CORTEX 70 (2015) 137–145

Available online at www.sciencedirect.com

ScienceDirect

Journal homepage: www.elsevier.com/locate/cortex

Special issue: Research report

Neural mechanisms of proactive and reactive cognitive control in social anxiety

Petra C. Schmid [a], Tali Kleiman [b] and David M. Amodio [a,*]

[a] Department of Psychology, New York University, 6 Washington Place, New York, NY 10003, USA
[b] Department of Psychology, The Hebrew University of Jerusalem, Jerusalem 91905, Israel

ARTICLE INFO

Article history:
Received 15 October 2014
Reviewed 17 February 2015
Revised 1 May 2015
Accepted 27 May 2015
Published online 16 June 2015

Keywords:
Social anxiety
Cognitive control
N2r
Prefrontal cortex
Anterior cingulate cortex

ABSTRACT

Social anxiety—the fear of social embarrassment and negative evaluation by others—ranks among people's worst fears, and it is often thought to impair task performance. We investigated the neurocognitive processes through which trait social anxiety relates to task performance, proposing a model of the joint contributions of reactive control, theoretically associated with conflict monitoring and activity of the dorsal anterior cingulate cortex (dACC), and proactive control, theoretically associated with top-down regulation and activity of the dorsolateral prefrontal cortex (dlPFC). Participants varying in their degree of trait social anxiety completed the Eriksen flanker task while electroencephalography (EEG) was recorded. Task-related left dlPFC activity was indexed by relative left prefrontal EEG (inverse alpha), and conflict-related dACC activity was indexed by the N2r component of the event-related potential. Stronger activity in both regions predicted better response control, and greater social anxiety was associated with worse response control. Furthermore, for all participants, greater left prefrontal EEG activity predicted better behavioral control, but for high social anxiety participants only, greater N2r responses also predicted behavioral control. This pattern suggests that low social anxiety individuals engaged a proactive control process, driven by dlPFC activity, whereas high social anxiety individuals relied additionally on a reactive control process, driven by conflict-related dACC activity. These findings support a model of control that involves different patterns of interplay between proactive and reactive strategies and may help to explain self-regulatory impairments in social anxiety.

1. Introduction

When people are asked to rank their greatest fears, the fear of public embarrassment often tops the list. According to some popular surveys, fear of public speaking even outranks fear of one's own death (Croston, 2012). Considering the importance of community support and social standing for human survival, the desire to avoid embarrassment, criticism and social rejection should not be surprising (Williams, 2007). What is ironic, perhaps, is that social anxiety, born out of the need to perform well in front of others, is often thought to undermine

* *Corresponding author*. Department of Psychology, New York University, 6 Washington Place, New York, NY 10003, USA.
E-mail address: david.amodio@nyu.edu (D.M. Amodio).
http://dx.doi.org/10.1016/j.cortex.2015.05.030

performance, especially on relatively difficult tasks that require a high degree of cognitive control.

Although the notion of "choking under pressure" has received empirical support (e.g., Baumeister, 1984; Beilock & Gray, 2007; Wittchen, Fuetsch, Sonntag, Mueller, & Liebowitz, 2000), the extent to which individuals' degree of dispositional social anxiety affects performance on tasks that require cognitive control has received less attention. This is an important question, as cognitive control—the process that governs one's ability to adapt to changing environments while maintaining goal directed behavior—is required in many situations in daily life. In the present research, we asked whether social anxiety is associated with impaired cognitive control and, to the extent that it is, whether different mechanisms of control are recruited depending on individuals' level of social anxiety when performing a task that requires cognitive control. To this end, we examined the relationship between trait social anxiety and cognitive control performance, and compared the roles of two major neural substrates of cognitive control—the left dorsolateral prefrontal cortex (dlPFC) and the anterior cingulate cortex (ACC), assessed using electroencephalography (EEG)—in individuals reporting relative high and low degrees of social anxiety.

1.1. *Cognitive control: psychological mechanisms and neural substrates*

People often encounter situations where they must override a dominant response in order to behave in an intended manner. Whether this involves sticking to one's diet despite a tasty dessert offering or treating someone fairly without the bias of implicit stereotypes, cognitive control is often critical to the pursuit of personal goals (Amodio et al., 2004; Devine, 1989; Heatherton, 2011). In the laboratory, cognitive control is typically investigated using response conflict paradigms such as the Stroop (Stroop, 1935) or flanker (Eriksen & Eriksen, 1974) tasks. These tasks manipulate the need for cognitive control by creating situations in which the attainment of a task goal is sometimes disrupted by task irrelevant distractors. For example, in the flanker task, participants are required to quickly and accurately identify a target letter placed in the middle of a letter string. The target stimulus is surrounded by non-target stimuli, which correspond either to the same response as the target (congruent trials; e.g., HHHHH) or to the alternative response (incongruent trials; e.g., SSHSS). Incongruent trials (but not congruent trials) elicit response conflict, and enhanced control is required to override the countervailing tendency in order to deliver an intended (i.e., correct) response.

A dominant neurocognitive model proposes that control involves two main components: conflict monitoring and response implementation (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Botvinick & Cohen, 2014). *Conflict monitoring* is the process by which conflict between higher-level goals and lower-level response tendencies is detected, and both fMRI and ERP studies have linked this process with activity in the dorsal ACC (dACC; e.g., Kerns et al., 2004). As conflict levels rise, the dACC is believed to increase its signaling to regions of the dorsolateral prefrontal cortex (dlPFC). The dlPFC supports goal representation and response selection, and aids in exerting top-down *regulative control* on behavior. This process generally describes a *reactive control* process, such that control is engaged in reaction to the detection of conflict (Botvinick et al., 2001; Braver, 2012).

Alternatively, control may be driven by a top-down goal-directed strategy, associated with expectancy, preplanning, and vigilance, that may operate independently of the conflict monitoring process. This *proactive control* process is associated with activity in the dlPFC but not the dACC (Amodio, 2010; Braver, 2012). The proactive/reactive framework described above builds on models that distinguish between the early selection of an intended response strategy and a late correction process that is triggered only when a response conflict is experienced (e.g., Botvinick et al., 2001; Gratton, Coles, & Donchin, 1992; Jacoby, 1991). A consideration of these two forms of control, and their neural substrates, has been useful for explaining why some individuals tend to succeed or fail in self-regulation on tasks requiring cognitive control (e.g., Amodio, 2010; Amodio, Devine, & Harmon-Jones, 2007; Amodio, Master, Yee, & Taylor, 2008).

1.2. *Social anxiety and cognitive control*

The existing links between social anxiety and impaired task performance, reviewed above, suggests that trait social anxiety may be associated with impairment in aspects of cognitive control. To date, research addressing this hypothesis has focused on the degree to which socially-threatening stimuli interfere with task performance, as compared with non-threatening stimuli. The general finding of this work is that socially anxious people perform worse in the presence of socially-threatening distractors compared with healthy controls (Amir, Freshman, & Foa, 2002; Becker, Rinck, Margarf, & Roth, 2001; Grant & Beck, 2006; Lundh & Öst, 1996; Maidenberg, Chen, Craske, Bohn, & Bystritsky, 1996). Although these studies revealed that highly socially anxious individuals are particularly sensitive to socially threatening distractors, they did not address our more general question of whether social anxiety is associated with worse cognitive control, and whether individuals with high versus low social anxiety tend to rely on different forms of control when performing tasks that entail response conflict.

Insights related to these questions come from research in cognitive neuroscience that has begun to examine the roles of ACC and dlPFC activity in control processing among anxious individuals. In particular, conflict-related ACC activity has been associated with some forms of anxiety. For example, patients with obsessive-compulsive disorder exhibited larger amplitudes of the error-related negativity (ERN) component of the ERP (which is primarily generated in the dACC) than non-anxious controls while responding to incongruent trials of the Stroop task (Gehring, Himle, & Nisenson, 2000; Hajcak & Simons, 2002; Soenke et al., 2001). This pattern has also been observed among individuals high in general anxiety (Hajcak, McDonald, & Simons, 2003; Moser, Moran, Schroder, Donellan, & Yeung, 2013). In the same vein, trait and state anxiety have been related to stronger conflict-related ACC activity, as assessed by the N2 ERP component during completion of the Go/No-Go task (Righi, Mecacci, & Viggiano,

2009). More relevant to the present research, Amodio, Master, et al., 2008 found larger No-Go N2 amplitudes among individuals reporting higher levels of behavioral inhibition sensitivity (BIS; Gray, 1982). In that study, BIS was assessed using Carver and White's (1994) popular questionnaire, in which items primarily reflect social anxiety concerns. These data suggest the possibility that conflict processing may play a greater role in response control among individuals with relatively greater social anxiety.

In another line of research, decreased left PFC activity has been linked to higher general trait anxiety (Davidson, Marshall, Tomarken, & Henriques, 2000) and to greater physiological reactivity in response to a social stressor (Master et al., 2009). Consistent with these findings, approach motivation—an orientation often associated with low anxiety—has been linked to heightened activity in the left dlPFC (Davidson, 1992; Harmon-Jones, 2003). This association has been observed between both trait and state assessments of approach motivation and relative left frontal EEG activity (Coan & Allen, 2003; Harmon-Jones & Sigelman, 2001). Although this body of work has not directly compared the roles of PFC and ACC activity in the context of social anxiety, these findings are consistent with the idea that lower trait anxiety may be associated with a relatively greater role of left PFC activity during task performance.

In light of prior research on the roles of the ACC and PFC in cognitive control, and their relations with anxiety, we propose that the distinction between PFC-mediated regulative control and ACC-mediated conflict monitoring can inform the effect of social anxiety on cognitive control. That is, whereas relatively low-anxiety individuals may rely primarily on PFC-mediated control processes for successful performance on a cognitive conflict task, highly socially anxious individuals would be more likely to rely on ACC-mediated control processes. Furthermore, we expect higher social anxiety to be associated with worse task performance, consistent with the proposal that a proactive control strategy is more efficient than a reactive strategy. These observations would provide a theoretical model for performance impairments associated with social anxiety and offer a new perspective on the interplay of ACC and PFC processes in cognitive control.

1.3. *Study overview*

The present study was designed to determine whether high dispositional social anxiety is associated with worse cognitive control and to examine the neurocognitive mechanisms involved in this effect. Participants ranging in their degree of social anxiety completed a response conflict task—a version of the Eriksen flanker task—while EEG was recorded. This EEG measure provided continuous indices of both dlPFC engagement throughout the task, as indicated by left frontal EEG asymmetry during task performance (Amodio, 2010), as well as trial-by-trial indices of conflict-related ACC activity, as indexed by the response-locked N2 component of the ERP (Amodio, Devine, & Harmon-Jones, 2008; Schmid, Kleiman, & Amodio, 2015).

We focused on the N2 locked to correct responses (N2r; also referred to as N2c, Pritchard, Shappell, & Brandt, 1991; or correct-response negativity (CRN) e.g., Amodio, Devine, et al., 2008), rather than the stimulus-locked N2, because the N2r corresponds more closely to response time than to stimulus onset (Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003; Ritter, Simson, Vaughan, & Friedman, 1979; Yeung, Botvinick, & Cohen, 2004) and thus the theoretical process of interest—response conflict processing—would be more evident in the N2r than in the more typically-reported stimulus-locked N2 ERP (Folstein & Van Petten, 2008; Nieuwenhuis et al., 2003).

Another frequently studied index of conflict-related ACC activity is the ERN, which represents the processing of a response error. That is, whereas the N2r is associated with response conflict, the ERN is associated with error detection (although early ERP research on conflict processing focused on the ERN, which could be interpreted as reflecting a combination of response conflict and error detection). Although the ERN is less directly relevant to our theoretical question, we added complementary analyses on the ERN in footnote 1. Our main analyses, however, focused on frontal EEG asymmetry and N2r responses, which permitted us to determine the degree of proactive versus reactive control processes engaged by participants during the task, and the extent to which each mode of cognitive control contributed to task performance.

2. Material and methods

2.1. *Participants and procedure*

Thirty-seven undergraduate students (70% females; $Mean_{age}$ = 19.49, SD_{age} = 1.22) participated in the study for course credit. After providing consent, participants were prepared for EEG recording. They then performed the response control task and completed questionnaires assessing social anxiety, personality traits, and demographic information.

[1] Supplementary analyses were conducted on ERN responses to incongruent error trials. ERN responses were scored at Fcz as the peak negative amplitude between stimulus onset and 100 msec after stimulus onset. Note that because error rates were relatively low, ERN scores were computed on the basis of relatively few trials (Olvet & Hajcak, 2009). Analyses revealed the typical ERN effect, such that amplitudes were greater on error trials ($M = -9.43$, $SD = 5.69$) than correct trials ($M = 2.25$, $SD = 3.67$), $t(24) = 11.58$, $p < .001$. A regression analysis, using the model described in the main text, revealed a main effect of social anxiety, such that greater social anxiety was associated with a marginally larger flanker effect in response accuracy, $\beta = .30$, $t = 1.85$, $p = .080$. In addition, greater left frontal asymmetry and ERN amplitudes were both associated with reduced flanker effects (for EEG asymmetry: $\beta = -.59$, $t = 3.91$, $p = .001$, for ERN amplitudes, $\beta = .42$, $t = 2.76$, $p = .012$). However, the social anxiety × EEG asymmetry interaction, $\beta = -.18$, $t = 1.15$, $p = .265$, and the social anxiety × ERN interaction, $\beta = .16$, $t = .98$, $p = .340$, were nonsignificant. When computing the regression model for RT flanker effect, no significant effects emerged, $ps > .243$. Thus, social anxiety did not moderate the contribution of the ERN on response control, a finding that is conceptually consistent with prior research (Amodio, Kubota, Harmon-Jones, & Devine, 2006).

2.2. Response control

The flanker task (Eriksen & Eriksen, 1974) was used as a measure of response control. Participants' goal in this task was to correctly identify the middle letter of a five-letter string. The flanker task includes two types of trials. On congruent trials, the middle letter (target) and the letters flanking it (distractors) are mapped to the same response (HHHHH, SSSSS). On incongruent trials, the target and distractors are mapped to alternative, conflicting responses (HHSHH, SSHSS). Hence, controlled processing is required to respond correctly to the target without being biased by the distractors (Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999; Gratton et al., 1992). Each trial began with a fixation cross appearing in the center of the screen for 800 msec, followed by a letter string (i.e., flanker trial), which remained on screen until response. Responses exceeding 450 msec were followed by a "Too slow!" message. Intertrial intervals were jittered (2000, 2500, or 3000 msec). Participants performed a total of 288 trials across 6 blocks, which included 144 congruent and 144 incongruent trials presented by DMDX software (Forster & Forster, 2003) in unique random trial orders.

During the flanker task, EEG was recorded from F7, F3, Fz, F4, F8, Fcz, Cz, CPz, P7, P3, Pz, P4, P8, and Oz with tin electrodes embedded in a nylon cap (ElectroCap, Eaton, OH), with left earlobe reference ($\Omega < 5$ k). Eye movements were recorded for use in artifact correction. Signals were amplified with a Neuroscan Synamps2 (El Paso, TX) with AC coupling, digitized at 1000 Hz and passed through a .15–100 Hz online filter.

2.3. Social anxiety

The mini-social phobia inventory (Mini-SPIN; Connor, Kobak, Churchill, Katzelnick, & Davidson, 2001) was used to measure social anxiety. Participants were instructed to indicate how much the following problems have bothered them during the past week by using a 5-point Likert scale (0 = not at all, 4 = extremely): "Fear of embarrassment causes me to avoid doing things or speaking to people.", "I avoid activities in which I am the center of attention.", and "Being embarrassed or looking stupid are my worst fears." The Mini-SPIN score is calculated by summing scores on the three items. A Mini-SPIN score of 6 or higher indicates potential risk for clinical social anxiety. The average Mini-SPIN score in normal populations is 2, whereas for individuals clinically diagnosed with social anxiety, it is approximately 9 (Seeley-Wait, Abbott, & Rapee, 2009). The Mini-SPIN is a validated and highly efficient measure; the scale is 90% accurate at diagnosing the presence or absence of a social anxiety disorder (Connor et al., 2001) and it discriminates cases of social phobia as reliably as more extensive measures of social anxiety (Seeley-Wait et al., 2009).

2.4. Personality

Participants filled out the ten-item personality inventory (Gosling, Rentfrow, & Swann, 2003), a validated brief assessment of extraversion, conscientiousness, emotional stability, openness to experiences and agreeableness. Each subscale comprised two items. On each item, participants indicated whether a pair of adjectives describes them accurately on a 7-point Likert scale (1 = disagree strongly, 7 = agree strongly). Analyses focused on the emotional stability subscale, which included the items "I often feel anxious, easily upset" and "I often feel calm, emotionally stable", with the latter reverse-coded. Scores on this subscale provided an index of general trait anxiety.

3. Data processing

3.1. Behavioral data

Accuracy rates and reaction times (RTs) were computed for valid congruent and incongruent trials. Flanker trials were considered valid if responses occurred within a 200–450 msec timeframe. Congruent/incongruent trial difference scores were computed for accuracy and RTs as indices of accuracy and RT-based flanker effects.

3.2. Questionnaires

For the social anxiety measure, internal consistency was acceptable, Cronbach $\alpha = .69$, and the three items were summed following the typical scoring of the Mini-SPIN. Greater values indicate higher degrees of social anxiety. The two items of the emotional stability scale correlated highly, $r = .55$, $p = .004$ and were averaged.

3.3. EEG asymmetry

Offline, EEG was re-referenced to average earlobes and submitted to regression-based blink correction. Two-second epochs were extracted from intertrial intervals prior to each trial and submitted to a fast-Fourier transformation using a 50%-overlapping Hamming window. Alpha power was extracted (8–13 Hz) and natural log-transformed. In line with previous work, alpha asymmetry was computed by subtracting left-sided alpha power (F7) from right-sided alpha power (F8) such that higher values indicate greater left-sided dlPFC activity (e.g., Amodio, 2010; Pizzagalli, Sherwood, Henriques, & Davidson, 2005). EEG asymmetry was used as an index of *proactive* control, which, by definition, should be active in preparation for both congruent and incongruent trial types and thus should represent activity from across all trial types within a task. Thus, frontal EEG asymmetry data were scored from intertrial intervals prior to both trial types.

3.4. N2r responses

To quantify the N2r, blink-corrected EEG was filtered through a 1–15 Hz bandpass to isolate the N2r waveform. To compute the ERPs, we extracted 1200 msec response-locked epochs starting 400 msec before stimulus onset. Average voltage during a baseline period (400–200 msec prior to stimulus onset) was subtracted from the entire epoch, and epochs representing congruent and incongruent trials were averaged separately. Following past research (e.g., Amodio, Devine, et al., 2008; Schmid et al., 2015), and based on visual inspection of the average waveform, the response-locked N2r was scored at Fcz as the peak negative amplitude between 150 and

80 msec prior to response for *correct* responses on incongruent trials. This scoring method captured conflict-related activity associated with successful response control. Because congruent trials are not expected to elicit response conflict, the N2r from such trials is difficult to interpret. Therefore, our index of conflict processing focused on the N2r for incongruent trials only. An additional analysis was conducted including a N2r difference score (N2r on incongruent trials minus N2r on congruent trials, scored during the same time window).

3.5. *Exclusions*

Data were excluded from eight participants with extensive EEG artifacts and impedance problems, one participant with below chance performance, and three with extreme flanker effect scores (values exceeded the 1.5 interquartile range).

4. Statistical analyses

Preliminary analyses included *t*-tests to determine the validity of the flanker task (i.e., whether it elicited greater conflict on incongruent versus congruent trials as indexed by accuracy scores, RT scores, and N2r responses). The main analyses tested the hypothesis that the different neurocognitive components of controlled processing, associated with proactive and reactive control, predict behavioral response control as a function of participants' degree of social anxiety. Social anxiety, EEG asymmetry across trial types, and N2r amplitudes associated with correct responses to incongruent trials were mean-centered and the social anxiety × EEG asymmetry and social anxiety × N2r interaction terms were computed. The mean-centered variables and interaction terms were entered simultaneously into separate regression models predicting flanker effect scores, based on either accuracy or RTs. Complementary analyses on error-related conflict processes, as indexed by the ERN component of the ERP, can be found in footnote 1.

5. Results

5.1. *Preliminary analyses*

A Kolmogorov–Smirnov test indicated that the SPIN scores of our sample were normally distributed, $D = .14$, $p = 20$. The mean SPIN score in our sample was 6.48 ($SD = 2.37$, *Median* = 6.0, *Range* 3–11). Scores of 6 and higher on this scale are interpreted as indicating high risk for clinical diagnosis, and thus our sample included a wide range of social anxiety scores, representing individuals with potential clinical problems of social anxiety as well as those within the low social anxiety range. This range is consistent with research showing that the prevalence of social anxiety is highest among individuals aged 18–24 years, which is the age range of our sample (Somers, Goldner, Waraich, & Hsu, 2006).

Analyses of the flanker task indicated the expected congruency effect: Participants were significantly less accurate on incongruent trials ($M = .81$, $SD = .09$) than on congruent trials ($M = .97$, $SD = .02$), $t(24) = 9.58$, $p < .001$, with congruent trial accuracy near ceiling. Responses were also slower on incongruent trials ($M = 386.14$, $SD = 17.39$) than on congruent trials ($M = 360.98$, $SD = 14.64$), $t(24) = 11.35$, $p < .001$. These results validate the flanker task as successfully eliciting cognitive control in our sample. Additionally, as expected, incongruent trials elicited greater (i.e., more negative) N2r amplitudes ($M = -2.44$, $SD = 2.46$) than congruent trials ($M = -.81$, $SD = 3.11$), $t(24) = 4.76$, $p < .001$, validating the N2r as a neural indicator of conflict processing (see Fig. 1). Furthermore, as expected, frontal EEG asymmetry associated with congruent and incongruent trials correlated highly ($r = .61$, $p = .001$, when computed across correct and error trials and $r = .91$, $p < .001$, when including correct trials only). This validates the frontal EEG measure as an index of proactive control as opposed to reactive control.

5.2. *Main analyses*

Our primary hypothesis regarding the roles of EEG asymmetry and N2r amplitude was tested using the multiple regression model described above. In the primary analysis, in which the outcome was the accuracy-based flanker effect, greater social anxiety was marginally associated with a larger flanker effect (i.e., reduced response control), $\beta = .28$, $t = 1.77$, $p = .093$. This analysis also revealed a contribution of proactive control to response control: greater left-sided EEG activity was associated with smaller flanker effect scores (i.e., better control), $\beta = -.40$, $t = 2.26$, $p = .036$. However, the effect of frontal asymmetry on response control was not moderated by social anxiety, $\beta = .11$, $t = .57$, $p = .576$, suggesting that proactive control processes contributed to performance regardless of participants' degree of social anxiety.

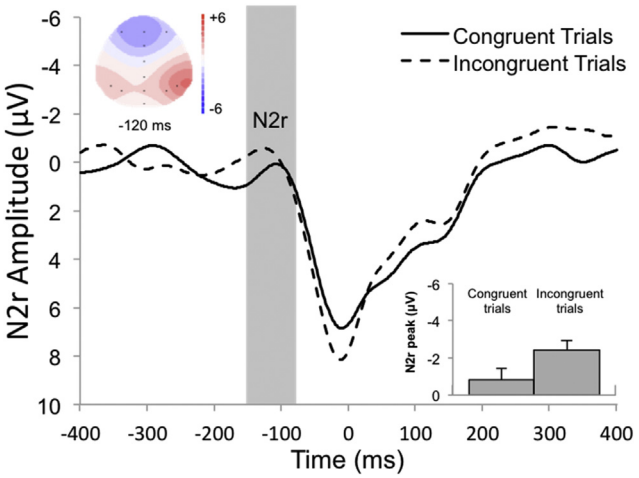

Fig. 1 – N2r amplitudes to congruent and incongruent trials. N2r amplitudes were greater (i.e., more negative) on incongruent than to congruent trials prior to a correct response, suggesting greater involvement of ACC activity in detecting and successfully overcoming response conflict. ERPs on correct trials were locked to response, represented by 0 msec. The shaded area indicates the scoring timeframe.

By comparison, although the main effect of N2r amplitude on flanker scores was nonsignificant, $\beta = .19$, $t = 1.06$, $p = .30$, the predicted Social Anxiety × N2r interaction was significant, $\beta = .39$, $t = 2.08$, $p = .051$ (see Fig. 2). Simple slope analyses (Aiken & West, 1991) revealed that for participants with relative high social anxiety scores, larger N2r amplitudes were associated with a reduced flanker effect (i.e., better response control), $\beta = .59$, $t = 2.10$, $p = .048$, but this was not the case for participants with relatively low social anxiety scores, $\beta = -.08$, $t = .34$, $p = .740$. This pattern suggests a reactive control pattern among participants with relatively high social anxiety but not low social anxiety.

To further test whether relatively high anxiety participants' performance was predicted more strongly by a reactive control process, we conducted a regression analysis in which N2r amplitudes and frontal asymmetry scores were entered simultaneously to predict flanker performance (i.e., simple slope effects with social anxiety scores set to 1 SD above the mean). This analysis revealed a marginal effect of N2r, $\beta = .57$, $t = 2.01$, $p = .079$, but no significant effect of EEG asymmetry, $\beta = -.19$, $t = .66$, $p = .525$. This pattern is consistent with the hypothesis that higher social anxiety individuals may rely more on reactive than proactive control strategies. Given the somewhat worse task performance among relatively high socially anxious participants, these results suggest that a reliance on reactive control, indexed by greater N2r responses was helpful but apparently less effective than a reliance on proactive control, as indexed by left frontal cortical activity.

Social anxiety scores were moderately correlated with emotional stability, an index of general trait anxiety, $r(25) = -.41$, $p = .042$. Therefore, to determine whether the observed effects of social anxiety were distinct from general trait anxiety effects, we conducted the regression model described above using residualized social anxiety scores, from which variance associated with emotional stability was removed. The results of this analysis replicated the key effects of the main analysis, including a significant main effect of EEG asymmetry, $\beta = -.41$, $t = 2.51$, $p = .021$ and the predicted Social Anxiety × N2r interaction, $\beta = .47$, $t = 2.41$, $p = .026$. The main effect of social anxiety on performance did not reach significance in this analysis, $\beta = .20$, $t = 1.26$, $p = .224$, and, as in the primary analysis, all other main effects and interaction effects were nonsignificant, all $ts < 1.72$, all $ps > .102$. Moreover, when entering emotional stability instead of social anxiety as a predictor, only a significant effect of frontal EEG asymmetry emerged. Together, these analyses suggest that the findings reported above represent effects of social anxiety and not general trait anxiety.

Finally, we examined effects on a reaction time-based flanker effect score, using the same regression model described above. This analysis did not yield any significant main effects or interactions, $ps > .143$.

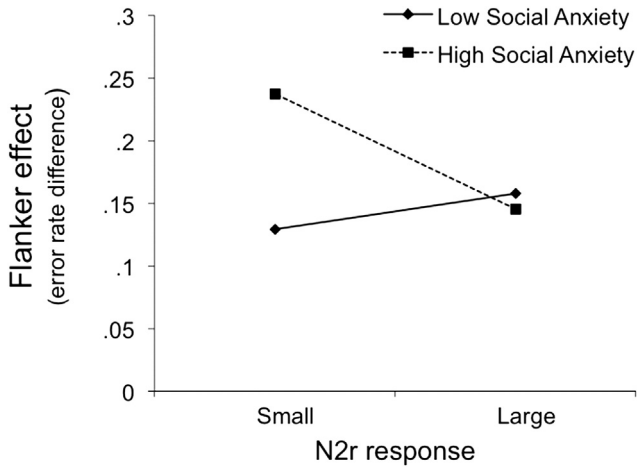

Fig. 2 – Effects of N2r amplitude on the flanker interference effect, as a function of trait social anxiety score. N2r amplitudes predicted a larger flanker effect (i.e., worse response control) among high social anxiety participants but were unrelated to response control among low social anxiety participants. N2r scores are represented as predicted values for relatively small magnitude (+1 SD, given that the N2r is a negative-going wave) and large magnitude (−1 SD) effects. Low and high social anxiety is represented by predicted values set at −1 and +1 SD from the mean, respectively. The flanker effect represents the difference in accuracy rate between congruent and incongruent trials.

6. Discussion

The goal of this research was to investigate the association between social anxiety and performance on a task requiring cognitive control, and to examine the relative contributions of proactive and reactive control processes, indicated by left prefrontal cortical activity and an ERP index of conflict-related ACC activity, respectively. As expected, participants reporting higher social anxiety performed comparatively worse on the response control task. More importantly, we found that contributions of proactive and reactive control processes to task performance differed as a function of social anxiety: whereas frontal cortical activity predicted better response control for all participants, N2r amplitudes predicted better response control only among participants with relatively greater social anxiety, independent of their degree of emotional stability. In other words, participants with lower social anxiety appeared to rely primarily on proactive control processes to control their responses, whereas participants with higher social anxiety relied primarily on reactive control processes.

These results suggest that people use different control strategies to enhance their performance depending on their levels of social anxiety. The predominant role of frontal cortical activity in response control among participants with relatively low social anxiety is consistent with a proactive control profile, characterized by attentional focus and the early selection of an intended response strategy. By comparison, the more prominent role of N2r amplitude on flanker performance among relatively high social anxiety participants is consistent with a reactive control profile, in which control is engaged only after a conflict is encountered and is driven by enhanced conflict monitoring activity. Together, these findings support a new account of cognitive control impairment in social anxiety based on a proactive/reactive control framework. Moreover, our findings may indicate that at least for some tasks, proactive rather than reactive control may be

more beneficial. Specifically, higher socially anxious individuals in our study, whose performance also relied on reactive control, performed worse on average than lower socially anxious individuals who relied primarily on a proactive control mechanism. These findings and interpretations are in line with social cognitive research on self-control suggesting that proactive strategies are more effective for implementing self-control in various domains (e.g., Amodio, 2010; Kleiman, Hassin, & Trope, 2014; Mendoza, Gollwitzer, & Amodio, 2010).

Social anxiety has been associated with a wide range of impairments in several domains including romantic relationships, education, and work performance (e.g., Wittchen et al., 2000). Despite its high prevalence and severe impact on people's lives, relatively little is known about how social anxiety affects self-regulation. Previous research has mainly focused on how social anxiety draws attention to socially threatening information, in a way that may interfere with performance on some tasks (e.g., Becker et al., 2001; Grant & Beck, 2006; Mattia, Heimberg, & Hope, 1993). It is notable that the task used in our study did not include social cues, such that the observed effects of social anxiety appeared to occur in the absence of social evaluative threat. However, the broader experience of participating in a psychology experiment is social, in the sense that a participant must follow the instructions of a research assistant who is often a member of their peer group (i.e., a fellow student) and attempts to perform the task as directed by the research assistant. Hence, we may interpret the experimental context as a socially-evaluative situation and, consequently, the cognitive control task as an opportunity to be socially evaluated. Considered in this light, it is possible that high anxiety participants devoted greater attention to socially-evaluative aspects of the experimental context, and this led them to rely more on reactive control processes during task performance. It is also possible that under more acute social evaluation, the situational induction of social anxiety would also lead low-anxiety individuals to rely more heavily on reactive control processes when performing a task that requires response control.

In our theoretical analysis, we examined the effect of trait social anxiety on neural mechanisms of cognitive control, which implies that social anxiety had a causal effect on participants' use of control strategies. However, our analyses cannot speak to the issue of causality, and either of direction of causality is plausible. Our analysis, which examined effects of social anxiety on control strategy, is consistent with prior research in which the manipulation of social anxiety was associated with worse response control (e.g., Amodio, 2009; Lambert et al., 2003; Ofan, Rubin, & Amodio, 2014; Richeson & Shelton, 2003) as well as with research showing that interventions that reduce social anxiety are associated with better task performance (e.g., Amir, Weber, Beard, Bomyea, & Taylor, 2008; Rehm & Marston, 1968; Schmid & Schmid Mast, 2013). However, it is also possible that the use of a particular control strategy could itself induce anxiety. For example, individuals who tend to rely on reactive control strategies could develop greater social anxiety due to their experience of relative poor performance. This alternative possibility suggests a new hypothesis for the pathogenesis of social anxiety disorder that could be tested in future research. Because of the correlational nature of the present work, our findings cannot speak to the direction of causality between social anxiety and the use of proactive versus reactive control processes.

The primary limitation of the present research is the relatively small sample, which may have limited our ability to observe more subtle effects while also increasing the possibility that any observed effects could be due to chance. However, several factors bolster our confidence in the reported findings. First, our study was powerful enough to detect a theoretically predicted pattern of effects and thus provided support for the complementary roles of proactive and reactive control as they relate to social anxiety. We were also careful to exclude participants with extreme or clearly invalid scores from the analysis to prevent the disproportionate effect of any one participant on the results. Nevertheless, it is possible that some patterns would emerge more strongly in a larger sample, such as the direct effects of social anxiety and N2r amplitude on response control. Although the present work provides a first look at the interplay of social anxiety with proactive and reactive forms of control, future research with larger and more representative participant samples will be needed to establish and understand these effects further.

7. Conclusions

More broadly, our research provides a novel perspective on the complementary roles of reactive and proactive control, vis-à-vis the conflict monitoring and regulative functions of the ACC and PFC, respectively, as they contribute to performance on a response conflict task. Prior work has examined the joint contributions of ACC and PFC activity to circumscribed instances of reactive control, such as when ACC reactivity to a response conflict recruits phasic PFC activity to aid in response control (e.g., Kerns et al., 2004). The present research assessed proactive control as the sustained tonic enhancement of left PFC activity across all task trials. Although traditional models of cognitive control have focused almost exclusively on reactive mechanisms, mounting evidence suggests that proactive processes play a major and, most likely, more efficient role in behavioral control (Amodio, 2010; Aron, 2011; Braver, Paxton, Locke, & Barch, 2009; see also, Gratton et al., 1992). Here, we provided new evidence for the role of proactive control over and above the more commonly observed role of reactive control processes, and we demonstrated how a consideration of these alternative profiles can inform impairments associated with the individual differences linked to affective disorders.

REFERENCES

Aiken, L. S., & West, S. G. (1991). *Multiple regression: Testing and interpreting interactions*. Newbury Park, CA: Sage.

Amir, N., Freshman, M., & Foa, E. (2002). Enhanced Stroop interference for threat in social phobia. *Journal of Anxiety Disorders, 16*, 1–9. http://dx.doi.org/10.1016/S0887-6185(01)00084-6.

Amir, N., Weber, G., Beard, C., Bomyea, J., & Taylor, C. (2008). The effect of a single-session attention modification program on

response to a public-speaking challenge in socially anxious individuals. *Journal of Abnormal Psychology, 117*, 860–868. http://dx.doi.org/10.1037/a0013445.

Amodio, D. M. (2009). Intergroup anxiety effects on the control of racial stereotypes: a psychoneuroendocrine analysis. *Journal of Experimental Social Psychology, 45*, 60–67. http://dx.doi.org/10.1016/j.jesp.2008.08.009.

Amodio, D. M. (2010). Coordinated roles of motivation and perception in the regulation of intergroup responses: frontal cortical asymmetry effects on the P2 event-related potential and behavior. *Journal of Cognitive Neuroscience, 22*, 2609–2617. http://dx.doi.org/10.1162/jocn.2009.21395.

Amodio, D. M., Devine, P. G., & Harmon-Jones, E. (2007). A dynamic model of guilt: implications for motivation and self-regulation in the context of prejudice. *Psychological Science, 18*, 524–530. http://dx.doi.org/10.1111/j.1467-9280.2007.01933.x.

Amodio, D. M., Harmon-Jones, E., Devine, P. G., Curtin, J. J., Hartley, S. L., & Covert, A. E. (2004). Neural signals for the detection of unintentional race bias. *Psychological Science, 15*, 88–93. http://dx.doi.org/10.1111/j.0963-7214.2004.01502003.x.

Amodio, D. M., Kubota, J. T., Harmon-Jones, E., & Devine, P. G. (2006). Alternative mechanisms for regulating racial responses according to internal vs external cues. *Social Cognitive and Affective Neuroscience, 1*, 26–36. http://dx.doi.org/10.1093/scan/nsl002.

Amodio, D. M., Devine, P. G., & Harmon-Jones, E. (2008). Individual differences in the regulation of intergroup bias: the role of conflict monitoring and neural signals for control. *Journal of Personality and Social Psychology, 94*, 60–74. http://dx.doi.org/10.1037/0022-3514.94.1.60.

Amodio, D. M., Master, S. L., Yee, C. M., & Taylor, S. E. (2008). Neurocognitive components of the behavioral inhibition and activation systems: implications for theories of self-regulation. *Psychophysiology, 44*, 1–9. http://dx.doi.org/10.1111/j.1469-8986.2007.00609.x.

Aron, A. R. (2011). From reactive to proactive and selective control: developing a richer model for stopping inappropriate responses. *Biological Psychiatry, 69*, e55–e68. http://dx.doi.org/10.1016/j.biopsych.2010.07.024.

Baumeister, R. F. (1984). Choking under pressure: self-consciousness and paradoxical effects of incentives on skillful performance. *Journal of Personality and Social Psychology, 46*, 610–620. http://dx.doi.org/10.1037/0022-3514.46.3.610.

Becker, E. S., Rinck, M., Margarf, J., & Roth, W. T. (2001). The emotional Stroop effect in anxiety disorders: general emotionality or disorder specificity? *Journal of Anxiety Disorders, 15*, 147–159. http://dx.doi.org/10.1016/S0887-6185(01)00055-X.

Beilock, S. L., & Gray, R. (2007). Why do athletes choke under pressure? In G. Tenenbaum, & R. C. Eklund (Eds.), *Handbook of sport psychology* (3rd ed., pp. 425–444). Hoboken, NJ: John Wiley & Sons.

Botvinick, M., Braver, T., Barch, D., Carter, C., & Cohen, J. (2001). Conflict monitoring and cognitive control. *Psychological Review, 108*, 624–652. http://dx.doi.org/10.1037/0033-295X.108.3.624.

Botvinick, M., & Cohen, J. D. (2014). The computational and neural basis of cognitive control: charted territory and new frontiers. *Cognitive Science, 38*, 1249–1285. http://dx.doi.org/10.1111/cogs.12126.

Botvinick, M., Nystrom, L. E., Fissell, K., Carter, C. S., & Cohen, J. D. (1999). Conflict monitoring versus selection-for-action in anterior cingulate cortex. *Nature, 402*, 179–181. http://dx.doi.org/10.1038/46035.

Braver, T. S. (2012). The variable nature of cognitive control: a dual mechanisms framework. *Trends in Cognitive Sciences, 16*, 106–113. http://dx.doi.org/10.1016/j.tics.2011.12.010.

Braver, T. S., Paxton, J. L., Locke, H. S., & Barch, D. M. (2009). Flexible neural mechanisms of cognitive control within human prefrontal cortex. *Proceedings of the National Academy of Sciences, 106*, 7351–7356. http://dx.doi.org/10.1073/pnas.0808187106.

Carver, C. S., & White, T. L. (1994). Behavioral inhibition, behavioral activation, and affective reponses to impending reward and punishment: the BIS/BAS scales. *Journal of Personality and Social Psychology, 67*, 319–333. http://dx.doi.org/10.1037/0022-3514.67.2.319.

Coan, J. A., & Allen, J. J. B. (2003). Frontal EEG asymmetry and the behavioral activation and inhibition systems. *Psychophysiology, 40*, 106–114. http://dx.doi.org/10.1111/1469-8986.00011.

Connor, K. M., Kobak, K. A., Churchill, L. E., Katzelnick, D., & Davidson, R. J. (2001). Mini-SPIN: a brief screening assessment for generalized social anxiety disorder. *Depression and Anxiety, 14*, 137–140. http://dx.doi.org/10.1002/da.1055.

Croston, G. (2012). *The thing we fear more than death*. Retrieved 10/14/14, from: http://www.psychologytoday.com/blog/the-real-story-risk/201211/the-thing-we-fear-more-death.

Davidson, R. J. (1992). Anterior cerebral asymmetry and the nature of emotion. *Brain and Cognition, 20*, 125–151. http://dx.doi.org/10.1016/0278-2626(92)90065-T.

Davidson, R. J., Marshall, J. R., Tomarken, A. J., & Henriques, J. B. (2000). While a phobic waits: regional brain electrical and autonomic activity in social phobics during anticipation of public speaking. *Biological Psychiatry, 47*, 85–95. http://dx.doi.org/10.1016/S0006-3223(99)00222-X.

Devine, P. G. (1989). Stereotypes and prejudice: their automatic and controlled components. *Journal of Personality and Social Psychology, 56*, 5–18. http://dx.doi.org/10.1037/0022-3514.56.1.5.

Eriksen, B. A., & Eriksen, C. W. (1974). Effects of noise letters upon identification of a target letter in a nonsearch task. *Perception & Psychophysics, 16*, 143–149. http://dx.doi.org/10.3758/BF03203267.

Folstein, J. R., & Van Petten, C. (2008). Influence of cognitive control and mismatch on the N2 component of the ERP: a review. *Psychophysiology, 45*, 152–170. http://dx.doi.org/10.1111/j.1469-8986.2007.00602.x.

Forster, K. I., & Forster, J. C. (2003). DMDX: a Windows display program with millisecond accuracy. *Behavior Research Methods, Instruments, & Computers, 35*, 116–124. http://dx.doi.org/10.3758/BF03195503.

Gehring, W. J., Himle, J., & Nisenson, L. G. (2000). Action-monitoring dysfunction in obsessive-compulsive disorder. *Psychological Science, 11*, 1–6. http://dx.doi.org/10.1111/1467-9280.00206.

Gosling, S. D., Rentfrow, P. J., & Swann, W. B., Jr. (2003). A very brief measure of the big five personality domains. *Journal of Research in Personality, 37*, 504–528. http://dx.doi.org/10.1016/S0092-6566(03)00046-1.

Grant, D. M., & Beck, J. G. (2006). Attentional biases in social anxiety and dysphoria: does comorbidity make a difference? *Journal of Anxiety Disorders, 20*, 520–529. http://dx.doi.org/10.1016/j.janxdis.2005.05.003.

Gratton, G., Coles, M. G. H., & Donchin, E. (1992). Optimizing the use of information: strategic control of activation of responses. *Journal of Experimental Psychology: General, 121*, 480–506. http://dx.doi.org/10.1037/0096-3445.121.4.480.

Gray, J. A. (1982). A theory of anxiety. *L'Encephale, 9*, 161B–166B.

Hajcak, G., McDonald, N., & Simons, R. F. (2003). Anxiety and error-related brain activity. *Biological Psychology, 64*, 77–90. http://dx.doi.org/10.1016/S0301-0511(03)00103-0.

Hajcak, G., & Simons, R. F. (2002). Error-related brain activity in obsessive–compulsive undergraduates. *Psychiatry Research, 110*, 63–72. http://dx.doi.org/10.1016/S0165-1781(02)00034-3.

Harmon-Jones, E. (2003). Clarifying the emotive functions of asymmetrical frontal cortical activity. *Psychophysiology, 40*, 838–848. http://dx.doi.org/10.1111/1469-8986.00121.

Harmon-Jones, E., & Sigelman, J. (2001). State anger and prefrontal brain activity: evidence that insult-related relative left-prefrontal activation is associated with experienced anger and aggression. *Journal of Personality and Social Psychology, 80*, 797–803. http://dx.doi.org/10.1037/0022-3514.80.5.797.

Heatherton, T. F. (2011). Neuroscience of self and self-regulation. *Annual Review of Psychology, 62*, 363–390.

Jacoby, L. L. (1991). A process dissociation framework: separating automatic from intentional uses of memory. *Journal of Memory and Language, 30*, 513–541. http://dx.doi.org/10.1016/0749-596X(91)90025-F.

Kerns, J. G., Cohen, J. D., MacDonald, A. W., Cho, R. Y., Stenger, V. W., & Carter, C. S. (2004). Anterior congulate conflict monitoring and adjustmentes in control. *Science, 13*, 1023–1026. http://dx.doi.org/10.1126/science.1089910.

Kleiman, T., Hassin, R. R., & Trope, Y. (2014). The control-freak mind: stereotypical biases are eliminated following conflict-activated cognitive control. *Journal of Experimental Psychology: General, 143*, 498–503. http://dx.doi.org/10.1037/a0033047.

Lambert, A. J., Payne, B. K., Jacoby, L. L., Shaffer, L. M., Chasteen, A. L., & Khan, S. R. (2003). Stereotypes as dominant responses: on the "social facilitation" of prejudice in anticipated public contexts. *Journal of Personality and Social Psychology, 84*, 277–295. http://dx.doi.org/10.1037/0022-3514.84.2.277.

Lundh, L.-G., & Öst, L. G. (1996). Stroop interference, self-focus and perfectionism in social phobics. *Personality and Individual Differences, 20*, 725–731. http://dx.doi.org/10.1016/0191-8869(96)00008-6.

Maidenberg, E., Chen, E., Craske, M., Bohn, P., & Bystritsky, A. (1996). Specificity of attentional bias in panic disorder and social phobia. *Journal of Anxiety Disorders, 10*, 529–541. http://dx.doi.org/10.1016/S0887-6185(96)00028-X.

Master, S. L., Amodio, D. M., Stanton, A. L., Yee, C. M., Hilmert, C. J., & Taylor, S. E. (2009). Neurobiological correlates of coping through emotional approach. *Brain, Behavior, and Immunity, 23*, 27–35. http://dx.doi.org/10.1016/j.bbi.2008.04.007.

Mattia, J. I., Heimberg, R. G., & Hope, D. A. (1993). The revised stroop color-naming task in social phobics. *Behaviour Research and Therapy, 31*, 305–313. http://dx.doi.org/10.1016/0005-7967(93)90029-T.

Mendoza, S. P., Gollwitzer, P. M., & Amodio, D. M. (2010). Reducing the expression of implicit stereotypes: reflexive control through implementation intentions. *Personality and Social Psychology Bulletin, 36*, 512–523. http://dx.doi.org/10.1177/0146167210362789.

Moser, J. S., Moran, T. P., Schroder, H. S., Donellan, M. B., & Yeung, N. (2013). On the relationship between anxiety and error monitoring: a meta-analysis and conceptual framework. *Frontiers in Human Neuroscience, 7*, 1–19. http://dx.doi.org/10.2289/fnhum.2013.00466.

Nieuwenhuis, S., Yeung, N., van den Wildenberg, W., & Ridderinkhof, K. R. (2003). Electrophysiological correlates of anterior cingulate function in a go/no-go task: effects of response conflict and trial type frequency. *Cognitive, Affective, & Behavioral Neuroscience, 3*, 17–26. http://dx.doi.org/10.3758/CABN.3.1.17.

Ofan, R. H., Rubin, N., & Amodio, D. M. (2014). Situation-based social anxiety enhances the neural processing of faces: evidence from an intergroup context. *Social Cognitive and Affective Neuroscience, 9*, 1055–1061. http://dx.doi.org/10.1093/scan/nst087.

Olvet, D. M., & Hajcak, G. (2009). The stability of error-related brain activity with increasing trials. *Psychophysiology, 46*, 957–961. http://dx.doi.org/10.1111/j.1469-8986.2009.00848.x.

Pizzagalli, D. A., Sherwood, R., Henriques, J. B., & Davidson, R. J. (2005). Frontal brain asymmetry and reward responsiveness: a source localization study. *Psychological Science, 16*, 805–813. http://dx.doi.org/10.1111/j.1467-9280.2005.01618.x.

Pritchard, W. S., Shappell, S. A., & Brandt, M. E. (1991). Psychophysiology of N200/N400: a review and classification scheme. In J. R. Jennings, & P. K. Ackles (Eds.), *Advances in psychophysiology: A research annual* (Vol. 4, pp. 43–106). London: Jessica Kingsley.

Rehm, L. P., & Marston, A. R. (1968). Reduction of social anxiety through modification of self-reinforcement: an instigation therapy technique. *Journal of Consulting and Clinical Psychology, 32*, 565–574. http://dx.doi.org/10.1037/h0026406.

Richeson, J. A., & Shelton, J. N. (2003). When prejudice does not pay: effects of interracial contact on executive function. *Psychological Science, 14*, 287–290. http://dx.doi.org/10.1111/1467-9280.03437.

Righi, S., Mecacci, L., & Viggiano, M. P. (2009). Anxiety, cognitive self-evaluation and performance: ERP correlates. *Journal of Anxiety Disorders, 23*, 1132–1138. http://dx.doi.org/10.1016/j.janxdis.2009.07.018.

Ritter, W., Simson, R., Vaughan, H. G., Jr., & Friedman, D. (1979). A brain event related to the making of a sensory discrimination. *Science, 30*, 1358–1361. http://dx.doi.org/10.1126/science.424760.

Schmid, P. C., Kleiman, T., & Amodio, D. M. (2015). Power effects on cognitive control: turning conflict into action. *Journal of Experimental Psychology: General, 144*, 655–663. http://dx.doi.org/10.1037/xge0000068.

Schmid, P. C., & Schmid Mast, M. (2013). Power increases performance in a social evaluation situation as a result of decreased stress responses. *European Journal of Social Psychology, 43*, 201–211.

Seeley-Wait, E., Abbott, M. J., & Rapee, R. M. (2009). Psychometric properties of the mini-social phobia inventory. *Primary Care Companion to The Journal of Clinical Psychiatry, 11*, 231–236.

Soenke, J., Wieringa, B. M., Nager, W., Rada, D., Dengler, R., Emrich, H. M., et al. (2001). Discrepant target detection and action monitoring in obsessive–compulsive disorder. *Psychiatry Research: Neuroimaging, 108*, 101–110. http://dx.doi.org/10.1016/S0925-4927(01)00117-2.

Somers, J. M., Goldner, E. M., Waraich, P., & Hsu, L. (2006). Prevalence and incidence studies of anxiety disorders: a systematic review of the literature. *Canadian Journal of Psychiatry, 51*, 100–113.

Stroop, J. R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology, 18*, 643–662.

Williams, K. D. (2007). Ostracism: the kiss of social death. *Social and Personality Psychology Compass, 1*, 236–247. http://dx.doi.org/10.1111/j.1751-9004.2007.00004.x.

Wittchen, H. U., Fuetsch, M., Sonntag, H., Mueller, N., & Liebowitz, M. (2000). Disability and quality of life in pure and comorbid social phobia: findings from a controlled study. *European Psychiatry, 15*, 46–58. http://dx.doi.org/10.1016/S0924-9338(00)00211-X.

Yeung, N., Botvinick, M., & Cohen, J. D. (2004). The neural basis of error detection: conflict monitoring and the error-related negativity. *Psychological Review, 111*, 931–959. http://dx.doi.org/10.1037/0033-295X.111.4.931.