



## Research report

# Inducing impulsivity leads high and low restrained eaters into overeating, whereas current dieters stick to their diet

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## ABSTRACT

Previous research has related impulsivity to overeating and obesity. However, the precise nature of this relation has not been examined yet. One possibility is that impulsivity causes overeating and hence contributes to overweight. To test this possibility we induced impulsivity versus inhibition to see whether this would affect food intake. In the first study participants were cognitively primed with the concepts “impulsivity” or “inhibition”. Caloric intake was significantly higher in the Impulsivity Condition compared to the Inhibition Condition. This effect was even stronger for highly restrained participants. In the second study impulsivity was manipulated via behavioural instructions. Restrained and unrestrained nondieters acted as expected: their caloric intake was significantly higher when impulsivity was induced compared to inhibition. Current dieters sharply reduced their caloric intake following the impulsivity induction. These results are in accordance with Lowe's model that, contrary to restraint theory, states that restraint and current dieting are different constructs that affect eating regulation differently. At least for nondieters it can be concluded that heightened impulsivity versus inhibition leads to a higher food intake in the lab.

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## Introduction

Impulsivity is a personality trait that has been found a causal and/or maintaining factor in numerous psychopathologies and behavioural disorders, ranging from addiction (Moeller & Dougherty, 2002) to AD/HD (Solanto et al., 2001). In general, impulsivity is defined as the tendency to think, control and plan insufficiently. In most cases this results in an inaccurate or maladaptive response (Solanto et al., 2001). Impulsivity is thought to be a multi-dimensional concept, so precise behaviours that are considered impulsive are very diverse. Logan, Schachar, and Tannock (1997), for example, see impulsivity as the diminished ability or inability to inhibit a prepotent or predominant response. Impulsivity thus results from weakened inhibition. Others (e.g. Gray, 1970) stress a greater likelihood of approach behaviour towards rewarding stimuli in impulsive individuals. Impulsivity has also been linked to major personality systems. Eysenck (Eysenck, Pearson, Easting, & Allsop, 1985), for example, sees impulsivity as a combination of impulsiveness (a lower order trait of psychoticism) and venturesomeness (a lower order trait of extraversion).

Recently, the quest for causal and maintaining factors of obesity has uncovered a link between certain aspects of impulsivity, overeating and obesity in populations that typically overeat, i.e. the obese and Bulimia Nervosa patients, and in populations that are typically impulsive, i.e. Attention Deficit Hyperactivity Disorder Patients (for an overview, see Guerrieri, Nederkoorn, & Jansen, 2008).

It has been shown that even in healthy, lean participants impulsivity is of importance when it comes to food. Healthy individuals who are more sensitive to reward according to a self-report questionnaire turned out to have more pronounced neural responses to images of appetizing food (Beaver et al., 2006). This could indicate that for high-impulsive people it is harder to resist food than for low-impulsive people. Indeed, Guerrieri, Nederkoorn, and Jansen (2007) found that high-impulsive, but normal-weight, healthy women ate significantly more in the lab compared to low-impulsive women. Guerrieri et al. (2007) demonstrated that impulsivity, measured by both a behavioural task and self-report, predicted a heightened cumulated food intake of three taste tests, spread over three days.

What these and most other studies on this topic have in common is that variables in the participants are observed in order to look for relationships between these variables. These kinds of studies cannot help us establish whether a causal link exists between impulsivity, overeating and obesity.

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In order to investigate whether impulsivity *causes* overeating, one needs to experimentally induce impulsivity and see whether this manipulation leads to a higher food intake compared to a control group. Something similar has been done by Rotenberg et al. (2005). They primed “lack of control” thoughts and these thoughts did indeed lead to greater food intake compared to priming “control” thoughts. Lack of control might be different from impulsivity in the sense that it puts more emphasis on the cognition of powerlessness, whereas impulsivity might be more related to behavioural activating. However, the concepts do show a great overlap and the results of the study do support the view that induced impulsivity might cause overeating, compared to induced control. The study of Guerrieri et al. (2007) also attempted to prime the concept of impulsivity in order to affect food intake in the lab. Although the priming group did report higher craving for the food, no differences in food intake between the priming group and a control group were found and it was concluded that the manipulation might not have lasted long enough. Thus, although there are indications that impulsivity might be a cause of overeating, solid proof is still lacking.

Another variable that is widely known and used in eating behaviour research is restraint (Herman & Polivy, 1980). Restrained eaters, identified by the Restraint Scale, are believed to be (chronic) dieters or weight suppressors (Herman & Polivy, 1980). It has been shown repeatedly that, compared to unrestrained eaters, restrained eaters consume *more* food after having consumed a high-caloric preload. This is called “counterregulation” (Herman & Polivy, 1980). This counterregulation is thought to occur because the consumption of the preload acts as a disinhibitor: it removes inhibitions and eliminates restraint. It turned out that it is not even necessary to consume food in order to disinhibit restrained eaters. Mere exposure to food stimuli is sufficient to break down dietary restraint (Jansen & van den Hout, 1991; Rogers & Hill, 1989). In other studies participants who were identified as restrained eaters were found to eat more than unrestrained eaters when they were given ad libitum access to palatable food, not preceded by a preload or cue exposure (e.g. Jansen, 1996).

Restraint theory implies that “restrained eating” and “dieting” can be used interchangeably (Herman & Polivy, 1980). More recently, Lowe (Lowe, Whitlow, & Bellwoar, 1991; Lowe, 1993) has postulated a multifactorial model in which restrained eating and dieting are different factors that have different effects on eating behaviour. The first factor describes the frequency of dieting and overeating in the past, with or without actual weight changes (Lowe, 1993). The second factor reflects whether a person is currently making an effort to restrict calories in order to lose weight. It does not matter what kind of diet is undertaken, or whether the person actually loses weight (Lowe, 1993).

We present two studies. The aim of these studies was mainly to influence food intake by inducing impulsivity, via a cognitive method (priming) and via a behavioural method (training). In the first study impulsivity as measured by certain self-report questionnaires was manipulated, whereas impulsivity as insufficient response inhibition was manipulated in the second study. Based on the findings of a previous study (Guerrieri et al., 2007) we expect both manipulations to influence food intake. This would empirically support the possibility that impulsivity is causally linked to overeating. Moreover, in the first study restraint (Herman & Polivy, 1980) was measured as a potential moderating factor. In the second study both restraint (Herman & Polivy, 1980) and current dieting (Lowe, 1993) were measured as potential moderating factors.

## Study 1

### Introduction

This study had two aims. First, we wanted to demonstrate that impulsivity is causally linked to overeating. For this purpose we used a cognitive priming task. Priming is a frequently used method in social cognition research. It is applied to enhance the cognitive availability of a psychological construct without conscious awareness. Research has shown that priming procedures affect people’s judgments and behaviour (Bargh, Gollwitzer, Lee-Chai, Barndollar, & Trötschel, 2001; Higgins, Rholes, & Jones, 1977). In the current study priming was used to manipulate the accessibility of the “impulsivity” construct for half of the participants (Impulsivity Condition) and the “inhibition” construct for the rest of the participants (Inhibition Condition). After this priming task participants did a bogus taste test. It was hypothesized that participants in the Impulsivity Condition would have a significantly higher caloric intake compared to the participants in the Inhibition Condition.

Second, it was hypothesized that the effect of impulsivity on overeating would be moderated by restraint (Herman & Polivy, 1980). Since the priming of impulsivity should bring participants in an impulsive, disinhibited state, it should have the same effect on eating regulation as a classic disinhibitor: a preload. Consequently, we expect an interaction between participants’ restraint status and the condition to which they are assigned (Impulsivity versus Inhibition). The restrained eaters in the Impulsivity Condition are expected to be extra vulnerable to overeating.

In summary, we had two hypotheses (1) the participants primed with “impulsivity” will consume more calories during a bogus taste test than the participants that were primed with “inhibition” and (2) the effect of impulsivity on overeating will be moderated by restraint status, meaning that in particular the restrained participants in the Impulsivity Condition will overeat.

### Methods

#### Participants

Forty-six female undergraduate students were recruited to participate in a study on “memory and taste perception” (mean age  $20.4 \pm 2$ ; mean BMI  $21.99 \pm 2.44$ ). All participants were tested individually and received course credit or a monetary reward of €7.50 for their participation. The procedure of the study was approved by the Ethical Committee of Maastricht University.

#### Materials

The *Priming Task* was presented to the participants in the form of a memory task. Both groups of participants read a story about New Year’s Resolutions. The participants were told that they would have to answer questions about the story at the end of the experiment. They were also told that it would help their memory if they imagined that they were the main character in the story. The Inhibition Condition received a story in which Andrea talks about her New Year’s resolutions: she will start studying for her exams in time and attend all lectures and she will save money regularly so that she will be able to go on that trip to Italy next summer. The Impulsivity Condition received a story in which Andrea says that one should not think too much about the future. Flexibility and spontaneity are more important. Andrea’s only New Year’s resolution is to enjoy life. In both stories, food, eating or drinking were not mentioned. The themes in the stories are believed to reflect impulsivity because they were based on items of two widely used impulsivity questionnaires: the Barratt Impulsiveness Scale (BIS; Patton, Stanford, & Barratt, 1995) and Eysenck’s  $I_7$  (Eysenck et al., 1985). The items used for the inhibition story were: “I am

restless at plays or lectures" (BIS item 28; item was inverted), "I save regularly" (BIS item 10), "I plan trips well ahead of time" (BIS item 7). The items used for the impulsivity story were: "I am more interested in the present than in the future" (BIS item 27), "I act on the spur of the moment" (BIS item 20), "An evening out is more successful if it is unplanned or arranged at the last moment" (I-7 item 38).

A *Bogus Taste Test* was used in order to measure food intake of the participants in an unobtrusive way. Participants were left alone for 15 min to taste and rate four sorts of food that were placed in front of them in large bowls: chocolate ( $\pm 1100$  g), wine gums ( $\pm 1500$  g), marshmallows ( $\pm 1000$  g) and a savoury nut mix ( $\pm 1100$  g). Participants had to rate odour, general palatability and, depending on the sort of food, sweetness, creaminess, saltiness and crunchiness. However, the primary interest of the authors was not how participants rated the taste of the food, but how much they ingested of the food that was offered. Without the participants' knowledge the bowls of food were weighed before and after the taste test in order to establish food intake. The amounts eaten of each food were converted to calories. The sum of these calories was the dependent variable: total caloric intake.

The *Restraint Scale* (RS; Herman & Polivy, 1980) collects information on attitudes towards weight, degree and frequency of dieting, loss of control overeating and weight fluctuations. Scores range from 0 to 40. The higher the RS score, the more restrained a person is believed to be.

#### Procedure

At recruitment participants were asked to eat something small like a cheese sandwich 2 h before the start of the experiment and to otherwise refrain from eating and drinking (except water). This was done in order to control for differing states of hunger between the conditions at the start of the experiment.

At their arrival in the lab, participants started with the "memory task", which was actually the priming task. Reading the story took about 1 min. Afterwards participants did the taste test. After the taste test the participants had to answer some questions on the priming story in order to confirm the cover story. Participants reported their height and weight, and they filled out the Restraint Scale. It was also checked whether participants had any relevant knowledge concerning the precise aims of the study. Afterwards, the participants were thanked and dismissed.

#### Results

##### Pre-existing differences

Independent-samples *t*-tests indicated that the Impulsivity Condition and the Inhibition Condition did not differ significantly in age,  $20.70 \pm 2.30$  years versus  $20.10 \pm 2$  years,  $t(44) = -0.98$ ,  $p > 0.3$ , and BMI,  $22.54 \pm 2.80$  versus  $21.38 \pm 1.86$ ,  $t(44) = 1.63$ ,  $p > 0.10$ .

Likewise age,  $20.67 \pm 2.62$  years versus  $20.14 \pm 1.46$  years,  $t(44) = -0.86$ ,  $p \geq 0.4$ , did not differ between high and low-restrained participants, but BMI did,  $22.76 \pm 2.66$  versus  $21.13 \pm 1.87$ ,  $t(44) = 2.43$ ,  $p < 0.05$ .

##### Effect of priming and restraint on caloric intake

The participants were divided into a restrained and an unrestrained group via a median split. The participants that scored below the median RS score 11 (a normal median for a Dutch sample) were considered to be unrestrained ( $n = 22$ ) whereas participants with an RS score of 11 or more ( $n = 24$ ) were considered to be restrained. Unrestrained participants had a mean RS score of  $7.91 \pm 2$ , whereas restrained participants had a mean RS score of  $14.33 \pm 3.1$ . An independent-samples *t*-test demonstrated that the two groups indeed differed significantly in mean RS score,  $t(44) = 8.35$ ,  $p < 0.001$ .

A 2 (Impulsivity versus Inhibition) by 2 (restrained versus unrestrained) between-subjects ANOVA with caloric intake as the dependent variable was conducted. Because the high restrained participants had a significantly higher BMI compared to the low-restrained participants, BMI was included as a covariate. BMI proved to be significant as a covariate,  $F(1, 41) = 5.32$ ,  $p < 0.05$ , partial  $\eta^2 = 0.12$ . The main effects of condition,  $F(1, 41) = 10.76$ ,  $p < 0.01$ , partial  $\eta^2 = 0.21$ , and restraint,  $F(1, 41) = 13.48$ ,  $p \leq 0.001$ , partial  $\eta^2 = 0.25$ , were significant. Caloric intake was on average higher in the Impulsivity Condition ( $432 \pm 29$  kcal) than in the Inhibition Condition ( $287.86 \pm 32$  kcal). Restrained eaters ( $443.76 \pm 30$  kcal) consumed significantly more calories than their unrestrained counterparts ( $276.61 \pm 32$  kcal). All means reported are Estimated Marginal Means  $\pm$  Standard Error of Mean. The interaction effect between impulsivity induction and restraint was marginally significant,  $F(1, 41) = 3.87$ ,  $p < 0.06$ , partial  $\eta^2 = 0.09$ . For each restraint status (restrained versus unrestrained) an ANOVA was conducted with condition as a factor and BMI as a covariate. These post hoc tests revealed that in for unrestrained participants there was no difference in caloric intake between the Inhibition Condition and the Impulsivity Condition,  $F(1, 19) = 1.5$ ,  $p > 0.2$ , partial  $\eta^2 = 0.08$ . However, the restrained women in the Impulsivity Condition ingested significantly more calories compared to the restrained women in the Inhibition Condition,  $F(1, 21) = 10.72$ ,  $p < 0.01$ , partial  $\eta^2 = 0.34$ . See Fig. 1 for an Estimated Marginal Means plot of the interaction effect between condition and restraint status.

#### Discussion

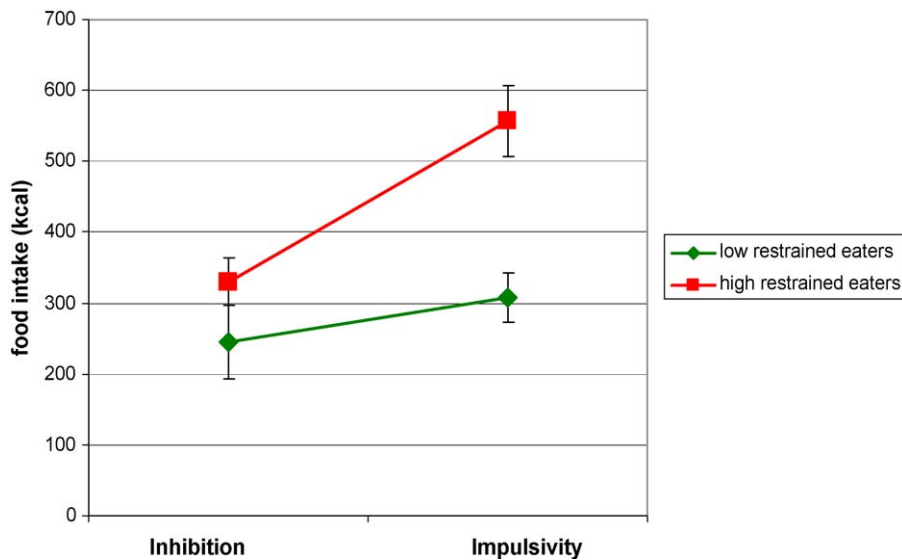
As predicted, priming healthy participants with "impulsivity" led them to ingest significantly more calories compared to priming them with "inhibition". In other words, the hypothesis that impulsivity is causally linked to overeating was supported. To the authors' knowledge this is the first time that this hypothesis was explicitly tested and supported. This means that replication of the results, preferably with different methods and/or in different populations is necessary. For this reason we decided to conduct a second study. In this study impulsivity was not manipulated by priming cognitions, but by a task that made the participants behave in an impulsive or controlling manner.

Also as expected, the effect of the impulsivity priming on caloric intake was moderated by restraint: the restrained participants in the Impulsivity Condition had the highest caloric intake. This outcome seems to support the notion that restraint is indeed an important variable to consider when it comes to eating regulation in healthy participants. In this case being restrained makes the participants sensitive to the impulsivity priming. This makes perfect sense in restraint theory, since bringing participants in an impulsive, disinhibited mood will make it more difficult for them to stick to their weight loss aspirations when confronted with tasty food.

A factor that was not taken into account in this study is current dieting. According to restraint theory "restrained eating" and "dieting" are essentially the same. However, Lowe (Lowe et al., 1991; Lowe, 1995) found that restrained eating and dieting have opposite effects on eating regulation in response to a disinhibitor. In the second study both restraint and current dieting were measured.

#### Study 2

In this study we tried to manipulate impulsivity through a behavioural task, instead of using a task to prime cognitions. We used a task that is normally used to measure impulsivity, the Stop Signal Task (SST; Logan et al., 1997), in order to train healthy participants to react in an impulsive ("Impulsivity Condition")



**Fig. 1.** Estimated Marginal Means plot of the interaction between condition (inhibition versus impulsivity) and restraint status (low restrained versus high restrained) on caloric intake,  $F(1, 41) = 3.87$ ,  $p < 0.06$ . Means are  $\pm$  SEM.

versus an inhibited (“Inhibition Condition”) manner. The advantages of this method are twofold. First, because no cognitions are used (like “enjoy life as much as possible”), possible demand characteristics are minimized. Second, the induction is closely related to a measurement of impulsivity, which has previously been linked to overeating and obesity (Guerrieri et al., 2007; Nederkoorn, Braet, Van Eijs, Tanghe, & Jansen, 2006). Thus, the relevant aspects of the broad domain of impulsivity are manipulated.

It was hypothesized that the participants in the Impulsivity Condition would eat more during a bogus taste test compared to the participants in the Inhibition Condition. Furthermore, as a manipulation check, it was expected that the participants in the Impulsivity Condition would score higher on an impulsive state measure compared to the Inhibition Condition. Other mood states such as anger, tension, depression and fatigue were expected not to differ between conditions.

In the study that was reported earlier the effect of restraint was examined. Supporting the division of dieting behaviour into restrained eating and current dieting, Lowe et al. (1991) and Lowe (1995) found that current dieters (CDs), unrestrained nondieters (UNDs) and restrained nondieters (RNDs) react differently to a disinhibitor in the form of a preload. Current dieters ate significantly more than restrained nondieters and unrestrained nondieters when not preloaded, whereas they sharply reduced their intake after a preload (Lowe et al., 1991). In the current study restraint and current dieting status were both measured. The impulsivity manipulation should bring participants in an impulsive, disinhibited state. Consequently, it should serve as a disinhibitor and have the same effect on eating regulation as the classic disinhibitor, a preload. If one sees the impulsivity manipulation in this study as a disinhibitor, then restraint theory’s predictions differ from Lowe’s. According to restraint theory both current dieters and high restrained nondieters should overeat in response to the disinhibitor, whereas low restrained nondieters should not. Based on the findings of Lowe et al. (1991) we would predict that current dieters should eat more than high and low restrained nondieters when not disinhibited. However, following the impulsivity induction current dieters should sharply reduce their intake.

In sum, this study looks at the effect of induced inhibition versus impulsivity and of dieting status on food intake. It is hypothesized that participants’ dieting status will moderate their

reaction to the behavioural priming of impulsivity or inhibition. Restraint theory and Lowe’s model differ in their predictions on the precise form of this interaction effect.

#### Methods

##### Participants

Sixty-six female undergraduate students (mean age: 20.8 years  $\pm$  2.64; mean BMI 22.32  $\pm$  2.55) were recruited to participate in a study on “cognitive performance and taste perception”. See Participants section in study 1 for further information.

##### Materials

The *Hunger Scale* contained four questions. Two questions were answered on a 100 mm VAS scale: “How hungry are you at the moment?” and “How much of your favourite food would you be able to eat at the moment?”. The sum of the scores on these questions was used to calculate a hunger score. The last two items were open questions: “What time did you last eat?” and “When do you next expect to eat?”.

The *Stop Signal Task* (SST; Logan et al., 1997) is a behavioural task that is normally used to measure impulsive behaviour in the form of insufficient response inhibition. In this study the Stop Signal Task served as a way to induce impulsivity versus inhibition. The SST consists of two concurrent, opposite tasks. First, participants are asked to push a button as quickly as possible in response to a stimulus (choice reaction time task). Second, it is their task to inhibit this response when they hear a tone that serves as a stop signal (inhibition task). It was our intention to mainly focus the participants’ attention on one of these two tasks. It was explained to all participants that the Stop Signal Task consists of two concurrent tasks. However, in the Impulsivity Condition, participants were told that the choice reaction time task was the most important task. In other words, for these participants responding as quickly as possible took precedence over inhibiting their response in reaction to a stop signal. This focus on reacting fast at the cost of inhibition was thought to bring the participants in an impulsive state. In the Inhibition Condition, participants were told that the inhibition task was the most important task. In other words, for these participants inhibiting their response took precedence over responding as quickly as possible. This focus on inhibiting at the cost of fast responses was thought to bring the participants in an inhibitive state. The Stop Signal Task was presented in four blocks of

96 trials which took about 20 min in total. Two variables are measured: go reaction time (RT) and stop delay. The main variable, stop signal reaction time (SSRT), is calculated by subtracting the stop delay from the go reaction time (Logan et al., 1997).

A *Bogus Taste Test* was used in order to measure food intake of the participants in an unobtrusive way. The test was conducted the same way as in study 1, but different food was used: salted peanuts ( $\pm 1450$  g), M&M's without peanuts ( $\pm 1800$  g), paprika flavoured crisps ( $\pm 850$  g) and marshmallows ( $\pm 950$  g).

The *Short Form of the Profile of Mood States* (POMS-SF; Curran, Andrykowski, & Studts, 1995; Schacham, 1983) is a 37-item questionnaire that aims to measure six kinds of moods on a five-point scale: Tension-Anxiety, Anger-Hostility, Depression-Dejection, Vigor-Activity, Fatigue-Inertia and Confusion-Bewilderment. In this study the Dutch translation of the POMS-SF (Wald & Mellenbergh, 1990) was used. This translation is shorter with 32 items that measure five moods (Confusion-Bewilderment was left out). Four items, chosen by the authors, were added to the POMS-SF. These items were thought to measure an "impulsive mood" and formed the subscale IMP. The items were: unconstrained, impulsive, relaxed, and uncontrolled.

The *Barratt Impulsiveness Scale* (BIS; Patton et al., 1995) is a self-report questionnaire that measures trait impulsiveness. It consists of 30 items to be rated on a four-point scale. Scores range from 30 to 120 with higher scores indicating more impulsiveness. The BIS consists of three subscales: motor impulsiveness (acting without thinking), attentional impulsiveness (not focusing on the task at hand, cognitive instability) and nonplanning impulsiveness (lack of orientation to the future).

#### Procedure

At recruitment participants received the same instructions as the participants in study 1.

At their arrival in the lab, participants started out by filling in the Hunger Scale. Then participants proceeded with the Stop Signal Task. Both conditions of participants were confronted with the exact same task, but the instructions that each condition received differed. In the Impulsivity Condition the participants were told that reacting as fast as possible during the go trials was more important than inhibiting their reaction during stop trials. In the Inhibition Condition the participants were told the opposite: being able to inhibit their reaction during stop trials took precedence over reacting as fast as possible during go trials. After the Stop Signal Task, which lasted approximately 20 min, participants did the taste test. After the taste test the participants filled in the POMS-SF. They also reported their height and weight and they indicated whether they were on a diet or not. It was also checked whether participants had any relevant knowledge concerning the precise aims of the study. Afterwards, the participants were thanked and dismissed.

The Restraint Scale (RS) was not administered during the study. The RS scores were extracted from data that had been collected during a large university screening session about six months earlier that year.

The Barratt Impulsiveness Scale (BIS), which measures trait impulsivity, was sent to the participants by e-mail after they had been tested. Participants were asked to fill it in at home and to send it back by e-mail. The BIS was not administered at the end of the experimental session because the impulsivity/inhibition induction might otherwise have distorted the participants' trait impulsivity score.

#### Results

##### Participant characteristics

Participants were divided into three dieting groups based on Lowe (1995). Those that claimed on the day of testing that they

**Table 1**

Means and standard deviations for relevant variables in Inhibition versus Impulsivity Condition in study 2.

	Inhibition		Impulsivity		p
	M	SD	M	SD	
Hunger	10.88	3.66	10.68	4.15	0.85
BMI	22.15	2.07	22.59	2.91	0.48
Age	20.85	2.80	20.69	2.62	0.82
Trait Imp (BIS)	66.47	11.93	62.65	10.12	0.25

were on a diet, were labeled "current dieters" (CDs;  $n = 15$ ). The nondieting participants that scored below the median RS score 12 (a normal median for a Dutch sample) were labeled "low restrained nondieters" (LRNDs;  $n = 25$ ), whereas those that scored 12 or more, were called "high restrained nondieters" (HRNDs;  $n = 26$ ). Low restrained nondieters had a mean RS score of  $7.44 \pm 3.3$ , whereas high restrained nondieters had a mean RS score of  $16.81 \pm 4.3$ . The current dieters had a mean RS score of  $15.78 \pm 4.6$ . Independent-samples *t*-tests demonstrated that the mean RS score of the low restrained nondieters was significantly lower than the mean RS score of the high restrained nondieters and the current dieters,  $t(49) = 8.7$ ,  $p < 0.001$ ,  $t(37) = 6.58$ ,  $p < 0.001$ .

The restraint scores of high restrained nondieters did not differ significantly of those of the current dieters,  $t(38) = 0.7$ ,  $p > 0.4$ .

##### Pre-existing differences

Independent-samples *t*-tests indicated that there were no significant differences in hunger,  $t(65) = 0.20$ ,  $p > 0.8$ , age,  $t(61) = 0.23$ ,  $p > 0.8$ , BMI,  $t(65) = 0.71$ ,  $p > 0.4$ , and trait impulsivity (BIS scores),  $t(43) = 1.16$ ,  $p > 0.2$  between the Impulsivity Condition and the Inhibition Condition. See Table 1 for descriptives.

One-way ANOVAs demonstrated that low restrained nondieters, high restrained nondieters and current dieters did not differ in hunger,  $F(2, 63) = 1.40$ ,  $p > 0.2$ , age,  $F(2, 59) = 0.02$ ,  $p > 0.9$ , and trait impulsivity scores,  $F(2, 41) = 0.84$ ,  $p > 0.4$ . BMI did differ  $F(2, 63) = 4.25$ ,  $p < 0.05$ . Post hoc analyses revealed that the BMI of the low restrained nondieters differed significantly from that of the high restrained nondieters,  $t(49) = 2.23$ ,  $p < 0.05$ , and the current dieters,  $t(38) = 2.61$ ,  $p < 0.05$ . High restrained nondieters' and current dieters' BMI did not differ significantly,  $t(39) = 0.96$ ,  $p > 0.3$ . See Table 2 for descriptives.

##### Adherence to the instructions that participants received

Independent-samples *t*-tests indicated that the participants in the Impulsivity Condition reacted significantly faster to the go trials compared to the Inhibition Condition (Mean Reaction Time:  $441.61 \pm 134.78$  ms for the Impulsivity Condition versus  $640.53 \pm 167.54$  ms for the Inhibition Condition;  $t(59) = 5.14$ ,  $p < 0.001$ ). The participants in the Inhibition Condition had a larger percentage of successful inhibitions during stop trials compared to the Impulsivity Condition, 62% versus 48%,  $t(64) = 3.90$ ,  $p < 0.001$ . This means that participants in both conditions did what they were asked to do: react as fast as possible for the Impulsivity Condition and inhibit as often as possible for the Inhibition Condition.

**Table 2**

Means and standard deviations for relevant variables for low restrained nondieters (LRNDs), high restrained nondieters (HRNDs), and current dieters (CDs).

	LRND		HRND		CD		p
	M	SD	M	SD	M	SD	
Hunger	11.24	4.42	9.84	3.74	11.77	3.17	0.25
BMI	21.29	2.29	22.68	2.19	23.46	2.93	0.02
Age	20.77	2.47	20.84	2.67	20.67	3.20	0.98
Trait Imp (BIS)	65.89	11.10	61.19	10.81	64.78	10.74	0.44

The Stop Signal Reaction Time (SSRT) was higher in the Impulsivity Condition compared to the Inhibition Condition,  $173.36 \pm 66$  ms versus  $115.05 \pm 85.7$  ms,  $t(59) = 3$ ,  $p < 0.01$ . When the Stop Signal Task is used to measure impulsivity, a higher SSRT indicates higher impulsivity. In this case a higher mean SSRT for the Impulsivity Condition indicates that the participants in the Impulsivity Condition certainly did behave in a more impulsive way during the task compared to the Inhibition Condition.

#### Effect of the Impulsivity versus Inhibition Condition and dieting status on caloric intake

We conducted a 2 (Impulsivity versus Inhibition) by 3 (LRNDs versus HRNDs versus CDs) between-subjects ANOVA with caloric intake as the dependent variable. Because BMI differed significantly among the dieting groups, BMI was included as a covariate. BMI was not significant as a covariate,  $F(1, 59) = 0.26$ ,  $p > 0.6$ , partial  $\eta^2 = 0.004$ . The main effects of condition,  $F(1, 59) = 0.72$ ,  $p > 0.4$ , partial  $\eta^2 = 0.012$ , and dieting status,  $F(2, 59) = 0.03$ ,  $p > 0.9$ , partial  $\eta^2 = 0.001$ , were not significant. However, the interaction between both factors was significant,  $F(2, 59) = 5.39$ ,  $p < 0.01$ , partial  $\eta^2 = 0.154$ . For each dieting status (LRND; HRND; CD) an ANOVA was conducted with condition as a factor and BMI as a covariate. These post hoc tests revealed that both low and high restrained nondieters in the Impulsivity Condition had a significantly higher caloric intake compared to the low and high restrained nondieters in the Inhibition Condition,  $F(1, 22) = 4.38$ ,  $p < 0.05$ , partial  $\eta^2 = 0.17$ ;  $F(1, 23) = 4.79$ ,  $p < 0.05$ , partial  $\eta^2 = 0.17$ . Current dieters on the other hand, did the opposite: the current dieters in the Impulsivity Condition had a significantly lower caloric intake compared to the current dieters in the Inhibition Condition,  $F(1, 12) = 8.01$ ,  $p < 0.05$ , partial  $\eta^2 = 0.40$ . See Fig. 2 for an Estimated Marginal Means plot of the interaction effect between condition and restraint/dieting status.

A 2 (low trait impulsivity: BIS score  $\leq 64$  versus high trait impulsivity: BIS score  $> 64$ ) by 2 (Impulsivity versus Inhibition) between-subjects ANOVA with caloric intake as the dependent variable was conducted. This was done to rule out an interaction between trait impulsivity, measured by the BIS, and the impulsivity induction. This interaction effect was not significant,  $F(1, 41) = 0.02$ ,  $p > 0.8$ .

#### Manipulation check: effect of the Impulsivity versus Inhibition Condition on state impulsivity and other mood states

As expected, the participants in the Impulsivity Condition scored higher (although marginally significantly) on the IMP subscale that was added to the POMS-SF compared to the Inhibition Condition, 6.5 versus 5.5,  $t(65) = 1.89$ ,  $p < 0.07$ .

IMP was not the only subscale in which the Impulsivity Condition and the Inhibition Condition differed. The Inhibition Condition scored significantly higher on Anger-Hostility, 2.42 versus 0.89,  $t(65) = 2.94$ ,  $p < 0.01$ , and marginally significantly higher on Depression-Dejection, 1.94 versus 1.05,  $t(65) = 1.73$ ,  $p < 0.10$ . In order to find out what mood state differences were responsible for the interaction effect between condition and dieting status, all subscale scores were centred and included as a covariate in the 2 by 2 ANOVA that yielded the significant interaction. Anger-Hostility and Depression-Dejection were not significant as covariates and did not affect the significant interaction (Anger-Hostility:  $F(1, 58) = 0.03$ ,  $p > 0.8$ , Interaction:  $F(2, 58) = 5.15$ ,  $p < 0.01$ ; Depression-Dejection:  $F(1, 58) = 0.09$ ,  $p > 0.7$ , Interaction:  $F(2, 58) = 4.99$ ,  $p \leq 0.01$ ). IMP was significant as a covariate ( $F(1, 57) = 4.16$ ,  $p < 0.05$ ). The interaction effect remained significant ( $F(2, 57) = 5.03$ ,  $p \leq 0.01$ ). It should be noted that the linearity and non-interaction assumptions for ANCOVA analyses were checked and were not rejected.

#### Discussion

In the current study we induced impulsivity versus inhibition by instructing participants to react in an impulsive or an inhibitory manner during the Stop Signal Task. Analyses showed that participants indeed did adhere to our instructions. Moreover, the Impulsivity Condition scored higher on an impulsive state measure compared to the Inhibition Condition.

The current data matched Lowe's findings and are not in line with restraint theory. Nondieters, high and low restrained, reacted as would be expected in the case of a causal connection between impulsivity and caloric intake: their caloric intake was the highest when impulsivity was induced. Current dieters reacted in the opposite way: the current dieters in the Impulsivity Condition ingested significantly less calories compared to the current dieters in the Inhibition Group. In other words, not restraint, as measured by the Restraint Scale (Herman & Polivy, 1980), but whether

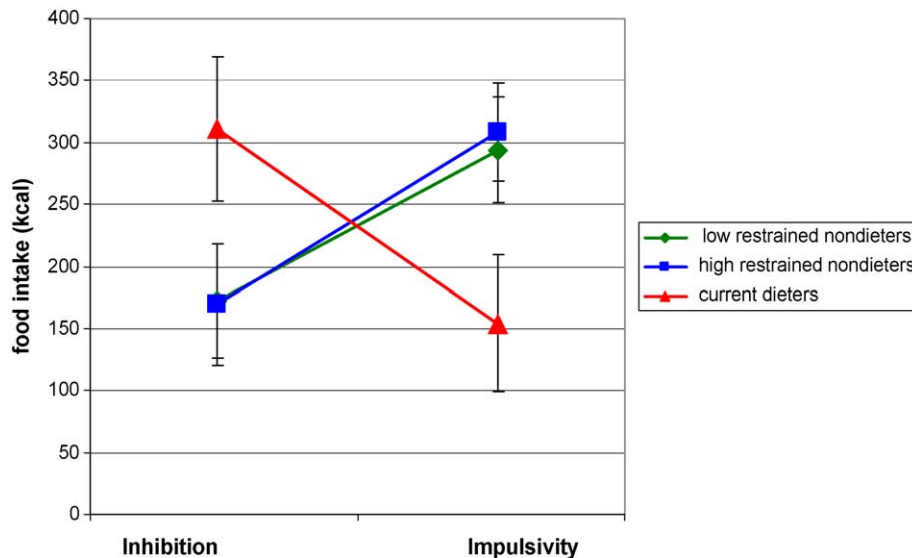


Fig. 2. Estimated Marginal Means plot of the interaction between condition (inhibition versus impulsivity) and restraint/dieting status (unrestrained nondieters versus restrained nondieters versus current dieters) on caloric intake,  $F(2, 59) = 5.39$ ,  $p < 0.01$ . Means are  $\pm$  SEM.

participants were on a diet or not had important implications for their reaction to the induction of impulsivity and inhibition. The outcome of this study is that restraint and dieting indeed seem to be different concepts that affect eating regulation in different ways.

The fact that current dieters increased their caloric intake in response to the inhibition induction compared to the impulsivity induction might seem a counterintuitive outcome. Why would dieters eat *more* in reaction to an induction of inhibition? If anything, should it not help them to stick to their diet? This is not so according to the Ego-Strength Model of Self-Regulation (Baumeister, Bratslavsky, Muraven, & Tice, 1998; Muraven, Tice, & Baumeister, 1998). This model may be able to explain the dieters' behaviour. It is proposed that people have a limited capacity for self-control. Consequently, if people have exerted great amounts of self-control during one task, they are not likely to have much self-control left. Hence, their performance of another task that also demands considerable self-control will be influenced in a negative way. In a classic experiment participants that were asked to only eat radishes when chocolate cookies were also present, persisted less long at a subsequent problem-solving task (Baumeister et al., 1998). In the current study, the dieters were presumably more ego-depleted than the nondieters. They had probably exerted more self-control by the time they arrived in the laboratory because they had a diet to stick to. The dieters in the Inhibition Condition were asked to inhibit their responses as often as possible. It is feasible that this task left them completely ego-depleted. Consequently they might not have had much self-control left to keep them from overeating when confronted with tasty food. During the impulsivity induction dieters were not asked to inhibit their reactions. This possibly left them with more self-control to resist the tasty food that was presented to them during the subsequent taste test. This would explain why the dieters ate *more* after the inhibition induction compared to when impulsivity was induced. However, this explanation would not account for Lowe's findings that current dieters showed increased consumption when they were not required to consume a preload before tasting ice cream (Lowe, 1995; Lowe et al., 1991).

## General discussion

The two studies that were presented here attempted to manipulate impulsivity and inhibition both in a cognitive and a behavioural way in order to find support for a causal link between impulsivity and overeating. In the first study cognitive priming was used and besides a main effect of the impulsivity/inhibition priming an interaction between impulsivity and restraint was predicted. The results indicate that impulsivity indeed causes overeating and that this effect is especially apparent in highly restrained participants. In the second study participants were categorized both according to their restraint status and their dieting status. High and low restrained nondieters reacted in the same way to the induction of impulsivity and inhibition: their caloric intake increased when impulsivity was induced. This again supports the hypothesis that state impulsivity *causes* overeating, albeit within a certain group of participants, the nondieters. Current dieters reacted in the opposite way: they ingested fewer calories following the impulsivity induction compared to the participants in the inhibition induction group. This outcome supports Lowe's model: restraint and dieting are different concepts that affect eating regulation in different ways.

It should be noted that, unlike the outcome of the first study, we did not find that restrained eaters were especially vulnerable for the impulsivity induction. We are not sure why this effect did not occur in study 2. Perhaps it has something to do with the use of a different manipulation compared to study 1. As stated before,

impulsivity is an umbrella concept, consisting of multiple aspects. It is possible that different aspects of impulsivity were manipulated in both studies (e.g. personality based impulsivity in study 1 versus deficient response inhibition in study 2) and that restrained eaters are more vulnerable to the first kind of impulsivity and not the latter. Considering the small effect size and marginal significance of the interaction effect in study 1 one could argue that there was no effect to begin with. However, since current dieting was not measured in study 1, it is likely that some of the restrained eaters were current dieters. Since current dieters reacted the opposite way in study 2, they could have undermined the effect shown by the restrained nondieters in study 1.

In sum, these studies lend support to the hypothesis that impulsivity is a cause of overeating. In the first study a main effect of the impulsivity induction on food intake was apparent with high restrained eaters being extra vulnerable. In the second study, when high restrained eaters were subdivided into high restrained nondieters and current dieters, all participants except the current dieters increased their caloric intake in response to the impulsivity induction. A second important outcome is that our results support Lowe's model in the sense that the reactions of high restrained nondieters and current dieters to the impulsivity induction mimic the reactions of Lowe's participants to a classic disinhibitor, a preload (Lowe et al., 1991).

However, there are some limitations to the studies that are presented. First, in both studies we included two experimental groups, but no neutral control group. Consequently it is not possible to establish whether the effects on food intake were due to heightened impulsivity in the Impulsivity Condition, to heightened inhibition in the Inhibition Condition or to a combination of both.

Second, there is the lack of a (validated) manipulation check. Ideally one would use a validated state measure of impulsivity as a manipulation check when trying to induce impulsivity. If participants score higher on this measure in the experimental condition compared to the control condition, then one is sure that the manipulation led to more food intake via increased impulsivity. In the first study no manipulation check was used, whereas in the second study we used a self-devised state impulsivity measure. Using a (validated) manipulation check was very difficult in the current studies for two reasons. First, to our knowledge, a validated state measure of impulsivity does not exist. Second, the timing of the manipulation check is very difficult to determine. If one does the manipulation check immediately after the manipulation, one risks that the priming effect is already weakened once participants get to the taste test. If one does the check after the taste test, chances are that the manipulation did work during the taste test, but that the priming effect is too weak by the time participants get to the manipulation check. In the second study we chose to administer the manipulation check after the taste test. We did find that the Impulsivity Condition scored higher on the state impulsivity measure compared to the Inhibition Condition, although the effect was marginally significant. In future studies the timing problem could be resolved by conducting two studies: one study in which the manipulation is validated and a second study in which the effect of the manipulation on food intake is measured. However, the need for a validated state measure of impulsivity remains.

Third, when inducing a state in order to establish a causal relationship between a trait and certain behaviours it is an issue whether the state that is induced is qualitatively comparable to the trait that it is supposed to represent. In other words, is the effect of being put in an "impulsive mood" comparable to the effect of having an impulsive personality? In the area of impulsivity little attention has been paid to this issue. More work has been done in the area of anxiety. Although trait and state anxiety are measured separately (Van der Ploeg, Defares, & Spielberger, 1980), they are

considered to be closely linked. The genes that influence state and trait anxiety are identical and people with high levels of trait anxiety are more likely to become state-anxious in reaction to a situation that is seen as threatening (Lau, Eley, & Stevenson, 2006). If this reasoning also applies to impulsivity, one could say that people with an impulsive personality are simply more prone to be in an impulsive mood. If it is indeed so that the effect of a trait on behaviour is mediated by its state, then it should not matter whether this state is induced or occurring naturally. However, further research is needed before this issue can be resolved.

Furthermore, a limitation of the second study is that weight and length were self-reported and not measured by the experimenter.

In conclusion, previous research has linked impulsivity to overeating and obesity. The current study takes it a step further: impulsivity could cause overeating. With the additional support of Rotenberg et al. (2005) – who found that priming “lack of control” thoughts led to more food intake – it becomes plausible that we have a causal mechanism towards overweight and obesity. If further research proves this mechanism to be a valid one, then this could have consequences for the prevention and treatment of overeating in obesity and possibly Bulimia Nervosa (Steiger, Lehoux, & Gauvin, 1999). The development and validation of a training method to decrease impulsive behaviour in high risk groups might be a promising contribution to the prevention of obesity. When it comes to treatment, things are more complicated. It could be the case that successful dieters are better able to manage state impulsivity. Finding out what coping strategies current dieters might use, could thus be useful in the search for treatment methods. However, before we go down that road a similar study should be done with the inclusion of a neutral control group that provides a baseline intake level and lets us interpret the results more clearly.

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