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High body mass index is associated with impaired cognitive control

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ABSTRACT

The prevalence of weight problems is increasing worldwide. There is growing evidence that high body mass index (BMI) is associated with frontal lobe dysfunction and cognitive deficits concerning mental flexibility and inhibitory control efficiency.

The present study aims at replicating and extending these observations. We compared cognitive control performance of normal weight (BMI < 25) and overweight (BMI ≥ 25) university students on a task tapping either inhibitory control (Experiment 1) or interference control (Experiment 2). Experiment 1 replicated previous findings that found less efficient inhibitory control in overweight individuals. Experiment 2 complemented these findings by showing that cognitive control impairments associated with high BMI also extend to the ability to resolve stimulus-induced response conflict and to engage in conflict-driven control adaptation. The present results are consistent with and extend previous literature showing that high BMI in young, otherwise healthy individuals is associated with less efficient cognitive control functioning.

1. Introduction

Weight problems are reaching epidemic proportions worldwide, with approximately 60% of American adults and 50% of European Union adults (in 46 of the 53 countries) being overweight or obese – rates that are expected to further increase over the next years (Flegal, Carroll, Ogden, & Curtin, 2010; World Health Organization, 2014). This increase represents a serious and costly challenge to public health given the well-known physical consequences associated with obesity, including type 2 diabetes (Medalie et al., 1974), hypertension and other cardio-vascular diseases (Avenell et al., 2004; Dyer & Elliott, 1989; Kuczmarski, Carroll, Flegal, & Troiano, 1997), stroke (Hubert, Feinleib, McNamara, & Castelli, 1983), several forms of cancer (Bostick et al., 1994; Chute et al., 1991), and depression (Annunziato & Lowe, 2007; Friedman, Reichmann, Costanzo, & Musante, 2002). Besides these physical issues, brain imaging and behavioral studies comparing overweight and normal-weight individuals have provided evidence that elevated body mass index (BMI) is also associated with neurological and metabolic modifications in the structure and function of the brain (including the frontal lobe) (Volkow et al., 2009; Walther, Birdsill, Glisky, & Ryan, 2010), cortical atrophy (Gustafson, Lissner, Bengtsson, Björkelund, & Skoog, 2004a), and white matter disease (Gustafson, Steen, & Skoog, 2004b), significant intellectual and cognitive deficits (Van den Berg, Kloppenborg, Kessels, Kappelle, & Biessels, 2009; Joseph, Alonso-Alonso, Bond, Pascual-Leone, & Blackburn, 2011; Smith, Campbell, & Trollor, 2011), high rates of attention-deficit hyperactivity disorder (ADHD) (Altfas, 2002:; Cortese et al., 2008), and increased risk of later-life dementia and Alzheimer disease (Elias, Elias, Sullivan, Wolf, & D'agostino, 2003; Gustafson, Rothenberg, Blennow, Steen, & Skoog, 2003; Nilsson & Nilsson, 2009; Sabia, Kivimaki, Shipley, Marmot, & Singh-Manoux, 2009).

The epidemic of obesity is partially caused by the increased availability of relatively cheap, high caloric, palatable food (e.g., fast-food, snacking, and sugar-sweetened beverages) that, combined with changes in lifestyles in response to the modern environment (e.g., advances in technology and transportation, increased use of television, electronic games and computers), have favored unhealthy eating and sedentary behavior (Hill & Peters, 1998). However, not all individuals become overweight or obese, and differences in metabolic efficiency between individuals and/or genetics factors are not sufficient to explain why some individuals tend to overeat and gain excessive weight while others do not.

Recently, it has been suggested that obesity can result from an imbalance between circuits that mediate reward-related and motivated behavior and those that control and regulate reward-

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driven responses (Volkow Wang, Fowler, & Telang, 2008a; Volkow, Wang, & Baler, 2011; Volkow, Wang, Fowler, Tomasi, & Baler, 2012). In vulnerable individuals, overeating is assumed to enhance, via conditioned learning (for details, see Volkow et al., 2008a), the rewarded value of food while weakening cognitive control functioning (Volkow et al., 2008a; 2011, 2012). Evidence supporting this hypothesis comes from studies using positron emission tomography (PET) to assess the role of dopamine (DA) in obesity, which have revealed that obesity is associated with impairments in dopaminergic pathways that regulate reward sensitivity and cognitive control (Volkow et al., 2008a; 2009). It has been found that, in overweight individuals, BMI correlates negatively with striatal dopamine D2 receptor levels (Haltia et al., 2007), and that lower-than normal striatal D2 receptor availability is associated with reduced metabolic activity in the prefrontal cortex (PFC) and anterior cingulate cortex (ACC) (Volkow et al., 2008b). Notably, DA functioning is reckoned to critically contribute to cognitive control efficiency (Cools & D'Esposito, 2011; Stock, Arning, Epplen, & Beste, 2014; Colzato, van den Wildenberg, van der Does, & Hommel, 2010), namely, to people's ability to control their thoughts and to orchestrate goal-directed behavior in a flexible and adaptive manner (Botvinick, Braver, Barch, Carter, & Cohen, 2001). This makes it plausible that overweight and obesity can either be caused or be the consequence (or both) of cognitive control deficits associated with DA dysfunction (Volkow et al., 2011). Consistent with that, research has revealed that excessive food intake and high BMI are not only associated with intellectual deficits (Joseph et al., 2011; Van den Berg et al., 2009), impairments in sustained attention (Cserjesi, Luminet, Poncelet, & Lenard, 2009), memory functioning (Volkow et al., 2009) and episodic memory (Cheke, Simons, & Clayton, 2016), but also are inversely related to decision-making (Brogan, Hevey, & Pignatti, 2010; Danner, Ouwehand, van Haastert, Hornsveld, & de Ridder, 2012; Davis, Patte, Curtis, & Reid, 2010, 2004; Pignatti et al., 2006), and cognitive control efficiency (for recent reviews, see Vainik, Dagher, Dubé, & Fellows, 2013; Fitzpatrick, Gilbert, & Serpell, 2013; Prickett, Brennan, & Stolwyk, 2015). Indeed, in spite of some inconsistencies among studies, overweight and obese people, compared to lean controls, have been found to show poorer performance on tasks tapping set-shifting and cognitive flexibility (Cserjesi et al., 2009; Gunstad et al., 2007; Mobbs, Iglesias, Golay, & Van der Linden, 2011), and inhibitory control (Nederkoorn, Smulders, Havermans, Roefs, & Jansen, 2006; Mobbs et al., 2011; Jasinska et al., 2012; but see; Hendrick, Luo, Zhang, & Li, 2011). These cognitive control impairments are not restricted to young and middle-age adults, but have also been observed in overweight or obese children and adolescents (e.g., Kamijo et al., 2012b, 2012a; Pauli-Pott, Albayrak, Hebebrand, & Pott, 2010; Reyes, Peirano, Peigneux, Lozoff, & Algarin, 2015; Verdejo-García et al., 2010), whose cognitive control system is likely not yet fully developed (Lenroot & Giedd, 2006). Interestingly enough, inhibitory control and cognitive flexibility impairments associated with high BMI have been found to lessen after weight loss (Smith et al., 2010).

The present study seeks to replicate and extend previous observations of impaired cognitive control in overweight people. The study includes two experiments in which we focused on two cognitive control functions that are crucial to guarantee efficient self-regulatory behavior, namely, inhibitory control and interference control. Importantly, in both experiments, we used well-validated tasks that, among the other things, have been previously found to be suitable to detect cognitive control impairments in recreational cocaine users, as compared to cocaine-free controls (Colzato, van den Wildenberg, & Hommel, 2007; Sellaro, Hommel, & Colzato, 2014).

In Experiment 1, we compared performance of overweight and normal-weight university students on a task tapping inhibitory control efficiency. The two paradigms that are frequently used to assess inhibitory control efficiency are the go/nogo paradigm (Donders, 1868/1969) and the stop-signal task (Logan & Cowan, 1984). All but one (Nederkoorn et al., 2006) of the previous studies (Jasinska et al., 2012; Kamijo et al., 2012b, 2012a; Pauli-Pott et al., 2010; Reyes et al., 2015) that have reported less efficient inhibitory control ability in overweight/obese, as compared to normal weight individuals, have used the go/nogo task. Although there is a tendency to consider the two tasks as equivalent and to generalize the results obtained in one task to the other, Verbruggen and Logan (2009) showed that the two tasks actually differ and concluded that the stop-signal task is to be preferred over the go/nogo task when assessing deficits in inhibitory control. This is because in go/nogo tasks the need for executive control processes is reduced throughout practice. As a consequence, go/nogo tasks may fail to reveal existing inhibitory deficits, or they may reveal impairments that actually reflect learning deficits.

Therefore in the present study, inhibitory control efficiency was assessed by means of the stop-signal task (Logan & Cowan, 1984). Participants were instructed to quickly react to the direction of a green left- or right-pointing arrow (i.e., the Go signal), by pressing a left- or right button. Occasionally, the color of the arrow suddenly changed to red, requiring participants to abort the ongoing response (i.e., stop trials). The task provides a measure of the efficiency of response execution, as reflected by reaction times (RTs) to go-signals, along with a quantitative estimation of the duration of the covert response-inhibition process (i.e., the efficiency of inhibitory control), as indexed by the stop-signal reaction times (SSRTs; i.e., the time required to stop the ongoing response), where longer SSRTs indicate a less efficient inhibitory control. Using this task, a previous study found obese middle-age women, as compared to lean controls, to show longer SSRTs but comparable RTs to go-signals, especially in the last part of the task (Nederkoorn et al., 2006). Replicating and extending these findings, we expect overweight, compared to normal-weight, university students to show less efficient inhibitory control (i.e., longer SSRTs).

In Experiment 2, performance of a different sample of overweight and normal-weight university students was compared on a well-established task tapping interference control, namely, the Simon task (Simon & Small, 1969). This task is known to index the ability to handle and resolve response conflict, that is, the capacity to select the proper response in face of other, competing pre-potent responses (Hommel, 2011). Participants were asked to press a left or right button on the basis of a non-spatial feature (i.e., the color) of a stimulus that could randomly appear to the left or to the right of a central fixation cross. Typically, performance is better (i.e., faster RTs and/or fewer errors) when the position of the stimulus spatially correspond to the position of the required response than when stimulus and response positions do not correspond – the Simon effect (Simon & Small, 1969). This effect reveals the difficulty of selecting the correct response among competing responses and, thus, provides a reliable measure of the efficiency of dealing with response conflict (Hommel, 2011; Kornblum, Hasbroucq, & Osman, 1990). Aside from measuring the ability to resolve stimulus-induced response conflict, this task also provides a measure of people's efficiency to engage in conflict-driven control adaptation. A consistent finding is that people's ability to resolve stimulus-induced response conflict is more efficient after conflict trials: i.e., the Simon effect in the present trial (I-C) is smaller after an incongruent trial (iI-iC) than after a congruent trial (cI-cC; Gratton, Coles, & Donchin, 1992). This effect, which is referred to as "conflict-adaptation effect" or Gratton effect, is used as an indication of

control fluctuation and resulting adaptation (Botvinick et al., 2001). To the extent to which cognitive deficits associated with high BMI generalize to other cognitive control functions, we expect high BMI students to show a less efficient ability to resolve stimulus-induced response conflict (i.e., a larger Simon effect) and to engage in conflict-driven control adaptation (i.e., a less pronounced Gratton effect).

2. General method

2.1. Participants

Overall, 82 undergraduate students of Leiden University with no hepatic, renal, cardiac, psychiatric or neurological disorders, personal or family history of migraine, depression, and medication or drug use participated in the study. To avoid possible confounding effects resulting from ego depletion (i.e., reduced self-control after an exhausting cognitive task; Baumeister, Bratslavsky, Muraven, & Tice, 1998), participants were randomly assigned to perform only one of the two tasks, namely, either the stop-signal task (Experiment 1) or the Simon task (Experiment 2). Thirty-nine participants (11 males; mean age = 22.2 years, SEM = 0.5, range 18–30; mean Body Mass Index = 24.4, SEM = 0.6. range 18.7–35) took part in Experiment 1, and 43 participants (13 males; mean age = 21.6 years, SEM = 0.5, range 18–31; mean Body Mass Index = 24.8, SEM = 0.6. range 17.6–33.5) took part in Experiment 2.

Participants were recruited via university message boards, social media outlets, and word of mouth. Once recruited, all participants were screened individually by the same lab-assistant using the Mini International Neuropsychiatric Interview (M.I.N.I.; Sheehan et al., 1998). The M.I.N.I. is a short, structured interview that screens for several psychiatric disorders and drug use, typically used in clinical and pharmacological research (Sheehan et al., 1998; Colzato, Kool, & Hommel, 2008; Colzato et al., 2010; Colzato et al., 2012). Participants were considered suitable to participate in this study if they fulfilled the following criteria: (i) age between 18 and 32 years; (ii) no history of neurological or psychiatric disorders; (iii) no history of substance abuse or dependence; (iv) no chronic or acute medications; (vi) not currently enrolled in a weight-loss program.

In both experiments, participants were categorized in normal-weight and overweight groups based on the criteria established by WHO (2014): participants with BMI equal or higher than 25 formed the high BMI group, whereas those with BMI lower than 25 formed the low BMI group (for detailed sample information, see Table 1).

The study conformed to the ethical standards of the Declaration of Helsinki, and the protocol was approved by the local ethical committee (Leiden University, Institute for Psychological Research). All participants were naïve to the purpose of the study, and gave their written informed consent.

Table 1
Sample information as a function of BMI group (high vs. low) and Experiment (Experiment 1 vs. Experiment 2). Standard errors are shown in parentheses.

Variables	Experiment 1		Experiment 2	
	High BMI	Low BMI	High BMI	Low BMI
N (F:M)	17 (11:6)	22 (17:5)	19 (11:8)	24 (19:5)
BMI	27.7 (0.6)***	21.9 (0.4)***	28.7 (0.6)***	21.7 (0.4)***
Age	23.4 (0.8)*	21.2 (0.6)*	22.9 (1.0)*	20.5 (0.5)*
Pleasure	1.7 (0.2)	1.5 (0.3)	2.1 (0.3)	1.8 (0.2)
Arousal	0.1 (0.3)	0.8 (0.3)	0.4 (0.4)	- 0.1 (0.3)
HR	85.9 (6.3)	79.6 (3.1)	75.4 (2.0)	70.4 (2.7)

*p < 0.05; ***p < 0.001.

2.2. Apparatus, stimuli and procedure

All participants took part in a single session and were tested individually. Upon arrival, after participants read and signed the informed consent, they were weighed and their body mass index (BMI) was measured using the OMRON Body Composition Scale Karada Scan. Immediately after, participants were asked to rate their mood on a 9 × 9 Pleasure × Arousal grid (Russel, Weiss, & Mendelsohn, 1989), with values ranging from −4 to 4. Heart rate (HR) was measured from the non-dominant arm using Medisana® Upper arm. Next, participants were asked to perform either the stop signal task (in Experiment 1) or the Simon task (in Experiment 2). After completed the experimental task, participants were debriefed and given course credits for their participation.

2.3. Stop signal task

Participants completed a version of the stop-signal task adapted from Colzato et al. (2007), Colzato, van den Wildenberg, & Hommel, (2013b), which lasted about 30 min. Each trial began with the presentation of a white fixation point (3 mm in diameter), which remained on the screen for a random period between 1750 and 2250 ms (in steps of 50 ms). Thereafter, a central green arrow pointing, with equal probability, to the left or to the right was shown, and remained visible until the response or 1500 ms had passed (i.e., go trials). Participants were instructed to respond as quickly and as accurately as possible to the direction (left or right) of the green arrow by pressing the "z" or "?" button of a QWERTY keyboard, respectively. On 25% of the trials, the green arrow changed to red signaling the participants to abort the ongoing response (i.e., stop trials). To control inhibition probability, the delay between the presentation of the go stimulus and the onset of the stop signal was dynamically adjusted using a staircase-tracking procedure (Levitt, 1971). Specifically, the stop-signal delay was increased by 50 ms after a successfully inhibited stop trial, and decreased by 50 ms after a trial in which the participant failed to stop on time. This procedure ensured to yield about a 50% probability of successfully inhibiting a motor action, thereby providing an accurate estimate of SSRTs and compensating for differences in choice RTs between participants (Band, Van Der Molen, & Logan, 2003). The task comprised 1 practice block and 4 experimental blocks of 104 trials each.

2.4. Simon task

The task, adapted from Colzato, Sellaro, Samara, and Hommel (2015), was about 20 min long. In each trial, participants were first presented with a central fixation point, for a random period between 1750 and 2250 ms (in steps of 100 ms). Immediately after, a target circle (either green or blue) was presented equiprobably to the left or to the right of the central fixation point. The target circle was shown until response, but no longer than 1500 ms. The task required participants to respond to the color of the target circle by pressing one of two lateralized keys on a QWERTY computer keyboard: the left key (i.e., "z") in response to the green circle, and the right key (i.e., "?") in response to the blue circle. Participants were instructed to react as fast and as accurately as possible to the color of the target circle, while ignoring its position.

The task consisted of 6 blocks of 60 randomly-presented trials, the first of which was a practice block. Stimulus and response positions did correspond in one half of the trials (congruent trials) but did not correspond in the other half (incongruent trials).

3. Statistical analyses

3.1. Participants

For both Experiment 1 and Experiment 2, separate independent sample t-tests were performed to verify whether the two groups (high BMI vs. low BMI) were comparable in terms of age, and baseline levels of arousal, pleasure and HR. A Chi-square test was performed to rule out possible differences between high and low BMI groups in terms of gender distribution.

3.2. Stop-signal task

For all participants, individual SSRTs for stop-signal trials (i.e., index of response inhibition) and mean RTs to go-signals (i.e, index of response execution) were calculated. SSRTs were calculated by subtracting the mean stop-signal delay from the mean RT to go-signals (cf. Logan & Cowan, 1984). Between-group (high BMI vs. low BMI) differences in terms of response inhibition (SSRTs) and response execution (RTs to go-signals) were analyzed by means of separate independent sample t-tests.

3.3. Simon task

Mean correct RTs and percentage of errors (PEs) were analyzed by means of separate repeated-measures analyses of variance (ANOVAs) with congruency in present trial [congruence (C) vs. incongruence (I)] and congruency in previous trial [congruence (c) vs. incongruence (i)] as within-subject factors, and group (high vs. low BMI) as between-subject factor. For all participants and for both RTs and PEs, Gratton effects were calculated by subtracting the Simon effect following correct incongruent trials (iI−iC) from the effect following correct congruent trials (cI−cC). Simon effects for RTs and PEs were calculated according to the following formula: [(cI + iI)/2 - (cC + iC)/2)]. The first trial of each block and post-error trials were excluded from all analyses.

A significance level of $p < 0.05$ was adopted for all statistical tests. In case of significant interaction, post hoc analyses were conducted using Tukey's HSD test.

4. Results

The data reported in this paper are archived in the Open Science Framework (OSF) and are available through https://osf.io/qk65s/?view_only=859ac58a92f64bc4abf379591c17b23b.

4.1. Experiment 1: BMI and inhibitory control

4.1.1. Participants

Sample information is shown in Table 1. No significant between-group differences were observed in terms of pleasure, $t(37) < 1$, $p = 0.71$, Cohen's $d = 0.12$, arousal, $t(37) = 1.5$, $p = 0.14$, Cohen's $d = 0.49$, HR, $t(37) < 1$, $p = 0.35$, Cohen's $d = 0.31$, and gender distribution, $\chi 2 < 1$, $p = 0.39$. In contrast, significant group differences were observed in terms of BMI, $t(37) = 8.51$, $p < 0.001$, Cohen's $d = 2.75$, and age, $t(37) = 2.24$, $p = 0.03$, Cohen's $d = 0.72$ (see Table 1).

4.1.2. Stop-signal task

All participants were unable to stop their responses on stop-signal trials successfully in about half of the time a stop signal required them to do so (M = 48.1%, SEM = 0.4 in the high BMI group, and M = 48.6%, SEM = 0.3 in the low BMI group), indicating that the dynamic tracking algorithm worked well for both groups, who did not differ significantly from each other on this measure, $t(37) = 1.13$, $p = 0.27$, Cohen's $d = 0.36$. The percentage of choice errors to go-signals was low and did not differ between high (M = 1.1%, SEM = 0.3) and low (M = 1.0%, SEM = 0.1) BMI groups, $t(37) < 1$, $p = 0.63$, Cohen's $d = 0.16$. More importantly, analyses of the stop-signal data revealed significant between-group differences for the SSRTs, $t(37) = 2.53$, $p = 0.02$, Cohen's $d = 0.82$, but not for the RTs to go-signals, $t(37) < 1$, $p = 0.90$, Cohen's $d = 0.04$. As expected, SSRTs were significantly longer in the high BMI (M = 253 ms, SEM = 5.8) than in the low BMI group (M = 234 ms, SEM = 4.6), who showed comparable performance in terms of RTs to go-signals (M = 433 ms, SEM = 10.3, and M = 431 ms, SEM = 6.5, for the high and low BMI group, respectively), see Fig. 1.

To further investigate the relation between BMI and stop-signal performance in terms of response execution and response inhibition, and the possible association between either measure and age, Pearson's correlations coefficients were computed between BMI, RTs to go-signals, SSRTs, and age. As shown in Table 2, no significant correlations were found between RTs to go-signals and either BMI or age. Significant positive correlations were observed between BMI and age, and, as expected, between BMI and SSRTs. Importantly, age did not significantly correlate with SSRTs (see also Fig. 2).

4.2. Experiment 2: BMI and interference control

4.2.1. Participants

Sample information is shown in Table 1. No significant between-group differences were observed in terms of pleasure, $t(41) < 1$, $p = 0.35$, Cohen's $d = 0.29$, arousal, $t(41) = 1.0$, $p = 0.32$, Cohen's $d = 0.31$, HR, $t(41) = 1.19$, $p = 0.24$, Cohen's $d = 0.37$, and gender distribution, $\chi 2 = 2.28$, $p = 0.13$. In contrast, significant group differences were observed in terms of BMI, $t(41) = 10.66$, $p < 0.001$, Cohen's $d = 3.27$, and age, $t(41) = 2.40$, $p = 0.02$, Cohen's $d = 0.74$ (see Table 1).

4.2.2. Simon task

ANOVAs revealed standard Simon effects in terms of both RTs and PEs. Indeed responses were faster and more accurate on congruent (M = 429 ms, SEM = 8.5 and M = 2.4%, SEM = 0.3) than on incongruent trials (M = 455 ms, SEM = 8.9 and M = 5.0%, SEM = 0.5), $F(1,41) = 133.51$, $p < 0.001$, $\eta 2p = 0.77$ (RTs), $F(1,41) = 34.04$, $p < 0.001$, $\eta 2p = 0.45$ (PEs). Moreover, a standard Gratton effect was obtained as well, as indicated by a significant

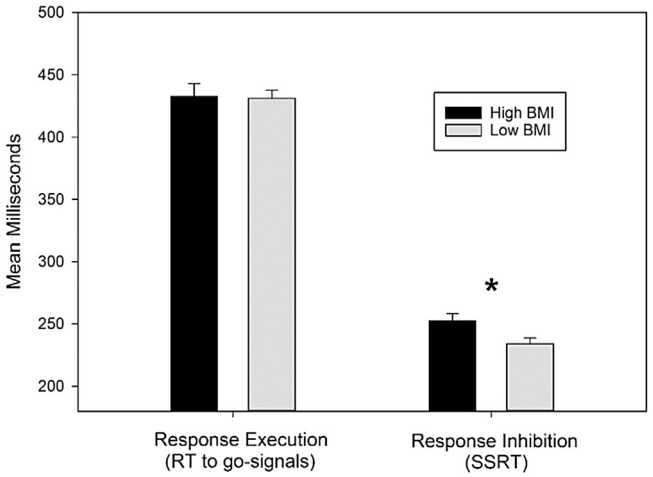

Fig. 1. Mean RT to go-signals (response execution) and mean SSRT (response inhibition) as a function of BMI group (high vs. low BMI). Asterisk indicates significant between-group difference (*$p < 0.05$). Vertical capped lines atop bars indicate standard error of the mean.

Table 2
Correlations between BMI, RTs to go-signals, SSRTs and age.

		BMI	RT to go signals	SSRT	Age
BMI	Pearson's r	–	0.115	0.321*	0.342*
	p-value	–	0.486	0.046	0.033
RT to go signals	Pearson's r		–	−0.015	−0.138
	p-value		–	0.929	0.401
SSRT	Pearson's r			–	0.237
	p-value			–	0.147
Age	Pearson's r				–
	p-value				–

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

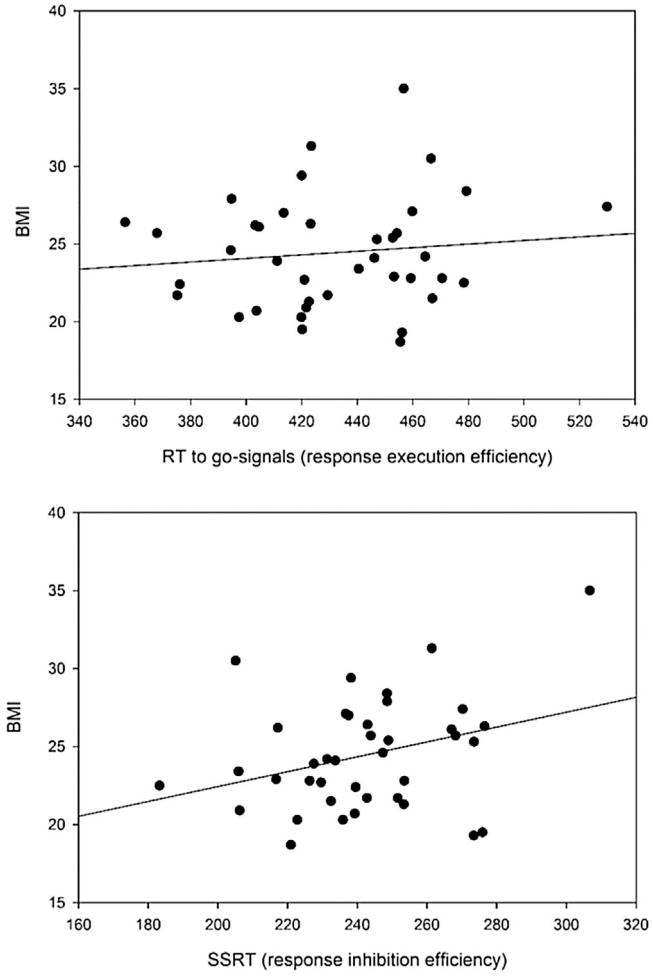

Fig. 2. Scatterplots of the RT to go-signals (top panel) and SSRT (lower panel) against individual BMI values.

interaction between congruency in previous trials and congruency in present trials for both RTs, $F(1,41) = 190.02$, $p < 0.001$, $\eta 2p = 0.82$, and PEs, $F(1,41) = 57.46$, $p < 0.001$, $\eta 2p = 0.58$. As usually observed, for both RT and PE data, post-hoc analyses indicated regular Simon effects (55 ms and 6.6%) after congruent trials ($p_s < 0.001$), which were reversed in sign (−3 ms and −1.4%), but not significantly so, after incongruent trials ($p_s \geq 0.18$). More interesting for our purposes, significant interactions between BMI group and congruency in present trial were observed for both RT and PE analyses, $F(1,41) = 5.63$, $p = 0.02$, $\eta 2p = 0.12$ (RTs), $F(1,41) = 4.94$, $p = 0.03$, $\eta 2p = 0.11$ (PEs), with high BMI group showing significantly larger Simon effects than low BMI group (31 ms and 3.6% vs. 21 ms and 1.6%; see Table 3). Also, as expected, between-group differences were observed for the Gratton effect in RT, but not in PE, as revealed by a significant three-way interaction involving the factors group, congruency in present trial, and congruency in previous trial, $F(1,41) = 7.60$, $p = 0.009$, $\eta 2p = 0.16$ (RTs), $F(1,41) < 1$, $p = 0.98$, $\eta 2p = 0.00$ (PEs). The RT effect indicated that the size of the Gratton effect was significantly smaller for the high BMI group than for the low BMI (see Table 3). The main effect of congruency in previous trial was significant for both RTs, $F(1,41) = 6.82$, $p = 0.01$, $\eta 2p = 0.14$, and PEs, $F(1,41) = 8.21$, $p = 0.007$, $\eta 2p = 0.17$: responses were faster but less accurate after congruent (M = 439 ms, SEM = 8.2 and M = 4.1%, SEM = 0.4) than after incongruent (M = 445 ms, SEM = 9.2 and M = 3.3%, SEM = 0.4) trials. This effect was modulated by BMI group for the PE, but not for the RT analysis, $F(1,41) < 1$, $p = 0.68$, $\eta 2p = 0.04$ (RTs), $F(1,41) = 8.6148$, $p = 0.01$, $\eta 2p = 0.17$ (PEs). Specifically, post-hoc analyses showed comparable PEs after congruent and after incongruent trials for the low BMI group (M = 3.6%, SEM = 0.6 vs. M = 3.7%, SEM = 0.5; $p = 1.0$); in contrast, for the high BMI group, PEs were significantly higher after congruent than after incongruent trials (M = 4.6%, SEM = 0.6 vs. M = 2.9%, SEM = 0.6; $p = 0.002$). Finally, both RT and PE analyses revealed no significant main effect of BMI group, $F_s < 1$, $p_s \geq 0.91$.

To further investigate the relation between BMI and Simon task performance in terms of Simon and Gratton effects, and the possible influence of participants' age in meditating it, Pearson's correlations coefficients were computed between BMI, Simon and Gratton effects (in terms of RTs and PEs), and age. As shown in Table 4, no significant correlations were observed between BMI and Simon and Gratton effects in terms of PEs, or between BMI and age. Also, age was not found to correlate with any variable. Notably, as expected, BMI correlated positively with the size of the Simon effect and negatively with the size of the Gratton effect (see also Fig. 3).

5. Discussion

The present study consisted of two experiments aimed to assess whether high BMI is associated with less efficient inhibitory (as indexed by performance on the stop-signal task; Logan & Cowan, 1984) and interference control (as indexed by performance on the Simon task; Simon & Small, 1969), respectively. The results of both experiments provide converging evidence corroborating previous observations of cognitive control impairments in overweight individuals (for reviews, see Vainik et al., 2013; Fitzpatrick et al., 2013; Prickett et al., 2015).

Results of Experiment 1 replicated, in a sample of university students, previous findings (Nederkoorn et al., 2006) that overweight, as compared to normal-weight individuals show comparable RTs to go-signals but longer SSRTs, which reflects a less efficient inhibitory control. This fits with the idea that the tendency

Table 3
Mean correct reaction times (RT; in ms), percentage of errors (PE; in %) and Simon and Gratton effects for both of these measures, as a function of BMI group (high and low). Standard errors are shown in parentheses.

Variables	RT (ms)		PE (in %)	
Simon Task	High BMI	Low BMI	High BMI	Low BMI
Congruent trial following a congruent trial (cC)	411 (12.0)	412 (10.7)	0.8 (0.3)	0.8 (0.3)
Incongruent trial following a congruent trial (cI)	466 (12.9)	468 (11.5)	8.4 (1.1)	6.5 (1.0)
Congruent trial following an incongruent trial (iC)	440 (13.8)	454 (12.3)	3.1 (0.8)	4.9 (0.7)
Incongruent trial following an incongruent trial (iI)	448 (14.2)	439 (12.6)	2.6 (0.7)	2.5 (0.6)
Simon effect [(cI + iI)/2−(cC + iC)/2)]	31 (3.3)*	21 (3.0)*	3.8 (0.7)*	1.7 (0.6)*
Gratton effect [(cI − cC) − (iI − iC)]	47 (6.3)**	70 (5.6)**	8.1 (1.6)	8.0 (1.4)

*p < 0.05; **p < 0.01.

Table 4
Correlations between BMI, Simon and Gratton effects (in terms of RTs and PEs), and age.

		BMI	Simon effect (RT)	Simon effect (PE)	Gratton effect (RT)	Gratton effect (PE)	Age
BMI	Pearson's r	–	**0.393****	0.278	**−0.394****	0.004	0.283
	p-value	–	**0.009**	0.071	**0.009**	0.978	0.066
Simon effect (RT)	Pearson's r		–	0.604***	−0.326*	−0.041	0.262
	p-value		–	<0.001	0.033	0.794	0.09
Simon effect (PE)	Pearson's r			–	−0.113	0.063	0.198
	p-value			–	0.47	0.687	0.204
Gratton effect (RT)	Pearson's r				–	0.081	−0.283
	p-value				–	0.608	0.066
Gratton effect (PE)	Pearson's r					–	−0.224
	p-value					–	0.149
Age	Pearson's r						–
	p-value						–

* p < 0.05, **p < 0.01, ***p < 0.001.

to overeat may depend on (i.e., it can either be the cause or the consequence of) a deficit in inhibiting the urge to engage in pre-potent behaviors (Becker, Jostmann, Wiers, & Holland, 2015; Wang et al., 2009). The presence of this kind of deficit may also account for the high prevalence of ADHD in the obese population (Altfas, 2002; Cortese et al., 2008), and for the observations that the administration of methylphenidate, a drug commonly used to treat ADHD to improve cognitive control, causes a weight loss especially in children with high BMI (Schertz, Adesman, Alfieri, & Bienowski, 1996; see also; Gurbuz et al., 2016).

Results of Experiment 2 complemented and extended these findings by showing that cognitive control impairments associated with high BMI also extend to the ability to resolve stimulus-induced response conflict and to engage in conflict-driven control adaptation. Indeed, compared to performance of normal-weight students, overweight students showed a larger Simon effect and a less pronounced Gratton effect. Therefore, the results of Experiment 2 suggest that, besides showing an impairment in response inhibition, overweight individuals have also deficits in overcoming misleading stimulus-induced response tendencies and in engaging in efficient control adaptation. In other words, overweight individuals may experience difficulties in prioritizing goal-relevant responses over automatic (reward-driven) responses, thereby systematically favoring the immediate hedonic gratification associated with eating over a healthier behavior.

The results of both experiments also provide indirect support for the hypothesis that overweight and obesity may critically depend on the activity in the PFC and on dopaminergic pathways that regulate it (Volkow et al., 2008a; 2011). It is assumed that frontal lobe circuits play a crucial role in the inhibition of pre-potent responses (Aron, Fletcher, Bullmore, Sahakian, & Robbins, 2003), and that these circuits are innervated by DA (Hershey et al., 2004). Likewise, response conflict efficiency is assumed to depend on regulatory feedback involving the PFC and ACC (Botvinick, 2007; Botvinick, Cohen, & Carter, 2004; Floden, Vallesi, & Stuss, 2011), and DA is believed to play a key role in both signaling and resolving response conflict (Botvinick, 2007; Holroyd & Coles, 2002). However, it remains to establish whether the cognitive control deficits and concomitant neuronal dysfunctions observed in high BMI individuals play a causal role in determining overweight and obesity or are rather the consequence of these conditions, though the presence of a "vicious circle" is more likely (Kanoski & Davidson, 2011; Sellbom & Gunstad, 2012). Therefore, more research is needed to ascertain the directionality of the association between elevated BMI and less efficient cognitive control. To this end, prospective studies in which cognitive control functioning is evaluated before and after weight loss may provide useful insight, which ultimately can help to develop effective weight loss interventions.

Some limitations of the present study need discussion. First, participants were categorized in normal-weight and overweight groups based on their BMI. Such a classification does not consider muscle composition and, thus, the fact that high BMI may just depend on large amounts of lean muscle mass – an issue that may have caused us to underestimate the association between BMI and cognitive control in the sample of participants we tested. Future studies should use more appropriate indices (e.g., waist-to-hip ratio, skinfold test) to determine whether an individual is overweight or not. Second, we did not screen our participants for the presence of a binge-eating disorder (BED), which would have allowed us to assess whether the cognitive control deficits we observed in the overweight groups were due to the presence of an eating pathology in addition to high BMI, or were independent from that. Follow-up studies should include such an assessment to compare performance of normal weight, overweight and BED overweight participants on measures of cognitive control. Third, and related to the previous point, in screening participants for the presence of physical, neurological and psychiatric disorders, and medication or drug use, we did not rely on a thorough medical and psychiatric examination. Animal and human studies have shown that overweight-related physical issues (e.g., diabetes and hypertension) *per se* can

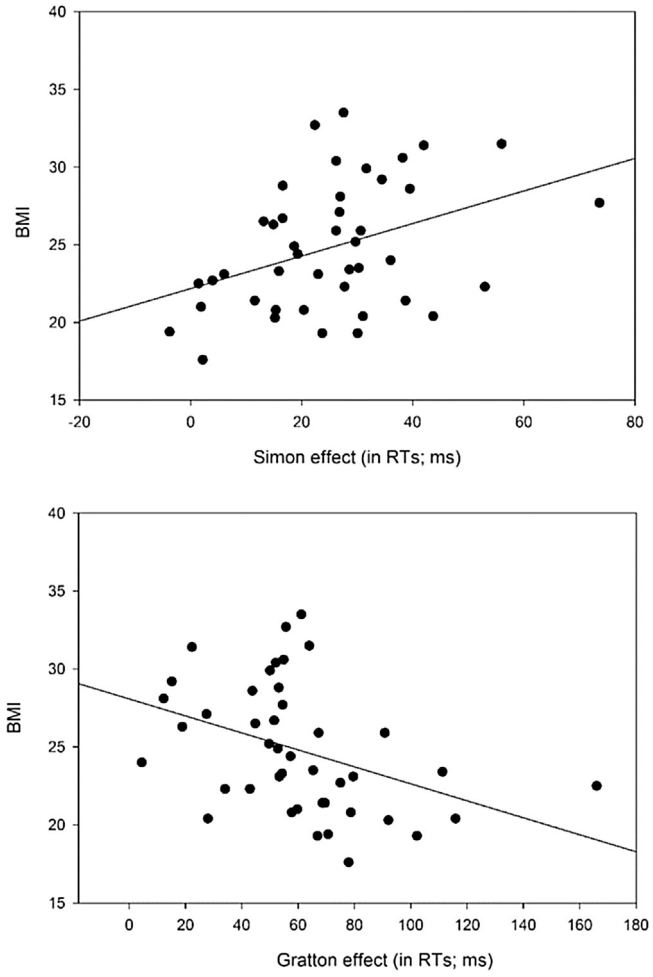

Fig. 3. Scatterplots of the Simon effect (top panel) and Gratton effect (lower panel) in RTs against individual BMI values.

cause significant neurological changes in the structure and function of the brain, and specific cognitive impairments via different mechanisms, such as neuronal degradation (Smith, Hay, Campbell, & Trollor, 2011), dysfunctional cerebral metabolism (Volkow et al., 2009), inflammation (Smith et al., 2011), and elevated leptin (Gunstad et al., 2008; Harvey, 2007). Furthermore, there is evidence that obesity and depression frequently co-occur (Annunziato & Lowe, 2007; Friedman et al., 2002), and that depressive mood can further aggravate the cognitive impairments associated with high BMI (Cserjesi et al., 2009). Therefore, although both overweight and normal-weight participants reported no medical or psychiatric disorder, and we made sure they were comparable in terms of subjective affective state and heart rate before performing the tasks, we cannot rule out the possibility that undetected disorders may have mediated the observed between-group differences on cognitive control measures. Fourth, in both experiments age was not statistically matched between groups, with overweight participants being slightly older than the ones whose weight was in the normal range. Both inhibitory control and interference control efficiency are known to decrease with increasing age (Kramer, Humphrey, Larish, Logan, & Strayer, 1994; Vallesi, Stuss, McIntosh, & Picton, 2009; Van der Lubbe & Verleger, 2002). However, the tiny between-group difference we observed in terms of age is unlikely to account for the observed differences in cognitive control efficiency, as further supported by the fact that, in both experiments, age did not significantly correlate with any dependent variable. This is not to deny, however, that it would be more appropriate for future studies to fully match the groups for age. Another limitation of the present study pertains to the small sample sizes, including mostly female participants, a common limitation for this kind of studies (Fitzpatrick et al., 2013). Therefore, more research is needed to verify the reliability and repeatability of our findings in larger samples, possibly balanced for gender. Finally, it is important to acknowledge that our conclusions that high BMI is associated with less efficient inhibitory and interference control rely on participants' performance on a single task, tapping either function. Complex cognitive functions are unlikely to be captured by a single task (Phillips, 1997). To obtain more reliable results, it would be ideal for future studies to compare high and low BMI participants on multiple tasks measuring cognitive control functioning, as required by confirmatory statistical analyses (e.g., confirmatory factor analysis and/or structural equation modelling analyses; see Miyake et al., 2000 and Jasinska et al., 2012, for examples of application of these methods).

Notwithstanding these limitations, our findings add to the growing evidence that high BMI in young, otherwise healthy individuals is associated with less efficient cognitive control. Interestingly enough, our findings also link to the emerging literature suggesting that cognitive control functioning in healthy individuals can be further ameliorate by consuming food rich in tyrosine, the biochemical precursor of DA (Colzato, Jongkees, Sellaro, van den Wildenberg, & Hommel, 2014, 2015, 2013a; Steenbergen, Sellaro, Hommel, & Colzato, 2015). Taken together, results from these related lines of research can be used to stimulate insightful investigations to develop preventive or corrective measures to possibly counteract the epidemic of obesity, by educating people to follow a healthy diet and simultaneously strengthening cognitive control functions in the attempt to promote a more efficient self-regulation aimed at maintaining a healthy weight. Specifically, interventions aimed at improving inhibitory control efficiency may help overweight individuals to resist temptations. Likewise, interventions aimed at improving response conflict management may allow overweight individuals to get rid of automatized stimulus-response associations (i.e., learned habits), thereby favoring goal-relevant behavior in condition of interference.

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