Neural correlates of stopping and self-reported impulsivity

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Abstract

Objective: To examine the relation between self-reported impulsivity, inhibitory control, and the neural correlates of stopping performance within the normal population.

Methods: Healthy individuals scoring high and low on trait impulsivity performed an auditory stop-signal task. Stopping performance and neural correlates of stopping (i.e. N1 and stop P3) were compared between the impulsive groups as well as between participants who were slow and fast in stopping.

Results: As expected, N1 and stop P3 were larger for successful relative to failed stops (i.e. N1 and stop P3 effects). Participants scoring high relative to low on impulsivity showed equal stopping performance, had larger stop P3, but similar N1 effects. Slow as compared to fast stoppers had reduced stop P3, but similar N1 effects.

Conclusions: Participants scoring high relative to low on impulsivity may need more effortful inhibitory control to yield equal stopping performance. Slow relative to fast stoppers may have weaker inhibition processes and abnormal error processing. In contrast to ADHD, both high impulsives as well as slow stoppers had an intact N1 effect.

Significance: Subjective impulsivity and slow stopping in healthy individuals cannot be generalized to ADHD.

Keywords: Stop-signal task; Impulsiveness; Inhibitory control; Attention deficit hyperactivity disorder; Frequency

1. Introduction

Impulsivity is associated with risky and spontaneous behavior, acting without thinking, and recklessness, but has also been specifically defined as the inability to inhibit inappropriate behavior or as an aversion to wait. Several measures have been developed to assess impulsivity, including questionnaires that rely on individuals’ perceptions of their own behavior. High levels of impulsivity have often been seen as dysfunctional. The construct impulsivity is also a defining characteristic of several psychiatric disorders, such as Attention Deficit Hyperactivity Disorder (ADHD; DSM-IV, American Psychiatric Association, 1994). It is an interesting question as to what the relation is between self-reported impulsivity in healthy individuals and impulsivity in psychopathology. The underlying deficits of impulsivity in ADHD patients may be an extreme form of normal subjective impulsivity. This may yield possibilities to use healthy individuals, characterized by extreme self-reported impulsivity, as model samples for ADHD.

Barkley (1997) argued that deficient inhibitory control may be the underlying deficit of impulsivity in ADHD. One important aspect of inhibitory control is the ability to stop an on-going or prepotent response. The stop-signal paradigm has often been used to study this variety of inhibitory control empirically (Logan, 1994). In the stop-signal paradigm, the primary task is typically a visual choice reaction time task. Participants have to respond as fast as possible to a specific stimulus (go-stimulus). On some trials, a stop-signal (auditory or visual) is presented and participants must stop their response to the go-signal. Inhibitory
control in the stop task, reflected in the time needed to stop the ongoing response (stop-signal reaction time; SSRT), can be estimated even though successful inhibitory control does not produce an overt response (Logan, 1994).

The stop-signal paradigm has often been used to investigate inhibitory control in psychiatric patients with presumed low impulse control (e.g. ADHD; Bekker et al., 2005c), but also in healthy individuals who are characterized by high self-reported impulsivity. This may provide clues as to whether the impulsivity in ADHD is an extreme form of self-reported impulsivity in healthy individuals. If so, we would expect differences between high and low impulsive healthy individuals similar to those between ADHD and controls. Robust differences have been found between ADHD patients and controls, especially in terms of speed of stopping, with slower stopping reaction times in ADHD, suggesting an inhibitory deficit (Lijffijt et al., 2005b; Oosterlaan et al., 1998). In high impulsive healthy individuals, as assessed by self-report measures, inconsistent findings have been reported regarding deficient stopping performance (Avila and Parcet, 2001; Lijffijt et al., 2004; Logan et al., 1997; Marsh et al., 2002; Rodriguez-Fornells et al., 2002; Vigil-Colet and Codorniu-Raga, 2004). One important factor here may concern the percentage of stop trials in the stop-signal task. Most studies that used a low probability of stop trials within their task (i.e. 25% as compared to 40–50%) have found significant correlations between impulsivity and stopping performance.

How this exactly relates to differences in SSRT is unclear. The general effect of decreasing stop probability is shortened reaction times to go-signals (Logan, 1994). Ramautar and colleagues (Ramautar et al., 2004, 2006b) showed that participants develop a stronger tendency to respond to a go-stimulus when there is a low probability of stop-signals, resulting in an impulsive response style that is characterized by faster reaction times to go-stimuli and more failed stops. In this situation, more inhibitory effort is necessary to stop the stronger tendency to respond to the go-signal. The stopping reaction time did not increase with lower probability of stop-signals. Consistently, Dimoska et al. (2006) demonstrated more failed stops, but no difference for stop-signal reaction time between fast and slow groups (median split of reaction time to go-signal). So, in a stop-signal task with a low probability of stop-signals, reaction times will be faster and the need for inhibitory control stronger, which may be especially problematic in individuals characterized by high levels of impulsivity.

Another reason for the inconsistent findings between poor stopping performance in ADHD and in healthy impulsive individuals may be the use of self-report measures to assess impulsivity in healthy participants. Self-report measures rely on participants’ own judgments which can be biased. For example, in adult ADHD it has been demonstrated that parental reports have greater validity than do self-reports (Barkley et al., 2002). Further, impulsivity is a complex and multidimensional construct and various self-report measures have been developed, each measuring one or more components of impulsivity. Accordingly, impulsivity as assessed by self-report and behavioral measures of inhibitory control may not capture similar processes (Reynolds et al., 2006). An alternative way to classify healthy individuals as either high or low impulsive is by using stopping performance as a criterion. Poor stopping behavior is the most robust finding in ADHD, especially in adult ADHD where longer stop-signal reaction times were found without differences in go reaction times (Bekker et al., 2005b; Lijffijt et al., 2005b). Moreover, stopping behavior can be more objectively assessed than subjective impulsivity. Slow stoppers, who are characterized by long stop-signal reaction times, may show similar deficits as individuals with ADHD, and subsequently may function as model samples for ADHD. It has already been demonstrated that groups divided by a median split of stopping behavior can differ in behavioral effects of medication (de Wit et al., 2000, 2002). Amphetamine shortened SSRTs in slow, but not in fast, stoppers.

The estimation of the SSRT is an indirect measure of stopping performance and depends on the assumption of independent go and stop processes, which is not always easy to verify. Event related potentials (ERPs) have been used to obtain more detailed insights into the nature of response inhibition mechanisms. In general, previous literature using the stop-signal task has focused on ERPs elicited by the stop-signal, and especially on the differences between stop-ERPs to successful and to failed stops. The most consistent finding thus far consists of a larger, centrally distributed positivity (150–250 ms after stop-signal onset) for successful, relative to failed, stops. This ‘stop P3’ has been interpreted as reflecting an inhibitory mechanism per se, but also as being related to error or conflict processing in case of failed stops (Bekker et al., 2005a; De Jong et al., 1990; Dimoska et al., 2006; Ramautar et al., 2004, 2006b; Schmajuk et al., 2006). A further recent finding, observed for auditory stop-signals among visual go-signals, is a larger negativity (‘N1’) for successful versus failed stops as early as 100 ms after stop-signal onset, thought to mainly reflect the enhanced activation of auditory cortex associated with successful stops (Bekker et al., 2005a). The N1 may reflect the trial-to-trial varying impact the stop-signal has in the auditory cortex. An enhanced N1 amplitude to successful relative to failed stops suggests that the probability of successful stopping at least partly depends on the amount of attention that is shifted to the auditory stop-signal (Bekker et al., 2005a,c; Kenemans et al., 2005). In addition to the stimulus-locked ERPs, recent studies have also examined response-locked ERP components, especially the error-related negativity (ERN or Ne) and the error positivity (Pe) (Dimoska et al., 2006; Dimoska and Johnstone, 2007; Liotti et al., 2005; Ramautar et al., 2004, 2006a; Stahl and Gibbons, 2007; van Boxtel et al., 2001). The response-locked ERN has been described as a negative deflection around 50–100 ms after an incorrect response and may reflect error detection.
or error monitoring (Falkenstein et al., 1991; Gehring and Knight, 2000; Ullsperger and von Cramon, 2006). It is
delayed by the Pe, thought to reflect the awareness of conflict
or the activation of compensatory processes after conflict
(Overbeek et al., 2005). It has been speculated that the
ERN and Pe may overlap the stop P3 elicited by failed
stops (Dimoska et al., 2006; Ramautar et al., 2004).

Previous studies have mostly found significantly reduced
stop related ERPs in children and adults with ADHD rela-
tive to controls. This especially holds for the stop P3 (Bek-
ker et al., 2005c; Liotti et al., 2005; Overtoom et al., 2002).
Regarding the N1, Bekker et al. (2005a) showed an N1
effect (i.e. greater negativity to successful than failed stops)
in healthy controls, but not in ADHD adults, indicating
decreased or even absent inhibition from the auditory on
the motor cortex. In healthy individuals, there is a link
between the impact of the stop-signal in the sensory cortex
and the probability of stopping the ongoing response,
which is absent in ADHD. With respect to the ERN and
Pe, a reduced ERN (Liotti et al., 2005) and a reduced rela-
tively late positivity (500–700 ms) in the ERP elicited by
failed stops, which may be related to the Pe (Overtoom
et al., 2002), have been found in ADHD relative to control
children.

Especially, the visibility of the N1 effect has been shown
to depend on the manner in which overlap between the
ERP to the stop-signal and the ERP to the preceding (often
a few hundreds of ms) go-stimulus is dealt with. Bekker
et al. (2005c) employed what is probably the most stringent
correction procedure (ADJAR level 2, Woldorff, 1993),
and showed that the stop N1 effect was only visible after
this correction, not without it. In the present study,
ADJAR level 2 was applied to correct for the overlap dis-
tortion. The first aim of the present study was to explore
stopping performance and the neural correlates of stopping
behavior in healthy individuals that vary on self-report
impulsivity. It was expected that individuals scoring high,
relative to individuals scoring low on impulsivity, have
longer SSRTs and smaller or even no N1 and stop P3
effects (i.e. greater ERP amplitudes for successful vs. failed
stops).

During preparation of the present paper, Dimoska and
Johnstone (2007) published a study that addressed the rela-
tion between stopping performance and the neural corre-
lates of stopping in healthy individuals, characterized by
low and high self-reported impulsivity scores. Whereas
the groups did not differ with respect to stopping perfor-
mance (i.e. SSRT), high relative to low impulsive partici-
pants showed a larger N1 and stop P3 to successful stops
as well as a larger ERN to failed stops. The enhanced N1
for high impulsive was interpreted as reflecting more
attention to the stop-signal (Dimoska and Johnstone,
2007). Given previous findings of an enhanced N1 to visual
and auditory stimuli with increased stimulus intensity in
healthy individuals scoring high on self-reported impulsiv-
ity and sensation seeking (Barrat et al., 1987; Carrillo-de-
la-Pena and Barrat, 1993), Dimoska and Johnstone
(2007) argued that the enhanced N1 for high impulsives
may reflect sensation seeking behavior. However, it should
be noted that the association between ERP augmenting
and sensation seeking behavior is far from clear (Carrillo-
de-la-Pena, 1992, 2001; Wang et al., 1999). Dimoska and
Johnstone (2007) interpreted the enhanced P3 and ERN
for high impulsive individuals as greater inhibitory activa-
tion in order to yield equal task performance, followed by
enhanced error-related processing. With regard to the
ERN, contradictory findings have also been reported: A
larger ERN to failed stops as well as to successful stops
in healthy individuals scoring low versus high on self-
reported impulsivity (Stahl and Gibbons, 2007).

The present study focused on the N1 and P3 components
to failed and successful stops. ADJAR level 2 was applied
to correct for overlap distortion. As already mentioned, self-
report measures are biased by participants’ own perceptions
and a more objective method may function better as a model
for ADHD. Therefore, the second aim of the present study
was to explore whether the neural mechanisms associated
with poor stopping performance in healthy individuals are
qualitatively similar to those observed in ADHD (i.e.
reduced N1 and P3 stop effects). To this end, within the
sample of subjects, two groups of healthy individuals, cre-
ated by applying a median split of SSRT, were compared
with respect to stop-related ERPs. It was expected that slow
relative to fast stoppers would show smaller or even no N1
and stop P3 effects. In the present study, two versions of
the stop task were used, one with a high (80%), and one with a
low (20%), stop-signal probability. It was expected that
when stop-signals are presented less frequently, participants
will develop an impulsive response style, resulting in faster
go reaction times. In addition, in the stop task with a low
probability of stop-signals more inhibitory effort is needed
to suppress the stronger tendency to respond, and
consequently the stop-related N1 and P3 effects may be
greater. Assuming that high impulsive individuals and slow
stoppers already have more problems in inhibiting their
response tendency, a stop task where stop-signals are pre-
sented less frequently may be a more sensitive test to detect
potential differences between high and low impulsives as well
as between slow and fast stoppers.

2. Methods

2.1. Participants

Participants were selected from a group of 435 psychol-
ogy students (357 females and 78 males) that filled out the
Dutch version of the I7 questionnaire (Eysenck and
Eysenck, 1978; Lijffijt et al., 2005a). Seventeen students with
a low impulsivity score (0 or 1) and 20 students with a high
impulsivity score (10–19) were tested. Three participants did
not complete the experiment and five participants were
excluded due to technical problems. Finally, 14 students with
low (3 males and 11 females) and 15 students with high
impulsivity scores (3 males and 12 females) participated in
the present study. Mean age was 20.71 (SD = 1.69) for the low and 21.53 (SD = 1.96) for the high impulsive group.

Slow and fast stoppers were created by a median split of the mean stop-signal reaction time across the high- and low-frequency stop tasks (SSRT; estimated as described by Logan, 1994). Eight out of 14 participants with low I7 scores and six out of 15 participants with high I7 scores were categorized as slow stoppers. Mean age was 21.13 (SD = 1.51) for fast stoppers (i.e. short SSRT) and 21.14 (SD = 2.21) for slow stoppers (i.e. long SSRT).

Participants had neither a current neurological or psychological disorder nor a history of one and were not on psychoactive medication. They reported to be right-handed and vision was normal or corrected-to-normal. They volunteered to participate in the study for course credit or could earn monetary compensation. All participants signed informed consent. The study was approved by the local Ethical Committee of the Faculty of Social Sciences.

2.2. Stop-signal task

The stop-signal task involves two types of trials: go trials and stop trials. Whereas go trials only contained go-signals, stop trials contained go- and stop-signals. Go-signals were square-wave, black-white vertical gratings of either a high (3.62 cycles per degree; cpd) or a low (0.46 cpd) fundamental spatial frequency (size was 7.6° × 7.6°). The go-stimuli were presented in the center of the screen one by one on a gray background. Participants were required to discriminate between the two gratings and press the correct (left or right) button. After the presentation of a fixation cross for 500 ms, a grating was presented for 750 ms. The variable time interval between the end of a grating and the start of the fixation cross was 1000–1250 ms. Stop trials consisted of a go- and a stop-signal (a 1000 Hz tone, 400 ms in duration, 83 dB intensity), generated by the computer and presented binaurally through earplugs. In stop-trials on which a grating was followed by a tone, the response to the grating had to be suppressed.

Two versions of the stop-signal task were administered. The percentage of stop-trials was 80% in the high-frequency stop task, and 20% in the low-frequency stop task. Each stop task contained 520 trials presented in 4 different blocks of 130 trials. Blocks in the high-frequency stop task consisted of 104 stop- and 26 go-trials, and blocks in the low-frequency stop task consisted of 26 stop- and 104 go-trials. Go-stop intervals were adjusted before each block according to a tracking algorithm to yield a performance of about 50% successful inhibitions (Pic) (De Jong et al., 1995; Logan et al., 1997). Before the go-stop interval for the next block was estimated, the percentage of successful stops was corrected for the amount of omissions on go-trials in the previous block (Tannock et al., 1989). To avoid strategies induced by the predictability of the timing of the stop-signal and to reduce the confounding effect of the overlap between the ERPs elicited by go- and stop-signals, the interval between the go-stimulus and the stop-signal was jittered in a range of 250 ms surrounding the calculated SOA (Pliszka et al., 2000) (i.e. 26 go–stop intervals ranging from −125 ms to 125 ms after the initial adjusted interval). Each of these intervals was used four times in the blocks of the high-frequency stop task and once in the blocks of the low-frequency stop task.

Gratulations of a high and a low fundamental spatial frequency were equally divided across stop- and go-trials. In half of the blocks, subjects had to react with the left finger to the grating of the high fundamental spatial frequency and with their right finger to the grating of the low fundamental spatial frequency. For the remaining blocks, stimulus-response mapping was reversed. All trials were pseudo-randomized within blocks. Whereas in the high-frequency stop task never more than two succeeding go-trials were presented without being followed by a stop-trial, in the low-frequency stop task never more than two succeeding stop-trials were presented without being followed by a go-trial. The order of presentation of the two stop tasks was counterbalanced between subjects.

Before the participants performed the stop tasks, two practice sessions were presented. In the first session, only go-trials (i.e. 30 gratings with narrow and 30 gratings with wide bars) were randomly presented and subjects had to press the correct button as quickly as possible. Second, before the participant performed each stop task, they practiced one block of the stop task. In these practice blocks, the go-stop interval was 250 ms.

2.3. Electrophysiological recordings

EEG and EOG activities were recorded using an Elec- trocap with 58 tin electrodes referenced to the right mastoid. The ground electrode was placed within the cap between Fpz and Fz. Vertical electrooculogram (VEOG) was recorded from electrodes attached above and below the left eye and the horizontal electrooculogram (HEOG) from the outer canthi of both eyes. Electrode impedance was kept below 5 kΩ. EEG and EOG were amplified with a Brain-Amp amplifier (Brain Products GmbH) with a bandwidth of 0.04–100 Hz. The sampling rate was 500 Hz.

2.4. Procedure

In the laboratory, participants filled out various self-report measures of impulsivity and performed three cognitive tasks in a dimly lit room: two stop-signal tasks that differed in the frequency of stop trials and an antisaccade task.

1 In the original experiment, 34 participants performed the stop-signal task. However, due to technical problems, the tracking algorithm did not always yield an average percentage of 50% successful inhibition. In the present study, only participants with more than 15% or less than 85% percentage of successful inhibition were included.

2 Relations between the self-report scales have been reported elsewhere.
The tasks were presented while EEG was recorded. The order of the stop-signal and antisaccade task was counterbalanced across participants. This article presents and discusses the behavioral and psychophysiological data from the stop task.

2.5. Data analysis

2.5.1. Performance data

Mean reaction times (MRT) to go-trials (not followed by a stop-signal) and overall choice error rate (Per) were recorded for both versions of the stop task (20% and 80% stop trials). MRT was based on correct responses between 150 ms and 1500 ms poststimulus. Overall choice error rate (in %) was calculated by dividing the number of incorrect choice responses by total number of incorrect choice responses + number of correct responses. The percentage of omissions (Pom) was calculated by dividing the number of omissions by the number of go-trials. Furthermore, the corrected percentage of inhibition (Pic) was calculated according to the procedure of Logan (1994) and after inspection of the grand-average waveforms and difference waves (Figs. 3, 4, and 7), we decided to analyze the stop P3 by calculating the mean amplitude for the 140–350 ms time window. The average number of trials used for deriving ERPs ranged from 19 to 67 and from 27 to 290 for the low- and high-frequency stop task, respectively.

Average response-locked ERPs (r-ERPs) were calculated, separately for failed stops and correct go-trials (only for responses faster than 750 ms), to investigate whether error-related processes contribute to the potential group differences in the stop P3 effect. It should be noted that ADJAR level 2 could not be applied to response-locked ERPs. The ERN was defined as the largest negative peak in the 20–120 ms time window after response onset (peak latency locked at FCz). The Pe was defined as the largest positive peak in the 175–450 ms time window after response onset (peak latency locked at Cz).

2.6. Statistical analyses

Fisher’s exact test was conducted to examine the association between I7 impulsivity and SSRT. Separate repeated measures analyses of variance (ANOVAs) were conducted for MRT, SSRT, Pic, Per, Pom, and SOA with probability (20% vs. 80% stop-trials) as within-subjects factor and impulsivity (high vs. low impulsivity) as between-subjects factor. Similar ANOVAs were performed for SSRT (fast vs. slow stoppers) as between-subjects factor.

Regarding the ERP components, repeated-measures ANOVAs were conducted separately for the N1 and P3, including three within-factors, i.e. frequency (low vs. high-frequency of stop-signals), stop (failed vs. successful stops), and lead (FCz vs. Cz) and one between-factor group (high vs. low impulsivity). Similar ANOVAs were conducted for the stop-related ERPs with SSRT as between-subjects factor (fast vs. slow stoppers).

Separate repeated measures ANOVAs were conducted for ERN and Pe including lead (FCz and Cz for ERN; FCz, Cz, and Pz for Pe), frequency (high vs. low), and trial (failed stops vs. correct go-trial) as within-subjects factors and group (high vs. low impulsivity) as between-subjects factor. Similar ANOVAs were performed with SSRT (or slow vs. fast stoppers) as between-subjects factor. Only group main effects or interactions with group effects are reported.

3. Results

3.1. Behavioral data

Fisher’s exact test revealed no significant association between I7 impulsivity and mean stop-signal reaction time (SSRT) across the high- and low-frequency stop task (p = 0.466). Hence, the complete group of 29 participants could be used to create a slow and fast stop group by applying a median split of SSRT.
Performance data with regard to differences between the two impulsive groups have been described elsewhere (Lansbergen et al., 2007) and will only be briefly reported here. Table 1 presents performance data for the high- and the low-frequency stop task, for slow and fast stoppers. Figs. 1 and 2 illustrate mean reaction time (MRT) and mean SSRT for the high-and low-frequency stop task, separately for individuals with high and low I7 impulsivity scores (left panels) and for slow and fast stoppers (right panels). In the low-frequency stop task, MRTs were faster ($F(1,27) = 39.53, p < .001$), standard deviations of the reaction time (SDRT) were smaller ($F(1,27) = 31.03, p < .001$), go-stop intervals (SOA) were shorter ($F(1,27) = 34.91, p < .001$), and percentage of omissions (Pom) were smaller ($F(1,27) = 16.23, p < .001$), relative to the high-frequency stop task. Although the tracking algorithm was used to yield a performance of about 50% successful inhibitions, the percentage of corrected inhibition (Pic) was smaller when stop-signals were presented less frequently ($F(1,27) = 18.70, p < .001$). SSRT and choice-reaction time errors did not differ between the high- and low-frequency tasks.

No differences were found between participants with high and low self-reported impulsivity for the parameters. Whereas slow stoppers had longer SSRTs ($F(1,27) = 27.86, p < .001$), greater SDRTs ($F(1,27) = 9.92, p = .004$), and made more choice-reaction time errors than fast stoppers ($F(1,27) = 10.85, p = .003$), no group differences were found for SOA, Pom, Pic, and choice reaction time. Further, no significant interaction between frequency and group was found.

3.2. Electrophysiological data

In the reported ERP results, the terms N1 and stop P3 refer to the negativity around 100 ms and the positivity around 200–300 ms after the presentation of the stop-signal, respectively. The greater N1 elicited by successful relative to failed stops will be labeled as the ‘N1 effect’. The enhanced stop P3 to successful as compared to failed stops will be labeled as the ‘stop P3 effect’.

### Table 1

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<tr>
<th></th>
<th>Fast stoppers</th>
<th>Slow stoppers</th>
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<tr>
<td></td>
<td>Low-frequency</td>
<td>High-frequency</td>
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<tr>
<td></td>
<td>task</td>
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<tr>
<td>MRT (in ms)</td>
<td>Mean</td>
<td>SD</td>
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<tr>
<td></td>
<td>388.3</td>
<td>64.0</td>
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<tr>
<td>SSRT (in ms)</td>
<td>137.8</td>
<td>17.4</td>
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<tr>
<td>SDRT</td>
<td>87.6</td>
<td>18.5</td>
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<tr>
<td>SOA</td>
<td>238.8</td>
<td>59.4</td>
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<tr>
<td>Pom</td>
<td>1.8</td>
<td>1.7</td>
</tr>
<tr>
<td>Pic</td>
<td>48.5</td>
<td>6.4</td>
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<tr>
<td>Per</td>
<td>2.7</td>
<td>1.9</td>
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**Note:** MRT = mean reaction time to go-stimulus; SSRT = stop-signal reaction time; SDRT = standard deviation of reaction times to go-stimulus; SOA = delay between go-stimulus and stop-stimulus Pom = percentage of omissions; Pic = percentage of successful inhibitions, corrected for estimated number of omissions on stop-trials; Per = percentage of choice errors.
3.2.1.2. Stop P3. Figs. 3 and 4 illustrate the enhanced stop-related P3 for successful relative to failed stops (i.e. stop P3 effect). Statistical analyses revealed a trend for a stop \times group effect \( F(1,27) = 3.08, p = .090 \), indicating a larger stop P3 effect for high than for low impulsives \( F(1,27) = 39.39, p < .001; F(1,27) = 13.12, p = .001 \), respectively; see Fig. 4, left panel). Group differences for successful stops, \( F(1,27) = 3.64, p = .067 \), rather than for failed stops, \( F < 1 \), might have contributed to the larger stop P3 effect for high as compared to low impulsive individuals. Further, a significant main effect of frequency indicated a larger frontal P3 for the low- relative to the high-frequency stop task \( F(1,27) = 49.52, p < .001 \).

3.2.1.3. Error-related ERP components. Fig. 5 presents the grand average response-locked ERPs at FCz, Cz, and Pz for failed stops and correct go-trials for high and low impulsive participants, separately for the low- and high-frequency stop task. As illustrated in Fig. 5, the impulsive groups did not differ significantly with respect to the ERN or Pe amplitude (see also the difference waves in Fig. 6, left panel).

3.2.2. Slow versus fast stoppers

Fig. 7 presents grand average ERPs elicited by stop-signal at FCz, Cz, and Pz for successful and failed stops for fast and slow stoppers, separately for the low- and high-frequency stop task. Mean amplitudes and standard deviations are summarized in Table 3. Effects of frequency and lead were qualitatively the same as those in previous analyses that included impulsivity as between-subjects factor (i.e. greater amplitudes in the low- vs. high-frequency task and greater amplitudes for successful vs. failed stops).

3.2.2.1. N1. With regard to the N1, a frequency \times lead \times group effect \( F(1.27) = 6.97, p = .014 \) was found. Post-hoc analyses per lead indicated a significantly larger N1...
in the low- than in the high-frequency stop task for fast, but not for slow stoppers at FCz and Cz (FCz: frequency \times group: F(1,27) = 5.31, p = .029; Cz: frequency \times group: F(1,27) = 8.43, p = .007). Furthermore, a marginally significant stop \times lead \times group effect (F(1,27) = 4.20, p = .050) was decomposed using post-hoc analyses for each group, revealing a stop \times lead effect for fast (F(1,27) = 16.20, p < .001), but not for slow, stoppers; this reflected an N1 effect for slow stoppers at both leads, but for fast stoppers only at Cz and not at FCz.

3.2.2.2. Stop P3. Regarding the stop P3, both fast and slow stoppers showed a stop P3 effect, but it was larger for fast than for slow stoppers (see Fig. 4 (right panel) and Fig. 7; stop \times group: F(1,27) = 8.70, p = .007; F(1,27) = 57.11, p < .001, and F(1,27) = 10.24, p = .003 for fast and slow stoppers, respectively). Post-hoc analyses indicated that this enhanced stop P3 for fast relative to slow stoppers was significant for successful (F(1,27) = 26.24, p < .001) as well as for failed stops (F(1,27) = 8.12, p = .008).

3.2.2.3. Error-related ERP components. Fig. 8 presents the grand average response-locked ERPs at FCz, Cz, and Pz for failed stops and correct go-trials for slow and fast stoppers, separately for the low- and high-frequency stop task. Statistical analyses for slow versus fast stoppers yielded no

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Table 2
Means and standard deviations of the mean amplitudes of the N1 (80–120 ms), and stop P3 (120–160 ms and 140–350 ms) at Cz, elicited by successful and failed stops, in the high- and low-frequency stop-signal task, separately for participants scoring high and low on impulsivity

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<th>Low I7</th>
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<td></td>
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<td>High-frequency stop task</td>
<td>Low-frequency stop task</td>
<td>High-frequency stop task</td>
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<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
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<td>6.0</td>
<td>-8.6</td>
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</tr>
<tr>
<td>N1-FS (80–120 ms)</td>
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<td>6.2</td>
<td>-7.7</td>
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<tr>
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<td>6.3</td>
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<td>5.0</td>
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</table>

Note: SS = successful stops; FS = failed stops.

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Fig. 4. Difference waves (successful minus failed stops) for the high- (80% stop trials) and low- (20% stop trials) frequency stop task at FCz, Cz, and Pz, for the impulsive (left panel) and SSRT groups (right panel).
Fig. 5. Grand-average ERPs for participants scoring high and low on I7 impulsivity, elicited by failed stops and correct go-trials at FCz, Cz, and Pz, separately for the high- (80% stop trials) and low- (20% stop trials) frequency stop task.

Fig. 6. Difference waves (failed stops minus correct go-trials) for the high- (80% stop trials) and low- (20% stop trials) frequency stop task at FCz, Cz, and Pz, for the impulsive (left panel) and SSRT groups (right panel).
group or interaction with group effects for ERN. Regarding the Pe amplitude, a significant frequency × trial × group was found ($F(1,27) = 10.36, p = .003$). Post-hoc analyses revealed a significant trial × group effect for the low ($F(1,27) = 8.54, p = .007$) but not the high frequency stop task version, indicating that the enhanced Pe amplitude for failed stops versus correct go-trials was greater for fast than for slow stoppers. ($F(1,27) = 81.24, p < .001$ and $F(1,27) = 21.56, p < .001$ for fast and slow stoppers, respectively).

3.2.3. Post-hoc analyses in 120–160 ms time window

Differences in stop P3 effects were accompanied by SSRT differences for slow versus fast stoppers, but not for high versus low impulsives. This combined pattern of results makes a unitary interpretation of the stop P3 effect as a manifestation of an inhibitory control process unattractive. Based on visual inspection (Figs. 3, 4, and 7), we formulated the hypothesis that the early phase of the stop P3 effect was different for slow versus fast, but not for high versus low impulsives. Such a result would be consistent with the notion that this early phase reflects inhibition, whereas the later phase would reflect more strategic processes like error monitoring. For the 120–160 ms time window (the early phase of the stop P3), we found no significant group × stop interaction for high versus low impulsives. For slow versus fast stoppers, a significant frequency × stop × group effect ($F(1,27) = 7.57, p < .010$)

![Fig. 7. Grand-average ERPs for slow and fast stoppers, elicited by successful and failed stops at FCz, Cz, and Pz, separately for the high- (80% stop trials) and low- (20% stop trials) frequency stop task.](image)

<table>
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<td>3</td>
<td>8.5</td>
<td>−1.7</td>
</tr>
</tbody>
</table>

Note: SS = successful stops; FS = failed stops.
reflected a significant stop × group effect for the low, but not the high-frequency task version ($F(1,27) = 11.22$, $p = .002$). As illustrated in Fig. 4 (right panel), the stop P3 effect in the low-frequency task was significant only for fast and not for slow stoppers ($F(1,27) = 16.20$, $p < .001$; $F < 1$, respectively).

4. Discussion

The present study examined the relation between self-report measures of impulsivity and stopping performance as well as the underlying neural mechanisms. Stopping performance was assessed using two stop-signal tasks that differed in the probability of stop-signals. Consistent with previous findings (Ramautar et al., 2004), participants in the present study developed an impulsive response style in the stop-signal task with a low probability of stop-signals, with faster reaction times to go-trials and a higher percentage of failed stops ($F(1,27) = 16.20$, $p < .001$; $F < 1$, respectively).

No differences were found in behavioral performance between individuals scoring low and scoring high on the I7 impulsiveness subscale, not even in the low-frequency stop task where an impulsive response style might have affected stopping performance more in participants scoring high on impulsivity. Together with the inconsistent findings of previous behavioral studies (e.g. Lijffijt et al., 2004; Logan et al., 1997), it seems that there is no clear relation between impulsivity, as assessed by self-report measures, and stopping performance in the stop-signal task. Consistently, previous studies did not provide evidence for an association between self-reported impulsivity in healthy individuals and other related forms of inhibitory control, such as stopping a response in a continuous performance task (Fallgatter and Herrmann, 2001; Horn et al., 2003) or interference control as quantified in the Stroop test (Avila and Parcet, 2001; Cheung et al., 2004; Kirkeby and Robinson, 2005; Visser et al., 1996).

With respect to event-related potentials, as expected, successful stops were associated with greater negativity around 80–120 ms and subsequently greater positivity beginning around 120 ms after the onset of the stop-signal, relative to failed stops (i.e. N1 and stop P3 effect, respectively). As discussed in the Introduction, the N1 effect may reflect a functional link between auditory-cortex activation and motor inhibition, and the P3 effect may be thought to reflect an inhibitory mechanism and/or error processing. Given the absence of behavioral differences between participants scoring high and low on self-reported impulsivity, the finding of similar N1 effects is not...
surprising. In contrast, Dimoska and Johnstone (2007) reported a larger N1 elicited by successful stops for individuals scoring high relative to low on the I7 impulsiveness subscale. They interpreted this as more attention to the stop-signal due to more sensation seeking in high versus low impulsives. Inconsistency between the findings of the present study and Dimoska and Johnstone (2007) may be explained by the difference in intensity of the stop tone (60 dB vs. 83 dB in Dimoska and in our study, respectively).

A positive relation between N1 amplitude and intensity of a stimulus (i.e., augmenting dimension of sensory performance) in individuals characterized by high self-reported impulsivity or more sensation seeking behavior has been explained as reflecting searching for an optimal level of stimulation for cognitive processes (Carrillo-de-la-Pena and Barrat, 1993). In this perspective, a stop tone of 83 dB sound pressure level may already be an optimal level of stimulation in contrast to a tone of 60 dB. Note that the ADHD study of Bekker et al. (2005c) used a stop tone of 80 dB.

Whereas the enhanced stop P3 for successful relative to failed stops was present in both groups, it was attenuated in participants scoring low, relative to high on self-reported impulsivity. Post-hoc analyses for each stop-trial indicated that probably the enhanced stop P3 elicited by successful in high relative to low impulsives, but not the stop P3 for failed stops, contributed to this effect, confirming the results of Dimoska and Johnstone (2007). In individuals with ADHD, it has consistently been found that the stop P3 effect is substantially reduced compared to controls (see Introduction). The reduced stop P3 effect in ADHD has always been reported in combination with deficient stopping performance (i.e., longer SSRTs). In the present study, stopping performance did not differ between individuals scoring low and high on self-reported impulsivity. To the extent that the stop P3 effect reflects the activation of an inhibition system, these results suggest that participants scoring high on self-reported impulsivity needed more inhibitory effort for equal stopping performance as compared to those scoring low on self-reported impulsivity (see also Dimoska and Johnstone, 2007). However, as discussed in the Introduction, the stop P3 effect may reflect both inhibition as well as error-related processing (Dimoska et al., 2006; Ramautar et al., 2004). As shown in Figs. 3 and 7, the ERN and Pe may overlap the stop P3 elicited by failed stops.

It can be speculated that the earlier phase of the stop P3 effect (before 200 ms-latency) reflects inhibition, and the later phase error-related processing. Accordingly, similar stopping performance in high and low impulsives was accompanied by differences in the later error-related phase, but not in the earlier inhibition phase. However, the impulsive groups did not differ with respect to the ERN and Pe amplitudes. For the time being, it is difficult to decide between these two alternatives, and we conclude that inhibition, rather than error processing, is different between individuals with high and low self-reported impulsivity (although in a manner opposite to what was expected). In the stop-signal task, participants scoring high on self-reported impulsivity may need more inhibitory effort for equal stopping performance.

The second aim of the present study concerned the neural basis of individual differences in stopping performance. There was no association between the efficiency of stopping (as indexed by stopping reaction time) and I7 impulsivity, justifying the division of the same sample into slow and fast stoppers. Slow stoppers also displayed more variable reaction times to go-signals and larger percentages of choice reaction time errors at the behavioral level. These results suggest lapses in attention (Castellanos and Tannock, 2002). However, larger percentage of choice reaction times errors in slow stoppers may also indicate a pattern of impulsive responding (Bekker et al., 2005b). In terms of the speed-accuracy trade-off, a larger percentage of choice reaction time errors without an increase in go RT may reflect favoring speed over accuracy, indicating a fast and impulsive response style. The dissociation between SSRT and go RT (no difference between slow and fast stoppers) is consistent with a recent report by Dimoska et al. (2006), who found no differences between fast and slow groups (median split of reaction time to go-signal) for stop-signal reaction time.

Contrary to our expectation, no differences were found between slow and fast stoppers with respect to the enhanced N1 for successful relative to failed stops. Consistent with our prediction, the increased stop P3 for successful relative to failed stops was attenuated in slow stoppers for the earlier (120–160 ms) as well as for the later phase (140–350 ms). These results suggest that in healthy individuals, impaired stopping performance is associated with weaker activation of the inhibition system (earlier phase) as well as less error processing (later phase), but the link between the amount of attention paid to the stop-signal and the probability of successful stopping, as reflected in the N1 effect, is not impaired.

Further, the contrast between fast and slow stoppers with regard to the stop P3 effect concerned a decreased stop P3 to successful as well as to failed ones. Assuming that the stop P3 elicited by successful stops reflects inhibitory control and the stop P3 elicited by failed stops reflects error-related processes (Dimoska et al., 2006; Ramautar et al., 2004), it may be concluded that differences between fast and slow stoppers refer to both inhibition and error-related processing. Comparing the error-related ERPs between the groups, no significant association between stopping performance and ERN, but a smaller Pe was found for slow stoppers relative to fast stoppers. In conclusion, present findings suggest that slow stoppers show deficient inhibitory control (as reflected in the stop P3 to successful stops), ‘normal’ error monitoring (ERN), but are less aware of conflict or engage less attentional control after conflict (Pe; stop P3 to failed stops). This pattern of ERP results only partly resembles the abnormal brain correlates of stopping performance in ADHD.

Note that the slow stoppers had similar stop P3 effects as the fast stoppers in the early time window (i.e.
120–160 ms) in the high-, but not in the low-frequency task (see Fig. 4; right panel). However, as illustrated in Fig. 4 (right panel), after approximately 180 ms the stop P3 effect was smaller in slow as compared to fast stoppers in both frequency versions.

The global frequency of stop-signals had an effect on processing the stop-signal in general. The N1 and stop P3 for both successful and failed stops were larger in the low than in the high-frequency stop task. The N1 is known to be sensitive to the global temporal probability, rareness of the evoking stimulus (Davis et al., 1966), or the intensity of the stimulus. Rare auditory stimuli and relatively intense auditory stimuli may evoke large N1s, which may reflect a larger impact of these stimuli in auditory cortex, or stated differently, more attention to these stimuli; the N1 can also be modulated by voluntary attention (Woldorff et al., 1993). Furthermore, the N1 is larger when participants are engaged in a task rather than relaxing (Näätänen, 1992). The enhanced N1 in the low- versus high-frequency task was much more pronounced and significant only for fast as compared to slow stoppers, suggesting an increase in sensory sensitivity to the auditory stop-signal in the low as compared to the high-frequency task, for fast, but not for slow, stoppers. However, it is not completely straightforward, as no interaction effects of group and frequency were found on task performance. A similar line of reasoning may apply to the larger stop P3 to low-frequency stop-signals observed specifically in fast stoppers. This frequency effect on the stop P3 may be viewed as a sequel of the N1 interaction, at the level of an inhibitory mechanism. However, the enhanced P3 to rare stop-signals may also reflect aspects of both the classical, frontal-centrally distributed P3a, related to orienting (Courchesne et al., 1975), as well as the classical, central-parietally distributed P3b, which responds especially to oddball stimuli (Donchin, 1981).

In summary, prototypical stopping deficits as repeatedly reported for ADHD could not be confirmed for questionnaire-based high impulsivity in healthy participants. Even stronger, at the level of neurophysiological correlates, healthy highly impulsive individuals exhibit a pattern opposite to that usually observed for ADHD. It may be speculated that individuals with relatively high self-reported impulsivity need more inhibitory effort to attain normal stopping levels, rather than having a stopping deficit. Our findings suggest that the underlying mechanisms of low impulse control in pathological samples (as in ADHD) are qualitatively different from that in healthy, highly impulsive individuals. Therefore, self-reported impulsivity within the normal population may not function as a model for impulsivity in psychopathology. So, conclusions about, for example, the genetics, the psychopharmacology, and the neurophysiological basis of inhibition that are based on comparing either high versus low subjective impulsive or slow versus fast stoppers cannot be generalized to ADHD, or vice versa.

With respect to stopping performance, stopping performance in ADHD may be an extreme form, but also qualitatively different from healthy individuals. Whereas the neural mechanism underlying the implementation of inhibitory control and/or error processing in ADHD patients may partly be the same as that in healthy volunteers, characterized by poor stopping performance, individuals with ADHD have an additional deficit, that is, the lack of a link between the amount of attention paid to the stop-signal and the outcome of the inhibition process. It is speculated that this discrepancy may be due to the additional attention deficits in ADHD patients, which may not be present in the slow stoppers in the present study.

Concluding, although we did not directly compare healthy individuals with ADHD patients, the findings of the present study in combination with a previous ADHD study (Bekker et al., 2005c) suggest at least three groups that can be differentiated according to stopping behavior in the stop-signal task and the neural correlates of stopping. The first group involves healthy participants scoring high relative to low on subjective self-report impulsivity. They had no deficiency in stopping behavior (similar speed of stopping), showed a normal relation between the amount of attention switched to the stop-signal and subsequent stopping performance (similar N1 effect), showed normal error processing (similar ERN and P3e), but may be less efficient in implementing the inhibition process (increased stop P3 effect). A second group of healthy individuals was characterized by slow relative to fast stop reaction times. They had a normal link between the amount of attention switched to the stop-signal and the probability of successful stopping (similar N1 effect), but may have weaker inhibition processes (reduced stop P3 effect) and may be less aware of conflict and/or engage less attentional control after conflict (reduced Pe). Third, Bekker et al. (2005a) reported on adults with ADHD (ADHD combined subtype), who were slow in stopping, showed a compromised link between attentional switching to the stop-signal and subsequent stopping (no N1 effect) as well as weaker inhibition processes (reduced stop P3 effect). Further research including an ADHD group, a healthy highly impulsive group, and a group with relatively slow stoppers is warranted to determine the exact differences between these groups with respect to stopping performance and the neural correlates of stopping.

We conclude that impulsivity in ADHD is qualitatively different from impulsive behavior within the normal population, as assessed by self-report measures. Healthy, but relatively deficient, stoppers only partly show the same abnormal neurophysiological correlates of stopping that characterize ADHD. Consequently, the neural mechanisms underlying impulsivity in ADHD should not be investigated in healthy highly impulsive volunteers or in healthy volunteers, characterized by poor stopping, and then extrapolated to an ADHD population.
Acknowledgements

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