Research Report

Subjective impulsivity and baseline EEG in relation to stopping performance

Marieke M. Lansbergen*, Dennis J.L.G. Schutter, J. Leon Kenemans

Departments of Experimental Psychology and Psychopharmacology, Utrecht University, Heidelberglaan 2, 3584 CS Utrecht, The Netherlands

ARTICLE INFO

Article history:
Accepted 14 February 2007
Available online 24 February 2007

Keywords:
Impulsiveness
Stop-signal paradigm
Stop-signal reaction time
Electroencephalogram
Theta/beta ratio
Attention deficit hyperactivity disorder

ABSTRACT

Impulsivity is a personality trait within the normal population, but also a feature of many psychiatric disorders that have been associated with poor inhibitory control. The aim of the present study was to examine the relation between subjective impulsivity, theta/beta EEG ratio, and inhibitory control in healthy individuals. In 15 high and 14 low impulsive healthy volunteers (as assessed by the I7 questionnaire), resting state EEG was recorded during an eyes open condition to obtain estimates for theta and beta activity. Subsequently, a stop-signal task was presented where participants responded to go-signals and had to stop their initiated response to stop-signals. Stopping performance and EEG activity were compared between the impulsive groups as well as between high vs. low theta/beta ratio groups. Results showed that subjective impulsivity was not related to stopping behavior or to theta/beta ratio. In contrast to our expectations that individuals with high theta/beta ratios would show relatively long stopping reaction times, analyses revealed that the low theta/beta ratio group had longer stopping reaction times. Given that increased theta/beta ratio may reflect reduced cortical inhibition over subcortical drives, it is proposed that healthy individuals with relative high theta/beta ratios are more motivated to maximize inhibition-related performance.

1. Introduction

Impulsivity is an important psychological trait in personality theories and implicated in psychiatric disorders. There is a lack of a precise definition of impulsivity and it has been suggested that impulsivity is multidimensional (Evenden, 1999). Impulsivity has been associated with various behaviors and characteristics, including impatience, restlessness, reward seeking, acting without thinking, and an inability to wait. Self-report measures have been developed to assess impulsivity, based on specific definitions, such as “a complete lack of looking ahead at the consequences of their actions” (Eysenck et al., 1985) and “the tendency to deliberate less than most people of equal ability before taking action” (Dickman, 1990).

In general, impulsive behavior is mostly viewed as depending on inhibitory control, that is, the ability to suppress undesirable response tendencies. Self-report measures, behavioral observations, cognitive tasks, and neurobiological measures have been used to study the construct impulsivity. In this paper, we distinguish three measures related to...
impulsivity. First, subjective impulsivity as assessed by self-report measures. Second, behavioral impulsivity, or impaired inhibitory control, that can be quantified objectively by cognitive tasks in which individuals must suppress the tendency to respond. Third, less cortical brain activity during rest as a possible biological marker of impulsivity. This baseline cortical brain activity can be assessed by recording electroencephalogram (EEG) during rest. Although the level of attention and state of alertness influence EEG, several studies have demonstrated that the human EEG is stable over time (e.g., Chi et al., 2005; Corsi-Cabrera et al., 2007; Gasser et al., 1985; Kendrac and Szabo, 1999; Salinsky et al., 1991; Williams et al., 2005).

Whereas impulsive behavior in healthy individuals has mostly been assessed by self report questionnaires, impulsive behavior in psychopathology (e.g., in attention deficit hyperactivity disorder; ADHD) is usually defined according to the DSM-IV (American Psychiatric Association, 1994). An interesting point is whether impulsivity in psychopathology is qualitatively different from impulsivity within the normal population, or whether it is an extreme variety within the continuous distribution of impulsive behavior. It has been argued that impulsive behavior in psychopathology, particularly in ADHD, may be attributed to deficient inhibitory control (Barkley, 1997). Moreover, individuals with ADHD have been characterized by a deviant pattern of baseline cortical activity, specifically increased slow-wave activity, primarily in the theta band, as well as decreased fast-wave activity, particularly in the beta band, often coupled together (i.e., an increased theta/beta ratio) (Barry et al., 2003). Assuming that impulsivity in psychopathology lies at the extreme end of a normal distribution, healthy individuals scoring extremely high on impulsivity, as assessed by self-report measures, may also show deficient inhibitory control and increased theta, decreased beta, and/or increased theta/beta ratio.

Inhibitory control can be assessed by several behavioral tests, such as the stop-signal task, which assesses the ability to stop an ongoing response pattern, with stop-signal reaction times (SSRT) reflecting inhibitory control. With respect to stopping performance in more vs. less impulsive healthy individuals, previous studies have reported inconsistent results. Whereas three studies reported impaired stopping in relatively high impulsive individuals (Avila and Parcet, 2001; Logan et al., 1997; Marsh et al., 2002), three other studies did not find a relation between impulsivity and stopping behavior (Lijffijt et al., 2004; Rodriguez-Fornells et al., 2002; Vigil-Colet and Codorniu-Raga, 2004). One reason for these contradictory findings may be the difference in the probability of stop-signals used. Studies that did find significant correlations between impulsivity and stopping performance used stop-signal tasks with a percentage of 25% stop-trials, whereas the null-result studies used stop-signal tasks with a relatively high percentage of stop-signals (40% and 50%). It has been demonstrated that in a version of the stop-signal task where there is a low probability of stop-signals, participants develop an impulsive response style, characterized by faster reaction times to go stimuli and more unsuccessful stops (Ramaurban et al., 2004, 2006). Ramaurban et al. (2004, 2006) suggested that in this situation more inhibitory control may be needed to overcome the stronger response tendency to the go-stimulus. This process may be more difficult for individuals with low inhibitory control. Consequently, differences in stopping performance between high and low impulsive individuals may be more evident in a stop task where stop-signals are presented less frequently. Therefore, in the present study, two versions of the stop-signal task were used, with a high probability (80%) and a low probability (20%) of stop-signals, respectively.

Resting state EEG studies suggest that deviant patterns of increased slow- and decreased fast-wave activity (i.e., increased theta, decreased beta, and/or increased theta/beta ratios) may be a biological marker of impulse control disorders, particularly ADHD. It has been suggested that this deviant pattern of brain activity is linked to cortical underarousal (Barry et al., 2003). Alternatively, given the sensitivity of theta/beta ratio to discriminate the occurrence of ADHD, together with the finding that theta/beta ratio was not related to the level of autonomic arousal (as assessed by skin conductance level), Barry et al. (2004) speculated that theta/beta ratio may represent the functionality of the cortical substrates of attentional processing rather than cortical arousal. In concordance, it has also been proposed that the balance between fast- and slow-wave frequencies is a biological correlate of corticosubcortical interactions (Schutter et al., 2006). From this perspective, impulsive behavior may be a consequence of reduced cortical control and increased subcortical drives.

To our knowledge, healthy individuals selected on high and low impulsivity, have never been compared in terms of theta, beta, or the ratio between theta and beta activity. The aim of the present study was to examine whether healthy individuals who score high on subjective impulsivity, as assessed by Eysenck’s I7 questionnaire (Eysenck and Eysenck, 1978; Lijffijt et al., 2005a) present deficiencies in inhibitory control and increased slow- and/or decreased fast-wave activity. Assuming that impulsive behavior in psychopathology is an extreme variety within the continuous distribution of impulsive behavior, it was hypothesized that individuals scoring high on the impulsiveness scale would show impaired inhibitory control comparable to psychopathological individuals (i.e., poor stopping performance). Furthermore, increased theta and decreased beta activity, as well as relatively high theta/beta ratios were expected in healthy individuals scoring extremely high as compared to low on subjective impulsivity.

2. Results

Table 1 presents performance data for the high- and the low-frequency stop task, for participants with low and high I7 impulsivity scores. Figs. 1 (left panel) and 2 (left panel) illustrate mean reaction time on go-stimuli (MRT) and mean stop-signal reaction time (SSRT) for the high- and low-frequency stop task, separately for individuals, scoring high and low on impulsivity. Whereas MRT on go-trials was significantly faster in the low-frequency stop task, where there is a 20% probability of stop-stimuli ($F(1,27)=39.53$, $p<0.001$), SSRT did not differ between the two stop task versions. Furthermore, in the low-frequency stop task, standard deviations of the reaction time (SDRT) were smaller
Table 1 – Mean values and standard deviation of the performance data obtained from the high and low frequency stop-signal task, separately for high and low impulsive participants

<table>
<thead>
<tr>
<th></th>
<th>High I7</th>
<th>Low I7</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Low-frequency</td>
<td>High-frequency</td>
</tr>
<tr>
<td></td>
<td>stop task</td>
<td>stop task</td>
</tr>
<tr>
<td>MRT (in ms)</td>
<td>389.6 (63.5)</td>
<td>517.9 (101.4)</td>
</tr>
<tr>
<td>SSRT (in ms)</td>
<td>161.5 (37.8)</td>
<td>154.2 (30.0)</td>
</tr>
<tr>
<td>SDRT</td>
<td>95.4 (27.7)</td>
<td>132.2 (33.6)</td>
</tr>
<tr>
<td>SOA</td>
<td>221.5 (58.8)</td>
<td>306.7 (74.5)</td>
</tr>
<tr>
<td>Pom</td>
<td>1.7 (1.7)</td>
<td>5.4 (4.3)</td>
</tr>
<tr>
<td>Pic</td>
<td>44.2 (8.9)</td>
<td>60.5 (15.7)</td>
</tr>
<tr>
<td>Per</td>
<td>3.8 (3.2)</td>
<td>3.6 (4.5)</td>
</tr>
</tbody>
</table>

Note. Standard deviations are given in parentheses; 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.

(F(1,27)=31.03, p<0.001), go–stop intervals (SOA) were shorter (F(1,27)=34.91, p<0.001), and percentages of omissions (i.e., a failure to respond to go-stimulus; Pom) and corrected inhibition (i.e., stop the ongoing response to a go-stimulus after a stop-signal; Pic) were smaller (F(1,27)=16.23, p<0.001 and F(1,27)=18.70, p<0.001, respectively), relative to the high-frequency stop task. No significant interaction effects with group or main group effects were found for these parameters. Choice-reaction time errors did not differ between the high- and low-frequency tasks or between participants with low and high I7 impulsivity scores.

To examine the relation between subjective impulsivity and stopping performance more extensively, additional repeated measures ANOVAs for MRT and SSRT were conducted to test the difference in stopping behavior between groups that were created by median splits of the total impulsivity score of the BIS-11 and the scores of the BIS-11 subscales (i.e., separate ANOVAs for non-planning, cognitive, motor, and total score on the BIS-11) and the BIS/BAS (i.e., separate ANOVAs for BIS and the total BAS score). Only the groups that were created by a median split of the motor subscale of the BIS-11 differed with regard to SSRT, with slower SSRTs observed in individuals scoring low as compared to high on BIS-11 motor impulsivity (F(1,26)=4.37, p=0.046).

Regarding EEG variables, no significant interaction with group or main group effects was found for theta and beta power, or theta/beta ratio. For participants with high I7 scores, mean theta and beta activity, and theta/beta ratio were 0.26 (SD=0.13), 0.049 (SD=0.02), and 5.91 (SD=2.38), respectively. For participants with low I7 scores, mean theta and beta activity, and theta/beta ratio were 0.23 (SD=0.09), 0.043 (SD=0.02), and 5.91 (SD=1.95), respectively.

Given the consistent findings of increased theta/beta ratios in ADHD (Barry et al., 2003), together with the findings that this EEG index can discriminate ADHD patients from controls with high accuracy (Monastra et al., 1999), post hoc analyses were conducted to compare performance on the stop-signal task in participants with relatively high to that of those with low theta/beta ratios. It should, however, be noted that the division of the present sample into two groups based on theta/beta ratios is only justified when I7 impulsivity scores and EEG ratios are independent and follow normal distributions. Statistical analyses revealed no significant correlation or association between I7 impulsivity scores and theta/beta ratios (rho(29)=-0.035, p=0.856; Fisher's exact test: p=0.715). Furthermore, theta/beta ratio followed a normal distribution (Kolmogrov–Smirnov Z-test: p=0.712).

Previous studies have shown that the highest degree of differentiation between individuals with ADHD and healthy controls using theta/beta EEG ratio is located over the vertex (Lubar, 1995; Lubar et al., 1995). For this reason, in the classification study of Monastra et al. (1999), theta/beta power ratios were obtained at Cz in the eyes open condition. In the present study, the highest theta/beta power ratio with maximum variance was found at Cz in the eyes open condition. To further investigate the difference between individuals with low and high theta/beta ratios regarding...
stopping performance, a median split of the theta/beta ratio at Cz in the eyes open condition was applied to create a high and a low theta/beta ratio group. Separate repeated measures ANOVA were conducted for MRT, SSRT, Pic, Per, Pom, and SOA with probability (20% vs. 80% stop-trials) as within-subjects factors and theta/beta ratio (high vs. low ratios) as between-subjects factor. In addition, separate repeated measures ANOVAs were performed for theta and beta power with hemisphere (left, midline, right) and area (frontal, central, parietal) as within-subjects factors and theta/beta ratio (high vs. low) as between-subjects factor to determine whether the theta/beta groups differed in theta, beta, or both power bands.

Table 2 presents performance data for the high- and the low-frequency stop task, for high and low theta/beta ratio groups.

Only significant main group and interaction with group effects are reported. Figs. 1 (right panel) and 2 (right panel) illustrate the mean MRT and mean SSRT for the high- and low-frequency stop task, separately for the high and low theta/beta ratio group. Repeated measures ANOVA for SSRT revealed a significant main group effect (F(1,27)=5.10, p=0.032), indicating longer SSRTs for the low theta/beta ratio group relative to the high theta/beta ratio group. Furthermore, the low theta/beta ratio group made significantly more omissions than the high theta/beta ratio group (F(1,27)=4.54, p=0.042).

Participants with increased theta/beta ratios relative to low theta/beta ratios had significantly less beta activity (F(1,27)=20.02, p=0.001; 0.03 vs. 0.06), but not significantly more theta activity (F<1; 0.24 for the high and 0.25 for the low theta/beta group). Finally, the factors hemisphere and area did not interact with group (for all interactions: F<1).

3. Discussion

The present study investigated the relation between subjective impulsivity, inhibitory control, and fast- as well as slow-wave EEG activity in healthy individuals. Inhibitory control was assessed using two versions of the stop task that differed in the probability of stop-signals. As expected, all participants developed an impulsive response style in the stop task version where there is a low probability of stop-signals, as indicated by faster reaction times to go stimuli and more unsuccessful stops.

Individuals, scoring high on I7 impulsivity, did not show impaired inhibitory control nor had elevated levels of theta activity, decreased beta activity, or increased theta/beta ratios, relative to individuals scoring low on I7 impulsivity. Given that impulsive behavior in ADHD has repeatedly been associated with deficient inhibitory control (Lijffijt et al., 2005b; Sergeant et al., 2002) and increased slow- and decreased fast-wave activity (Barry et al., 2003; Snyder and Hall, 2006), the present results are not in line with the notion that impulsive behavior in psychopathology is an extreme end of a continuous distribution of impulsivity, as quantified by the I7 questionnaire. However, impulsivity is a complex construct and many self-report measures have been developed, each probably measuring a slightly different component of impulsivity. Whereas I7 impulsivity may not be associated to impaired inhibitory control or to increased theta, decreased beta, and increased theta/beta ratios, other dimensions of the construct impulsivity may be related with inhibition and EEG activity, for instance, motor, cognitive, or non-planning components of impulsivity (as can be assessed by BIS-11). In the present study, it appeared that two groups created by a median split of motor impulsivity of the BIS-11 questionnaire differed on stopping performance. However, contrary to our expectations, participants scoring low on this subscale had longer stop-signal reaction times relative to participants scoring high. This result is in agreement with a previous study on the relation between subjective impulsivity and Stroop interference control (i.e., an inhibition-related variable) which found an effect of conflict manipulation on interference in terms of errors for individuals with low, but not for individuals with high I7 impulsivity scores (Lansbergen et al., in press). We have suggested that high impulsive participants might have had some degree of insight into their impulsive characteristic and consequently were more motivated to maximize their performance and allocated more attention to the task, resulting in better performance, especially regarding inhibition-related performance.

The lack of evidence for an association between subjective impulsivity and inhibitory control might also be due to limited construct validity of self-report measures. Self-report measures rely on participants’ own judgments which can be biased by lack of honesty or social desirability. A more objective measure which has consistently been demonstrated to be related with impulse disorders, particularly with ADHD, may be increased theta/beta ratio (Barry et al., 2003; Snyder and Hall, 2006). From this perspective, the present sample was divided into two groups based on a median split of theta/beta ratio and were compared with respect to performance on the stop-signal task. Assuming that theta/beta ratio in psychopathology is an extreme end of a continuum of the balance between fast- and slow-wave cortical activity within the normal population, it can be expected that healthy individuals, characterized by increased theta/beta ratio will show

| Table 2 - Mean values and standard deviation of the performance data obtained from the high and low frequency stop-signal task, separately for the high and low theta/beta ratio group |
|----------------------------------|----------------------------------|----------------------------------|----------------------------------|----------------------------------|
|                                  | High theta/beta ratio             | Low theta/beta ratio             |                                  |
|                                  | Low-frequency stop task           | High-frequency stop task         | Low-frequency stop task          | High-frequency stop task         |
| MRT (in ms)                      | 406.4 (74.5)                      | 531.4 (124.7)                    | 394.2 (59.6)                     | 509.4 (131.0)                    |
| SSRT (in ms)                     | 150.8 (27.0)                      | 152.2 (25.0)                     | 181.5 (46.3)                     | 181.4 (50.4)                     |
| SDRT                             | 99.1 (27.8)                       | 137.7 (42.9)                     | 107.5 (24.7)                     | 135.4 (31.30)                    |
| SOA                              | 240.9 (61.5)                      | 316.5 (74.6)                     | 209.9 (58.3)                     | 284.4 (98.35)                    |
| Pom                              | 1.7 (1.7)                         | 3.5 (3.4)                        | 2.5 (2.5)                        | 7.0 (5.0)                        |
| Pic                              | 48.0 (8.6)                        | 61.1 (14.2)                      | 42.5 (10.5)                      | 55.2 (22.3)                      |
| Per                              | 3.7 (2.8)                         | 4.8 (5.5)                        | 6.3 (5.6)                        | 5.1 (6.0)                        |

Note. Standard deviations are given in parentheses; 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.
impaired inhibitory control. Recently, Schutter and Van Honk (2005) used the method of creating two groups by a median split of theta/beta power ratio in normal individuals to compare their performance on the Iowa gambling task. In this study, healthy individuals with high theta/beta ratios made more disadvantageous choices. This is consistent with the impaired performance on the Iowa Gambling task in ADHD (Toplak et al., 2005), revealing first evidence for an association between healthy individuals, characterized by increased theta/beta ratios, and ADHD-related impulsivity.

In contrast to our hypothesis, participants in the present study, characterized by relatively high theta/beta ratios had faster instead of slower stop-signal reaction times relative to participants with lower theta/beta ratios. One possible explanation for the contradictory findings may be that in the present study, the majority of participants included were females between the ages of 19 and 26 years, whereas previous studies on baseline EEG in psychopathology have included mostly male children. However, regarding ADHD, it has been demonstrated that increased theta/beta ratio persists into adulthood (Bresnahan et al., 1999, 2006; Hermens et al., 2004; Hobbs et al., 2007). Moreover, findings of normalization of decreased beta, but not of increased theta activity with age (Bresnahan et al., 1999, 2006; Hermens et al., 2004; Hobbs et al., 2007) even further contradict the present finding (i.e., the theta/beta groups differed in beta, but not in theta activity). Furthermore, despite the fact that only female volunteers were tested, it is also unlikely that our unexpected results can be explained by gender bias as theta/beta ratio does not differ between males and females (Hermens et al., 2005). Important to note is that although ADHD males and females, relative to controls, did not differ on theta/beta ratios, elevated levels of theta activity have been demonstrated in ADHD males as compared to females (Hermens et al., 2004, 2005). Moreover, ADHD female adolescents (not adults) relative to their controls showed decreased beta at posterior sites, but slightly increased beta power at frontal sites, whereas ADHD males showed a slight decrease in beta power across all sites (Hermens et al., 2005). Hermens and colleagues (2004, 2005) have suggested that different psycho-physiological processes may underlie ADHD in males and females. Following this suggestion, in our sample with mostly females, decreased mean beta activity across frontal, central, and parietal sites, and not increased theta activity may account for the elevated theta/beta ratio. However, the findings of Hermens et al. (2005) regarding beta activity, may also be interpreted in terms of gender differences in gyrality folding.

The present findings may also be explained by assuming that stopping reaction time varies as a U-shaped function of the theta/beta ratio. In this case, individuals with extreme high as well as those with extreme low theta/beta ratios may have deficient stopping performance (i.e., longer SSRTs), whereas individuals with theta/beta ratios in between may show a good performance on the stop task. Following the theory that theta/beta ratio reflects cortical arousal, we speculate that, in the present study, participants in the low theta/beta group who had deficient stopping performance might have been relatively hyperaroused, whereas the participants in the high theta/beta group might have been within the normal range of cortical arousal. Thus, it may be possible that there are two ‘deficient’ groups, a hypoaroused and a hyperaroused group. Consistently, in a group of boys with ADHD combined type, different EEG subtypes have been identified (Clarke et al., 2001). Whereas one subtype had elevated theta/beta ratios which may be associated with cortical hypoarousal, another subtype had excess beta activity and lower theta/beta power ratios, which may be associated with cortical hyperarousal. In the present study, the participants with higher theta/beta ratios might have shown “normal” levels of theta/beta ratio, whereas the participants with lower theta/beta ratios might have been cortico-hyperaroused, resulting in deficient stopping behavior. Correlation studies in a large sample of healthy individuals that vary on the continuum of theta/beta activity may be able to clarify a possible U-shaped relation between theta/beta ratio and stopping behavior. In line with the ADHD subgroup that have an excess of beta activity, we found that individuals with low theta/beta ratios had significantly more beta power, rather than less theta power, relative to the high theta/beta ratio group. This suggests that individuals with low theta/beta ratios are characterized by an excess of beta activity rather than a reduction in theta activity.

As already mentioned in the Introduction it has been proposed that theta/beta ratio does not reflect arousal, but rather the functionality of the cortical substrates of attentional processing (Barry et al., 2004). In concordance, several measures of attention (e.g., reaction time in the Test of Variables of Attention and errors in an auditory oddball task) have been reported to have a negative relation with theta activity (Hermens et al., 2005; Lazzaro et al., 2001; Swartwood et al., 2003) and a positive relation with beta activity (Loo et al., 2004) in ADHD. However, the present data seem to contradict the view that theta/beta ratio is inversely related to attentional processing. Assuming that impaired stopping performance in ADHD may be related to attentional processing of the stop-signal rather than impaired inhibitory control (see Bekker et al., 2005), it would be expected that individuals with high theta/beta ratios would show longer, but not shorter stop-signal reaction times.

An alternative interpretation may be that theta/beta ratio reflects reduced cortical inhibition over subcortical structures (Schutter et al., 2006). At first, the result of deficient stopping in individuals with relatively low theta/beta ratio scores seems not to be consistent with the findings of impaired performance on the Iowa gambling task in participants with relatively high theta/beta ratio scores (Schutter and Van Honk, 2005). However, in the Iowa gambling task (IGT) participants learn to inhibit choosing from card decks that produce immediate large rewards but eventually lead to even larger punishments. It has been suggested that high theta/beta ratios are associated with a relatively poor inhibitory control over motivational drives causing increased reward dependency and reduced punishment sensitivity (Schutter and Van Honk, 2005). This motivational stance will lead to risky and disadvantageous decision making. In the present study, the increased reward drive associated with the high theta/beta EEG ratio may also provide a plausible explanation for the observed faster reaction times and fewer omissions in the stop-trials as participants with high theta-beta ratios are
more motivated to maximize their performance than participants with low theta-beta EEG ratios.

A possible limitation of the present study is that for most participants, baseline EEG was recorded in a previous experiment. However, various studies investigated the intra-individual stability of human EEG, revealing that human EEG is stable over a time period, varying from several days to a couple of years (e.g., Chi et al., 2005; Corsi-Cabrera et al., 2007; Gasser et al., 1985; Kondacs and Szabo, 1999; Salinsky et al., 1991; Williams et al., 2005). This suggests that EEG is a stable electrophysiological trait. In addition, the possible confound of state-related differences in arousal between the two sessions was minimized by using equal test procedures and environments.

A second limitation of the present study relates to our group EEG analyses that only included theta/beta ratios recorded over Cz in the eyes open condition. However, the most robust finding in ADHD is theta/beta ratio (Barry et al., 2003) and, in addition, Monastra et al. (1999) demonstrated that this EEG index can discriminate ADHD patients from controls with high accuracy. Furthermore, the goal was to use comparable settings as the baseline condition used in the study of Monastra et al. (1999) who recorded EEG activity at Cz in an eyes open condition. Moreover, as has been argued by Monastra and colleagues (1999), using more than one condition to create groups needs multiple statistical comparisons, and thereby increases the probability of false positives.

4. Experimental procedure

4.1. Participants

Participants were selected from a group of 435 psychology students (357 females and 78 males) that filled out the Dutch version of the I7 questionnaire (Eysenck and Eysenck, 1978; Lijffijt et al., 2005a,b). From the group of 435 students, 308 were willing to participate in an experimental study. Students with an impulsivity score on the I7 questionnaire of 0 or 1 were ‘defined’ as low impulsive (30 females and 5 males) and students with an impulsivity score that exceeded 9 were defined as high impulsive (47 females and 12 males). We tried to recruit all male students, but aimed at including an equal proportion of female vs. male participants in each group. Seventeen students with low impulsivity scores and 20 students with high impulsivity scores were tested, but 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 (3 males and 12 females) impulsivity scores were included 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. 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. Participants were required to abstain from the use of caffeine in the morning of the experiment. 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.

4.2. 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-waves, black-white vertical gratings (7.6°×7.6°) with a high (3.62 cycles per degree; cpd) or a low (0.46 cpd) fundamental spatial frequency. 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) 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. The interval between the grating and the stop-signal (SOA) was adjusted by means of a tracking algorithm to yield a performance of about 50% corrected successful inhibitions (i.e., stop the ongoing response to a go-stimulus after a stop-signal; Pic) (De Jong et al., 1995; Logan et al., 1997). Participants were instructed to respond as quickly and accurately as possible to the primary task and to stop their response if they heard the stop-signal. Participants were also told they should not wait for the tones as they would be unable to withhold their response on each trial.

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 version 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. Before the go-stop interval for the next block was estimated, the percentage of successful stops was corrected for the amount of omissions (i.e., a failure to respond) on go-trials in the previous block (Tannock et al., 1989). To increase the unpredictability of the stop tone, the interval between the go-stimulus and the stop-signal was jittered in a range of 240 ms surrounding the calculated SOA (Pilzka 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.

Narrow and wide bars were equally divided across stop- and go-trials. In half of the blocks, subjects had to react with the left finger to the grating with the narrow bars and with their right finger to the grating with the wide bars. 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 stop-trials were presented without being followed by a go-trial, in the low-frequency stop task never more than two
succeeding go-trials were presented without being followed by a stop-trial. The order of presentation of the two versions of the stop-task was counterbalanced across 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.

4.3. **Electrophysiological recordings**

For 22 participants in the present study, EEG activity was recorded using a BrainCap with 58 Ag/AgCl electrodes referenced to the Cz electrode. The ground electrode was placed within the cap between Afz and Fz. For seven participants, EEG activity was recorded using an Electrocap with 58 electrodes referenced to the right mastoid. Here, the ground electrode was placed within the cap between Fpz and Fz. For all individuals, 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 BrainAmp amplifier with a bandwidth of 0.04–100 Hz. The sampling rate was 200 Hz and 500 Hz for the EEG recording using the BrainCap and Electrocap, respectively.

4.4. **Procedure**

In the laboratory, various self-report measures of impulsivity were administered to each participant, for example, the Dutch version of the I7 questionnaire, the Barrat Impulsiveness Scale (BIS-11; Patton et al., 1995), and The Behavioral Inhibition System/Behavioral Activation System Scale (BIS/BAS; Carver and White, 1994). As already mentioned, impulsivity is a multidimensional construct and various self-report measures may measure different components of impulsivity. Several one-way analyses of variances were conducted to test whether the high impulsive group in the present study, as assessed by the first measure of the I7, also has higher impulsivity scores on the second measure of the I7, on the BIS-11 or on the BIS/BAS, relative to the low impulsive group. The results indicated significantly higher impulsivity scores on the second measure of the I7 on Barrat’s motor and cognition subscales, and on the BAS-fun subscale from the BIS-BAS in individuals scoring high as compared to low on the first measure of I7 impulsivity: F = 36.95, p < 0.001; motor: F = 28.99, p < 0.001; cognition: F = 7.32, p = 0.012; BAS-fun: F = 18.08, p < 0.001), suggesting that these subscales reflect comparable components of impulsivity. No significant differences between the impulsive groups were found for Barrat’s nonplanning subscale (p = 0.088) and for BIS (p = 0.498), BAS-drive (p = 0.760), and BAS-reward (p = 0.079) subscales from the BIS–BAS.

Participants were placed in a dimly lit room and performed three cognitive tasks: two versions of the stop-signal task that differed in the frequency of stop-trials and an antisaccade task. The order of the stop-signal and antisaccade tasks was counterbalanced across participants. For seven participants, baseline EEG was also recorded in this experiment. Before the performance of the stop-signal tasks, they were asked to sit quietly for 4 min, 2 min with eyes open, and 2 min with eyes closed, during which baseline EEG was recorded. The resting EEG for the other 22 students was already recorded in a previous experiment.1

4.5. **Data analysis**

4.5.1. **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 was calculated by dividing the number of incorrect choice responses to go-trials without a stop-signal by total number of incorrect choice responses. The percentage of omissions (Pom) was calculated by dividing the number of go-trials a participant failed to respond by the number of go-trials. Furthermore, the corrected percentage of inhibition (Pic) was calculated according to the procedure described by Tannock et al. (1989). The average time interval between the grating and the stop-signal (SOA) was also calculated. The stop-signal reaction time (SSRT) was estimated as described by Logan (1994).

4.5.2. **EEG analyses**

EEG and EOG data were analyzed using Analyzer software. EEG signals were rereferenced off-line to the average of all electrodes and the sampling-rate was changed to 256 Hz with a low-pass filter of 100 Hz. For the participants where EEG activity was recorded using electrodes referenced to the Cz electrode, the current activity at Cz was calculated using Analyzer software. The continuous EEG data were segmented into 2-s epochs, separately for the eyes open and eyes closed condition. Trials with artifacts were rejected from further analyses (absolute amplitude criterion of 100 μV; low activity criterion of 0.3 μV within a 50 ms time window) and ocular artifact correction was conducted according to the Gratton et al. algorithm (1983). EEG data were Fourier transformed (Hanning window: length 20%) and subsequently ln-transformed. Power estimates were derived from the average for the theta (4–7.5 Hz) and the beta (12.5–25 Hz) frequency bands at frontal (Fz, F3, F4), central (Cz, C3, C4), and parietal (Pz, P3, and P4) sites in the eyes open condition. Theta/beta power ratios were calculated between the frequency bands by dividing the power of the slower frequency band by the power of the faster frequency band.

4.5.3. **Statistical analysis**

Separate repeated measures analyses of variance (ANOVA) were conducted for MRT, SSRT, Pic, Per, Pom, and SOA with probability (20% vs. 80% stop-trials) as within-subjects factors and impulsivity (high vs. low impulsivity) as between-subjects factors.

---

1 Note that the intra-individual stability of human EEG is very high, even over a time period of 25 to 62 months (Kondacs and Szabo, 1999).
factor. In addition, separate measures (ANOVA) were performed for the eta and beta power, and theta/beta ratio with hemisphere (left, midline, right) and area (frontal, central, parietal) as between-subjects factors and impulsivity (high vs. low) as between-subjects factor. The alpha level of significance was set at 0.05 two-tailed.

Acknowledgments

This study was supported by a grant from the Dutch Organization for Scientific Research (NWO: 425-20-302). The authors are grateful to Erica van Hell for help in data recording.

References