Reactive and proactive control adjustments under increased depressive symptoms: Insights from the classic and emotional-face Stroop task.

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Abstract

The current research investigated differences in reactive and proactive cognitive control as a function of depressive symptomatology. Three participant groups with varying symptom levels (BDI-II score) completed both the classic and an emotional-face Stroop task separately under speed and accuracy instructions. All groups made equivalent speed-accuracy trade-offs independent of task, suggesting that proactive adjustments are unaffected by depressive symptoms. Additionally, groups made equivalent reactive control adjustments (Stroop effects, congruency sequence effects) in the classic Stroop task, suggesting that these reactive control adjustments are spared across a wide range of BDI-II scorers. In contrast, the high BDI-II group displayed a selective impairment in the resolution of conflict in the emotional-face Stroop task. Thus, while proactive control and many aspects of reactive control were unaffected by the level of depressive symptoms, specific impairments occurred when current task demands required the trial-to-trial regulation of emotional processing.

Key words: conflict monitoring; depression; proactive control; reactive control; control adjustments

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The human cognitive system possesses a remarkable ability to exert control over information processing, allowing us to make efficient, goal-directed behaviours in complex and challenging environments. This regulation of cognition is a dynamic process, initiated either proactively by pre-determined, external goals or reactively after unexpected challenges to on-going performance (Botvinick, Braver, Barch, Carter & Cohen, 2001; Braver, Gray & Burgess, 2007; Braver, 2012; Wühr & Kunde, 2008). *Proactive* control processes facilitate the sustained maintenance of future-oriented goal-states by optimizing performance through the prolonged biasing of information processing systems. In contrast, *reactive* control is past-oriented; triggering the transient up-regulation of control after spontaneous challenges to performance, such as response conflict. Importantly, reactive control adjustments require internal performance monitoring processes (putatively located within the anterior cingulate cortex (ACC), see Botvinick, Braver, Barch, Carter & Cohen, 2001; Braver et al., 2007; Braver, 2012), which signal the need for increased control after processing challenges (this top-down control is achieved by other brain structures, e.g., the dorsolateral prefrontal cortex (DLPFC), Botvinick et al., 2001; Ridderinkhof, Ullsperger, Crone & Nieuwenhuis, 2004).

In addition to this variation in the expression of cognitive control, executive impairments have been reported in various psychopathologies such as schizophrenia (Barch & Ceasar, 2012; Kerns et al., 2005), attention deficit/hyperactivity disorder (e.g., King, Colla, Brass, Heuser, & Cramon, 2007) and,

of importance for the current research, depression (Holmes & Pizzagalli, 2007; West, Choi & Travers, 2010). In addition to the profound and sustained low mood which often characterises depression, the disorder is associated with subjective feelings of impaired concentration and general loss of interest (Diagnostic and Statistical Manual of Mental Disorders IV (DSM-IV), American Psychiatric Association, 2000), suggesting that attentional and goal-motivated cognitive control processes might be impaired as a function of increased depressive symptoms. Furthermore, functional neuroimaging research consistently implicates the dysfunction of the fronto-cingulate structures which underlie cognitive control in the neuropsycholgy of depression and low mood (Liotti & Mayberg, 2002; Mayberg, 1997; Pizzagalli, 2011). Consequently, several authors have suggested that both proactive and reactive control processes might be impaired in participants with elevated depressive symptoms (Holmes & Pizzagalli, 2008; West, Choi & Travers, 2010).

Studies of reactive cognitive control often focus on interference tasks such as the Stroop paradigm. Once described as "the gold standard of attentional measures" (Macleod, 1992), the classic colour-word Stroop protocol (Stroop, 1935) has been used extensively in experimental psychology, contributing to both the formulation of current models of cognitive control (e.g., Botvinick et al., 2001; Braver et al., 2007) and investigations of disordered processing in various psychopathologies (e.g., Holmes & Pizzagalli, 2008; Kerns et al., 2005; King et al., 2007; Moritz et al., 2002). In the classic task responses are reliably slower and more error-prone during incompatible trials in which participants must suppress a pre-potent response (e.g., word reading when the word "BLUE" is written in red ink) as opposed to compatible trials in which both task relevant and irrelevant stimulus dimensions are associated with the same response alternative (e.g., colour naming when the word "BLUE" is

written in blue ink). Furthermore, Stroop interference effects are modulated dynamically as a function of conflict experienced in the previous trial: The Stroop effect is typically reduced following high compared to low conflict trials. This congruency sequence effect (CSE) is commonly explained by a conflict adaptation mechanism where the experience of conflict recruits increased attentional control, reducing the influence of irrelevant stimulus dimensions on post-conflict trials (Botvinick et al., 2001; Egner, 2007; Kerns et al., 2004; Ullsperger, Bylsma, & Botvinick, 2005). Thus, in addition to classic Stroop interference, CSEs provide further evidence that cognitive control is reactively up-regulated after processing interference (see Botvinick et al., 2001; di Pellegrino, Ciaramelli & Làdvas, 2007; Duthoo & Notebaert, 2012; Ullsperger et al., 2005; Wühr & Kunde, 2008). Importantly, the CSE has been shown to diminish severely as both inter-stimulus- and response-stimulus-intervals increase (Egner, Ely & Grinband, 2010; see also West et al., 2010; Wühr & Ansorage, 2005), suggesting that CSEs reflect interference resolution processes oriented to the recent experience of conflict, and not to control processes which develop over time in expectation of upcoming conflict (cf. Egner et al., 2010; for similar a suggestion see Alpay, Goerke & Stürmer, 2009). It should be noted, however, that within one computational framework (Dipisapia & Braver, 2006), the CSE might be considered a reactively triggered, short-term increase in proactive control (see Dipisapia & Braver, 2006; Braver et al., 2007; but see Duthoo & Notebaert, 2012; Scherbaum, Dshemuchadse, Fischer & Goschke, 2010).

In relation to depression, studies of Stroop interference have often used blocked designs in which Stroop interference is operationalized as the difference between the time to read a list of incompatible stimuli minus the reading time for

compatible or neutral word lists. While these studies often found increased Stroop interference in depressed groups (e.g., Moritz et al., 2002; Trichard et al., 1995; but see Egeland et al., 2003), it is important to note that proactive control can dominate performance in circumstances where upcoming conflict is entirely predictable (De Pisapia & Braver, 2006). Conversely, studies employing the randomised presentation of compatibility levels within blocks have often failed to find depression realted differences in Stroop interference (e.g., Holmes & Pizzagalli, 2007; Meiran, Diamond, Toder & Nemets, 2011; Wagner et al., 2006, but see Holmes & Pizzagalli, 2008). Addittionally, randomised designs permit the investigation of the CSE. While some studies have reported impaired CSEs in depressed groups using the classic Stroop task (Holmes & Pizzagalli, 2007, Meiran et al., 2011), many others have not found such depression-related impairments in a variety of conflict paradigms (*Stroop-type*: Holmes & Pizzagalli, 2008; West et al., 2010, *Simon task*: Holmes & Pizzagalli, 2007; Ng, Chan & Schlaghecken, 2012; *Flanker task*: Pizzagalli, Peccoralo, Davidson, & Cohen, 2006). Finally, it is important to note that many of these studies also biased the ratio of compatible to incompatible stimuli, altering both task difficulty and conflict-expectancy (Holmes & Pizzagalli, 2007; Holmes & Pizzagalli, 2008; Meiran et al., , 2011; West et al., 2010). Thus, the current study investigated the operation of reactive control processes (Stroop interference, CSEs) as a function of increasing depressive symptomatology when trial compatibility levels are equiprobable and presented in a randomised order. For these purposes we used the classic colour-word Stroop task due to its prevalence in the existing literature, allowing comparison with existing results.

While the classic Stroop task provides a reliable laboratory paradigm to investigate individual differences in attentional control, the task cannot inform us on

the ability to detect and resolve conflict which arises from affective environmental distractors. Depression has consistently been associated with impairments in tasks which require the regulation, inhibition or manipulation of affective content (Gotlib & Joormann, 2010; Joormann, 2004; Koster, De Raedt, Goeleven, Frank & Crombez, 2005), thus, it might be predicted that elevated depression would be associated with an impaired ability to resolve interference from affective distractors. The emotional-face Stroop task (Etkin, Egner, Kandel & Hirsch, 2006) is a laboratory protocol specifically designed to investigate such affective conflict. In this paradigm participants must selectively respond to a relevant emotional stimulus dimension (affective face) whilst avoiding the processing of irrelevant, sometimes conflicting, emotional distractors (affective word). Thus, akin to the classic Stroop task, conflict occurs when relevant and irrelevant stimulus dimensions are in semantic opposition (e.g., negative facial expression; word: “HAPPY”). Additionally, CSEs similar to those observed in the classic Stroop task occur in the emotional-face Stroop task, reflecting the ability to resolve emotional conflict on a trial-to-trial basis. Important for current concerns, past research suggests that a distinct mechanism facilitates executive, top-down control over this emotional conflict by resolving processing interference caused by simultaneously activated affective representations (Egner et al., 2008; Etkin et al., 2006; Etkin et al., 2011; Maier and di Pellegrino, 2012; Monti, Weintraub and Egner, 2010; Soutschek & Schubert, 2013). Given that depression has previously been related to particular impairments in control adjustments when tasks or events require the regulation, inhibition and manipulation of affective information processing (e.g., Elliott, Sahakian, Herrod, Robbins & Paykel, 1997; Elliott, Sahakian, McKay, Herrod, Robbins, & Paykel, 1996; Gotlib & Joormann, 2010; Holmes & Pizzagalli, 2007; Joormann et al., 2011), we hypothesised that

increased depressive symptoms would be associated with particular impairment in the trial-to-trial resolution of interference arising from affective distractors.

Unlike reactive control, much less is known about the relationship between sustained, proactive control processes and depression. In a recent ERP study, West et al. (2010) explored proactive control processes as a function of increasing negative affect. The authors observed that during a counting Stroop task, their electrophysiological measure of proactive control (the pre-stimulus slow wave) was reduced. From these results, West et al. (2010) concluded that proactive processes were impaired as a function of increasing depressive symptoms. It is important to note, however, that no behavioural difference was related to the level of depressive symptoms in their study. In order to provide a behavioural measure of preparatory, sustained control processes, we instructed participants to prioritise either speed or accuracy during task performance. Importantly, many authors consider the ability to adjust response-thresholds in order to trade-off speed for accuracy and vice versa to be a fundamental component of cognitive control (e.g., Botvinick et al., 2001; Jentzsch & Leuthold, 2006; Jones, Cho, Nystrom, Cohen, & Braver, 2002; Laming, 1979; Saunders & Jentzsch, 2012; Verbruggen & Logan, 2009). In contrast to the transient modification of control settings observed in reactive control, successful compliance with externally provided Speed-Accuracy Trade-off (SAT) instructions requires the proactive modulation of response strategy over a number of trials. Furthermore, although several models of SAT exist, almost all theories suggest that such trade-offs are created by adapting the response threshold of decision-related mechanisms to create either more or less conservative responding (see Bogacz, Wagenmakers, Forstmann & Nieuwenhuis, 2009; van Veen, Krug & Carter, 2008; Wenzlaff, Bauer, Maess, & Heekeren, 2011). Therefore, in line with previous

suggestions (West et al., 2010), we hypothesised that increased depressive symptoms would be related to the impaired implementation of these explicit, proactive adjustments.

Method

Participants

72 individuals (mean age = 21.4, S.D. = 3.1, 53 females), recruited from the University of St Andrews subject pool (~50% answering advertisement seeking "low mood" participants), participated for course credits or cash reimbursement (£5/hour). Nine participants were excluded due to either non-compliance with experimental procedure (3 participants); withdrawal before study completion (1 participant) or currently taking psychoactive medication (5 participants).

Three groups were formed depending on Beck Depression Inventory-II (BDI-II; Beck, Steer & Brown, 1996) score. The low BDI-II group included all participants scoring below 7 (25 participants; mean BDI-II = 2.7, range = 0-6). Participants scoring ≥ 17 were included in the high BDI-II group (19 participants; mean BDI-II = 25.7, range = 18-38) as this score yields a high true-positive rate (see Beck et al., 1996). Importantly, this method ensured that our highest symptom group had similar mean BDI-II scores as Major Depressive Disorder groups selected by clinical interview in similar experiments (e.g, Holmes & Pizzagalli, 2008). While previous research has often discarded medium scorers (e.g., Compton et al., 2008; Watson, Dritschel, Jentzsch, & Obonsawin, 2008) we collected a sufficient number to permit their inclusion in the analyses (19 participants, mean BDI-II = 10.6, range = 7-16). Groups did not differ on mean age, $F(2, 60) = 1.60$, $p > .10$, or gender, $\chi^2(2, N = 63) = 4.08$, $p > .10$. Additionally, the State and Trait Anxiety Inventory-Trait (STAI-T;

Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) served as a measure of trait anxiety. For our participants BDI-II scores were highly correlated with STAI-T scores, r = .878, p <.01. All participants gave informed consent, spoke English fluently and were tested in a single session.

Apparatus and Stimuli

The stimuli were presented centrally on a 17-in. CRT monitor controlled by an IBM-compatible personal computer. Two response keypads were used with one keypad assigned to each participant. Each keypad had two response keys, mounted 15 cm apart in the horizontal plane of the participant. The classic Stroop task consisted of the words 'BLUE' and 'RED' presented in either blue or red font. The emotional-face Stroop comprised 16 face stimuli (4 male, 4 female, showing either happy or sad expressions; Ekman & Friessen, 1976). Images were masked to remove hair, body and background details. Either the word "HAPPY" or "SAD" was printed beneath each face. All words were presented in capital letters, each letter measuring approximately 10 x 7 mm. The masked faces measured approximately 50 x 30 mm.

Procedure

Task (classic or emotional) and SAT instruction (fast or accurate) was manipulated within-subjects and between blocks (4 block conditions). Each block condition contained 384 trials (with rests after every 96 trials). Half of the participants first completed the classic Stroop task, the remainder began with the emotional-face Stroop. SAT instructions were balanced across participants within each task. In the classic Stroop task participants were instructed: "*Please respond to the PRINTED colour of the words as ACCURATELY as possible".* For blocks in which speed was instructed the word "ACCURATELY" was replaced with "QUICKLY". In the emotional-

face Stroop task the instructions were: "*Please respond to the EMOTION expressed by the face as… QUICKLY* [or] *ACCURATELY…as possible".* After being instructed participants completed 10 practice trials per condition and then completed the main experimental blocks.

Participants were seated in a darkened testing booth approximately 80cm from the computer screen. Responses were made with left and right key presses using the left and right index fingers, respectively. Responses were made to the font colour of the word (classic Stroop task) and to the emotional expression of the face (emotional-face Stroop task). Assignment of targets to response alternatives was balanced across participants. Stimuli were presented until response (max. 1500 ms), after which a fixation point appeared for 1650 ms between trials. Lastly, the BDI-II and STAI-T were administered.

Data Analysis

Only RTs between 100 and 1500 ms in trials N-1, and N were considered correct. Trials with missing, too slow or too fast responses (< 1%) in trials N-1 and/or trial N were discarded. Error responses in trial N or N-1 were discarded from RT analyses.

An initial omnibus ANOVA revealed an interaction between Task, Group, Previous Compatibility and Current Compatibility, $F(2, 60) = 4.00$, $p = .023$, $\eta p^2 = .12$, suggesting that performance in the two paradigms was not equal across groups. Thus, subsequent analyses were conducted for each task separately. Importantly, in order to report the critical speed-accuracy manipulation we retained the SAT factor in the task-wise analyses. Consequently, RTs and choice-error rates were subjected to separate repeated measures ANOVAs for the emotional and classic Stroop tasks[1]. The within-subjects variables were SAT instructions (speed vs. accuracy); Previous

Compatibility (compatible vs. incompatible); and Current Compatibility. The between-subjects factor was Group (Low, Medium, High BDI-II score). Bonferroni corrected *p*-values are presented for post-hoc tests.

Results

Classic Stroop task

RTs: Responses were faster under speed (366 ms) than accuracy instructions (427 ms); $F(1, 60) = 61.09$, $p < .001$, $\eta_p^2 = .50$. Importantly, this difference was not modulated by group, $F(2, 60) < .70$, $p > .50$ (see Figure 1).

RTs were higher for incompatible (405 ms) than compatible trials (388 ms) $F(1, 60) = 56.15$, $p < .001$, $\eta_p^2 = .48$ and this compatibility effect was larger under accuracy than speed instructions, $F(1, 60) = 16.16$, $p < .001$, $\eta_p^2 = .21$. RTs also showed a main effect of previous compatibility, $F(1, 60) = 5.57$, $p = .022$, $\eta_p^2 = .09$, due to slightly faster RTs after compatible than incompatible trials. The effect of current compatibility did not interact with group, $F(2, 60) < .03$, $p > .97$, indicating that Stroop effects (incompatible *minus* compatible) did not differ as a function of group in the classic Stroop task.

Importantly, previous and current trial compatibility interacted, $F(1, 60) = 28.77$, ps $< .001$, $\eta_p^2 = .34$. That is, the Stroop effect was smaller following incompatible (11 ms) than compatible trials (25 ms). This congruency sequence effect (CSE) was further modulated by SAT, $F(1, 60) = 10.96$, $p = .002$, $\eta_p^2 = .15$; the CSE was present only under accuracy, $F(1, 60) = 30.26$, $p < .001$, $\eta_p^2 = .34$, but not speed instructions, $F(1, 60) = 3.69$, $p = .12$, $\eta_p^2 = .06$. Importantly, the CSE did not interact with group, $F(2, 60) = .16$, $p > .85$[3] (this was also the case when CSE analyses only included the accuracy condition: Group x CSE, $F < 1$, ns.). Therefore, groups did not differ in their

ability to make these reactive cognitive control adjustments, see Figure 2 (gray bars), and Table 1. No other effects were significant.

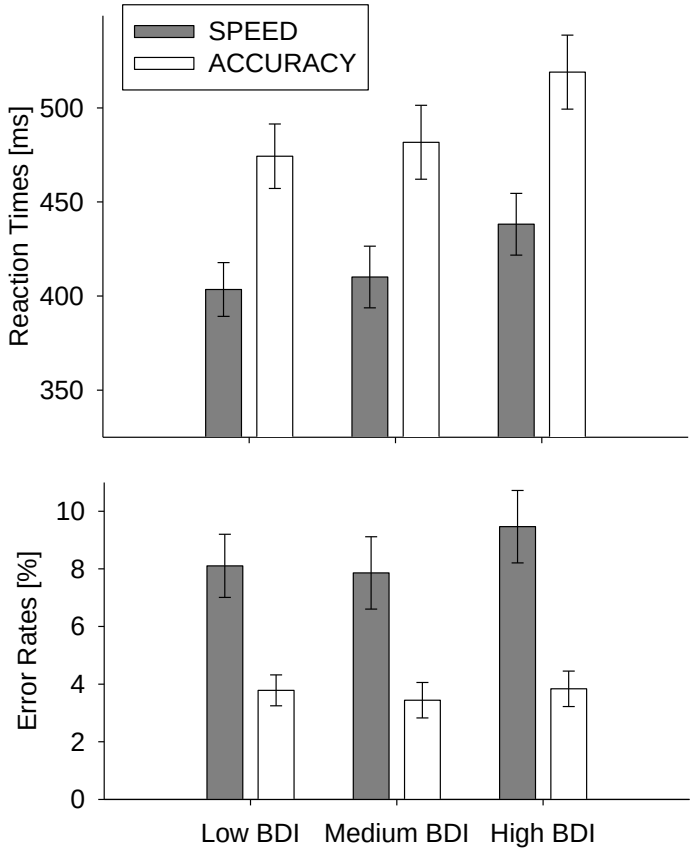

Figure 1: Mean RT (top) and error rates (bottom) as a function of SAT instruction (Speed vs. Accuracy) and Group. Error bars depict S.E.M. As no group differences emerged in the SAT effect for either task, means were collapsed across tasks for purposes of these figures only.

Choice error rates: Responses were more error prone under speed (6.2%) than accuracy instructions (2.6%); $F(1, 60) = 40.65$, $p < .001$, $\eta_p^2 = .40$. Together with the RT findings, this result confirms that our manipulation of SAT was successful in the classic Stroop task. Importantly, this SAT effect was not modulated by group, $F(2, 60) = .77$, $p > .47$, suggesting the high BDI-II group had no detectable impairment in implementing these proactive adjustments (see Figure 1).

Error rates were higher for incompatible (5.1%) than compatible trials (3.7%), $F(1, 60) = 15.69$, $p < .001$, $\eta_p^2 = .21$. This Stroop effect did not interact with group,

$F(2, 60) = .71$, $p > .49$. Importantly, previous and current trial compatibility interacted, $F(1, 60) = 5.94$, $p = .018$, $\eta_p^2 = .09$; the Stroop effect was smaller following incompatible (0.9%) than compatible trials (2.0%). As with RTs, this CSE did not interact with group, all $F(2, 60) = 1.23$, $p > .49$ (see Table 1). No other effects reached significance.

Table 1 : Summary of mean RTs [ms] and choice error rates [%] in the Classic Stroop task

		BDI-II GROUP					
		LOW		MEDIUM		HIGH	
Instruction	Seq.	RT [ms]	errors [%]	RT [ms]	errors [%]	RT [ms]	errors [%]
Speed	cC	349	6.5	348	4.9	381	6.3
	cI	358	8.7	365	5.9	396	8.1
	iC	349	7.6	353	5.0	381	6.5
	iI	358	6.3	365	4.4	384	8.6
Accuracy	cC	405	2.3	392	1.5	439	1.5
	cI	443	4.4	425	3.3	474	3.8
	iC	408	2.7	398	1.8	448	2.4
	iI	421	3.8	410	2.6	464	3.1

Note. 'Seq.' denotes trial compatibility sequence, lower case i/c and upper-case I/C indicate the compatibility level on trial N-1 and N, respectively

Emotional-face Stroop task

RTs: Responses were faster under speed (469 ms) than accuracy instructions (556 ms), $F(1, 60) = 71.85$, $p < .001$, $\eta_p^2 = .55$, however, this effect did not interact with group, ($F < 1$, $p > .10$; see Figure 1). RTs were higher for incompatible (523 ms) than compatible (502 ms) trials, indicating the presence of a Stroop effect (incompatible *minus* compatible), $F(1, 60) = 60.10$, $p < .001$, $\eta_p^2 = .50$, which was slightly larger under accuracy (24 ms) than speed instructions (17 ms), $F(1, 60) = 5.64$, $p = .021$,

η_p^2 = .09. This compatibility effect did not interact with group, $F(2, 60) = 1.63$, $p > .20$. Previous and current trial compatibility tended to interact, $F(1, 60) > 2.87$, $p = .095$, η_p^2 = .05, indicating the presence of CSE effect. This CSE further interacted with SAT, $F(1, 60) = 5.13$, $p = .027$, $\eta_p^2 = 0.08$; due to control adjustments occurring only under accuracy, $F(1, 60) = 6.68$, $p = .024$, $\eta_p^2 = .10$, but not speed instructions, $F < 1$, $p > .10$.

Importantly for the current hypotheses, BDI-II group interacted with the CSE, $F(2, 60) = 6.26$, $p = .003$, $\eta_p^2 = 0.17$[4], indicating that emotional conflict resolution differed between groups. Although the inclusion of SAT was not strictly justified by a 5 way interaction in the original omnibus ANOVA, it is clear from inspection of the means (see Table 2) that the group difference was restricted to the accuracy instructions. This observation was qualified by a significant 4-way interaction between SAT, Group and the CSE, $F(2, 60) = 4.12$, $p = .021$, $\eta_p^2 = 0.12$[5]. Further post-hoc tests confirmed that the interaction between CSEs and group was only present under accuracy, $F(2, 60) = 8.32$, $p = .002$, $\eta_p^2 = .22$, but not speed instructions, $F(2, 60) = 0.82$, $p = .45$, $\eta_p^2 = .026$. Specifically, CSEs ([Stroop effect after previous compatible trials] minus [Stroop effect after previous incompatible trials]) in the accuracy condition were reduced in the high (-13 ms) compared to both the low (15 ms), $t(42) = 2.62$, $p = .036$, $d = 0.80$, and medium BDI-II group (31 ms), $t(36) = 4.24$, $p < .001$, $d = 1.44$, no difference between the low and medium BDI-II group was found ($p > .10$), see Figure 2 and Table 2. No other main effects or interactions were significant.

Choice error rates: Responses were more error prone under speed (10.7%) than accuracy instructions (4.7%), $F(1, 60) = 60.09$, $p = .001$, $\eta_p^2 = .50$, indicating that our SAT manipulation was also successful in the emotional-face Stroop task. Importantly,

this SAT effect was not modulated by group, (F(2, 60) =.47, p > .62; see Figure 1). Error rates were higher for incompatible trials (9.6%) than compatible trials (5.9%), indicating the presence of a Stroop effect, F(1, 60) = 72.78, p < .001, η_p^2 = .55, which was larger under speed than accuracy instructions, F(1, 60) = 21.38, p < .001, η_p^2 = .26. Error rates also showed a main effect of previous compatibility, F(1, 65) = 5.25, p = .026, η_p^2 = .08 due to very slightly higher error rates after compatible (8.0%) than incompatible (7.4%) trials. Neither the Stroop nor the effect of previous compatibility interacted with group, both Fs(2, 60) < .43, ps > .64. Previous and current trial compatibility interacted in error rates, F(1, 60) > 27.94, p < .001, η_p^2 = .31, indicating the presence of a CSE, however, this did not interact with group, see Table 2. No other main effects or interactions were significant.

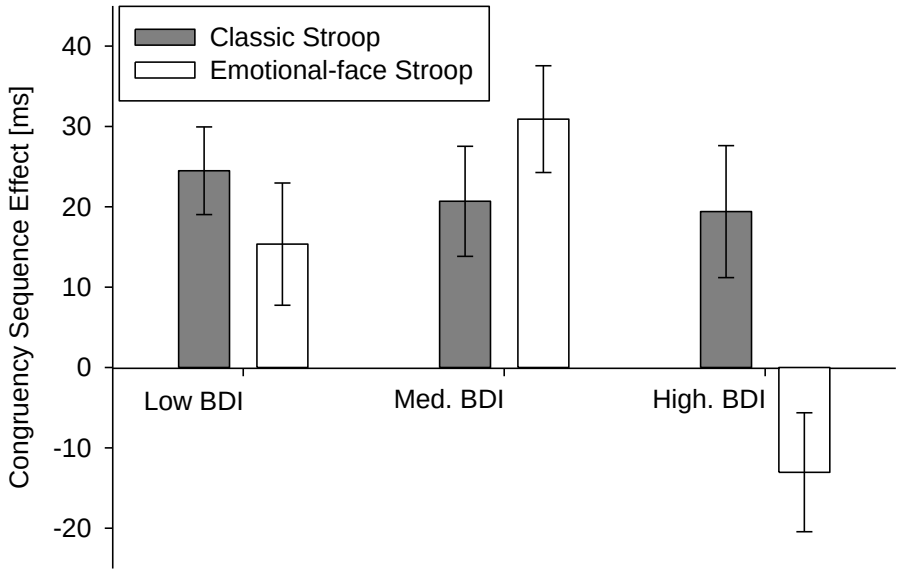

Figure 2: Mean RT CSEs in the accuracy condition of both the classic (gray bars) and the emotional-face Stroop (white bars) task. CSEs were calculated as (Stroop effect after previous compatible trials) minus (Stroop effect after previous incompatible trials), therefore, positive values indicate a reduction in the Stroop effect after conflict. Error bars depict the standard error of the mean.

Table 2: Summary of mean RTs [ms] and choice error rates [%] in the Emotional-face Stroop task

		BDI-II GROUP					
		LOW		MEDIUM		HIGH	
Instruction	Seq.	RT [ms]	errors [%]	RT [ms]	errors [%]	RT [ms]	errors [%]
Speed	cC	448	7.9	450	6.8	488	9.3
	cI	460	13.1	475	13.7	498	16.3
	iC	444	9.1	452	7.0	482	10.2
	iI	463	11.1	473	11.0	495	13.3
Accuracy	cC	513	3.5	532	3.5	572	3.7
	cI	540	7.1	579	6.2	589	8.0
	iC	526	4.3	551	3.6	567	4.2
	iI	537	5.3	567	4.7	597	5.5

Note. 'Seq.' denotes trial compatibility sequence, lower-case i/c and upper-case I/C indicate the compatibility level on trial N-1 and N, respectively.

Discussion

The current study assessed the operation of reactive and proactive cognitive control processes as well as the resolution of emotional conflict as a function of increasing depressive symptoms. The first novel finding of the current experiment was that SAT adjustments did not differ across groups, suggesting the spared implementation of these proactive adjustments across the range of participants included in our study. Secondly, as most aspects of reactive control (Stroop interference in both tasks and CSEs in the classic Stroop paradigm) were unimpaired in all groups, the current results do not support the idea of a general impairment in reactive control in those reporting elevated depressive symptoms. In contrast to these findings, participants in the highest BDI-II group showed selectively impaired CSEs in the affective task. More specifically, while high BDI-II scorers did not differ from low and medium range participants on overall Stroop effects in the emotional-face task, they demonstrated a

highly selective impairment in the ability to resolve conflict arising in this affective paradigm.

Given prior suggestions that negative moods might impair proactive control processes (Braver et al., 2007; West et al., 2010), we predicted that SAT adjustments would be impaired in the high symptom group, however, this was not the case. These results appear to challenge recent interpretations of ERP findings (West et al., 2010) where increasing depressive symptoms were associated with the reduced amplitude of an ERP (pre-stimulus slow wave) relating to proactive control. Importantly, unlike the former study, we gave participants explicit, external performance goals to maintain over time (e.g., “respond as accurately as possible”), allowing preparatory control processes to be quantified behaviourally. Conversely, West et al. (2010) interpreted the reduction of a pre-stimulus slow wave as reflecting impaired proactive control, however, they found no behavioural differences as a function of increasing depressive symptoms. Importantly, pre-stimulus slow waves may be related to both motoric (preparatory activation preceding an imperative stimulus, Leuthold, Sommer & Ulrich, 2004), and non-motoric processes (general anticipatory processes preceding a stimulus, van Boxtel & Bocker, 2004), therefore, a finding of reduced pre-stimulus slow wave alone does not necessarily permit the conclusion that preparatory control processes are compromised in a particular group of individuals.

Secondly, we found little evidence for generally impaired reactive control as a function of increasing depressive symptoms. These results are consistent with previous reports of unimpaired Stroop interference in a range of clinical and subclinical cohorts using non-emotional Stroop paradigms (Holmes & Pizzagalli, 2007; Meiran et al., 2011; West et al., 2010). Similarly, CSEs were unimpaired as a

function of increasing BDI-II score in the classic Stroop task, which is consistent with similar findings reported in other studies (e.g., Holmes & Pizzagalli, 2008; Ng et al., 2012; West et al., 2010). Importantly, when our analyses controlled for potential mnemonic contributors to the CSE (Hommel, Proctor & Vu, 2004; Mayr, Awh & Laurey, 2003) or restricted analyses to post-conflict performance benefits on incompatible trials alone (i.e., RT cI *minus* iI; Holmes & Pizzagalli, 2007), significant and BDI-II group independent CSEs remained, suggesting that control processes made a significant contribution to these effects (see also, Egner, 2007; Ullsperger et al., 2005). Finally, it has recently been suggested that instead of speeding on iI relative to cI trials, CSEs are mainly driven by faster responses on cC compared to iC trials, suggesting that CSEs may be driven mainly by speeding on sequences of conflict-free trials and not adaptations after conflict rich-trials (Schlaghecken & Martini, 2012). However, in the current study we found no evidence for asymmetrical effects of previous conflict on subsequent compatible or incompatible trials[6]. Thus, despite finding robust Stroop interference effects and CSEs, we were unable to find significant depressive-symptom related impairments in reactive control in the classic Stroop task.

Importantly, as previous studies have uncovered impaired CSEs in those with increased depressive symptoms (e.g., Holmes & Pizzagalli, 2007; Holmes & Pizzagalli, 2008; Meiran et al., 2011), it is necessary to consider why discrepant results might occur between investigations. First, regarding our sample, we studied a larger cohort of high symptom scorers than previous investigations which uncovered CSE impairment (Holmes & Pizzagalli, 2007: N = 13; Meiran et al., 2011: N = 9). Furthermore, our High BDI-II sample also reported numerically higher depressive symptoms than sample of Holmes and Pizzagalli (2007). Therefore, simple

variations in sample size and symptom severity between cohorts provide unlikely explanations for the divergence of results across investigations. Similarly, previous studies biased the proportion of conflict trials either towards compatible (Holmes & Pizzagalli, 2007) or incompatible stimuli (Meiran et al., 2011). While we avoided such frequency manipulations as they bias performance towards reactive and proactive control, respectively (Braver et al., 2007; Braver, 2012; De Pisapia & Braver, 2006), studies with such frequency manipulations have also often failed to find general depression-related CSE impairment in the classic Stroop task (Holmes & Pizzagalli, 2008), the Simon task (Holmes & Pizzagalli, 2007) and a counting Stroop task (West et al., 2010). Thus, given the results from both our study and that of prior investigations, depressive symptomatology does not appear to be associated with particularly robust or easily replicable impairments in reactive cognitive control.

Most important, however, for present purpose, we found the CSEs to be reduced in the highest depressive symptom group in the emotional-face Stroop task compared to participants with low levels of depressive symptoms. Interestingly, affect-specific mechanisms are proposed to underlie the top-down regulation of interference caused by emotional distractors (Etkin et al., 2006; Egner et al., 2008; Maier et al., 2012; Soutscheck & Schubert, 2013). Therefore, a specific impairment in these affective-regulatory processes might explain the observed differences between groups in our study. In line with this hypothesis, Holmes and Pizzagalli (2007) reported CSE impairments especially for elevated BDI-II scorers when blocks were preceded by negative emotional feedback. Therefore, converging evidence appears to suggest that impaired reactive control adjustments may occur as a function of increasing depressive symptomatology selectively in tasks which require the concurrent processing of affective information. On a neuroanatomical level,

evidence (Etkin et al., 2006; Egner et al., 2008; Maier et al., 2012) suggests that the functioning of the rostral ACC is central to the resolution of emotional conflict in the emotional-face Stroop task (see also Etkin et al., 2011). Interestingly, studies have observed the dysfunction of the rostral ACC in depressed groups in tasks which require the inhibition of affective information processing (e.g., Eugène, Joormann, Cooney, Atlas, & Gotlib, 2010; Mitterschiffthaler et al., 2008). In light of these prior findings, it seems plausible that the impairment observed in our high BDI-II group may stem from the dysfunction of this neural structure. Future functional neuroimaging research should aim to clarify this relationship. It is also important to note that despite frequently being associated with the dysfunction of the neural correlates of cognitive control (George et al., 1997; Holmes & Pizzagalli, 2008; Pizzagalli, 2011; West et al., 2010), studies often do not find behavioural differences between groups on classic conflict tasks. Therefore, it seems imperative that future research aims to account for which neural abnormalities correlated with depressive symptoms are predictive of measurable impairments.

Some differences between the classic and emotional-face Stroop are worthy of discussion when evaluating the effects of depressive symptomatology on CSEs in each task. As stimulus repetitions occurred more frequently in the classic compared to the emotional-face Stroop, the spared ability to make control adjustments across BDI-II range in the classic Stroop may in part be attributable to the higher number of exact stimulus repetitions in this paradigm. Importantly, group differences did not emerge on the classic Stroop even when such repetitions were excluded. Therefore, stimulus-specific priming effects cannot explain spared CSE in the high BDI-II group. Additionally, RTs and error-rates were generally higher in the emotional than classic Stroop task, indicating that the emotional-face Stroop was more difficult. Thus, group

differences perhaps emerge on the more difficult task, irrespective of emotion. As an alternative to the classic Stroop, a “matched” non-emotional face-Stroop task (i.e., naming the sex of a face with either compatible/incompatible irrelevant words) may be recommended to resolve this problem. However, existing research comparing such non-emotional and emotional face-Stroop performance has consistently found increased RT and error rates in the emotional compared to the non-emotional task (see Egner et al., 2008; Maier et al., 2012; Monti et al., 2010; Soutschek & Schubert, 2013, see also similar differences occur in the non-emotional vs. emotional flanker task, Samanez-Larkin, Robertson, Mikels, Carstensen, & Gotlib, 2009). Thus, regardless of their apparent similarities, emotional interference tasks may be generally more difficult than their non-emotional counterparts. Importantly, while demonstrating impaired CSEs in the emotional-face Stroop task, overall RTs, error rates, and Stroop effects did not differ between the High BDI-II and other participant groups, suggesting that the impairment we observed was highly specific to interference resolution and not to a general impairment in task performance.

As a final point, some characteristics of our sample must be noted when relating the current results to existing literature. Firstly, BDI-II scores were highly correlated with trait anxiety (STAI-T), meaning that it is currently not possible to make very strong distinctions between a depression-specific result and one driven by general negative affect (common to both depression and anxiety). Such correlations between depressive and anxious symptoms are common, and clinical depression uncomplicated by such comorbidity is particularly rare (see Hirschfeld, 2001). Thus, our high BDI-II group is not unrepresentative of typical cohorts with increased depressive symptoms. Additionally, as we used a sub-clinical sample some differences not apparent in our experiment may only emerge in more severely

depressed or clinical samples. However, although sub-clinical, our high BDI-II group reported moderate to severe depressive symptoms (Beck et al., 1996). Thus, given that BDI-II scores correlate highly with other measures used to generate clinical diagnoses of depression in a number of samples (Beck et al., 1996; Sprinkle et al., 2002), our data suggests that impairments in reactive control might only occur in those reporting very severe symptoms. Secondly, it has been demonstrated that antidepressant medication can have a profound effect on emotional information processing (see Merens, Van der Does & Spinhoven, 2007). As none of the participants in the current experiments were taking anti-depressant medication at the time of testing, emotional processing was studied un-confounded by concurrent medication status. This factor may explain differences between our study and that of De Lissneyder et al., (2012) who found general cognitive control impairments in the Internal Shift Task in a clinical sample with no emotion-specific impairment, however, their study included a highly medicated depressed group (85%), perhaps explaining the lack of emotion-specific effects.

In conclusion, the current results provide important insights into the operation of executive processes as a function of increasing depressive symptoms. Firstly, to our knowledge the current study is the first to report that the maintenance of proactive speed-accuracy trade-offs are unimpaired across a range of depressive symptoms. Secondly, as most aspects of reactive control (e.g., Stroop interference and CSEs in the classic task) were unimpaired in all groups, the current findings do not support the idea of generally impaired reactive control in depression. Importantly, high BDI-II scorers demonstrated a selective impairment in the ability to resolve emotional conflict. Together with a prior report of reduced post-conflict adjustments in depression when the task included negative emotional feedback (Holmes &

Pizzagalli, 2007), converging evidence appears to suggest that reactive, trial-to-trial control impairments occur in depression particularly when task demands require concurrent emotional information processing. Future work should investigate the nature of such emotion-cognition interactions in relation to vulnerability to the onset and subsequent duration of depressive episodes.

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Footnotes

1. As depression has been associated with mood-congruent attentional biases, it may be suggested that elevated BDI-II groups would have higher interference effects than the lower BDI-II participants when the conflicting, irrelevant word in the emotional-face Stroop task is negative (e.g., positive face, negative word compared with positive face, positive word), but not when positive (negative face, positive word compared with negative face, negative word). However, we found no differences between groups on interference effects when distractor words were negative (ps > .10) or positive (ps > .10). Similarly, when previous target emotion and current target emotion were entered as factors in the analyses we found no emotion-specific influence on the CSEs (all ps > .10).

2. CSEs remained when repetitions were excluded, $F = 6.74$, $p = .012$, no interactions with group emerged during this analysis (ps > .10).

3. This effect remained when repetitions were excluded from the analyses, $F = 4.69$, $p = .013$.

4. This four-way interaction persisted when the medium BDI-II group was removed from the analyses, $F(1, 42) = 6.39$, $p = .015$, and when the participants were split into minimal (BDI-II <13) and elevated scorers (BDI-II ≥ 13), $F(1, 61) = 4.17$, $p = .046$, as in Holmes & Pizzagalli (2007).

5. We re-analysed our data using the method of Schlaghecken and Martini (2012), focusing on the critical response alternation conditions. Importantly, we did not find any significant interactions between compatibility sequence and current compatibility for either task (all *p*s > .10). Therefore, significant support for context adaptation did not exist within our current data.