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Reliability of triggering inhibitory process is a better predictor of impulsivity than SSRT

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ARTICLE INFO	ABSTRACT
	The ability to control behaviour is thought to rely at least partly on adequately suppressing impulsive responses to external stimuli. However, the evidence for a relationship between response inhibition ability and impulse control is weak and inconsistent. This study investigates the relationship between response inhibition and both self-report and behavioural measures of impulsivity as well as engagement in risky behaviours in a large community sample ($N = 174$) of healthy adolescents and young adults (15–35 years). Using a stop-signal paradigm with a number parity go task, we implemented a novel hierarchical Bayesian model of response inhibition that estimates stop-signal reaction time (SSRT) as a distribution and also accounts for failures to react to the stop-signal (i.e., "trigger failure"), and failure to react to the choice stimulus (i.e., "go failure" or omission errors). In line with previous studies, the model reduced estimates of SSRT by approximately 100 ms compared with traditional non-parametric SSRT estimation techniques. We found significant relationships between behavioural and self-report measures of impulsivity and traditionally estimated SSRT, that did not hold for the model-based SSRT estimates. Instead, behavioural impulsivity measures were correlated with rate of trigger failure. The relationship between trigger failure and impulsivity suggests that the former may index a higher order inhibition process, whereas SSRT may index a more automatic inhibition process. We suggest that the existence of distinct response inhibition processes that may be associated with different levels of cognitive control.

1. Introduction

Our capacity to successfully adapt within an ever-changing environment relies partly on an ability to control impulses and suppress inappropriate responses. This ability to cancel prepotent responses when they are contextually inappropriate is known as response inhibition, a core cognitive control process (Miyake et al., 2000),[1] which has been theoretically linked to impulse control (Bari & Robbins, 2013). However, empirical studies have yet to find strong evidence for a relationship between an individual's response inhibition ability and the extent to which they act on impulse (for a review, see Sharma, Markon, & Clark, 2014). This failure to find empirical evidence to support a relationship between response inhibition and impulsivity has been

partly attributed to measurement issues with both constructs. Response inhibition is most commonly measured as the latency to inhibit a prepotent response, using stop-signal reaction time (SSRT) derived from the stop-signal task (Logan & Cowan, 1984). In this paper, we use a novel hierarchical Bayesian modelling approach (Heathcote et al., 2018; Matzke, Curely, Gong, & Heathcote, in press) to extract measures that differentiate between the latency to inhibit a response (i.e., stop-signal reaction time; SSRT), the failure to begin to inhibit the response altogether (i.e., trigger failure), and the failure to begin responding to the go stimulus (i.e., go failure). We describe self-report and behavioural measures of impulsivity and examine their association with estimates of response inhibition using both frequentist and Bayesian methods. The evidence presented here suggests that the weak or null

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[1] Note that in more recent formulations of Miyake and Friedman's model, response inhibition is an integral part of a common-executive function cognitive control component (Miyake & Friedman, 2012).

relationships found throughout the literature cannot readily be attributed to invalid measurement of response inhibition.

1.1. Inhibition and impulsivity

In the laboratory, response inhibition can be measured using the stop-signal task (Logan & Cowan, 1984; for a recent review, see Matzke, Verbruggen, & Logan, 2018), where a prepotent response to a 'go' stimulus (e.g., in a 2-choice decision task) needs to be inhibited upon the subsequent presentation of a 'stop signal' (e.g., a loud tone). In the stop-signal task, the main measure of response inhibition is stop-signal reaction time (SSRT), an estimate of the time required to inhibit the response to the 'go' stimulus. SSRT shows an inverted U-shape across the lifespan, speeding up during childhood and slowing down again in old age (Bedard et al., 2002; van de Laar, van den Wildenberg, van Boxtel, & van der Molen, 2011). Efficient response inhibition (i.e., reduced SSRT) during adolescence is considered a major factor contributing to greater impulse control in adulthood (Casey, Jones, & Hare, 2008; Shulman et al., 2016). Likewise, the co-occurrence of reduced inhibitory ability and heightened impulsivity in many clinical conditions, such as ADHD (Lijffijt, Kenemans, Verbaten, & van Engeland, 2005), schizophrenia (Badcock, Michie, Johnson, & Combrinck, 2002; Hughes, Fulham, Johnston, & Michie, 2012), and substance use disorder (Smith, Mattick, Jamadar, & Iredale, 2014; for a review, see Lipszyc & Schachar, 2010), has resulted in a theoretical link between poor impulse control and inefficient response inhibition (i.e., increased SSRT) in both healthy and clinical cohorts (Bari & Robbins, 2013; Jentsch & Taylor, 1999).

The link between response inhibition and impulse control is made explicitly within the personality literature, where SSRT is often used as a behavioural measure of impulsivity. However, Stahl et al. (2014) among others have questioned the direct correspondence between the construct of response inhibition (i.e., the inhibition of a well-established behavioural response) and constructs such as delay aversion (i.e., a preference for smaller immediate rather than larger later rewards), reflection impulsivity (i.e., acting without thought of consequence or adequate information), and even broader constructs such as propensity towards risk-taking and sensation seeking (Bari & Robbins, 2013; Caswell, Bond, Duka, & Morgan, 2015; Dalley & Robbins, 2017; Evenden, 1999).

Previous literature shows a clear distinction between self-report and behavioural measures of impulsivity, and advocates using measures from both domains to obtain an accurate description of individual variability in impulsivity (Cyders & Coskunpinar, 2011; Sharma et al., 2014; Stahl et al., 2014). Attempts to identify the factor structure of impulsivity have been more successful in the self-report than the behavioural domain. Self-report questionnaires measure impulsivity as a stable trait, asking questions about propensity towards urgency, sensation seeking, lack of premeditation, and lack of perseverance (Whiteside & Lynam, 2001). On the other hand, behavioural impulsivity measures are characterised by substantial variability (Sharma et al., 2014; Stahl et al., 2014). In an empirical study, Stahl et al. (2014) reported a five-factor model of behavioural impulsivity with a sixth separate behavioural inhibition factor loading on the stop-signal and Go/No-go tasks. In a meta-analysis of 98 studies, Sharma et al. (2014) found that the stop-signal task loaded onto both inhibition and impulsive decision-making factors. This and other meta-analyses indicate that the relationship between response inhibition (e.g., SSRT) and self-reported impulsivity measures is weak ($r \approx 0.1$), if present at all (Cyders & Coskunpinar, 2011; Sharma et al., 2014; Stahl et al., 2014). Likewise, the relationships between SSRT and outcome behaviours, such as alcohol and drug use, are poor (Sharma et al., 2014).

There exist a number of possible causes for these weak relationships and ambiguous findings (see Sharma et al., 2014). Many behavioural measures of impulsivity (which often include SSRT) are not reliable over time (Cyders & Coskunpinar, 2011; Kindlon, Mezzacappa, & Earls,

1995). However, other studies suggest that SSRT can be quite stable over time (Wostmann et al., 2013) and therefore reliability alone is unlikely to explain the weak relationship between SSRT and self-report measures of impulsivity.

Alternatively, methodological differences across versions of the stop-signal task may explain variability in the effect size of correlations between response inhibition and impulsivity. For example, Lansbergen, Schutter, and Kenemans (2007) showed that varying the inhibitory demands of the stop-signal task (e.g., adjusting the proportion of inhibition trials) changes the relationship between SSRT and self-reported impulsivity scores. Although some studies have also shown that methodological variations in stop-signal task parameters may alter relationships with measures of impulsivity (Billieux, Gay, Rochat, & Van der Linden, 2010; Burkard, Rochat, & Van der Linden, 2013; Malesza & Ostaszewski, 2016; Van den Wildenberg & Christoffels, 2010), many other studies do not (Aichert et al., 2012; Allom, Panetta, Mullan, & Hagger, 2016; Broos et al., 2012; Burkard et al., 2014; Caswell et al., 2015; Crunelle, Veltman, van Emmerik-van Oortmerssen, Booij, & van den Brink, 2013; Farr, Hu, Zhang, & Li, 2012; Fernie, Cole, Goudie, & Field, 2010; Gorlyn, Keilp, Tryon, & Mann, 2005; Havik et al., 2012; Huddy et al., 2013; Lawyer, Boomhower, & Rasmussen, 2015; Moreno et al., 2012; Roberts, Fillmore, & Milich, 2011; Stahl & Rammsayer, 2007). For instance, Castro-Meneses, Johnson, and Sowman (2015) showed that the relationship between SSRT and impulsivity did not differ in a within-subjects manipulation of high and low inhibitory demands. Therefore, methodological variations of the stop-signal task are unlikely to explain the fragility of the relationship between SSRT and impulsivity measures.

Here we explore whether the task-impurity problem, that is, the fact that executive function tasks such as the stop-signal task are likely to engage more than a single underlying process (Miyake & Friedman, 2012; Scherbaum, Frisch, Holfert, O'Hora, & Dshemuchadse, 2018), may contribute to this weak and inconsistent relationship between response inhibition and impulsivity. In particular, we suggest that the traditional way in which SSRT is measured may not provide a pure measure of response inhibition latency. We will examine whether improved estimation of the latent variables associated with response inhibition in the stop-signal task can improve our ability to identify relationships between measures of response inhibition and impulsivity.

1.2. Improved estimation of response inhibition

The prominent horse-race model of the stop-signal task assumes that SSRT is representative of the efficiency of response inhibition (Verbruggen & Logan, 2009). The horse-race model (Logan & Cowan, 1984) attributes the outcome of any stop trial (i.e., stop failure or stop success) to a race between two independent processes: the *go process* that is measured by go RT and the *stop process* that is measured by the sum of SSRT and the delay between go stimulus onset and the stop signal. Most studies use SSRT to describe an individual's inhibitory ability, with faster SSRT representing more effective response inhibition. Nevertheless, it has been argued that effective inhibitory control may rely not only on fast SSRT, but also on the reliability of triggering the stop process itself (i.e., probability of trigger failure; Band, Van Der Molen, & Logan, 2003; Logan, 1994). Although traditional methods are unable to estimate trigger failures unambiguously (Band et al., 2003), a recent advancement in stop-signal task modelling has overcome this problem.

Matzke, Dolan, Logan, Brown, and Wagenmakers (2013) recently developed the *Bayesian Estimation of Ex-Gaussian Stop-Signal Reaction Time Distribution* (BEESTS) approach, which models SSRT as a distribution (rather than a single summary measure, thus encompassing its variance). Later extensions of the BEESTS approach also accounted for other parameters that can influence SSRT estimation, i.e. trigger failure, go omissions, and go errors. Not accounting for the presence of trigger failure (i.e., the failure to trigger an inhibitory response on a stop trial)

has been shown to result in overestimation of SSRT (Band et al., 2003; Matzke, Love, & Heathcote, 2017). Because traditional methods (see Verbruggen & Logan, 2009) are not able to account for trigger failure, they are susceptible to overestimating SSRT when it is present. Matzke, Hughes, Badcock, Michie, and Heathcote (2017) applied the version of BEESTS that accounts for trigger failure to stop-signal data from a clinical sample of schizophrenia patients and matched controls. Patients had increased SSRT compared to controls, an expected and common effect (Lipszyc & Schachar, 2010). However, the model showed that it was the leading edge of the SSRT distribution that differed, not the tail or variance. This suggested that the difference arose, not because of generally slower processing in patients, but because they had slower stop-signal encoding or action selection. Patients also had higher estimated trigger failure probabilities, which were related to early components of simultaneously acquired event-related potentials (ERPs) to the auditory stop signal, suggestive of attentional deficits. These findings suggest that estimating both SSRT as a distribution and the probability of trigger failure may improve characterisation of an individual's inhibitory ability.

Although there is not a way to account for trigger failure using traditional methods, there are methods to account for failures on the go task using the integration technique. Tannock, Schachar, Carr, Chajczyk, and Logan (1989) describe an adjustment to the integration technique that avoids biases in SSRT estimates caused by go omissions (i.e. failure to trigger a response to the go stimulus). Matzke et al. (in press) showed that a BEESTS model ignoring go omission also produces biased SSRT estimates and provided an extension that addressed this problem. Likewise, choice errors (i.e., incorrect responses on the go task) can also bias SSRT estimates. Matzke et al. (in press) proposed an extended BEESTS model for difficult choice tasks where error rates are not negligible. This "EXG3" model avoids bias by modelling errors. It does so by introducing a second go runner, so that the race is among three runners: a stop runner and two go runners, one for correct and one for error go responses, where the finishing times of all three runners are characterised by ex-Gaussian distributions. This model also explicitly incorporates go-omissions. Details of how to fit the EXG3 model, incorporating both trigger failure and go failure are reported in Heathcote et al. (2018). The model fits data hierarchically, so that the estimation of each individual's model parameters is informed by data from the entire sample, resulting in more precise and, on average, more accurate estimates of the true parameters (e.g., Farrell & Ludwig, 2008). As the paradigm used here also contains a relatively difficult go task, we use the EXG3 model to obtain estimates of SSRT, trigger and go failure, as well as the finishing times of the correct and incorrect go processes. We used this improvement in estimation of inhibitory performance to investigate whether the weak and inconsistent relationship between response inhibition and impulsivity in non-clinical groups may result partly from the fact that traditional SSRT estimates do not account for trigger failure. Here, we examine whether the potentially more process-pure estimates of SSRT obtained by the EXG3 model produce stronger relationships between response inhibition and impulsivity when compared to traditional estimation methods (see Verbruggen & Logan, 2009).

1.3. The present study

In a large sample of healthy adolescents and young adults (*N* = 174), we examine whether different SSRT estimates of response inhibition derived from the hierarchical EXG3 model and the traditional integration method alter the interpretation of relationships between SSRT and both self-report and behavioural measures of impulsivity. Self-report measures were derived from two prominent questionnaires: the Barratt Impulsivity Scale (BIS-11; Stanford et al., 2009) and the Zuckerman Sensation Seeking Scale (SSS; Zuckerman, Kolin, Price, & Zoob, 1964). Behavioural impulsivity was quantified using two Cambridge Automated Neuropsychological Assessment

Battery tasks (CANTAB, Cambridge Cognition, 2017). The Information Sampling Task was used to measure impulsive decision making and the Cambridge Gambling Task was used to measure risk-taking behaviour. We also examined whether individual differences in measures of response inhibition were associated with real-world risky behaviours, such as drug and alcohol use, and risky sexual behaviour measured via self-report questionnaires.

Following Matzke et al. (in press), we expected that the EXG3 model would provide attenuated, but more valid, estimates of SSRT than the traditional integration method. Estimates of SSRT obtained using the integration method were expected to be weakly associated with measures of impulsivity (Stahl et al., 2014), with stronger relationships for behavioural than self-report measures of impulsivity. As SSRT estimates derived from the EXG3 model are theoretically more pure measures of response inhibition, we expected that they may be more strongly related with impulsivity. Finally, we explored whether failure to trigger the go or the stop processes may be related to impulsivity measures.

2. Methods

2.1. Participants

Participants aged 15–35 years (*N* = 282) were recruited from the community via local community groups, schools, and post-secondary and tertiary education centres, after screening for neurological or psychiatric conditions (Karayanidis et al., 2016). Participants were recompensed AUD$20/h. This study conforms to the Declaration of Helsinki and was approved by the University of Newcastle Human Research Ethics Committee (HREC: H-2012-0157). A subset of 207 participants who attempted the stop-signal task were included in this study. The final sample included 174 participants (see Section 2.4.2, Stop-signal task quality control for exclusion criteria).

2.2. Measures

2.2.1. Stop-signal task

As shown in Fig. 1, the primary go task was a two-choice number parity task. A number between 2 and 9, inclusive, was presented for 100 ms in the centre of a grey rectangle ($\approx 2.4°$ x 3.3° visual angle). Participants responded with the index finger of the left or the right hand to indicate whether the number was odd or even. Response-hand mapping was counterbalanced across participants. The inter-trial interval for go stimuli was randomised between 1300 and 4800 ms, irrespective of whether participants responded. On 29% of trials, the go stimulus was followed by an auditory stop signal (binaural, 1000 Hz, 85 dB tone, 100 ms duration), after a variable stop-signal delay (SSD). A stop trial was always followed by a go trial.

As recommended by Band et al. (2003), an adaptive staircase was used to adjust SSD on a trial-by-trial basis to optimise the estimation of SSRT, targeting a 50% failure rate on stop trials. The SSD ranged from 50 to 800 ms and increased or decreased by 50 ms after every successful or failed stop trial, respectively. There were four independent staircases, two for each hand, which started at either 100 or 350 ms. Following a single practice block, behavioural responses were recorded for 700 trials across five blocks.

Stimuli were presented on a PC (Intel dual core Windows 7, 3.1 GHz) via Presentation (version 18.1, http://www.neuro-bs.com) stimulus delivery software. Participants were seated 120 cm from a 23″ monitor. The room was lit with two 40 W halogen bulbs and a screen was placed between the participant and researcher during the task to reduce distraction.

2.2.2. Demographic, impulsivity, and outcome covariates

Information on participants' demographic characteristics was collected using self-report questionnaires. Participants responded to questions regarding their level of income, educational attainment, as
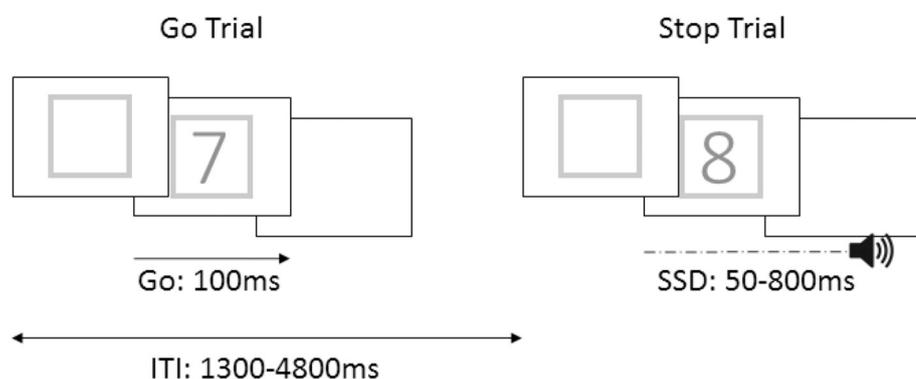
Fig. 1. Stop-signal task – example of go and stop trials. Each trial consisted of a number (2–9) that was displayed briefly (100 ms) to which participants made a parity response within a jittered inter-trial interval (ITI) of 1300–4800 ms. A stop signal (binaural, 1000 Hz, 85 dB tone, 100 ms duration) was presented on 29% of trials after a stop-signal delay (SSD) of 50–800 ms. The SSD was adapted to increase or decrease by 50 ms after each successful and failed inhibition trial, respectively.

well as past and current employment. Socio economic status was obtained by referencing postcodes against the Australian Bureau of Statistics' index of relative socio-economic advantage and disadvantage (Australian Bureau of Statistics, 2013). Additionally, a neuropsychological battery including the Digit Span and Matrix Reasoning subtest of the Wechsler Adult Intelligence Scale-IV (Wechsler, 2014), and the verbal fluency test (FAS; Lezak, 2004) was used to characterise general cognitive ability.

Impulsivity was assessed using both self-report and behavioural measures. The Barratt Impulsivity Scale (BIS) and Sensation Seeking Scale (SSS) were included in a take-home self-report packet. The BIS-11 (Stanford et al., 2009) is a 30-item self-report questionnaire that measures total impulsivity as well as three impulsivity sub-traits: Attention, Motor, and Non-Planning. Items are scored on a four-item Likert type scale from 'rarely/never' to 'almost/always'. Internal consistency in the final sample, as measured by Cronbach's alpha, was 0.85. The SSS (Zuckerman et al., 1964) is a 40-item questionnaire that derives a total sensation seeking score, as well as four subscales: Thrill and Adventure Seeking, Disinhibition, Experience Seeking, and Boredom Susceptibility. Across U.S and U.K samples, males scored higher on the total score and some subscales when compared to females, with significant age decline in both sexes. Internal consistency of the scale was 0.85. The take-home questionnaire packet also included assessment of outcome behaviours using the past frequency (past 6 months) subscales of the Cognitive Appraisal of Risky Events (CARE, Fromme, Katz, & Rivet, 1997): Risky Sexual Behaviour, Heavy Drinking, Illicit Drug Use, Aggressive and Illegal Behaviours, and Irresponsible Academic/Work Behaviours.

The CANTAB Cambridge Gambling Task (CGT; Cambridge Cognition, 2017) is designed to measure risk-taking behaviour when controlling for impulsivity. It differentiates between decision-making and risk-taking while minimising the effects of learning and working memory demands. Each trial presents a row of ten boxes; some are blue and some red. On each trial, the number of red vs. blue boxes differs (e.g., six:four, nine:one). Participants decide whether a hidden yellow token is most likely to be hidden under a red or a blue box and place a bet to win points if they are correct or lose points if they are incorrect. In the ascending condition, this bet starts at a low value and increases in five-second intervals, so that participants must wait longer to place a large bet. In the descending condition, the starting bet is large, and participants can immediately select large bets (for confident choices, e.g., 9:1) or wait for the bet to reduce (for ambiguous choices, e.g., 6:4). These two conditions differentiate between impulsivity and risk-taking, as risky bets require restraint in the ascending, but not the descending condition (Rogers et al., 1999).

Delay Aversion is estimated as the difference in percentage bet between ascending and descending conditions when controlling for risk level and is used to assess impulsivity. Participants with larger scores wait less across both conditions and are considered impatient – not waiting long enough to appropriately bet on each trial. The score is calculated on each bet proportion, i.e., 9:1, 8:2, 7:3, and 6:4. Risk

Adjustment is the extent to which the behaviour is moderated by the ratio of red to blue boxes, when controlling for delay and is considered a measure of risk-taking. The most appropriate behaviour is to increase bets for more certain outcomes (e.g., 9:1) and reduce bets for ambiguous trials (e.g., 6:4). Higher scores indicate better risk adjustment.

The Information Sampling Task (IST; Cambridge Cognition, 2017) measures reflection impulsivity, or the tendency to evaluate information before deciding (Clark, Robbins, Ersche, & Sahakian, 2006). Participants are presented with a 5 × 5 array of grey tiles and can click on any box to reveal a yellow or blue reverse surface. They are asked to open as many boxes as they want before deciding whether there are more yellow or blue boxes in the array. In the fixed win condition, the participant earns 100 points for selecting the correct colour, regardless of how many boxes they open. In the decreasing win condition, the maximum points available start at 250, but decrease by 10 with every box opened, so that if all boxes are opened there is no reward. Higher scores indicate participants are sampling more and require a higher probability of being correct before deciding, therefore being more cautious and making less impulsive decisions. Recent work has shown that the traditional estimation of probability of being correct may overestimate levels of impulsivity (Bennett et al., 2017). Therefore, we use the Bayesian equation described by Bennett et al. (2017) that controls for the order in which the participant opens boxes.

As some measures have overlapping subscales and some have multiple conditions, we assessed the relationships within each impulsivity measurement using Pearson product moment correlations. Subscales of the BIS and SSS were not related strongly enough to consider collapsing the subscales (all $r < 0.55$) and are therefore interpreted separately using the total score for comparison with previous studies. Likewise, the correlations between bet proportions of delay aversion scores, conditions of risk adjustment, and probability correct scores of the CANTAB tasks were not consistently strong ($r < 0.79$, except delay aversion 7:3 bet proportions with 6:4, $r = 0.84$), and were also assessed separately (see Table A.1 in Appendix for the correlation matrix and associated statistics). During analysis, impulsivity and outcome variables were calculated so that positive correlations indicated that poor inhibition was associated with higher levels of impulsivity or risk-taking behaviour.

2.3. Procedure

In the first session, participants completed the neuropsychological tests and collected their take-home package. Almost all participants had complete data from the CANTAB, but some had missing self-report data as these measures were completed at home. Treatment of missing information is detailed below (see Section 2.4.1, Missing data and outliers). During the second session, participants also practiced and then performed a task-switching paradigm (Cooper et al., 2015) with concurrent EEG recording for approximately 45 min before completing the stop-signal task. Both the task-switching and the stop-signal paradigms included a number parity task, and as participants completed the two

Table 1
Summary statistics (number of cases, minimum, maximum, mean, and standard deviation) of the impulsivity and outcome behaviour covariates.

Measure (variable)	*n*	Min	Max	Mean	SD
Self-report Impulsivity					
BIS (Total)	159	41	92	63	10.6
BIS (Attentional)	159	8	28	16.7	4
BIS (Motor)	158	14	35	22.7	4.1
BIS (Non-Planning)	159	14	38	23.6	4.7
SSS (Total)	157	2	36	20.0	6.8
SSS (Boredom Susceptibility)	155	0	9	2.8	2.2
SSS (Disinhibition)	154	0	9	4.7	2.5
SSS (Experience Seeking)	154	0	10	5.9	2.1
SSS (Thrill Seeking)	156	0	10	6.7	2.8
Outcome variables					
CARE (Drug)	153	0	17	1.0	2.8
CARE (Aggr/Illegal)	155	0	14	0.9	2.1
CARE (Sex)	154	0	27	1.5	4.3
CARE (Drink)	155	0	56.7	3.8	8.3
CARE (Work)	155	0	36	2.8	4.6
Behavioural Impulsivity					
Information Sampling Task					
P(Correct) - Decreasing	174	0.6	1	0.8	0.1
P(Correct) - Fixed	174	0.7	1	0.9	0.1
Cambridge Gambling Task					
Delay Aversion - 9:1	172	−0.4	0.9	0.2	0.2
Delay Aversion - 8:2	172	−0.2	0.9	0.3	0.2
Delay Aversion - 7:3	172	−0.2	0.9	0.3	0.2
Delay Aversion - 6:4	172	−0.2	0.9	0.3	0.2
Risk Adjustment - Ascending	172	−1.7	5	2	1.2
Risk Adjustment - Descending	172	−0.8	4.3	1.3	0.9

Note. Risk Adjustment and *P*(Correct) are coded in later analysis so that larger values indicate more impulsivity, but are presented here in their original form. CARE subscales: Heavy Drinking (Drink), Illicit Drug Use (Drug), Aggressive and Illegal Behaviours (Aggr/Illegal), Risky Sexual Behaviour (Sex), Irresponsible Academic/Work Behaviours (Work).

tasks consecutively (with only a short break), hand mapping for odd/even digit responses was kept consistent across tasks. Therefore, participants were well practiced on the number parity task, which is a more difficult task than those used commonly in stop-signal task paradigms.

2.4. Data analysis

2.4.1. Missing data and outliers

No adjustment for missing data was undertaken on variables from outcome measures or the neuropsychological assessments. For self-report measures, we used recommended approaches to handle missing items. For the BIS-11 and SSS, missing responses to questionnaires were replaced with the subscale mean, provided that there was only one missing item per subscale, and total scores were computed with the replaced values. If there was more than one missing item, the subscale was set to missing value. On the CARE, participants' responses of the frequency of risk behaviours that were over 10 standard deviations from the mean were coded as missing values (one case of risky sexual behaviour +13 SD and one case of drug use +10 SD). Any further missing cases in the self-report data were removed pairwise from analysis (see Table 1 for *n* per task).

2.4.2. Stop-signal task quality control

Some participants failed to inhibit on a substantial proportion of trials possibly because they did not understand the task or had poor stop trial compliance. Other participants appeared to deliberately slow their go RT to increase the likelihood of detecting the stop signal before initiating a response. Consequently, 33 of the 207 (≈16%) participants that attempted the stop-signal task were removed from the current analysis. A total of 15 unique participants' mean go RT was faster than their mean RT on failed stop trials, violating the context independence

assumption of the horse-race model. An additional eight participants progressively slowed their mean go RT over trials by > 300 ms, in an apparent attempt to beat the SSD tracking algorithm[2] and biasing SSRT estimates (Verbruggen, Chambers, & Logan, 2013). Four participants had response rates > 75% on stop trials. One participant had both context independence and response rate violations, another had both context independence violations and error rates on go trials reflecting chance performance (> 45%). To avoid estimation issues, one participant with no errors on go trials was removed. Another three were removed because their mean RT on failed stop trials showed a strong decrease with increasing SSD, a pattern that suggests violations of the assumptions of the horse-race model. The remaining 174 participants were used in the present analyses.

During the quality control process, we found six participants with a block of behavioural data that differed from the rest of their performance. One participant made no responses with their right hand across the entire 5th block, and one reported forgetting to stop to the signal in the first block, but corrected this for the remaining blocks. Three participants made no responses at all in one block (1st, 2nd, or, 4th), and the final participant made no successful stops in the 1st block. For each participant, this block with the problem performance was removed from further analysis. In all cases the SSD tracking recovered quickly.

Given the exclusion criteria on the stop-signal task, it is possible that high levels of impulsivity interfered with participants' ability to comply with the inhibition demands of the task, thus biasing the results. Alternatively, non-compliance may be due to a range of contextual factors differentially impacting participants on the test occasion resulting in difficulty with completing the task (e.g., level of fatigue, amount of sleep, frustration threshold, etc.). To examine possible sampling bias, we used a logistic regression model to assess if any impulsivity or outcome variables predicted exclusion of participants' stop-signal data. Due to issues of multicolinearity, we averaged the delay aversion bet proportions and excluded the BIS and SSS total scores. Otherwise, model predictors included all variables in Table 1, as well as socioeconomic status, gender, and the neuropsychological test measures (i.e., matrix reasoning, verbal fluency total, and digit span length). The model predictors did not significantly improve the explanatory power compared to the null model containing only the intercept, $\chi^2_{(26)} = 15.967$, $p = .947$. Likewise, no individual measure was a significant predictor of stop-signal task completion. Variance inflation factors ranged from 1.27–2.77, with only three variables > 2 (SSS Disinhibition = 2.12; BIS Motor = 2.27; BIS non-planning = 2.77), suggesting limited issues with multicolinearity.

We also used a group approach to compare participants who were included or excluded from modelling due to stop-signal task non-compliance. A two-sample Kolmogorov-Smirnov test was used to compare the empirical distributions for included vs. excluded participants and test the null hypothesis that the samples are drawn from the same distribution. Unlike traditional two-sample tests, which focus on the mean and standard deviation, this test is sensitive to all characteristics of a distribution including location, dispersion, and shape. The tests were conducted on the same variables from the logistic regression except gender. The empirical cumulative distribution functions of each group, for each variable is presented online at osf.io/dhy6b. No significant differences were found between the distributions of included and excluded participants (all uncorrected *p* values > .1). In summary, there is no evidence that a difference in impulsivity, demographics, or risk-taking behaviours accounts for compliant and non-compliant performance on the stop-signal task.

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[2] Slowing was estimated by a linear regression of RT by trial number. The slope of each participant's line was then multiplied by the total number of trials to estimate their slowing over the experiment in milliseconds.

2.4.3. Traditional estimation of SSRT

The go task in the stop-signal paradigm used here consisted of a number parity task (i.e., classifying single digit numbers as odd or even) rather than the more common simple classification tasks (e.g., object categorisation (circle or square) or arrow direction (left or right)). Therefore, our go task resulted in higher rates of choice errors (e.g., incorrectly responding that the stimulus was odd, when it was even) on go trials in comparison to simpler go tasks used in other stop-signal studies. Likewise, the increased cognitive load may lead to increased omission errors. The EXG3 model is designed to model both choice errors, as well as errors of omission, whereas traditional methods of SSRT estimation cannot. This is important, because in the presence of high omission errors on go trials, the omission of a response to the go stimulus on a stop trial does not necessarily indicate a successful inhibition. Tannock et al. (1989) described an approach for dealing with omission rates by adjusting the probability of response inhibition using the following formula:

$$P(Inhibit \mid Signal)_{adj} = \frac{P(Inhibit \mid Signal)_{obs} - O}{1 - O} \qquad (1)$$

where O is a correction term equal to the probability of omissions on go trials. We utilised this adjusted probability of responding in the integration technique to calculate SSRT. The integration method is described in Verbruggen and Logan (2009) and is used here on those stop-signal delays with > 10 occurrences for a given participant.

2.4.4. Bayesian modelling of behavioural data

The EXG3 model (Heathcote et al., 2018; Matzke et al., in press) assumes a race between three stochastically independent runners: one runner that corresponds to the stop process and two runners that correspond to the two possible response options in the go task. Correct go responses occur when the "matching" go runner (i.e., runner that matches the choice stimulus) finished before the "mismatch" go runner (i.e., runner that mismatches the choice stimulus); error responses occur when the mismatch runner finishes before the matching runner. The model assumes that the finishing times of the three runners can be described by an ex-Gaussian distribution. The ex-Gaussian distribution is a convolution of a Gaussian (i.e., normal) and an exponential distribution. For each of the go processes and the stop process, the ex-Gaussian distribution has three parameters (μ, σ, and τ), which characterizes the mean and standard deviation of the normal component, and the mean of the exponential component (i.e., the long slow tail of the distribution), respectively. The mean and variance of each finishing time distribution can then be described as $\mu + \tau$ and $\sigma^2 + \tau^2$, respectively. The probability of go and trigger failures were first projected from the probability scale to the real line with a probit transformation (i.e., standard normal cumulative distribution function).

To determine the effect of incorporating the trigger failure parameter, we estimated two models independently using the Dynamic Models of Choice (DMC) software (Heathcote et al., 2018), one with a trigger failure parameter and one without. Both models included a go failure parameter as Matzke et al. (in press) demonstrated that this parameter may be included in the model without causing estimation problems even if go omissions are infrequent. The Deviance Information Criterion (DIC; Spiegelhalter, Best, Carlin, & Van Der Linde, 2002) was used to formally compare hierarchical Bayesian models. Smaller DIC values indicate a better model in terms of providing an accurate yet parsimonious fit to the data. When comparing models, a difference in DIC of 10 or more is taken as substantial evidence in favour of the model with the smaller DIC. DIC values for the two models were compared to establish the most appropriate model for the data, and therefore whether non-negligible trigger failure rates were present. Furthermore, we assessed the absolute goodness-of-fit using posterior predictive model checks (Gelman, Meng, & Stern, 1996). The process of posterior predictive checks involves randomly selecting a set of, in this case 100, parameter vectors from the joint posterior of the participant-

level model parameters. Following this, 100 stop-signal data sets are generated using the parameter vectors. If the model provides an adequate representation of the data, these predictions should closely resemble the observed data. The output of these checks is presented in the supplementary materials at osf.io/dhy6b.

In hierarchical modelling, the population-level mean and standard deviation parameters characterise the population-level distribution for each model parameter. Weakly informative uniform priors were set for the population-level parameters, which closely match those of Heathcote et al. (2018). We establish two sets of population-level priors, one for the mean (location) and one for the standard deviation (scale) of the distributions. For the population means, we specified broad normal hyper-prior distributions truncated below at zero and above by 4000 ms. However, the tau distributions were truncated at 8000 ms to allow for considerable variability in these estimates. For the μ parameters (both go and stop), we set the mean of the hyper-priors to 500 ms and the standard deviation to 1000 ms. For σ and τ, we set the mean to 100 ms and the standard deviation to 1000 ms. For the probability of trigger and go failure we set the mean at -1.5 ($\approx 6.7\%$) with a standard deviation of 1. For the population standard deviations, we chose identical exponential distributions for all parameters. These priors are provided in the supplementary materials found at osf.io/dhy6b. Posterior distributions of the parameters were obtained using Markov Chain Monte Carlo (MCMC) sampling, with steps closely mimicking Heathcote et al. (2018). For each model parameter, we used 33 chains in the model with trigger failure and 30 in the model without (e.g., three times as many chains as model parameters). Participants were initially modelled separately until the MCMC chains reached convergence, with thinning of every 10th sample. Convergence was identified with visual inspection and Gelman-Rubin $\widehat{R}$ (Gelman & Rubin, 1992) values below 1.1. These participant fits were then used as the start values for the hierarchical fits. Each MCMC chain was thinned, retaining every 10th sample. During the burn-in period, we set a 5% probability of migration for both participant and hierarchical levels. After burn-in, migration was turned off and only crossover steps were performed until chains were converged and stable. After this, an additional 200 samples per chain were taken as the final set from which further analysis is undertaken.

The population distributions describe the between-subject variability of the parameters and are appropriate for population inference, analogous to frequentist random-effects analysis. On the other hand, individual participant parameters are useful for examining individual differences, and are used here to examine the relationships between response inhibition and impulsivity. Our analysis of stop parameters focused on the model's estimate of mean SSRT (i.e., $\mu_{stop} + \tau_{stop}$) to maintain comparability with the majority of previous studies that have examined SSRT.

2.4.5. Plausible value analysis

Traditional tests of correlations between model parameters and both impulsivity and outcome covariates ignore the uncertainty of the parameter estimates, and so tend to be overconfident. Here we use a plausible value analysis to evaluate these relationships, which calculates a distribution of correlations between covariates (e.g., BIS-11 Total) and model parameters and mean SSRT using each MCMC sample from the posterior distribution of the parameters for each participant. Ly, Marsman, and Wagenmakers (2018) provide a detailed rationale for this fully Bayesian and more conservative approach, which is more appropriate for the novel effects examined here (Benjamin et al., 2018; Open Science Collaboration, 2015). This process results in a set of 'plausible' values of the sample correlation, r, for n individuals. As described by Ly, Boehm, et al. (2018), the sample correlation can be transformed into the posterior distribution of the population correlation (ρ), which is a function of r and n. Repeating this process for all plausible sample correlations and averaging each population correlation distribution yields the estimated posterior distribution of the

population correlation.

2.5. Statistical procedures

In line with the previous literature, we examined the relationship between the integration method estimate of SSRT (see Verbruggen & Logan, 2009) and impulsivity, risk-taking, and outcome covariates. We provide frequentist statistics most typically used in prior literature, with all frequentist *p*-values False Discovery Rate (FDR) corrected at alpha = 0.05. However, as we expected weak, non-significant results, Bayesian correlations were also used (JASP Team, 2018), with the *r* values reported being the mean of the posterior distribution. Bayes factors were computed to quantify evidence in favour of a null relationship. We used default uniform (i.e., scaled beta (1,1)) priors for the Bayesian correlations. The uniform prior on the correlation assigns equal likelihood for all values of ρ between −1 and 1. To examine sex differences in the measures of impulsivity we used a Bayesian *t*-test, as implemented in JASP with a Cauchy prior with a scale of 0.707. Associated *p* values are also FDR corrected at 0.05. Effect sizes are given using the posterior median effect size from JASP (δ). A two-sided Bayes factor (BF_{10}) of 2 tells us that the data are 2 times more likely to occur under the alternative hypothesis than the null. The inverse can be taken to describe the level of evidence for the null (e.g., 0.5 times more likely to occur under the null hypothesis). We use the Kass and Raftery (1995) conventions to describe the strength of evidence the data provide for the alternative hypothesis. A Bayes factor between 1/3 and 3 is considered 'Equivocal' (i.e., indicating more data are needed to obtain a clear outcome), between 1/20–1/3 and 3–20 is considered 'Positive', between 1/150–1/20 and 20–150 'Strong', and Bayes factors < 1/150 and > 150 are considered 'Very Strong'.

We use the plausible values approach in order to capture the wealth of information that is provided by the posterior distributions from the hierarchical modelling. The posterior distribution of the population correlation ρ is summarized by its median, alongside the 95% credible interval (i.e., an interval from the 2.5th to 97.5th percentiles of the distribution). If the 95% credible interval does not include zero, we consider a reliable correlation to be present, as 95% or more of the plausible correlations are greater (in case of a positive correlation) or lower (in case of a negative correlation) than zero. We quantify this using Bayesian *p* values, which quantify the area of the posterior distribution of the population correlation above or below zero and are the basis for inference about correlations that would occur in a new sample of participants. As some of our relationships are with novel parameters, we use two-sided tests so that a relationship is considered reliable if the Bayesian *p* < .025.

3. Results

3.1. Demographics

Participants (*n* = 174, 55% female) were aged 21.3 ± 4.8 years (mean ± SD, 15–35 year range), and were targeted to have a higher representation of younger age groups (median age = 20 years). Education level was 12.67 ± 1.2 years, with 40.8% of participants enrolled in or having completed a Bachelor degree or higher. The socioeconomic attributes of most participants were average with 49% living in postal codes above the fifth decile of socioeconomic advantage.

Overall, general cognitive ability was high and showed large individual variability. Span length was within the normal range on the Digit Span Forwards (6.99 ± 1.27; Iverson & Tulsky, 2003; Miller, 1956) and variability remained consistent across the sequencing and backwards span conditions. Matrix Reasoning scores were almost half a standard deviation above the norm (mean T score of 53.6). The total Verbal Fluency score was 40.27 ± 11.12, which is within the typical average range (Tombaugh, Kozak, & Rees, 1999).

3.2. Outcome measures

On average, participants reported low to moderate levels of risk behaviours in the past 6 months on the CARE, with highest means on hazardous drinking and level of irresponsible work behaviours (Table 1). These scores did not vary with age or behavioural risk-taking measures (Table A.1 contains the full description of these correlations and their associated statistics) but were related to some of the self-report impulsivity measures. Higher levels of illicit drug taking was moderately correlated with higher scores on SSS scales of disinhibition (*r* = 0.37, BF_{10} > 150), experience seeking (*r* = 0.33, BF_{10} > 150), and thrill seeking (*r* = 0.32, BF_{10} = 7.07), as well as the BIS subscales (except attentional; *r* = 0.22–0.27, BF_{10} = 3.8–22.7). However, note that while these relationships were all significant in the frequentist sense at p_{FDR} < 0.05, they had varying levels of support from the Bayes factors ranging from 'Positive' to 'Very Strong'.

Risky drinking behaviour was correlated with higher self-report impulsivity and sensation seeking. Larger total scores on the BIS were correlated with more risky drinking behaviour, *r* = 0.28, p_{FDR} < 0.001, BF_{10} = 51.52. While there was positive relationships between risky drinking and the motor (*r* = 0.28, p_{FDR} < 0.001, BF_{10} = 38.34) and attention (*r* = 0.23, p_{FDR} < 0.001, BF_{10} = 6.93) subscales of the BIS, the support for these relationships was quite different. More irresponsible work and academic behaviour was associated with all BIS scales (*r* = 0.24–0.31, p_{FDR} < 0.001, BF_{10} = 8.6–150) but no other measures or impulsivity or risk-taking.

3.3. Self-report measures of impulsivity

Consistent with the literature, we find no gender effect on total ($t_{(157)}$ = 0.872, p_{FDR} = 0.673, BF_{10} = 0.245) or subscale BIS scores (Stanford et al., 2009). However, the evidence for the data under the null (i.e., that there is no gender difference) was weak. Mean total score on the BIS was 62.96 ± 10.64 on a scale ranging from 30 to 120. A total BIS score larger than 71 is often considered as highly impulsive. In our sample 34 (19.5%) participants fit this criterion. Conversely, 25 (14%) participants scored below 52, which suggests that they either had a very high level of self-control or were not compliant in completing the questionnaire (Stanford et al., 2009). The remaining majority of participants fell within the normal ranges of impulsive behaviour. Older age was weakly associated with lower scores on the BIS Attentional Impulsivity subscale and found to be significant, but there was little support for this effect from the Bayes factor analysis (*r* = −0.21, p_{FDR} < 0.05, BF_{10} = 2.78; see Table A.1). No other supported correlations were found between BIS scores and age.

Scores on the Sensation Seeking Scale (SSS) were largely within normal range (Ball, Farnill, & Wangeman, 1984; Zuckerman, Eysenck, & Eysenck, 1978). Males scored higher than females on the total score (Males: 22.36 ± 6.6; Females: 18.16 ± 6.3), $t_{(155)}$ = 4.06, p_{FDR} = 0.002, BF_{10} = 261.67, δ = 0.61, and this effect was present for most subscales, i.e., Disinhibition ($t_{(152)}$ = 2.94, p_{FDR} = 0.02, BF_{10} = 8.64, δ = 0.44), Thrill Seeking ($t_{(154)}$ = 3.05, p_{FDR} = 0.02, BF_{10} = 11.7, δ = 0.46), and Boredom Susceptibility ($t_{(153)}$ = 3.14, p_{FDR} = 0.02, BF_{10} = 14.93, δ = 0.47). Scores on the SSS scales did not vary with age (Table A.1).

3.4. Behavioural measures of impulsivity and risk-taking

On the CGT, mean deliberation time (time to make red or blue choice) was 2007 ± 649 ms and the quality of decision-making (proportion of times the most probable option was selected) was high with 79% of participants choosing the most likely outcome at least nine out of 10 times. On the IST, the majority of participants (96%) made less than two discrimination errors on either condition and did so in an appropriate amount of time (13,467 ± 7272 ms). These scores indicate that participants were compliant on both tasks.

Table 2
Median, standard deviation, and 95% credible intervals of the population-level mean parameters from the selected EXG3 model.

Population mean	Posterior median	Credible interval	
		2.5%	97.5%
SSRT (ms)	131.5	124.4	138.6
Mean Matching Go (ms)	600.5	586.4	614.2
Mean Mismatch Go (ms)	2228.2	581.8	4320.7
Go Failure (%)	1.6	1.3	2
Trigger Failure (%)	17.9	15.6	20.4

Note. SSRT = Stop-signal reaction time.

Mean CGT risk adjustment scores on ascending and descending trials indicate that participants adjusted their betting in accordance with the certainty of each trial's outcome (Table 1). Both conditions showed a broad range, with good individual variability. Delay aversion scores showed substantial variability among participants, with the largest range on the 8:2 bet proportion being −0.16 to 0.9. Since delay aversion is calculated as the betting behaviour on descending minus ascending trials, the average of ≈0.25 across bet proportions suggests that many participants engaged in some level of impulsive decision-making.

On the IST, there was a moderate ceiling effect in the fixed win condition - almost all participants opened every box and made a decision only when certain, thus yielding an average probability of being correct of 89.5%. However, when the points available decreased with every box opened, participants were far more likely to make a decision without certainty. The average probability of being correct was 0.81 (ranging from 0.64–1). Age was weakly negatively correlated with delay aversion on 7:3 ($r = -0.23$, $p_{FDR} < 0.001$, $BF_{10} = 10.47$) and 6:4 ($r = -0.21$, $p_{FDR} < 0.05$, $BF_{10} = 3.95$) bet proportions. However, note the weak support for the correlation between age and delay aversion 6:4. No other behavioural impulsivity scores were related with age (Table A.1) and none were found to differ by sex.

3.5. Stop-signal task

3.5.1. Sample characteristics
In the final sample, participants responded on average to 48 ± 5% (mean ± SD) of stop trials, which is consistent with the 50% adaptive algorithm. Errors of omission and choice occurred on 2.92 ± 3.8% and 7.07 ± 6.3% of go trials, respectively. The adjusted integration technique returned an average $SSRT_{int}$ of 247.1 ± 132.85 ms.

3.5.2. Model fits and comparison
For both models, the Gelman-Rubin $\widehat{R}$ (Gelman & Rubin, 1992) diagnostics for convergence were below the recommended criterion of 1.10 for all parameters. Furthermore, visual inspection suggested that the MCMC chains achieved convergence. The chains for the hierarchical and individual level parameters and the results of the posterior predictive checks for both models can be found at osf.io/dhy6b. The posterior predictive data generated from the EXG3 model with both failure parameters (i.e., trigger and go failure), more closely matched the observed data than the model without trigger failures. A DIC difference of 1899 between the models strongly supports that inclusion of the trigger failure and suggests the non-negligible presence of trigger failures. Hence, we used the EXG3 model with both failure parameters in all further analyses.

Table 2 summarises the posterior distribution of the population-level mean parameters for the selected EXG3 model. The median of the posterior distributions and 95% credible intervals are given for the mean (i.e., μ + τ) of the SSRT and finishing times of both go process distributions, their standard deviation (SD), as well as the probability of

both go and trigger failure. Probability of go and trigger failure is transformed from the probit scale used in estimation to percentages for reporting and analysis using an inverse-probit transform (see the *pnorm* function in the R statistical language; R Core Team, 2018).

The posterior distributions of the population-level mean go time for the matching runner, probability of go failure, probability of trigger failure, and mean SSRT are presented in Fig. 2 in black, with individual participant-level posterior distributions in grey. In line with the DIC results, the median of the trigger failure posterior distribution was high at 17.9%. The median of the probability of go failure posterior was 1.6%. Samples for the plausible values analysis are taken from each individual's posterior distribution.

3.6. Relationships between traditional estimates of SSRT and measures of impulsivity and outcome behaviours

Results for correlations between the $SSRT_{int}$ estimate and measures of impulsivity, risk-taking, as well as outcome behaviours were mixed (Table 3). From the 22 covariates, 6 had Bayes factors in the 'Strong' range of evidence for the relationship. The behavioural measures of impulsivity that fell into this category were delay aversion subscales ($r = 0.33$–0.34, $p_{FDR} < 0.001$, $BF_{10} > 150$) and risk adjustment on descending trials ($r = 0.25$, $p_{FDR} < 0.001$, $BF_{10} = 22.94$). Relationships with delay aversion suggested that increased aversion to delay was associated with increased SSRT. Likewise, participants who had poorer risk adjustment had increased SSRT. For the self-report measures, increased BIS total and BIS non-planning scores were significantly correlated with increased $SSRT_{int}$ (both $p_{FDR} < 0.05$). However, differing levels of support were found, with a 'Strong' Bayes factor of 27.36 for BIS Non-planning, but a 'Positive' 6.22 for the total BIS score. For the majority of the other relationships tested there was at least positive evidence (i.e., $BF_{10} < \frac{1}{3}$) in favour of a null relationship with $SSRT_{int}$, except for BIS Attention, SSS Experience Seeking, CARE Drinking, and risk adjustment on ascending trials where the result was equivocal (i.e., all $BF_{10} = \frac{1}{3} - 3$).

3.7. Relationships between EXG3 model parameters and measures of impulsivity and outcome behaviours

Table 4 displays summary statistics of the plausible values analysis for the relationship between SSRT and the trigger failure parameters estimated from the EXG3 model, and measures of impulsivity and outcome behaviours. The posterior distribution of the population correlation for each covariate can be found at osf.io/dhy6b. SSRT estimates did not reliably correlate with any self-report and behavioural measures of impulsivity, or with any outcome variables as indicated by Bayesian *p* values > .025.

In contrast, the probability of trigger failure was related to the same behavioural impulsivity variables found to be correlated with the traditional estimate of SSRT in Table 3. As shown in Table 4, a higher probability of trigger failure was correlated with higher delay aversion (for all bet proportions). The relationships with delay aversion scores were very reliable with 100% of the posterior distribution of the population correlation falling above zero. The strength of the correlations ranged from 0.258 (9:1 bet proportion) to 0.311 (6:4 bet proportion), with the upper bound of the credible interval reaching a moderate 0.445 with the 6:4 bet proportion. Likewise, the relationship with risk adjustment suggested increased probability of trigger failure was associated with poorer risk adjustment. On the other hand, self-report measures of impulsivity which previously correlated with $SSRT_{int}$ (i.e., BIS Non-Planning and Total scores) were not found to relate to trigger failure.

As shown in Table 4, the mean of the matching go parameter and the probability of go failure showed a similar pattern of positive relationships with delay aversion and risk adjustment scores as the probability of trigger failure. Specifically, slower finishing time of the
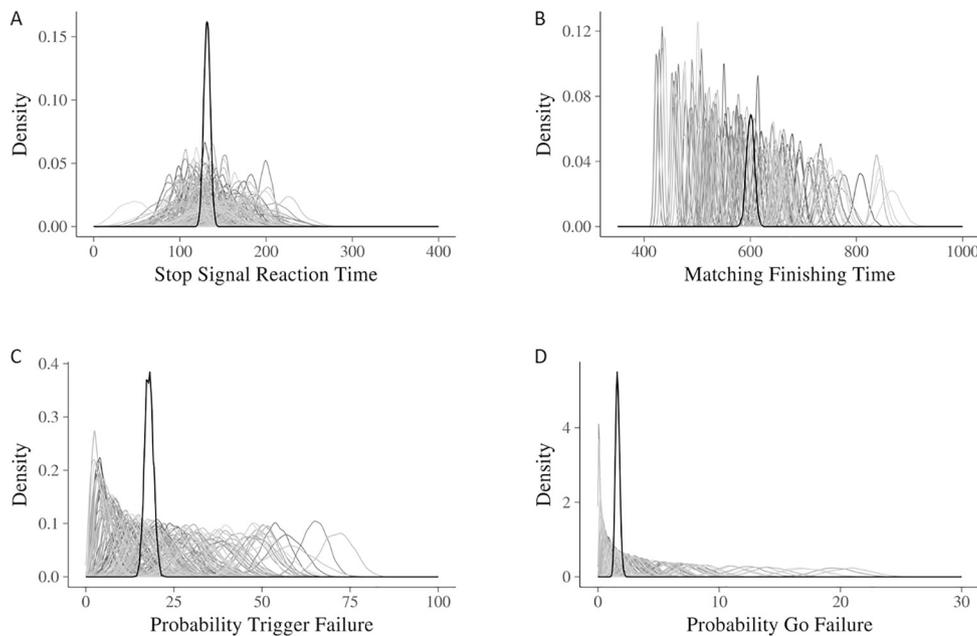
Fig. 2. Posterior distributions of the individual (grey density lines) and group-level mean (black density lines) parameters. Mean Stop Signal Reaction Time (Milliseconds; Panel A). Mean finishing time for the matching go runner (Milliseconds; Panel B). Probability of Trigger Failure (Panel C). Probability of Go Failure (Panel D). Each grey shaded distribution represents the posterior of a single participant. The black distribution represents the posterior distribution of the population-level mean parameter.

Table 3

Summary statistics for the correlations between $SSRT_{int}$ and the impulsivity, risk-taking, and outcome covariates. Pearson product moment correlation coefficient (r), frequentist significance, and associated Bayes factors are given.

	r	BF_{10}
Self-report Impulsivity		
BIS (Attention)	.2	2.5
BIS (Motor)	.1	.2
BIS (Non-Planning)	.26	27.36*
BIS (Total)	.23	6.22*
SSS (Boredom Susc)	.08	.16
SSS (Disinhibition)	.03	.11
SSS (Exp Seek)	-.17	.84
SSS (Thrill Seeking)	-.03	.11
SSS (Total)	-.03	.11
Outcome Variables		
CARE (Drug)	.02	.1
CARE (Aggr/Illegal)	.05	.12
CARE (Sex)	-.01	.1
CARE (Drink)	-.13	.38
CARE (Work)	0	.1
Behavioural Impulsivity		
P(Correct) Fixed	0	.09
P(Correct) Decreasing	-.05	.12
Delay Aversion (9:1)	.34	2625.89**
Delay Aversion (8:2)	.35	6667.28**
Delay Aversion (7:3)	.33	1401.26**
Delay Aversion (6:4)	.38	36553.93**
Risk Adjustment (Asc)	.13	.38
Risk Adjustment (Des)	.25	22.94

Note. The number of participants changes for each correlation: BIS subscales ($N = 159$). BIS Motor ($N = 158$). SSS Boredom ($N = 154$). SSS Disinhibition ($N = 153$). SSS Experience Seeking (N = 153). SSS Thill Seeking ($N = 155$). SSS total ($N = 156$). Probability Correct ($N = 172$). CARE Drug (N = 154). CARE Aggression (N = 156). CARE Sex (N = 155). CARE Drink (N = 156). CARE Work (N = 156). Age ($N = 173$). All other variables ($N = 171$). Cases were removed for pairwise complete observations. ** = $p_{FDR} < 0.001$, * = $p_{FDR} < 0.05$. Estimation of $SSRT_{int}$ is detailed in Section 2.4.3, Traditional Estimation of SSRT. Correlations with at least 'positive' evidence for the alternative hypothesis are shaded grey.

matching go parameter and higher probability of go failure were associated with more delay aversion. However, the relationship between the matching go parameter and delay aversion was not reliable on the 9:1 bet proportion. Furthermore, the while the Bayesian *p*-value indicated that the relationship between the matching go parameter and delay aversion 6:4 was reliable, the lower bound of the credible interval does cross zero. Therefore, the relationships between delay aversion and the matching go parameter are not as consistent as with trigger failure. Poorer risk adjustment was associated with both slowing in the matching go parameter and a higher probability of go failure. Although both parameters correlated with risk adjustment on descending trials, risk adjustment on ascending trials was only correlated with go failure. However, the lower bound of the latter relationship crossed zero and therefore shouldn't be considered as reliable.

Both the matching go parameter and go failure correlated with BIS non-planning impulsivity, and go failure correlated with BIS total. These were the same self-report measures which correlated with $SSRT_{int}$, but not trigger failure. In other words, only go parameters were found to correlate with self-report measures of impulsivity, unlike the stop related parameters. Although these correlations were weaker than those found with probability of trigger failure (Table 4), they were highly reliable (e.g., < 1% of the population plausible distribution was below zero).[3]

4. Discussion

This study set out to examine the relationship between response inhibition and impulsivity in a large healthy adolescent and young adult sample. In order to comprehensively characterise performance on the stop-signal task, we used both traditional estimates of SSRT as well as a novel hierarchical Bayesian approach to model the speed of the inhibition process (i.e., SSRT), and in the latter case, the reliability of this process (i.e., trigger failure) and go-task parameters. In addition, we used multiple measures of both self-reported and behavioural

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[3] See Appendix for relationship between impulsivity and outcome measures and other EXG3 parameters. Table A.2 shows that the variance of SSRT did not reliably correlate with impulsivity or outcome measures. However, the variance of the matching go parameter essentially mirrored the relationships found with trigger failure and go RT. Table A.3 shows that the mismatching go parameter and its variance showed few reliable correlations with these variables.

impulsivity, as well as risk-taking outcome behaviours typically believed to be associated with poor inhibition and high impulsivity.

As predicted, the traditional estimate of SSRT (i.e., SSRT$_{int}$) was weakly correlated with self-report impulsivity measures from the BIS (i.e., total score, non-planning subscale) and more strongly correlated with behavioural impulsivity measures, especially the delay aversion measure of the CGT. Bayesian analyses showed evidence favouring the absence of a relationship for most of the remaining measures of self-report and behavioural impulsivity, as well as most outcome behaviours (e.g., risky sexual activities and illicit drug use). However, there were a number of relationships with ambiguous or weak evidence in either direction and further examination is needed to determine the presence or absence of a relationship for those variables (e.g., SSS thrill seeking and self-reported risky drinking).

It is important to note that, although we found a relationship between traditional SSRT and both self-report and behavioural impulsivity measures, this relationship was not ubiquitous. For the self-report measures, it was stronger and most supported for the BIS non-planning impulsivity subscale, and weaker but still in the 'Positive' range of evidence for the BIS total score. There was 'Positive' evidence in favour of a null relationship with the motor subscale and ambiguous evidence for the relationship with BIS attention. Sensation seeking subscales had at least 'Positive' evidence in favour of a null relationship, except the experience seeking subscale, which had ambiguous evidence.

For the behavioural impulsivity measures, relationships with traditional SSRT were strongest and most supported for CGT delay aversion scores, but less so for risk adjustment – descending (i.e., adjusting bet size to match chance of winning). The Bayes factors provided ambiguous evidence for the relationships between traditional SSRT and the corresponding ascending condition, but null evidence was supported by IST probability of being correct measures that more directly target impulsive decision-making. This pattern of findings suggests that discrepancies in the literature are likely to arise, at least partly, from the type of impulsivity being targeted by the specific self-report and behavioural measures of impulsivity used. These findings are consistent with the findings from the Cyders and Coskunpinar (2011) meta-analysis that a prepotent response inhibition factor correlates with factors representing delay aversion and lack of planning.

Using traditional estimates of SSRT, the ability to withhold a response (i.e., delay aversion) and the ability to cancel a response (i.e., response inhibition) have been suggested to rely at least partly on a similar underlying process (Bissett, Nee, & Jonides, 2009; Wessel, 2018b). Sharma et al. (2014) showed that SSRT cross loaded with factors representing go/no-go based response inhibition and a delayed gratification task. Sharma et al. proposed that an interplay between personality traits, generic impulsivity, and the ability to both inhibit and delay responses are core to the general construct of impulse control in the real world. Other evidence suggests that combining measures of delay aversion and response inhibition can better discriminate children with ADHD from controls than either measure alone (Solanto et al., 2001).

In contrast to our findings with the traditional SSRT estimate, the mean SSRT derived using the EXG3 model was not reliably correlated with any impulsivity or outcome measure. Instead, the exact same behavioural impulsivity measures (but not BIS scales) that correlated with the traditional estimate of SSRT, were now reliably related with the estimated probability of trigger failure. This novel finding has important implications for our understanding of the structure of response inhibition as well as the relationship between response inhibition and impulsivity. It is possible that a common higher order mechanism may mediate both the ability to withhold a response (delay aversion) and the resources necessary to trigger an inhibitory response. Such a mechanism may be similar to that has been previously attributed to SSRT (e.g., Robbins & Dalley, 2017). Alternatively, an inability to withhold a response (delay aversion) could lead to inappropriately withholding the stop response (trigger failure).

4.1. Stop-signal reaction time as a proxy for effortful inhibitory control

Consistent with the findings of Matzke, Hughes, et al. (2017) and Matzke, Love, and Heathcote (2017) using the BEESTS model, accounting for trigger failure substantially attenuated the estimate of mean SSRT. Specifically, mean SSRT from the EXG3 model was over 100 ms faster than the traditional SSRT derived using the Tannock et al. (1989) method. In their seminal paper, Logan and Cowan (1984, p. 302) reported SSRT estimates in the 150–300 ms range with a mean of 200 ms, which they noted is analogous to simple RT to a tone presented by itself. Our mean SSRT estimate of around 130 ms – when not contaminated by trigger failures – is certainly faster than this, but not greatly so. It is also not inconsistent with simple RT to loud tones (Luce, 1986), especially considering that simple RT also includes response execution time (which is not included in SSRT). Likewise, recent electromyography (EMG) work suggests that SSRT may also be shorter than previously reported. Sub-threshold EMG activity to the go stimulus was interrupted as early as 150 ms after the stop stimulus (Raud & Huster, 2017). Hence, the fast mean SSRT estimate found here is plausible and suggestive of a reflexive type of action control that is activated by the stop signal in a fairly automatic manner, rather than a slow effortful cognitive control process that is more likely to subserve any link between response inhibition and impulsivity.

Both the fast estimates of mean SSRT produced by the EXG3 model and the absence of evidence for a relationship between this more valid estimate of SSRT and measures of impulsivity, are consistent with growing evidence that, under some conditions at least, response inhibition may be accomplished without a strong need for cognitive control. Recent evidence also suggests that such automatic inhibitory responses (i.e., responses that do not require intentional inhibition) can be produced by associative pairing of specific 'go' stimulus with a stop signal (Verbruggen, Best, Bowditch, Stevens, & McLaren, 2014) or by presenting an unexpected stimulus (Wessel, 2018a). Although our paradigm did not have either manipulation, it is possible that our loud auditory stop stimulus quickly became associated with a fast and automatic stop response.

The present finding that the mean SSRT derived from the EXG3 model is over 100 ms faster than that derived from traditional SSRT estimates suggests that, by not taking account of trigger failures, traditional approaches of estimating SSRT may mask the reflexive nature of at least some of the inhibitory processes involved in standard stop-signal paradigms. If SSRT indexes a process that is driven by more automatic than controlled processes, it may help explain the inconsistency of the relationship between SSRT and impulsivity within the literature, especially with paradigms that are unlikely to produce high levels of trigger failure. As noted by Logan and Cowan (1984), such automatic processes, so long as they are enacted to achieve or maintain a goal, can still be themselves considered a control mechanism. The present findings are not inconsistent with this position. Instead, they qualify this position by indicating that, when differentiating between distinct response inhibition processes, individual variability in impulsivity is not associated with the more process-pure SSRT measure, but rather with processes that control the activation of the goal to inhibit a response, that are indexed by trigger failure.

Although a number of other studies have found that allowing for trigger failures attenuates estimates of SSRT, only one has explored the possible covariates associated with trigger failure. Matzke, Hughes, et al. (2017) reported higher rates of trigger failure in people with schizophrenia relative to controls and examined the relationship between trigger failure and event-related potential (ERP) responses elicited during the SST task. Trigger failure was correlated with the latency of the N1 to the stop-signal, an ERP component associated with early attentional processes. They interpreted trigger failure as reflecting a type of goal-neglect, or attentional failure to utilize the auditory stop

Table 4

Summary statistics for plausible values analysis between SSRT and trigger failure derived from the EXG3 model and covariates. Median of the posterior distribution of the population correlation, corresponding Bayesian p-value, and credible interval are given.

	SSRT			Trigger Failure			Matching Go RT			Go Failure		
	Median ρ	Bayes P value	Credible Interval [2.5%, 97.5%]	Median ρ	Bayes P value	Credible Interval [2.5%, 97.5%]	Median ρ	Bayes P value	Credible Interval [2.5%, 97.5%]	Median ρ	Bayes P value	Credible Interval [2.5%, 97.5%]
Self-report Impulsivity												
BIS (Attention)	.090	.149	[-.09, .264]	.070	.183	[-.093, .229]	.022	.366	[-.134, .177]	.089	.123	[-.07, .243]
BIS (Motor)	.076	.185	[-.102, .249]	.080	.153	[-.084, .24]	.102	.090	[-.055, .253]	.150	.028	[-.009, .3]
BIS (Non-Planning)	.075	.187	[-.102, .247]	.123	.061	[-.04, .278]	.218	.002	[.065, .361]	.178	.012	[.021, .326]
BIS (Total)	.097	.132	[-.082, .269]	.110	.083	[-.053, .267]	.145	.029	[-.011, .293]	.170	.015	[.013, .319]
SSS (Boredom Susc)	-.017	.426	[-.197, .163]	.151	.031	[-.013, .307]	.071	.173	[-.087, .226]	.124	.057	[-.036, .278]
SSS (Disinhibition)	.032	.341	[-.145, .207]	.007	.444	[-.157, .17]	.059	.214	[-.1, .215]	-.009	.458	[-.168, .151]
SSS (Exp Seek)	.040	.308	[-.137, .214]	-.101	.113	[-.261, .063]	-.032	.345	[-.189, .126]	-.148	.036	[-.302, .013]
SSS (Thrill Seeking)	-.068	.229	[-.242, .111]	.010	.429	[-.153, .172]	.023	.365	[-.134, .179]	.002	.463	[-.156, .161]
SSS (Total)	-.004	.484	[-.178, .17]	.022	.373	[-.14, .183]	.046	.261	[-.111, .201]	-.001	.480	[-.159, .157]
Outcome Variables												
CARE (Drug)	.075	.173	[-.092, .237]	-.099	.114	[-.256, .063]	-.027	.370	[-.183, .131]	-.072	.185	[-.227, .086]
CARE (Aggr/Illegal)	.161	.033	[-.016, .328]	.047	.271	[-.119, .21]	-.041	.303	[-.197, .116]	-.032	.346	[-.19, .127]
CARE (Sex)	.117	.089	[-.062, .287]	-.027	.373	[-.189, .137]	.030	.331	[-.128, .187]	-.048	.276	[-.205, .111]
CARE (Drink)	.025	.365	[-.145, .192]	-.067	.210	[-.227, .096]	-.030	.353	[-.187, .127]	-.050	.269	[-.207, .109]
CARE (Work)	.056	.247	[-.12, .226]	.010	.427	[-.153, .173]	-.039	.315	[-.195, .119]	-.038	.319	[-.196, .121]
Behavioural Impulsivity												
P(Correct) Fixed	-.062	.238	[-.229, .108]	.031	.323	[-.124, .185]	-.109	.075	[-.254, .04]	.010	.421	[-.141, .161]
P(Correct) Decreasing	.019	.389	[-.146, .183]	-.091	.123	[-.241, .063]	-.031	.340	[-.179, .118]	.120	.052	[-.031, .266]
Delay Aversion (9:1)	.027	.358	[-.146, .197]	.258	.001	[.106, .398]	.131	.037	[-.019, .275]	.202	.004	[.051, .343]
Delay Aversion (8:2)	.052	.261	[-.122, .222]	.268	0	[.117, .407]	.169	.011	[.02, .31]	.209	.003	[.059, .35]
Delay Aversion (7:3)	-.007	.469	[-.18, .166]	.279	0	[.128, .417]	.179	.008	[.031, .32]	.229	.001	[.08, .369]
Delay Aversion (6:4)	.007	.446	[-.166, .179]	.311	0	[.162, .445]	.147	.023	[-.003, .29]	.198	.004	[.047, .339]
Risk Adjustment (Asc)	.053	.252	[-.118, .221]	.091	.114	[-.066, .242]	.098	.089	[-.052, .244]	.149	.024	[-.003, .294]
Risk Adjustment (Des)	-.030	.363	[-.197, .138]	.187	.008	[.032, .333]	.154	.019	[.005, .296]	.187	.006	[.037, .328]

Note. Bayesian *p* value is the proportion of the posterior distribution shifted away from zero. For example, if only 2.5% of the posterior distribution is below zero than the *p* value = .025. Those deemed reliable on this basis are shaded grey.

signal. As the auditory signal was well above threshold (i.e., ≈ 85 dB), they argued that trigger failure was unlikely to reflect a failure of sensory processing, and so more likely to represent an attentional deficit. This interpretation of trigger failure is compatible with a failure at an action-selection stage (as opposed to the stimulus encoding or action execution stages) as discussed by De Jong, Coles, Logan, and Gratton et al. (1990) and Verbruggen, McLaren, and Chambers (2014). In other words, although the signal is encoded, its context is not interpreted and the appropriate action (i.e., inhibition) is not triggered. Although trigger failure refers only to the failure to execute a stop response, go failures may consist of a mixture of both 'true' failures to respond and sensory lapses. The very brief (100 ms) go stimuli used here may have increased the frequency of such sensory lapses (due to blinks or eye saccades). If a common mechanism underlying stop and go failures is driving the relationship with delay aversion, the failure to find a matching relationships between both failure parameters and impulsivity may be due sensory lapses adding noise to go failures.

In sum, the present findings supports the idea that response inhibition may involve distinct mechanisms (Chatham et al., 2012; Logan, Van Zandt, Verbruggen, & Wagenmakers, 2014; Logan, Yamaguchi, Schall, & Palmeri, 2015; Munakata et al., 2011; Schall, Palmeri, & Logan, 2017; Verbruggen & Logan, 2009; Verbruggen, McLaren, & Chambers, 2014): An automatic process captured by SSRT and a process indexed by trigger failure (and possibly both failure parameters) which better reflects impulse control. However, further work is needed to determine the cognitive and neural basis of these response inhibition mechanisms and examine whether trigger and go failures share a common underlying process.

4.2. Limitations and future research

The demanding go task paradigm used here to increase cognitive load (number classification task) and encourage speeded responding (100 ms go stimulus exposure), combined with the fact that the stop-signal task was completed at the end of a long testing session (after completing a task-switching paradigm), may have inadvertently resulted in a performance pattern that differs from typical stop-signal studies. The demanding paradigm and testing conditions may have contributed to the exclusion of 16% of participants using the EXG3 approach (Heathcote et al., 2018; Matzke et al., in press) that directly models go errors and go omissions. Importantly, there was no evidence of differences between included and excluded participants in impulsivity, outcome, and demographic variables. Therefore, the exclusions are unlikely to have impacted the relationships between SSRT, trigger failure and impulsivity reported here. Nevertheless, it is possible that the demanding task and context may have contributed to the relatively high trigger failure. Future work is needed to examine whether a similar pattern of findings is found with typical stop-signal paradigms that use a simpler go task and can be replicated with other go-tasks yielding high omission and/or error rates.

The go task used here may also have affected the ability to fit the data to the EXG3 model. As shown in the posterior predictive checks (osf.io/dhy6b), there was a quite small but systematic tendency for the inhibition function predicted by the EXG3 model to be steeper than the observed inhibition function, even though all other aspects of the data were well fit. This tendency contrasts with the fits reported by Matzke et al. (in press) where the inhibition function for a stop-signal task with a difficult go choice was well fit. One potential explanation of the misfit is a minor violation of context independence in the present task because the go RT distribution was more variable when a stop signal occurred than when it did not. One potential difference between the two difficult choice paradigms is that, in the present study, the go stimulus was only presented briefly, so that the choice was based on a representation in visual short-term memory. It is possible that this visual representation was vulnerable to interference from the stop signal, resulting in greater variability in the time course of processing the go stimulus on stop trials

compared to trials without a stop signal. In contrast, in Matzke et al. (in press) the choice stimulus remained on until the response was elicited, as typical in stop-signal paradigms. Clearly, however, this explanation is speculative and requires further investigation.

The large number and variety of measures used here as covariates allowed us to investigate relationships across a range of different facets of impulsivity and risk taking. However, this meant that we also conducted a large number of tests. In the frequentist analysis, we attempted to compensate for this by using a false discovery rate correction. Unfortunately, there is no consensus on how to apply a similar correction to the Bayesian analysis (especially to posterior inference using Bayesian *p*-values). Promisingly, frequentist and Bayesian analysis produced consistent findings, with only few instances of significant relationships not being supported by Bayes factors (see Table A.1). Likewise, the consistency of relationships across different EXG3 parameters suggests that the findings are less likely to be due to multiple testing. In particular, the relationships with delay aversion and risk adjustment, and the BIS non-planning and total scales were supported consistently, although in different parameters for traditional and EXG3 analyses. Nevertheless, these findings need to be treated with caution until replicated.

Finally, the much faster estimates of SSRT derived from models that account for trigger failures have important implications for previous interpretations of the temporal link between SSRT and ERP activity time-locked to the stop signal (for a review, see Aron, Robbins, & Poldrack, 2014; Huster, Enriquez-Geppert, Lavallee, Falkenstein, & Herrmann, 2013). If, as suggested here, after accounting for trigger failure, SSRT latency is substantially reduced, this would require a re-evaluation of interpretations that based on the temporal link between traditional estimates of SSRT and P3 latency (Wagner, Wessel, Ghahremani, & Aron, 2018; Wessel & Aron, 2015). For instance, based on the common finding that SSRT latency typically coincides with the onset latency of the P3 component, the P3 has often been interpreted as representing the execution of the inhibition process (e.g., Wessel & Aron, 2015). However, others have argued that the P3 occurs too late and is more likely related to a later context updating process (for a review, see Huster et al., 2013). We are currently examining whether the improved estimation of SSRT after accounting for trigger failure shows similar relationships with P3 latency and other ERP components as the traditional SSRT (Skippen et al., in preparation).

In conclusion, our findings in healthy young participants, together with those of Matzke and colleagues in participants with schizophrenia and their controls (Matzke, Hughes, et al., 2017; Matzke, Love, & Heathcote, 2017; Matzke et al., in press), strongly suggest that traditional summary measures of SSRT may need to be reconceptualised. These findings support the existence of at least two distinct processes contributing to response inhibition ability: relatively more automatic processes represented in mean SSRT and more higher-order processes represented in trigger failure, and that these processes differentially map to behavioural measures of impulsivity in healthy young people. Further work is needed to replicate this multiple process account of response inhibition, define the underlying cognitive and neural processes, and examine their impact on outcome behaviours in clinical as well as healthy populations.

members of the Age-ility Project past and present for assistance with data collection/entry. Special thanks to Montana McKewen and Patrick Cooper for their contributions to project management. We would also like to thank participants for their time.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.actpsy.2018.10.016.

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