is reflective of compulsivity. Set-shifting tasks or tasks assessing proactive strategic adjustment of behaviour may be more appropriate for investigations of compulsivity, as perseveration of inappropriate behaviour is explicitly assessed. However, this review has sought to include comment on compulsivity by including findings regarding both go and stop accuracy, and exploration of the relationship between SST performance and a number of compulsive eating and weight-control behaviours, e.g., binge eating and LOC eating. Thus, it can be used as a starting point for discussions of the involvement of compulsivity in unhealthy eating behaviours.

4.6. Methodological considerations

There are a few methodological issues to consider with respect to both the included studies and the present review. With regards to the included studies, a gender bias was most prominent amongst studies assessing inhibitory control in EDs (with the exception of Mole et al., 2015), and in relation to other non-clinical eating behaviours. Thus it is unclear whether findings can be generalised to clinical or non-clinical eating behaviours in males. Similarly, all of the non-clinical adult studies were conducted on university students and staff, which may introduce sampling bias and prohibit generalisation to the whole community. Manipulation studies were also composed of predominantly female university students and staff. Given their years in education, these individuals are expected to already possess good executive skills (including inhibition), leaving little room for improvement through behavioural training: this may explain the lack of a direct effect of training on response inhibition. Studies in non-clinical populations did not consistently distinguish individuals who were within the normal weight range (BMI of 18.5-25kg/m^2) to those outside of this range. Some included individuals who were underweight in their healthy weight group (Houben et al., 2014), whereas others included individuals who were obese, with BMIs of over 30kg/m^2 (e.g., Houben, 2011; Houben and Jansen, 2014). In one study in older adults, almost half of the sample (47.6%) were overweight/obese (Hall et al., 2015). Moreover, some studies did not report the BMI range of the sample (e.g., Guerrieri et al., 2007a; Nederkoorn et al., 2004; van Strien et al., 2014). This is important, as it obscures our ability to assess the association between weight and inhibitory control.

With respect to the design of the present review, as a consequence of the few limitations placed on the inclusion criteria (with regard to study design, outcome measures, study sample and study quality), there was large heterogeneity in the studies and this hampered the ability to compare and integrate findings and to draw meaningful conclusions. For example, this review has included studies of children and adolescents, as well as adults. While a similar number of studies assessing the relationship between SST performance and weight status were conducted in adult and youth

samples, very few studies assessed the SST in relation to EDs or non-clinical eating behaviours in youths, or in healthy youths. Thus, it is unclear whether findings are specific to children, adolescents or adults. However, with respect to weight status, similar findings were observed in youth and adult samples, suggesting that the association between SST performance and BMI/weight status is consistent across ages. In addition, this review did not include animal studies, those that were not reported in English, conference abstracts or dissertations not published in journals, and therefore may have missed findings that might have influenced the present conclusions. Furthermore, this review did not assess the possible impact of other psychiatric problems characterised by poor inhibitory control, such as ADHD, addiction or substance abuse. As these are often comorbid with EDs and obesity, and because symptoms of inattention, hyperactivity and substance addiction (e.g., smoking) are quite common in the general public, it is important to establish whether these comorbid symptoms explain the presence of alterations in inhibitory control and/or the relationship between inhibitory control and eating behaviours or weight.

4.7. Conclusions

There is evidence for impaired reactive inhibition in restrained eaters, and to some degree in EDs that are characterised by binge episodes. In obesity, poor inhibitory control may be specific to the *maintenance* of inhibitory control, rather than a general impairment. Altering inhibitory control by manipulating SST instructions can generalise to eating/weight behaviours, particularly in restrained eaters. Only one study has assessed the impact of manipulating inhibitory control using the SST in obese individuals, while no such studies have been conducted in ED populations. It is suggested that studies should examine whether training to improve inhibitory control can be used as a cost-effective treatment adjunct that can reduce bulimic symptoms in ED and/or improve weight loss in obesity. However, for EDs in particular, the inconsistent findings suggest that reactive inhibition alone does not explain the lack of control associated with symptom presentation. It is likely that a combination of inhibitory control components contribute, including, for example, proactive

inhibition or motivational inhibitory control (e.g., delaying gratification). Future reviews could assess the independent contribution of other inhibitory control components to various eating and weight-related states and behaviours. In addition, future studies may wish to assess multiple inhibitory components using several neuropsychological paradigms to explore which types of inhibitory control are related to the different eating/weight behaviours. Inhibitory control should also be examined using a longitudinal approach to explore whether altered reactive inhibitory control is a trait marker of various eating and weight-related behaviours, such as in restrained eaters. Finally, more neuroimaging studies of reactive inhibition in EDs are warranted as the limited number of existing neuroimaging studies using the SST suggest that the neural systems underlying inhibitory control may be altered, which may not be reflected at a behavioural level. Thus, altered neural activity associated with performance on the SST may be an endophenotype that has use, for example, as a construct that could aid diagnostic classification, or be a biomarker of future illness development (Bartholdy et al., 2015b; Bartholdy et al., 2015c) or treatment response (Bartholdy et al., 2015a; Kulendran et al., 2014; Nederkoorn et al., 2006a; Nederkoorn et al., 2006b).

5. Authors' contributions

SB conceived the study and drafted the manuscript. SB and BLD conducted the search and extracted the data. BLD, OOD, ICC and US revised the manuscript critically for important intellectual content. All authors contributed to the design, conception, drafting, critiquing and approving of the manuscript, and accept responsibility for the accuracy and integrity of this work.

6. Conflicts of interest

The authors report no conflicts of interest.

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Figure 1. PRISMA (preferred reporting items for systematic reviews and meta-analysis) (Moher et al., 2009) flowchart highlighting the number of records identified at each stage of the search and final total included in the review.

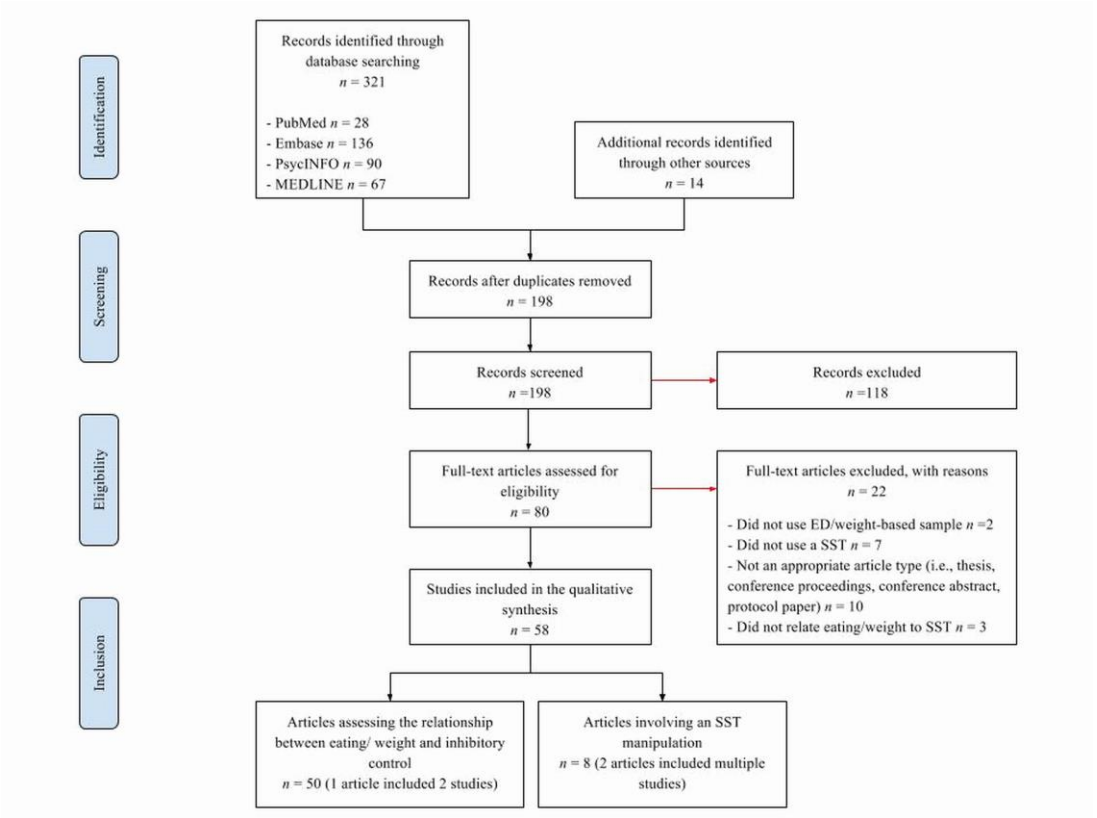

Table 1. Studies assessing the relationship between eating, weight and performance on the SST

			Measures			Findings		
Author	**N[a]**	**Population**	**Measure of eating/ weight**	**SST main outcomes**	**SSRT calculation**	**Impact of eating/ weight on SST**	**Impact of inhibitory control on eating/weight**	**Associations between eating/weight and SSRT**
EATING DISORDERS								
ADULTS								
(Boisseau et al., 2012)	53	Females with an ED (n=21; 12 BN, 9 EDNOS), OCD (n=16) and HC females (n=16)	ED diagnosis	SSRT, mean RT, % omission errors	Mean [e]	All SST measures: ED = HC.		
(Claes et al., 2006)	139	Female ED in/outpatients (n=56 (20 ANR, 14 ANBP, 22 BN)) and female HC (n=83)	ED diagnosis	SSRT, SSD, mean RT, % incorrect inhibition	Mean [e]	All SST measures: ED (ANR=ANBP=BN) = HC		
(Galimberti et al., 2012)	92	Females with ANR (n=24), ANBP (n=12), BN (n=16) and HC females (n=40)	ED diagnosis	SSRT, mean RT, Number of inaccurate responses	Mean [e]	SSRT: AN (R and BP)>HC, ANBP>BN. Inaccurate responses: BN>HC. Mean RT: AN(R and BP) = BN = HC.	AN: Good performers (+1SD SSRT z-score) = bad performers (-1SD SSRT z-score) for BMI, disorder onset, illness duration and other SST variables	No significant correlations between SST and BMI, age at onset, illness duration or illness severity.

			Measures			Findings		
Author	**N[a]**	**Population**	**Measure of eating/ weight**	**SST main outcomes**	**SSRT calculation**	**Impact of eating/ weight on SST**	**Impact of inhibitory control on eating/weight**	**Associations between eating/weight and SSRT**
(Mole et al., 2015)	180	Obese BED (n=30, 17 f), obese without BED (n=30, 11 f), and abstinent alcohol-dependent participants (EtOH, n=30, 12 f) and matched HC (c-BED: n=39, 17 f; c-Obese: n=30, 11 f; c-EtOH: n=30, 12 f)	ED diagnosis, Binge eating scale	SSRT, mean RT	Median	SSRT: BED=c-BED Obese>c-Obese Obese>BED. Mean RT: BED=c-BED Obese=c-obese Obese=BED.		Trend for positive correlation between BMI (but not binge eating scale) and SSRT in BED and Obese groups.
(Oberndorfer et al., 2011) [b]	24/ 17	Females recovered from AN (Rec-AN, neuroimaging data: n=12, behavioural data: n=7) and HC females (neuroimaging data: n=12 and behavioural data: n=11)	ED diagnosis	Accuracy, mean RT, fMRI during hard and easy trials	N/A	Mean RT and accuracy (easy and hard trials): AN = HC. Neural: Rec-AN < HC (mPFC activity on hard trials only).		

			Measures			Findings		
Author	**N[a]**	**Population**	**Measure of eating/ weight**	**SST main outcomes**	**SSRT calculation**	**Impact of eating/ weight on SST**	**Impact of inhibitory control on eating/weight**	**Associations between eating/weight and SSRT**
(Svaldi et al., 2014) [c]	60	Females with BED (n=31) and weight-matched HC (n=29)	ED diagnosis, DEBQ, food vs. neutral trials	SSRT, mean RT, commission errors	Mean (but found comparable results when median was used)	SSRT: BED>HC. SST stimuli: Mean RT: Food>Neutral for both BED and HC (BED=HC). Commission errors: BED>HC (food trials only).		SSRT positively correlated with eating pathology (DEBQ total score, external eating and emotional eating). Increase in commission errors for food>neutral trials was correlated with DEBQ emotional eating and BMI.
(Wu et al., 2013a)	124	Patients with BN (n=16, 15 f), overweight/obese BED patients (n=44, 40 f) and two separate groups of age- and BMI-matched HC (c-BN (n=25, 24 f) and c-BED (n=39, 38 f), respectively)	ED diagnosis, EDEQ	SSRT, SSD, mean RT, % correct responses (go), RT on signal trials	Mean	SSRT: BN>c-BN BED=c-BED. Other SST measures: BN=c-BN BED=c-BED.		No correlation between SSRT and ED pathology in BN; In BED, SSRT only correlated with EDEQ restraint.
YOUTH								

			Measures			Findings		
Author	**N[a]**	**Population**	**Measure of eating/ weight**	**SST main outcomes**	**SSRT calculation**	**Impact of eating/ weight on SST**	**Impact of inhibitory control on eating/weight**	**Associations between eating/weight and SSRT**
(Wierenga et al., 2014a) [b]	23	Adolescent females with ANR (n=11) and HC (n=12)	ED diagnosis	Mean RT, inhibition errors, fMRI during easy/hard blocks (split by individual mean RT)	NA (no SSRT)	Any behavioural SST measure: AN = HC. Neural: AN<HC (middle frontal regions [right dACC, right middle FG and left PCC] on hard trials only).		
WEIGHT STATUS								
ADULTS								
(Allom and Mullan, 2014)	115	Normal weight to overweight undergraduate students (83 f, 85% of sample within the normal BMI range)	Block Food Screener, BMI	SSRT	Mean			SSRT correlated with and predicted saturated fat intake, but not fruit and vegetable consumption or BMI.
(Bongers et al., 2015)	319 [d]	Obese (n=169) and healthy weight (n=116) participants.	Attention bias for food, weight status	SSRT	Mean		No main effects or interaction between weight status and SSRT on attention bias for high/low-calorie food.	

			Measures			Findings		
Author	**N[a]**	**Population**	**Measure of eating/ weight**	**SST main outcomes**	**SSRT calculation**	**Impact of eating/ weight on SST**	**Impact of inhibitory control on eating/weight**	**Associations between eating/weight and SSRT**
(Chamberlain et al., 2015)	511	Obese (n=55, 33 f), Overweight (n=110, 32 f) and HC adults (n=346, 118 f). (Note: obese participants had higher rates of maladaptive gambling behaviour).	Weight status	SSRT	NR	SSRT: Obese>HC.		SSRT predicted weight status.
(Grant et al., 2015)	207	Obese (n=22, 12 f), overweight (n=49, 20 f), and normal weight (n=136, 44 f) young adults with subsyndromal gambling disorder	Weight status	SSRT, median go RT	NR	SSRT: Obese = normal weight. Median RT: Obese>Normal weight.		
(Hendrick et al., 2012)[b]	43	Lean (n=18, BMI<22), intermediate weight (n=12, BMI between 22-30) and obese (n=13, BMI>30) females	Weight status, BMI	SSRT, mean RT, post-error slowing, fMRI (stop>go)	Median	Any SST behavioural measure: Obese = Lean. Neural: Obese<Lean (cuneus, insula, SMA and IPC bilaterally on stop compared to go trials).		BMI negatively correlated with activity in all regions more active during stop (vs. go) trials.

			Measures			Findings		
Author	**N[a]**	**Population**	**Measure of eating/ weight**	**SST main outcomes**	**SSRT calculation**	**Impact of eating/ weight on SST**	**Impact of inhibitory control on eating/weight**	**Associations between eating/weight and SSRT**
(Houben et al., 2014) [c]	87	Females of a range of BMIs (underweight: n=13, overweight: n=17)	BMI, food/neutral trials of the SST	SSRT	Mean	SST*BMI on SSRT: No main effect of SST type or BMI, significant interaction: higher BMI = higher SST on food-specific but not general SST.		
(Lawyer et al., 2015)	291	Obese (n=56, 35 f) and non-obese (n=235, 126 f, underweight: n=10, healthy weight: n=147, overweight: n=78) participants.	BMI	SSRT	Mean	SSRT: Obese = non-obese.		No association between SSRT and BMI/weight status.
(Nederkoorn, 2014)	118	Overweight (n=45, 38 f) and healthy weight (n=73, 64 f) participants.	Purchased calories from a virtual (internet) supermarket with (n=48) or without (n=70) sales promotion.	SSRT	Mean	SSRT: Overweight = HC.	SSRT * weight status * promotion on calories purchase: higher SST = more calories purchased in promotion condition (only) in overweight but not HC participants.	
(Nederkoorn et al., 2006c)	59	Obese (n=31) and lean (n=28) females.	Weight status	SSRT, mean RT, SST block	Mean [e]	Mean RT and overall SSRT: Obese = Lean. Weight status * SST block on SSRT: Obese>Lean in later SST blocks only.		

			Measures			Findings		
Author	**N[a]**	**Population**	**Measure of eating/ weight**	**SST main outcomes**	**SSRT calculation**	**Impact of eating/ weight on SST**	**Impact of inhibitory control on eating/weight**	**Associations between eating/weight and SSRT**
YOUTH								
(Fields et al., 2013)	61	Obese (n=21, 11 f), overweight (n=20, 11 f) and healthy weight (n=20, 12 f) adolescents.	Weight status	SSRT	Mean	SSRT: Obese = Overweight = Healthy Weight.		
(Fliers et al., 2013)	232 [d]	Children with combined subtype ADHD	BMI Standard Deviation scores (BMI-SDS)	SSRT	Mean [e]		No effect of inhibitory control on BMI SDS score.	
(Guerrieri et al., 2008b)	78	Overweight (n=15) and normal weight (n=63) primary school children (33 females in the total sample)	Food intake (bogus taste test)	SSRT, SST block	Mean [e]	Weight status * SST block on SSRT (trend): Overweight>Normal weight in later (third) but not earlier, blocks.	No main effect of SSRT or interaction with variety on food intake.	
(Kulendran et al., 2014)	103	Obese adolescents (n=53, 32 f) attending a residential treatment camp for obesity and non-obese adolescents (n=50, unknown gender proportions)	Weight status, BMI	SSRT, Mean RT, SSD, proportion of successful stops, number of inaccurate responses, change in SSRT over time.	Mean [e]	SSRT, number of inaccurate responses: Obese>Normal weight. SSD, proportion of successful stops: Obese<Normal weight. Mean RT: Obese = Normal weight.		SSRT significantly predicted weight category. Initial SSRT and change in SSRT over treatment predicted change in BMI. Longer stay in camp = greater reduction in SSRT.

			Measures			Findings		
Author	**N[a]**	**Population**	**Measure of eating/ weight**	**SST main outcomes**	**SSRT calculation**	**Impact of eating/ weight on SST**	**Impact of inhibitory control on eating/weight**	**Associations between eating/weight and SSRT**
(Levitan et al., 2015)	193	Preschool children (90 f)	BMI-z scores, snack test: fat, carbohydrate and protein intake	logSSRT	Mean			Higher logSSRTs predicted higher BMI-z scores (females only), and carbohydrate and sugar intake (not total/protein intake).
(Lokken et al., 2009)	25	Extremely obese adolescents (15 f).	Weight status	SST z-score compared to the mean of a normative sample (n=79, 52 f)	NR	SST measures: Obese = Normative sample.		No correlation between BMI and SST measure.
(Nederkoorn et al., 2006a)	63	Obese children (n=32, 19 f [15 binge eaters, 10 f]) from a residential treatment centre for obesity, and normal weight children (n=31, 19 f) from a secondary school.	% weight loss	SSRT, mean RT	Mean [e]	SSRT: Obese (binge eaters = non-binge eaters)>Normal weight. Mean RT: Obese=Normal weight.		SSRT negatively correlated with % weight loss during treatment.

			Measures			Findings		
Author	**N[a]**	**Population**	**Measure of eating/ weight**	**SST main outcomes**	**SSRT calculation**	**Impact of eating/ weight on SST**	**Impact of inhibitory control on eating/weight**	**Associations between eating/weight and SSRT**
(Nederkoorn et al., 2006b)	25	Overweight children (17 f) receiving a behavioural treatment for overweight	% overweight, change in overweight	SSRT	Mean		Higher SST = higher % overweight and least weight lost (at all time-points).	SSRT predicted change in overweight after 12 months (after controlling for baseline overweight), but overweight did not predict weight change after controlling for SSRT.
(Nederkoorn et al., 2012)	89	Overweight (n=14, 11 f) and lean (n=75, 38 f) children	Weight status, food vs. neutral trials of the SST	SSRT	RTs ranked from fastest to slowest: nth reaction (where n= probability of responding given a stop signal.	SSRT: Overweight (categorical or continuous)>Lean. Group (categorical) *condition on SSRT: Overweight>SSRT on food but not neutral SST trials.		
(Verbeken et al., 2009)	81	Overweight (n=41, 25 f) children recruited from a paediatric centre for obesity treatment and lean children (n-=40, 22 f) recruited from schools	Weight status	SSRT, mean RT	Mean	SSRT: Overweight>Lean. Mean RT: Overweight = Lean.		
OTHER EATING BEHAVIOURS								
ADULTS								

			Measures			Findings		
Author	**N[a]**	**Population**	**Measure of eating/ weight**	**SST main outcomes**	**SSRT calculation**	**Impact of eating/ weight on SST**	**Impact of inhibitory control on eating/weight**	**Associations between eating/weight and SSRT**
(Dong et al., 2014) [b]	52	Female undergraduate students categorised as unrestrained eaters (URE: n=30) or restrained eaters (RE: n=22)	Eating restraint (RS)	SSRT, mean RT, correlations: regional homogeneity (ReHo)	Mean [e]	SSRT: RE>URE Mean RT: RE=URE		ReHo correlated with SSRT for REs only: positive correlation with left insula, negative correlation in left DLPFC.
(Jansen et al., 2009)	63	Female university students categorised according to high/low restraint (HR/LR, RS cut off score of 13) and high/low impulsive (HI/LI, SSRT median split): HR-HI (n=12), HR-LI (n=12), LR-HI (n=20), LR-LI (n=19)	Eating restraint (RS), Calorie intake (taste test), three food exposure manipulations: preload (2 milkshakes), exposure (smell of high caloric foods), control (Sensation seeking questionnaire)	SSRT	Mean [e]		Food intake: (Exposure=control) > preload for HR-HI only. Restraint correlated with caloric intake (all conditions) for participants with high (HI) but not low SSRT (LI).	

			Measures			Findings		
Author	**N[a]**	**Population**	**Measure of eating/ weight**	**SST main outcomes**	**SSRT calculation**	**Impact of eating/ weight on SST**	**Impact of inhibitory control on eating/weight**	**Associations between eating/weight and SSRT**
(Leitch et al., 2013)	75	Normal weight female students categorised on eating restraint (low/high restraint = LR/HR) and disinhibition (low/high disinhibition = LD/HD): HDHR (n=18), HDLR (n=20), LDHR (n=18), LDLR (n=19)	Eating restraint and disinhibition (TFEQ), eating condition (controlled/unrestricted eating instructions prior to task completion)	SSD	NA (no SSRT)	SSD: LR>HR, LD=HD, No restraint *disinhibition interaction, no impact of eating condition.		
(Meule et al., 2014) [c]	50	Normal weight female university students who were dieting (n=15) or not dieting (n=35)	Food/neutral of the SSRT, food craving (FCQ-S)	SSRT	SSRT= $RT_{(m)}$ – SSD, where m=*n* (number of responses in the go RT distribution) x $P_{(respond\|signal)}$	SST performance: Dieters = Non-dieters.	Mean RT: Food>Neutral. All other SST measures: Food = Neutral.	Mean RT and SSRT in food trials (but not neutral trials) positively predicted food craving scores (FCQ-S total scores and desire/lack of control subscale scores). BMI, dieting success, food addiction and food deprivation did not correlate with any SST measure.

			Measures			Findings		
Author	**N[a]**	**Population**	**Measure of eating/ weight**	**SST main outcomes**	**SSRT calculation**	**Impact of eating/ weight on SST**	**Impact of inhibitory control on eating/weight**	**Associations between eating/weight and SSRT**
(Nederkoorn et al., 2004)	56	Female undergraduate students categorised as restrained eaters (RE, n=31) and unrestrained eaters (URE, n=25)	Eating restraint (RS)	SSRT, mean RT, food exposure half-way through SST blocks	Mean [e]	SSRT: RE>URE (not influenced by food exposure). Mean RT: RE=URE.		
(van Strien et al., 2014)	54	Female university students high (n=29) or low (n=25) in emotional eating	Food intake (taste test), emotional eating (DEBQ), hunger	SSRT	Mean	SSRT: LEE=HEE.	No effect of SSRT on hunger or total food/snack intake. SSRT*emotional eating on food intake: emotional eating affected food/snack intake only in individuals with low SSRT.	SSRT did not correlate with emotional eating, or food/snack intake.
YOUTH								
(Hartmann et al., 2013)	88	Children with LOC eating (n=43, 14 f), children with ADHD (n=33, 11 f), and HC children (n=32, 18 f)	LOC eating	GRTV before and after negative mood induction	NA (no SSRT)	GRTV pre-mood induction: LOC = HC. GRTV: LOC: pre<post induction.		
HEALTHY INDIVIDUALS								
ADULTS								

			Measures			Findings		
Author	**N[a]**	**Population**	**Measure of eating/ weight**	**SST main outcomes**	**SSRT calculation**	**Impact of eating/ weight on SST**	**Impact of inhibitory control on eating/weight**	**Associations between eating/weight and SSRT**
(Fay et al., 2015)	50	50 adults who either participants initiated optional snacking ("initiators": n=38, 23 f) or not ("non-initiators": n=12, 5 f)	Initiation of opportunistic snacking in taste test	Stop accuracy (mean % correct inhibition on stop trials)	NA (no SSRT)	Stop accuracy: initiators>non-initiators.		
(Giesen et al., 2012)	70	Undergraduate students (53 f) categorised as high/low impulsive (HI/LI) determined by SSRT (median split), randomly assigned to a tax/subsidy condition: 15 Tax-LI, 16 Tax-HI, 20 Subsidy-LI, 19 Subsidy-HI	Difference in calories (total, high (HED)/medium (MED)/ low (LED) energy dense products) purchased in an internet supermarket task.	SSRT	Mean [e]		SSRT did not affect calories purchased. Tax reduced and subsidy increased total calories and HED products purchased by those with high SSRT, and LED products purchased by those with low SSRT.	
(Guerrieri et al., 2007a)	86	Female undergraduate students categorised as high/low impulsive by SSRT (median split)	Taste test: sugar beans, eating pathology (EDEQ, RS)	SSRT	Mean [e]		No effect of SSRT on food intake or eating pathology. SSRT did not interact with colour variety to influence food intake.	

			Measures			Findings		
Author	**N[a]**	**Population**	**Measure of eating/ weight**	**SST main outcomes**	**SSRT calculation**	**Impact of eating/ weight on SST**	**Impact of inhibitory control on eating/weight**	**Associations between eating/weight and SSRT**
(Guerrieri et al., 2007b)	38	Normal weight female undergraduate students	Food intake (taste test). Impulsivity priming: form impulsivity-related /neutral sentences.	SSRT	Mean [e]		SSRT did not interact with state impulsivity manipulation to influence food intake.	SSRT significantly correlated with total food intake and was a significant predictor of total food intake.
(Hall et al., 2015) Study 1	43	Older adults (29 f) (note: 47.6% were overweight or obese)	High calorie food consumption (taste test). Manipulated eating restraint: facilitation (no restriction), restriction (eat bare minimum to make ratings) or control (no specific instructions).	% accuracy	NA (no SSRT)		There was no effect of SST performance on snack food consumption in any of the manipulations (facilitation, restraint, control).	

			Measures			Findings		
Author	**N[a]**	**Population**	**Measure of eating/ weight**	**SST main outcomes**	**SSRT calculation**	**Impact of eating/ weight on SST**	**Impact of inhibitory control on eating/weight**	**Associations between eating/weight and SSRT**
(Haynes et al., 2015)	134	Female undergraduate students motivated to manage weight through healthy eating	Snack consumption (taste test). Evaluative conditioning intervention: Unhealthy food words (critical trials) and neutral words (filler trials) were paired with positive or negative images.	SSRT	Mean [e]		Inhibitory control moderated the effect of training on food intake, and this was mediated by temptation. Snack intake: negative<positive food pairing (for individuals with high SSRT only). Mediation: higher temptation = greater snack consumption.	No correlation between inhibitory control and BMI, hunger, temptation or snack consumption.
(Hermans et al., 2013)	85	Normal weight (n=75) and overweight (n=10) women who completed two study sessions: one on their own and one with an experimental confederate	confederate food intake (no/low/high intake), total food intake	SSRT	Mean [e]		No main effect of response inhibition or interaction with confederate intake on participants' food intake.	Behavioural impulsivity was not significantly correlated with food intake or BMI.

			Measures			Findings		
Author	**N[a]**	**Population**	**Measure of eating/ weight**	**SST main outcomes**	**SSRT calculation**	**Impact of eating/ weight on SST**	**Impact of inhibitory control on eating/weight**	**Associations between eating/weight and SSRT**
(Hofmann et al., 2009)	118	Healthy females	Candy consumption (taste test)	(Mean go RT - Mean SSD) * -1	NA (SSRT not explicitly reported: multiplied by -1 to provide an index of inhibitory control)		SSRT moderated the impact of automatic affective reactions (IAT) on candy consumption: High SSRT>low SSRT (impact of IAT).	
(Houben et al., 2012) [c]	50	Female participants assigned to a food exposure (n=26) or control (n=24) condition	PSRS (weight regulation success), RS, food/general SST, food intake (taste test)	SSRT	Mean [e]	SSRT: Exposure>control for unsuccessful regulators (not successful regulators) in the food-specific SST only.		Higher SSRT on the food-specific SST (but not the general SST) was a significant predictor of food intake.
(Lattimore and Mead, 2015)	50	Female participants who were categorised as low (LI: n=27) or high impulsive (HI: n=23) based on BIS-11 scores	Food cue exposure (LI: n=14, HI: n=11) vs control (LI: n=13, HI: n=12) completion of a filler task unrelated to food).	SSRT	Mean [e]		SSRT: No main effect of impulsivity or condition, but significant interaction: High impulsive: exposure>control Low impulsive: Exposure=control.	

			Measures			Findings		
Author	**N[a]**	**Population**	**Measure of eating/ weight**	**SST main outcomes**	**SSRT calculation**	**Impact of eating/ weight on SST**	**Impact of inhibitory control on eating/weight**	**Associations between eating/weight and SSRT**
(Lowe et al., 2014)	34	Undergraduate students (23 f) assigned to one of three exercise conditions (minimal, moderate, vigorous)	Food intake (taste test)	Stop accuracy (proportion of failed stops)	NA (no SSRT)		Stop accuracy did not interact with exercise condition to affect food intake.	Accurate inhibition was positively correlated with BMI, but not with food intake.
(Nederkoorn et al., 2009) Study 1	57	Female participants in a hunger (n=25) or sated (n=32) condition	Caloric intake (taste test)	SSRT	Mean [e]		Impulsivity interacted with state hunger, with participants who were both hungry and impulsive consuming the greatest number of calories.	SSRT positively correlated with and marginally predicted food intake.
(Nederkoorn et al., 2009) Study 2	94	Undergraduate students (77 f)	Total, snack and non-snack calories purchased from a virtual (internet) supermarket	SSRT	Mean [e]	SSRT: Overweight>Lean (trend)	Participants who were both hungry and impulsive purchased more snack calories, however hunger and impulsivity did not affect the total or non-snack calories purchased.	BMI and SSRT were not significantly correlated. SSRT positively correlated with intake of snack calories, but not total calories or non-snack calories.

			Measures			Findings		
Author	**N[a]**	**Population**	**Measure of eating/ weight**	**SST main outcomes**	**SSRT calculation**	**Impact of eating/ weight on SST**	**Impact of inhibitory control on eating/weight**	**Associations between eating/weight and SSRT**
(Nederkoorn et al., 2010)	51	Female undergraduate students	Weight change (over 1 year), SC-IAT (implicit preference for snack food)	SSRT	Mean [e]		Weight gain over a year: high SSRT>low SSRT (only for individuals with high implicit preferences for snack foods).	SSRT did not correlate with weight change, BMI or implicit preference for snack food.
(Papachristou et al., 2013)	50	Adult volunteers (39 f)	Acquisition and extinction of liking and craving of chocolate as a conditioned stimulus	SSRT	Mean [e]		No effect of SSRT on acquisition or extinction of a learned craving or liking response.	
(Wang et al., 2013)	60	Male university students	BMI	SSRT	Integration method: calculated using distribution of go RT and response probability for a given SSD			BMI was not associated with SSRT.
YOUTH								

			Measures			Findings		
Author	**N[a]**	**Population**	**Measure of eating/ weight**	**SST main outcomes**	**SSRT calculation**	**Impact of eating/ weight on SST**	**Impact of inhibitory control on eating/weight**	**Associations between eating/weight and SSRT**
(Nederkoorn et al., 2015)	88	Children categorised as high/low impulsive (HI/LI, median SSRT split), tested before or after lunch: LI-before (n=23, 17 f), LI-after (n=21, 13 f), HI-before (n=22, 16 f), HI-after (n=22, 11 f)	Taste test (low/medium /high energy dense foods)	SSRT	Mean		Significant interaction between SSRT and food type on food intake: HI>LI for HED food only. This was not influenced by time of eating.	No correlation between BMI z-score and impulsivity.

[a] Final number of participants included in the study
[b] Included neuroimaging (magnetic resonance imaging) measures.
[c] Included food-specific and neutral/general versions/trials of the SST.
[d] Gender ratio not provided after participants were excluded from the final analysis.
[e] Use of mean/median RT in the SSRT was not explicitly reported (often referred to as "reaction time"): cited other articles for method of calculation.
Abbreviations: f = number of females in sample. ED = eating disorder. BN = bulimia nervosa. EDNOS = eating disorder not otherwise specified. OCD = obsessive compulsive disorder. HC = healthy controls. SSRT = stop signal reaction time (calculated by subtracting the stop signal delay by mean or median go reaction time). RT = reaction time. % = percentage. SST = stop signal task. ANR = anorexia nervosa restrictive subtype. ANBP = anorexia nervosa binge-purge subtype. SSD = stop signal delay. R = restrictive subtype. BP = binge-purge subtype. AN = anorexia nervosa. SD = standard deviation. z-score = standardised score. BMI = body mass index. BED = binge eating disorder. EtOH = abstinent alcohol-dependent participants. c- = controls. Rec-AN = recovered from anorexia nervosa. fMRI = functional magnetic resonance imaging. mPFC = medial prefrontal cortex. DEBQ = Dutch Eating Behaviour Questionnaire. EDEQ = Eating Disorder Examination Questionnaire. dACC = dorsal anterior cingulate cortex. FG = frontal gyrus. PCC = posterior cingulate cortex. NR = not reported. SMA = supplementary motor area. IPC = inferior parietal cortex. vs = versus. * = interaction with. BMI-SDS = body mass index standard deviation scores. CANTAB = Cambridge Neuropsychological Test Automated Battery. logSSRT = log transformed stop signal reaction time. URE = unrestrained eater. RE = restrained eater. RS = Restraint Scale. ReHo = regional homogeneity. DLPFC = dorsolateral prefrontal cortex. HR = high restraint. LR = low restraint. HI = high impulsive. LI = low impulsive. LD = low disinhibition. HD = high disinhibition. TFEQ = Three-Factor Eating Questionnaire. NA = not available. FCQ-S = State Food Cravings Questionnaire. LEE = low emotional eating. HEE = high emotional eating. LOC = loss of control. GRTV = go reaction time variability. HED = high energy dense. MED = medium energy dense. LED = low energy dense. PSRS = perceived self-regulatory success. BIS-11 = Barratt's Impulsiveness Scale. SC-IAT = The Single Category Implicit Association Test.

Table 2. Studies assessing the impact of SST manipulations on eating and weight

Author	N[a]	Population	Manipulation	SSRT calculation (mean/ median)	Main outcomes	Findings
WEIGHT STATUS						
YOUTH						
(Verbeken et al., 2013)	44 [b]	Overweight children in an inpatient treatment program (20 f) assigned to care as usual (baseline: n=22, 9 f; 8-weeks post-treatment: n=15; 12-weeks post-treatment: n=18) or care as usual plus executive function training (baseline: n=22, 11 f; 8-weeks post-treatment: n=18; 12-weeks post-treatment: n=18)	Care as usual (CAU) vs. CAU + executive function training (CAU-EF: inhibition training (SST) and working memory training).	NR	Executive function and weight loss maintenance	Baseline SSRT, change in SSRT: CAU-EF = CAU Weight loss maintenance (8 weeks post-treatment): CAU-EF>CAU
OTHER EATING BEHAVIOURS						
ADULTS						
(Guerrieri et al., 2009) Study 2	66	Female undergraduate students: 15 current dieters (CD), 25 low restrained non-dieters (LRND), 26 high restrained non-dieters (HRND)	Manipulated SST instructions to promote impulsivity (prioritise go RT) or inhibition (prioritise accurate stopping).	Mean [c]	Caloric intake	Caloric intake: No main effect of SST condition but interaction with dieting status: Impulsivity>Inhibition for HRND and LRND Inhibition>Impulsivity for CD.
(Houben and Jansen, 2014)	35	Female participants randomly assigned to one of two conditions: reward (n=16) or control (n=19)	Manipulated SST instructions: Reward condition (participants rewarded for accurate performance [fast RT, correct stops]) or control condition (no additional instructions).	Mean [c]	Taste test (energy intake = weight*caloric density), SSRT	SSRT: RE: Reward>Control URE: Reward = Control Food intake and craving: RE: Reward>Control URE: Control>Reward
HEALTHY INDIVIDUALS						

Author	N[a]	Population	Manipulation	SSRT calculation (mean/ median)	Main outcomes	Findings
ADULTS						
(Allom and Mullan, 2015) Study 1	82 [b]	Undergraduate students (66 f) assigned to one of three conditions: food specific inhibition (pre-intervention: n=29; post-intervention: n=26), general inhibition (pre intervention: n=25, post-intervention: n=21), and control (pre-intervention: n=28, post-intervention: n=25).	Participants completed one of three SSTs daily for 10 days: food-specific inhibition (stop trials only presented after unhealthy food images), general inhibition (stop signal presented randomly either after healthy/unhealthy food), or control (no stop signals presented).	NR	Block food screener (saturated fat intake), BMI	Saturated fat intake: No main effect of condition or time, no significant interaction. BMI: Pre>Post (food-specific inhibition intervention only).
(Allom and Mullan, 2015) Study 2	78 [b]	University staff and students (61 f) assigned to one of three conditions: food specific inhibition (pre-intervention: n=27; post-intervention: n=24), general inhibition (pre intervention: n=26, post-intervention: n=23), and control (pre-intervention: n=25, post-intervention: n=23).	Participants completed one of three SSTs daily for 10 days: food-specific inhibition (stop trials only presented after unhealthy food images), general inhibition (stop signal presented randomly either after healthy/unhealthy food), or control (no stop signals presented).	NR	BMI, % daily energy intake from fat (NCI screener)	BMI, % energy from fat: No main effects of time or condition, no significant interaction.
(Guerrieri et al., 2012)	61	Normal weight female undergraduate students assigned to one of three conditions: inhibition (n=21), impulsivity (n=20) and control (n=20)	Changed the proportion of stop trials across SST blocks to promote inhibition (increasing proportion of stop trials) or impulsivity (decreasing proportion of stop trials). The control group completed a neutral task (read and summarised text) but no SST.	NR	Calorie intake (taste test)	Calorie intake: Impulsivity > (Inhibition = Control)
(Houben, 2011)	29	Female undergraduate students (note: 17.2% of sample were	3 SST conditions (within-subjects design): inhibition (one food type always paired with	Mean [c]	Taste test (energy intake =	Energy intake: No main effect of SSRT or SST

Author	N[a]	Population	Manipulation	SSRT calculation (mean/ median)	Main outcomes	Findings
		overweight)	stop signal), impulsivity (one food type was never paired with stop signal) and control condition (third type of food was presented with stop signal on half the trials).		weight*caloric density)	condition but significant interaction: High SSRT: stop food<control food Low SSRT: (trend) go food>control food Control food: High SSRT>Low SSRT
(Lawrence et al., 2015b) Study 1	54	University staff and students (32 f) semi-randomly assigned to either the stop group (n=29, 18 f) or double-response group (n=25, 14 f)	Modified SST instructions to either withhold responses (stop-group) or provide an extra response (double-response group) on signal trials. Stimuli included food/neutral images, and signal trials occurred predominantly on food trials.	NR	Calorie consumption (crisps)	Calorie intake: Double response > Stop group (this was not influenced by self-reported dietary restraint). No association between calorie consumption and overall or improvement in accuracy in food-stop trials.
(Lawrence et al., 2015b) Study 2	136	University staff and students (100 f) semi-randomly assigned to the stop group (n=44, 33 f), double-response group (n=46, 33 f) or ignore group (n=46, 34 f).	Modified SST instructions to either withhold responses (stop-group), provide an extra response (double-response group), or respond as usual (ignore group) on signal trials. Signals appeared equally on trials of images of food and neutral items. One food was nearly always associated with the signal (signal food) while another was rarely associated with signals (no-signal food).	NR	Intake of signal and no-signal food	Food intake: No main effect of training, food type or interaction. Training interacted with dietary restraint: Stop food: Stop group < Double-response group (only for individuals with high dietary restraint). No correlation between food intake and overall/improvement in stop accuracy on food trials.
(Lawrence et al., 2015b) Study 3	146	University staff and students (111 f) semi-randomly assigned to a stimulus-specific stop group (n=47, 33 f), a stimulus-	Modified SST instructions to either withhold responses (stop-group), provide an extra response (double-response group) on stimulus-specific signal trials, or withhold a	NR	Chocolate and crisp consumption	Food intake: No main effect of training. Absence of training effect was not influenced by dietary restraint.

Author	N[a]	Population	Manipulation	SSRT calculation (mean/ median)	Main outcomes	Findings
		specific double-response group (n=51, 39 f) or a general stop group (n=46, 34 f).	response on a general SST (general stop group; no association between pictures and stop signals). In the stimulus-specific conditions, one neutral stimulus category was almost always associated with the signal, whereas another neutral stimulus category was rarely associated with the signal.			
(Sellitto and di Pellegrino, 2014)	40	Young adult females	Two SST conditions: One food rated by the participant as high in wanting was associated with low error likelihood (LEF; SSD varied to enable ~15% errors) and another highly-wanted food associated with high error likelihood (HEF; ~50% errors).	NR	Food temporal discounting task (preference between smaller number of bites now or larger number of bites later of a hypothetical food).	Impulsive choices (preference for smaller sooner): LEF>HEF (only for participants low in hunger)

[a] Final number of participants included in the study (at the study baseline assessment)

[b] Gender ratio not reported for follow-up assessments.

[c] Use of mean/median RT in the SSRT was not explicitly reported: cited other articles for method of calculation.

f = number of females in sample. SSRT = stop signal reaction time (calculated by subtracting the stop signal delay by mean or median go reaction time). RT = reaction time. SST = stop signal task. CAU = care as usual. CAU-EF = care as usual with executive function training. CD = current dieters. LRND = low restrained non-dieters. HRND = high restrained non-dieters. RE = restrained eaters. URE = unrestrained eaters. * = interaction with. NR = not reported. BMI = body mass index (kg/m^2). % = percentage. NCI screener =. LEF = food associated with low error likelihood. HEF = food associated with high error likelihood.