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Women are more sensitive than men to prior trial events on the Stop-signal task

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Sexual dimorphism in the brain and cognition is a topic of widespread interest. Many studies of sex differences have focused on visuospatial and verbal abilities, but few studies have investigated sex differences in executive functions. We examined two key components of executive function – response inhibition and response monitoring – in healthy men (n = 285) and women (n = 346) performing the Stop-signal task. In this task, participants are required to make a key press to a stimulus, unless a tone is presented at some delay following the initial stimulus presentation; on these infrequent trials, participants are instructed to inhibit their planned response. Response inhibition was assessed with an estimate of the latency needed to inhibit a response (stop-signal reaction time), and response monitoring was measured by calculating the degree to which participants adjusted their reaction times based on the immediately preceding trial (e.g., speeding following correct trials and slowing following errors). There were no sex differences in overall accuracy or response inhibition, but women showed greater sensitivity to trial history. Women sped up more than men following correct 'Go' trials, and slowed down more than men following errors. These small but statistically significant effects (Cohen's d = 0.25–0.3) suggest more flexible adjustments in speed–accuracy trade-offs in women and greater cognitive flexibility associated with the responsive control of action.

Sexual dimorphism in the brain and cognition is a topic of great public and scientific interest. Findings from human and animal studies have indicated sex differences ranging from molecular to behavioural levels, although these differences are often subtle and inconsistent (Eliot, 2011). A more thorough understanding of putative differences

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between the brain functions of men and women has implications for education, clinical neuropsychiatry, and many other fields. For example, research into sex differences in the brain and cognitive functioning has already fuelled education policy making (e.g., same–sex classrooms; Eliot, 2011) and in theory could be used to leverage unique strengths and learning styles of girls and boys. Furthermore, given large sex differences in the incidence and severity of many psychiatric illnesses (e.g., schizophrenia and attention deficit hyperactivity disorder) and evidence for sex differences in pathophysiological mechanisms of disease, an understanding of sex differences in brain and behaviour could impact both theory and clinical decision making.

With respect to cognitive functioning, most previous studies have examined sex differences in visuospatial and verbal abilities, with women typically outperforming men on verbal tasks and men performing better on spatial tasks (Hyde, 1988; Voyer, Voyer, & Bryden, 1995). Although these differences are relatively small and might have some basis in social biases (Baenninger & Newcombe, 1989) and experience, correlations of these differences with both prenatal sex hormone exposure (see Hampson, 1995, for review) and circulating levels in adulthood suggest a biological basis. For example, variability in testosterone and estradiol levels during the menstrual cycle has been found to predict changes in cognitive ability, particularly spatial skills (Broverman *et al.*, 1981; Hampson, 1990, 1995; Hausmann, Slabbekoorn, Van Goozen, Cohen-Kettenis, & Güntürkün, 2000). Interestingly, there is also evidence that sex hormones mediate the effect of social biases and stereotypes on spatial abilities (Hausmann, Schoofs, Rosenthal, & Jordan, 2009).

Surprisingly neglected in studies of sex differences in cognition is executive functioning, which refers to the diverse cognitive abilities, largely subserved by frontal cortex, involved in the control of thought and action. Despite clear sex differences in psychiatric disorders that are associated with pronounced impairments in executive functions and putative effects of sex hormones on executive functioning via their contribution to prenatal brain development and modulation of dopamine, few studies have examined sex differences in executive functioning in healthy populations. Executive dysfunction has been posited to be a central feature of both schizophrenia and attention deficit/hyperactivity disorder (ADHD; Barkley, 1997; Goldman-Rakic & Selemon, 1997). As previously noted, sex differences in the epidemiology and illness severity of both schizophrenia and ADHD have been well documented. Although there is evidence for androgenic modulation of neurodevelopment (Wisniewski, 1998) and behaviour, which may help explain sex differences in the epidemiology of these diseases, these differences still remain poorly understood. Importantly, circulating oestrogen is a significant neuromodulator of dopamine (Becker, 1990; Pasqualini, Olivier, Guibert, Frain, & Leviel, 1995; Xiao & Becker, 1994), a neurotransmitter that has been implicated both in executive control (Robbins & Arnsten, 2009) and pathophysiological mechanisms of both ADHD and schizophrenia (Iversen & Iversen, 2007).

Results from the few studies that have investigated sex differences in executive functioning using standard neuropsychological measures have been mixed. A handful of studies have reported a female advantage (Baroun & Alansari, 2006; Kalkut, Han, Lansing, Holdnack, & Delis, 2009; Mekarski, Cutmore, & Suboski, 1996; Sarmany, 1977; von Kluge, 1992), others reporting a male advantage (Clayson, Clawson, & Larson, 2011), and most reporting no difference across the sexes (Brocki & Bohlin, 2004; Daniel, Pelotte, & Lewis, 2000; Houx & Jolles, 1993; Schirmer & Kotz, 2003; Silveri *et al.*, 2006; Swerdlow, Filion, Geyer, & Braff, 1995). There are a few factors that contribute to the lack of consensus on sex differences in executive functioning. First, there is significant variability in both the putative executive function constructs examined and the specific paradigms used to

measure these. Although there is no clear consensus about the most valid subdivisions of executive function (e.g., Sabb *et al.*, 2008), some studies suggest that discrete constructs can be identified at the behavioural level (Miyake *et al.*, 2000) or as defined by distinctive neural circuitry (Bilder, 2012; Rushworth, Noonan, Boorman, Walton, & Behrens, 2011). Second, many measures of executive functioning place considerable demands on multiple cognitive processes, which might obscure sex differences in specific components of executive functioning. Third, most previous studies used relatively small samples and therefore lacked statistical power to detect subtle sex differences.

Sex differences in response monitoring and response inhibition, two key components of executive functioning, have been examined with the Stop-signal task. Response inhibition refers to the ability to *deliberately* suppress inappropriate motor responses and response monitoring involves the ability to evaluate actions and use feedback signalling success or failure to guide future performance. The Stop-signal task has emerged as one of the leading methods to examine these two constructs. In this task, subjects are presented with a stimulus that requires executing a speeded motor response unless a signal is presented following initial stimulus presentation. In this case, subjects are instructed to inhibit their response. A measure of the latency of the covert inhibitory process, stop-signal reaction time (SSRT), can be estimated (Logan, Cowan, & Davis, 1984). Along with measures of inhibition, well-characterized RT adjustments as a function of trial history have been described in this task. Participants tend to slow down following errors and correctly inhibited responses, and to speed up with consecutive trials that do not require inhibition (Emeric *et al.*, 2007; Rieger & Gauggel, 1999; Verbruggen, Logan, Liefooghe, & Vandierendonck, 2008). An additional advantage of this task is the relatively low demands it places on other cognitive processes, for example, working memory. Furthermore, using a 'tracking' version of the task, in which task difficulty is manipulated to ensure equal inhibition success across subjects, allows measures of response inhibition and response monitoring that are not confounded by differences in success rate.

Although most studies have failed to observe sex differences in Stop-signal task performance, reduced functional asymmetry of inhibition-related event-related potentials (ERPs) in women (Huster, Westerhausen, & Herrmann, 2011) and different networks of fMRI activity in men and women during inhibition and error processing (Li, Huang, Constable, & Sinha, 2006; Li *et al.*, 2009) have been reported. Colzato, Pratt, and Hommel (2011) reported less efficient inhibition in women, but only in the follicular stage of their menstrual cycle. In the current study, sex differences in response inhibition and response monitoring were examined in a large sample of healthy adults performing the Stop-signal task.

Method

Participants

Participants were drawn from the first 1,000 enrollees in a project of the Consortium for Neuropsychiatric Phenomics at UCLA (www.phenomics.ucla.edu), approved by the UCLA Institutional Review Board. The participants, ages 21–50 years, were recruited by community advertisements from the Los Angeles area and completed the Stop-signal task. To be included individuals had to be either 'White, Not of Hispanic or Latino Origin' or 'Hispanic or Latino, of Any Race' following National Institutes of Health (NIH) designations of racial and ethnic minority groups, and have completed at least 8 years of education (other racial and ethnic minority groups were excluded because this was

Table 1. Demographic data

	Males (n = 285)		Females (n = 346)		Analysis	
	n	%	*n*	%	Test statistic	*p*-Value
Race						
White	212	74.7	246	71.1	$\chi^2 = 4.0$	.55
Native American[a]	66	23.2	84	24.3		
Multiracial	4	1.4	10	2.9		
African American	1	0.4	4	1.2		
Asian	0	0	1	0.3		
Not reported	1	0.4	1	0.3		
Ethnicity						
Hispanic origin	122	43	150	43	$\chi^2 = 0.02$	.89
Not of Hispanic origin	163	57	196	57		
Language testing administered						
English	244	86	290	84	$\chi^2 = 0.39$	.53
Spanish	41	14	56	16		
	M	*SD*	*M*	*SD*		
Age	31.2	8.9	31.3	8.5	$t = 1.32$	.19
Edinburgh Handedness Quotient[b]	0.73	0.54	0.72	0.57	$T = 0.40$	.69
Education (years)	14.7	2.0	15.2	2.1	$t = 2.71$	.007

[a]A high proportion of these individuals who identified themselves as Native Americans was of Mexican origin.
[b]Higher scores indicate greater right-handedness.

thought to increase risk of confounding planned genetic studies). For participants who spoke both English and Spanish, language for testing was determined by a verbal fluency test. Participants were screened for neurological disease, history of head injury with loss of consciousness or cognitive sequelae, use of psychoactive medications, substance dependence within past 6 months, history of major mental illness or ADHD, and current mood or anxiety disorder. Self-reported history of psychopathology was verified with the SCID-IV (First, Spitzer, Gibbon, & Williams, 1995). Urinalysis was used to screen for drugs of abuse (cannabis, amphetamine, opioids, cocaine, benzodiazepines) on the day of testing, and participants were excluded if results were positive. These primary inclusion/exclusion criteria yielded 640 participants who completed the study. We then applied additional exclusion criteria based on Stop-signal task performance (see Statistical analyses), yielding 285 males and 346 females in the final analysis. Demographic data are outlined in Table 1. Males and females were similar on age, race, handedness, and ethnicity. Compared with Hispanic or Latino individuals who were examined in English, Hispanic or Latino individuals who were examined in Spanish were slower on both Go trials: $t(270) = 3.36$, $p = .0009$, and Stop-error trials: $F(270) = 2.48$, $p = .01$, and had a higher proportion of Go trials in which they failed to respond: $t(270) = 2.21$, $p = .03$. However, men and women were matched on the language in which the test was administered. Women averaged about 0.5 more years of education than men. Written informed consent was obtained from all subjects prior to testing. Subjects were paid $15 per hr and received compensation for transportation and parking. After receiving a thorough explanation, all participants gave written informed consent according to

procedures approved by the University of California Los Angeles Institutional Review Board, and a certificate of confidentiality was obtained from the NIH.

Data acquisition

E-Prime software (Psychology Software Tools Inc., Pittsburgh, PA, USA) was used for task presentation and response collection; the E-prime programs used are available on request. Data were collected on Dell workstations with Intel Core Duo processors, 2.4 or 2.2 GHz processing speed, 1 GB memory, and Dell 17' LCD monitors. Responses were indicated using a standard Dell keyboard. Subjects were positioned so that their eyes were approximately 20' from the monitor.

Task design

Subjects performed the Stop-signal task (Figure 1; Lappin & Eriksen, 1966). On each trial, participants were shown a visual stimulus ('X' or 'O') in the centre of the screen. On 75% of the trials (Go trials), they were instructed to press the left arrow button on the keyboard when they saw an 'X' and to press the right arrow button on the keyboard when they saw an 'O', as quickly and accurately as possible. On the remaining 25% of trials (Stop trials), a 500 Hz tone lasting 250 ms was presented through headphones at a variable delay following onset of the visual stimulus (Stop-signal delay [SSD]). On these trials, participants were instructed to withhold their response to the visual stimulus. Trials in which the participant successfully inhibited the response were labelled Stop-correct, and trials in which the participant erroneously responded to the visual stimulus were labelled Stop-error. Participants were instructed that stopping and going were equally important.

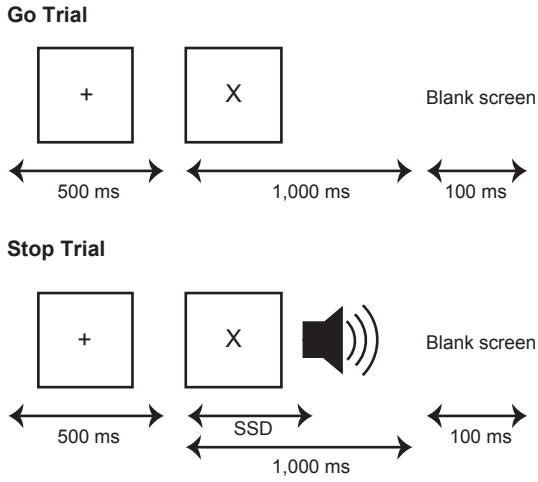

Figure 1. Stop-signal task. All trials began with the presentation of a central fixation spot. After 500 ms, the fixation spot disappeared, and, simultaneously, an 'X' or 'O' appeared at a central location. On 75% of the trials (Go trials), they were instructed to press the left arrow button on the keyboard when they saw an 'X' and to press the right arrow button on the keyboard when they saw an 'O', as quickly and accurately as possible. On the remaining 25% of trials (Stop trials), a 500 Hz tone lasting 250 ms was presented through headphones at a variable delay following onset of the visual stimulus Stop-signal delay (SSD). On these trials, subjects were instructed to withhold their response to the visual stimulus.

All trials began with a 500 ms fixation cross in the centre of the screen. Then, the Go stimulus was presented for a 1,000 ms fixed-response interval. Participants were allowed to respond at the start of stimulus presentation until the end of the 1,000 ms fixed-response interval. Each trial was separated by a fixed, 100 ms delay.

Response inhibition becomes more difficult as the SSD increases. SSDs were dynamically adjusted using two independent 1-up/1-down tracking procedures (or 'staircases'), thereby ensuring successful inhibition on approximately 50% of the Stop trials. The initial SSD for each staircase was based upon performance in a practice block. SSD was increased or decreased by 50 ms when the participant succeeded or failed to inhibit, respectively. The testing session included at least one practice block of 22 Go trials and 10 Stop trials. Participants were required to inhibit on at least 20% of Stop trials and have a mean Go RT of < 750 ms to continue. Two experimental blocks of 128 trials each then followed.

Performance measures

Behavioural performance was evaluated through measurements of RT on Go and Stop-error trials, and of mean SSD. Performance in the Stop-signal task can be accounted for by a mathematical model that assumes a race between independent processes that generate (GO) and inhibit (STOP) the movement (Logan & Cowan, 1984). The response is executed if the GO process finishes first and inhibited if the STOP process finishes first. The latency of the GO process can be measured directly from the observable RTs, but the latency of the STOP process can only be estimated from performance (considering both proportion of successful inhibition on Stop trials and SSD). The estimate of time needed to respond to the Stop signal and to cancel the movement is referred to as the Stop-signal Reaction Time (SSRT; Figure 2). SSRT was calculated using a quantile method (for more detailed description of this method, see Band *et al.*, 2003; Congdon *et al.*, 2012). First, the proportion of failed inhibition trials, which are the proportion of Stop trials in which the participants responded, was calculated across both of the two independent 1-up/1-down staircases used to manipulate task difficulty. Then, correct RTs from Go trials were sorted in ascending order. The quantile RT was determined by finding the RT corresponding to

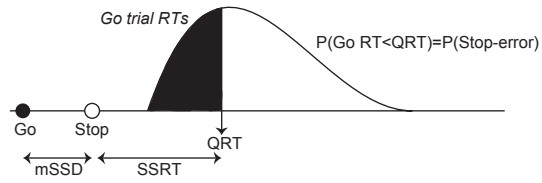

Figure 2. Stop-signal reaction time (SSRT) calculation. The proportion of failed inhibition trials, P (Stop-error), was calculated for each participant. The mean SSD (mSSD), represented by the delay between the stimulus to respond (filled circle) and the Stop signal (empty circle), was calculated for the same participant. Then, RTs from correct Go trials were sorted in ascending order, represented by the probability distribution in the figure. The quantile RT (QRT) was determined by finding the RT corresponding to P (Stop-error). Under race model assumptions, those Go trials that were left of the QRT (represented by the shaded area under the curve) would have been too fast to be inhibited, provided a Stop signal was presented at mSSD following the Go signal. Likewise, the Go trials to the right of the QRT (represented by the empty area under the curve) would have been slow enough to be inhibited. The average SSD (mSSD) was then subtracted from the QRT to calculate SSRT.

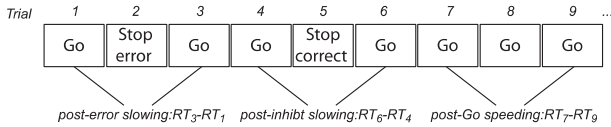

Figure 3. Representation of how trial history effects were calculated using an example trial sequence. For each critical trial type (Stop-correct, Stop-error, and Go), triads of trials were identified such that the critical trial was preceded and followed by a correct Go trial. Post-error and post-inhibit slowing were calculated by subtracting the reaction time (RT) preceding a Stop-error or Stop-correct trial, respectively, from the trial following it. Likewise, post-Go speeding was calculated by subtracting the RT following a Go trial from the RT preceding it. For each critical trial, change in RT between the preceding and following Go trial was averaged to arrive at one aggregate measure of post-error slowing, post-inhibit slowing, and post-Go speeding for each subject.

the proportion of failed inhibition. For example, if the proportion of inhibition for a particular subject was 0.6, the quantile RT would be the RT for which 60% of trials were faster and 40% of trials were slower. The average SSD (across both staircases) was then subtracted from the quantile RT to calculate SSRT.

To index response monitoring, RT was examined as a function of trial history (Figure 3). Triads of trials were identified for each of our trials of interest (Stop-correct, Stop-error, and Go) such that the trial of interest was preceded and followed by a correct Go trial. The middle trial of the triad is referred to as the critical trial. Post-error and post-inhibit slowing were calculated by subtracting the RT preceding a Stop-error or Stop-correct trial, respectively, from the trial following it. Likewise, post-Go speeding was calculated by subtracting the RT following a Go trial from the RT preceding it. For each critical trial type, change in RT between the preceding and following Go trial was averaged to arrive at one aggregate measure of post-error slowing, post-inhibit slowing, and post-Go speeding for each subject.

Statistical analyses

The following rules were used to exclude subjects on the basis of performance: (a) ineffective tracking procedure as defined by per cent inhibition > 75% or < 25%; (b) Go trial accuracy < 60% (see Congdon *et al.*, 2012 for justification of these criteria). These criteria resulted in the exclusion of two males and seven females.

Analysis of covariance was conducted to measure the effect of sex on directional accuracy, omission errors, Stop-trial accuracy, and SSRT. To measure the effect of sex and current trial type (Go or Stop-error) on current trial RT, a mixed-model analysis was conducted, using sex as a between-subject variable and trial type as a within-subject variable. To assess the effects of trial history on Go trials, a mixed-model analysis was conducted on Go RTs with sex as a between-subject variable and critical trial (Go, Stop-correct, and Stop-error) and history ($n - 1$ or $n + 1$, relative to critical trial) entered as within-subject variables (Figure 3). In addition, sex differences in post-inhibit slowing, post-error slowing, and post-Go speeding, as defined in the previous section, were examined using analysis of covariance. In all analyses that included sex, we included years of education as a covariate, as this measure differed significantly between men and women. Levene's test indicated no significant difference in within-group variability between sexes on any of the dependent measures (all p's > .09). All tests were two-tailed except as otherwise specified. Eta-squared (η^2), Cohen's *d,* and correlation coefficients (r)

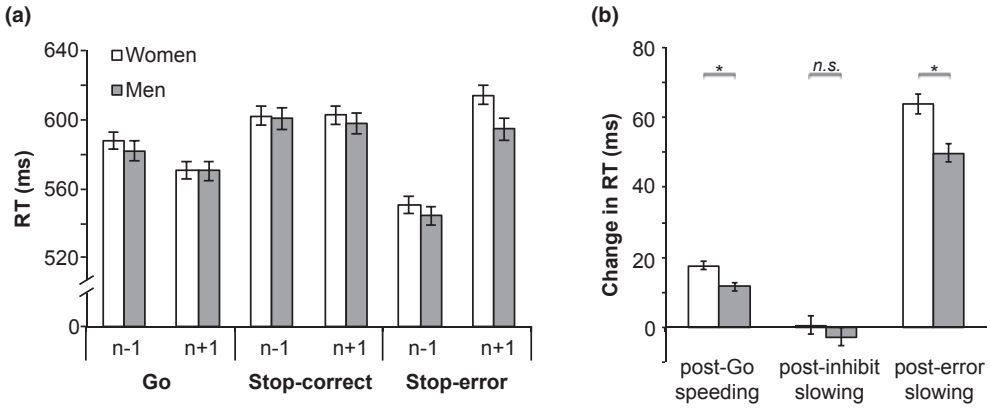

Figure 4. (a) Mean Go RT (with standard error) for trials following ($n + 1$) and preceding ($n - 1$) Go, Stop-correct, and Stop-error trials for women (empty bars) and men (filled bars). (b) Mean post-Go speeding, post-inhibit slowing, and post-error slowing.

Table 2. Stop-signal task performance

	Males ($n = 285$)		Females ($n = 346$)	
	M	*SD*	*M*	*SD*
Go trial directional errors (%)	0.39	0.58	0.32	0.81
Go trial no response (%)	1.03	1.52	1.02	2.06
Probability of inhibition (%)	52.95	5.43	53.53	5.26
No-stop signal reaction time (RT; ms)	588.94	108.19	595.71	104.89
Signal-respond RT (ms)	490.57	88.18	491.68	87.34
Stop-signal RT (ms)	210.13	39.51	213.00	41.12
Post-Go speeding (ms)	11.62	20.90	17.63	21.91
Post-inhibit slowing (ms)	−2.82	44.73	0.51	44.22
Post-error slowing (ms)	49.89	47.00	63.70	52.81

were used where appropriate to report effect sizes. Generalized eta-squared (η^2_G) measures of effect size are reported for repeated measures analyses, as they provide comparability across within- and between-subjects designs and have been recommended for such analyses (Bakeman, 2005).

Results

Table 2 shows Stop-signal task performance and RT adjustments for men and women.

Accuracy

Directional accuracy on Go trials was high and did not differ between sexes: $F(1, 628) = 0.10$, $p = .75$, $d < 0.10$. The proportion of Go trials in which participants did not respond within the deadline was low and also did not differ between groups: $F(1, 628) = 0.56$, $p = .45$, $d < 0.10$. On Stop trials, the dynamic tracking procedure was successful, and the mean proportion of Stop-correct trials was 53%. The two groups did

not differ significantly in the proportion of Stop-correct trials: $F(1, 628) = 1.65$, $p = .20$, $d = 0.11$.

No-stop signal and signal-respond RTs

Consistent with race model logic, there was a significant effect of trial type: $F(1, 628) = 4{,}099.88$, $p < .0001$, $\eta^2_G = 0.21$, with Go trial responses being slower than Stop-errors. There was no significant main effect of sex: $F(1, 628) = 0.43$, $p = .51$, $\eta^2_G = 0.0007$, nor sex-by-trial type interaction: $F(1, 628) = 3.16$, $p = .08$, $\eta^2_G = 0.0002$.

Stop-signal reaction time

There was no sex difference in SSRT: $F(1, 628) = 1.31$, $p = .25$, $d < 0.1$.

Trial history effects

There was a significant effect of history: $F(1, 628) = 318.09$, $p < .0001$, $\eta^2_G = 0.005$, and critical trial: $F(2, 1{,}256) = 128.79$, $p < .0001$, $\eta^2_G = 0.013$, on Go RT (see Figure 4). Notably, there was a significant history-by-critical trial interaction: $F(2, 1{,}256) = 468.61$, $p < .0001$, $\eta^2_G = 0.024$. The significance of *post-hoc* tests were Bonferroni corrected for the seven comparisons planned to disentangle this three-way interaction, which included: (1) Go RT when preceded by Stop-error versus Go trial, (2) Go RT when preceded by Stop-correct versus Go trial, (3) Go RT when preceded by Stop-error versus Stop-correct trial, (4) Go RT preceding a Stop-error versus Stop-correct trial, and (5–7) Go RT preceding versus following either a Stop-error, Stop-correct, or Go trial. Comparisons revealed that Go trials following Stop-correct: $t(630) = 13.2$, adjusted $p < .0001$, and Stop-error: $t(630) = 14.4$, adjusted $p < .0001$, trials were slower than those following another Go trial. There was no difference in RT for trials following Stop-correct versus Stop-error: $t(630) = 1.8$, adjusted $p = .49$. There were also differences in Go trials preceding Stop–correct and Stop-error trials. Go trials preceding a Stop-correct trial were slower than those preceding Stop-error trials: $t(630) = 25.1$, adjusted $p < .0001$. That is, when subjects are responding more slowly, they are more likely to inhibit. Relative to the immediately preceding trial, participants slowed following a Stop-error trial: $t(630) = 28.5$, adjusted $p < .0001$, and sped up following a Go trial: $t(630) = 17.3$, adjusted $p < .0001$. However, no difference between RTs for Go trials preceding and following a Stop-correct trial was observed: $t(630) = 0.54$, adjusted $p > .99$. To summarize, Go trials following Stop trials were slower than Go trials following another Go trial. Additionally, Go trials preceding Stop-correct trials were slower than Go trials following Stop-error trials. When compared to the immediately preceding Go trial, we observed speeding following Go trials (post-Go speeding) and slowing following Stop-errors (post-error slowing). No change in RT between Go trials preceding and following Stop-correct trials was observed.

In examining the effect of sex on history-based adjustments, there was no main effect of sex: $F(1, 628) = 1.0$, $p = .32$, $\eta^2_G = 0.001$. However, the sex-by-history: $F(1, 628) = 5.64$, $p = .02$ $\eta^2_G = 0.00009$, sex-by-critical trial: $F(2, 1{,}256) = 5.32$, $p = .0005$, $\eta^2_G = 0.0004$, and three-way sex-by-history-by-critical trial interaction effects: $F(2, 1{,}256) = 6.2$, $p = .002$, $\eta^2_G = 0.0004$, were significant. To investigate the differences in means giving rise to this three-way interaction, the following comparisons were planned: (1–3) sex differences between Go RT when preceded by either a Go, Stop-error,

or Stop-correct trial; (4–5) sex differences between Go RT when followed by either a Stop-error or Stop-correct trial. Planned comparisons revealed no sex difference in RT for Go trials preceding either Stop-correct or Stop-error trials (all adjusted p-values > .99); however, Go trials following a Stop-error were slower in women: $F(1, 628) = 6.62$, adjusted $p = .05$. No sex differences in trials following Go trials: $F(1, 628) = 0.01$, adjusted $p > .99$, or Stop-correct trials: $F(1, 628) = 0.56$, adjusted $p = .99$, were observed.

Finally, sex differences in the magnitude of change between RTs following and preceding each trial of interest were also examined. Compared with the immediately preceding trial, females slowed down significantly more than males following Stop-errors: $F(1, 628) = 14.55$, $p = .0001$, $d = 0.28$, and sped up significantly more following consecutive Go trials: $F(1, 628) = 13.20$, $p = .0003$, $d = 0.28$. There was no significant sex difference in post-inhibit slowing: $F(1, 628) = 0.75$, $p = .39$, $d < 0.10$.

Within-subject variability

As greater trial history-based adjustments might give rise to greater within-subject variability in RT, we also examined the within-subject standard deviation of Go RT and its relationship with the magnitude of post-error slowing and post-Go speeding as a *post-hoc* analysis. Controlling for years of education, women showed greater inter-individual variability in RT: $F(1, 628) = 11.3$, $p = .0008$, $d = 0.25$. Furthermore, the magnitude of post-error slowing was significantly related to within-subject variability in Go RT in both men ($r = 0.27$, $r < 0.0001$) and women ($r = 0.20$, $p = .0001$). A small relationship was also observed between within-subject variability and post-Go speeding in men ($r = 0.14$, $p = .01$), but not in women ($r = 0.01$, $p = .87$). Finally, we examined differences between men and women in within-subject variability in the magnitude of post-Go speeding and post-error slowing. Controlling for education, women showed greater variability in both post-error slowing: $F(1, 628) = 14.6$, $p = .0001$, $d = 0.39$, and post-Go speeding: $F(1, 628) = 13.2$, $p = .0003$, $d = 0.28$.

Age effects

As an exploratory analysis, we investigated sex differences in the relations between age and Stop-signal task measures of response inhibition and response monitoring in men and women. After controlling for years of education, only post-Go speeding showed a differential relationship with age between men and women, evidenced by an age-by-sex interaction effect: $F(1, 626) = 7.46$, $p = .007$, $\eta^2 = 0.01$. In men, age was associated with greater speeding following Go trials: $F(1, 283) = 13.31$, $p = .0003$, $r = 0.21$. There was no significant relationship between age and post-Go speeding in women: $F(1, 344) = 0.04$, $p = .84$, $r = 0.01$.

Discussion

This study found differences between men and women on a measure of response monitoring derived from the Stop-signal task, even though the sexes did not differ in overall accuracy, response speed, or the efficiency of inhibitory control. Data from this study of a large group of healthy individuals performing the Stop-signal task indicated no sex differences in overall response speed or in the efficiency of inhibitory control. Men and women differed, however, on a more subtle aspect of task performance. Specifically,

women made greater adjustments to their performance based on the outcome of the preceding trial. Both men and women sped up following consecutive trials in which they were not required to inhibit a response, and slowed down on the trial following an error, consistent with prior reports. But compared with men, women showed a larger response to trial history: They sped up more following consecutive Go trials and slowed down more following Stop-error commission. Combined, these findings support the hypothesis that women manifest more flexible adjustments to speed–accuracy trade-offs in response to recent experience of success and failure.

We are not aware of prior studies that explicitly examined sex differences in speed–accuracy adjustments. Several prior studies of sex differences in post-error slowing reported no difference (Larson, South, & Clayson, 2011; Li *et al.*, 2006, 2009), but these studies were underpowered to detect subtle differences.[1] Increased speeding in women following successive Go trials has also not been previously reported in the literature, but may be consistent with findings from studies showing greater RT variability in women. In a large sample of individuals performing a choice RT task, Der and Deary (2006) found that the most robust sex difference was increased RT variability in women, which was present even after controlling for mean RT. In a follow-up study, Reimers and Maylor (2006) replicated these findings and observed that increased RT variability in women was systematic in nature and due to women being slower initially, but speeding up with experience. In the current study, the variability in Go RT was greater in women than men and significantly related to post-error slowing. Thus, in this study, larger performance-based adjustments appear to contribute to increased overall RT variability in women.

There are several theories about the origin and significance of speed–accuracy trade-off (SATO; Schouten & Bekker, 1967) and post-error slowing. Computational accounts of SATO posit that evidence accumulates until a decision threshold is crossed, and that shifts in SATO reflect changes in decision threshold, baseline information, or rate of information accrual (e.g., Reddi & Carpenter, 2000). Post-error slowing is a robust phenomenon (Rabbitt & Rodgers, 1977) widely interpreted as reflecting bias towards a more cautious response strategy (Dutilh *et al.*, 2012). Recent work shows that slowing is not specific to errors, and may generally follow rare events due to attention being oriented away from current task demands (Notebaert *et al.*, 2009). Another theory posits that errors delay the start of information accrual on the following trial due to task-irrelevant factors like affective responses (Rabbitt & Rodgers, 1977). Recent computational work using diffusion models supported the idea that increased response caution gives rise to post-error adjustments (Dutilh *et al.*, 2012), but other task-related factors and individual differences can also affect the psychological process that result in post-error slowing, including error-related distraction away from task demands and delayed accumulation of sensory information (Dutilh, Forstmann, Vandekerckhove, & Wagenmakers, 2013). Although not explicitly accounted for in drift diffusion models, it remains plausible that affective response to errors underlies response caution. Indeed, it has been observed that individuals high in negative affect show more post-error slowing (Robinson, Meier, Wilkowski, & Ode, 2007), and individuals high in anxiety were more cautious after committing an error, as indexed by diffusion model parameters (White, Ratcliff, Vasey, & McKoon, 2010), possibly to avoid further negative affect associated with error commission.

[1] *The statistical powers of Li* et al. *(2006, 2009) to detect a difference with alpha of .05 (two-tailed) were 23% and 17%, respectively. Statistical power could not be calculated for Larson* et al. *(2011). In contrast, the statistical power for the current study was 91%.*

Although it is possible that more cautious adjustment in women is related to emotional reactions to errors, given that women tend to report more negative affect (Feingold, 1994), our data do not indicate that sex differences in affect can fully account for the present results as women also sped up more than men following consecutive correct Go trials. Furthermore, post-error slowing was significantly related to post-Go speeding in both men ($r = 0.48, p < .0001$) and women ($r = 0.50, p < .0001$). Rather, we favour the explanation that women show greater flexibility than men in their cognitive control adjustments.

With regard to the mechanisms underlying trial history adjustments, extensive research suggests that error processing involves anterior cingulate cortex (ACC) in an integrated circuit involving prefrontal cortex, thalamus, and basal ganglia (see van Veen & Carter, 2006, for review). The error-related negativity (ERN; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring, Goss, Coles, Meyer, & Donchin, 1993), which peaks after an error, is localized to the ACC (Dehaene, Posner, & Tucker, 1994; Van Veen & Carter, 2002), and the error positivity (Pe), which also follows errors and peaks later than the ERN, has been linked to post-error slowing (Hajcak, McDonald, & Simons, 2003; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001) and error awareness (Endrass, Franke, & Kathmann, 2005; Endrass, Reuter, & Kathmann, 2007; Nieuwenhuis *et al.*, 2001). Theories about error-related ACC activity emphasize comparisons of motor output and representations of the correct response (Falkenstein *et al.*, 1991; Gehring *et al.*, 1993), conflict between incorrect and correct responses (Botvinick, Cohen, & Carter, 2004; Carter *et al.*, 1998), phasic decreases in dopaminergic activity resulting from violations in expectations based on prior reinforcements (Holroyd & Coles, 2002), and affective responses to error commission (Luu, Flaisch, & Tucker, 2000; Luu, Tucker, Derryberry, Reed, & Poulsen, 2003). It has been suggested that monitoring-related activity in the ACC signals the need for additional control processes, which are implemented in lateral prefrontal cortex (PFC; see Ridderinkhof, van den Wildenberg, Segalowitz, & Carter, 2004, for review); this argument is supported by findings that ACC activity on error trials is associated with post-error slowing and greater activity in lateral PFC on the subsequent trial (Garavan, Ross, Murphy, Roche, & Stein, 2002; Kerns *et al.*, 2004; Li *et al.*, 2008). A role of the lateral PFC has also been highlighted in a recent study of speed–accuracy trade-off (Ivanoff, Branning, & Marois, 2008). While so far there is insufficient evidence to falsify any of these theories, further tests might consider sex-related moderating factors associated with neuroanatomical or neurochemical mechanisms.

Despite the absence of prior evidence for sex differences in error-related behavioural measures, some studies found sex differences in the magnitude of error-related brain activity. A larger ERN and Pe in men has been reported (Larson *et al.*, 2011), which would be contrary to the current results, to the extent that these ERPs are related to post-error slowing. Li *et al.* (2006, 2009) report different networks of error-related fMRI activation in men and women in a Stop-signal task. Unfortunately, methodological limitations obscure the significance of these findings as the authors contrasted successful versus unsuccessful Stop trials, which differ in the presence of a motor response. Thus, it remains unclear whether sex differences are related to error processing or motor output. Li *et al.* (2009) also compared trials following Stop-error trials in which participants did and did not show post-error slowing, and found that women exhibited greater posterior cingulate cortex activation than men. Although we currently lack compelling evidence, further study of sex differences in the ERN and Pe appears warranted, specifically as these relate to post-error adjustments.

Interestingly, neuroanatomical differences between men and women in the midcingulate region have been reported, with men having greater left-asymmetric doubling of the midcingulate gyri (Yucel *et al.*, 2001). With regard to the functional significance of variability in cingulate morphology, greater leftward folding has been associated with more accurate Stroop performance (Hausmann *et al.*, 2009; Huster, Enriquez-Geppert, Pantev, & Bruchmann, 2012), spatial working memory (Fornito *et al.*, 2004), and verbal fluency (Fornito *et al.*, 2004), as well as augmented conflict-related ERPs (Hausmann *et al.*, 2009; Huster *et al.*, 2012). Thus, the observed sex differences in history-based RT adjustments might have some basis in differences in cingulate morphology. Furthermore, sex differences in morphology should also be considered when interpreting sex differences in ERP amplitude, as they might reflect differences in brain structure rather than differences in neural activity (Luck, 2005).

Other possible neuroanatomical distinctions that might help explain the response monitoring sex differences reported here include: differences in interhemispheric connections and functional asymmetries (Davatzikos & Resnick, 1998; Voyer, 1996); and sex differences in functions mediated by archicortical and paleocortical systems (Bilder, 2012) as suggested by sex differences in dorsal and ventral visual stream functions (Alexander, 2003; Lewin & Herlitz, 2002; Voyer *et al.*, 1995), evidence for a greater bias towards novelty detection in women (Colzato *et al.*, 2011), and larger volumes of frontoorbital cortex volume in women but frontomedial cortex in men (Cosgrove, Mazure, & Staley, 2007). The paleocortical system has been hypothesized to play a special role assigning emotional salience to external stimuli (Christensen & Bilder, 2000), thus increased paleocortical activity in women might promote bonding and empathy – processes for which women are argued to be more specialized (De Vries & Panzica, 2006; Hoffman, 1977).

Levels of circulating oestrogen may also help explain sex differences in response monitoring. Estradiol enhances dopaminergic activity via effects on dopamine synthesis, release, and turnover (Becker, 1990; Pasqualini *et al.*, 1995; Xiao & Becker, 1994). Furthermore, one of the more compelling theories of error-related ACC activity posits that it is related to phasic dopamine signalling (Holroyd & Coles, 2002), and dopamine antagonists decrease the ERN amplitude and post-error slowing in healthy volunteers (de Bruijn, Sabbe, Hulstijn, Ruigt, & Verkes, 2006; Zirnheld *et al.*, 2004). Thus, a relationship between oestrogen and reactive behavioural adjustments via oestrogen's neuromodular effects on dopamine is possible, and exaggerated trial history effects in women might be dependent on menstrual cycle stage and circulating estradiol levels. Unfortunately, the current study cannot speak to this hypothesis, as hormonal status of participants was not controlled.

In terms of implications, the current findings are relevant for understanding the nature of sex differences in executive function and their underlying neural mechanisms. They also highlight the advantage of using tasks from the cognitive neuroscience literature over standard neuropsychological measures of executive function to understanding sex differences in cognition, as they are better suited to isolate specific functions. These findings of larger RT adjustments as a function of trial history in women also have potential implications for understanding cognitive dysfunction in psychiatric diseases, particularly those with robust sex differences in incidence and severity. For example, intra-individual differences in RT across different tasks have been reported in both ADHD and schizophrenia. We are not aware of any studies that have explored the extent to which increased variability in RT is related to exaggerated performance-based adjustments. Although most studies have failed to find differences in post-error slowing between

individuals with schizophrenia and ADHD compared to healthy controls (O'Connell, Bellgrove, Dockree, & Robertson, 2004; van Meel, Heslenfeld, Oosterlaan, & Sergeant, 2007; but see Schachar *et al.*, 2004), patients with schizophrenia have been found to show exaggerated adjustments in RT following correctly inhibited responses during oculomotor tasks (Barton, Cherkasova, Lindgren, Goff, & Manoach, 2005; Thakkar, Schall, Boucher, Logan, & Park, 2011). To our knowledge, changes in reaction time following correctly performed trials in ADHD have not been investigated.

In interpreting the significance of the current findings, it should be noted that, like with most findings of sex differences in cognitive functions, men and women are more alike than different. The current findings reflect a small (Cohen's $d = 0.25$–0.3) difference in magnitude of trial history adjustments, rather than qualitative differences in how men and women are performing the task. Nevertheless, these novel findings of enhanced adjustments in reaction time based on trial history in women, in the context of otherwise similar performance on the Stop-signal task, help clarify the specific aspects of executive functioning that differ between men and women and suggest an alternate framework for interpreting sex differences in cognition that may provide compelling new insights for future studies.

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