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# Are electrophysiological correlates of response inhibition linked to impulsivity and compulsivity? A machine-learning analysis of a Go/Nogo task

Kerstin Dück[1], Rebecca Overmeyer[1], Holger Mohr[1], and Tanja Endrass[2]

[1]Technische Universität Dresden
[2]Technische Universitat Dresden

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**Abstract**

Heightened impulsivity and compulsivity are often found in association with both dysfunctional everyday behavior as well as with psychopathology. Impulsivity and compulsivity are also linked to alterations in behavioral response inhibition and its electrophysiological correlates. However, they are rarely examined jointly and their effect outside of clinical samples is still disputed. This study assesses the influence and interaction of impulsivity and compulsivity as measured by questionnaires on behavioral performance and event-related potentials (N2, P3a and P3b) in a visual Go/Nogo task. Data from 250 participants from the general population (49% female; age M = 25.16, SD = 5.07; education level: 94% high school or higher; self-reported lifetime diagnosis of any mental disorder: 12%) were collected. We used robust linear regression as well as regression tree analyses, a type of machine learning algorithm, to uncover potential non-linear effects. We did not find any significant relationship between the self-report measures and behavioral or neural inhibition effects in either type of analysis, with the exception of a linear effect of the premeditation scale of the UPPS on behavioral performance. The current sample size was large enough to uncover even small effects. We discuss potential explanations for this current null finding. One possibility is that inhibitory performance was unimpaired in the current sample and that associations between inhibitory performance and self-report measures might only be seen in samples with mental disorders.

## Introduction

Both impulsivity and compulsivity are psychological concepts that have increasingly gained research interest over the last years, as they are driving human behavior and are presumably involved in dysfunctional or even psychopathological behavior. While initially, they were often considered opposing ends of one spectrum , impulsivity and compulsivity are now conceptualized as two distinct, albeit correlated, personality traits .

Impulsivity can be characterized as a "predisposition toward rapid, unplanned reactions to internal or external stimuli without regard to the negative consequences of these reactions to the impulsive individual or to others" . While there are different conceptualizations of impulsivity, it is commonly described as multidimensional and encompassing cognitive (e.g., attention, planning), behavioral (e.g., motor control, risk taking) and sometimes affective (dependency on strong emotions) domains. The concrete operationalization mostly depends on the impulsivity scales used, some of which will be further described below. Compulsivity does not match the mere opposite of this definition. Instead, compulsive behavior comprises repetitive acts carried out while under a subjective loss of control thereover . Their implementation serves the purpose of preventing a negative outcome (i.e., specific feared consequences or stress and anxiety), although the agent is also on some level aware that it goes against their longer-term goals or the behavior is not suitable for reaching or even related to the intended outcome. Thus, compulsivity itself can be described as the trait facilitating this behavior.

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Due to their dimensional nature, both impulsivity and compulsivity are represented to varying degrees in the general population and can have substantial effects, since they are by definition associated with potential negative outcomes: Heightened impulsivity is correlated with substance use, aggressive behavior, delinquency, gambling and risky sexual behavior, potentially leading to personal as well as societal costs . As of such, heightened impulsivity is associated with disorders like substance use disorder (SUD; , borderline personality disorder and eating disorders , primarily those associated with purging and/or bingeing. Compulsivity is predominantly examined in the context of psychopathology, as it is one of the key diagnostic criteria of obsessive-compulsive disorder (OCD; , which makes non-clinical findings scarce. A longitudinal study in Switzerland found a one-year prevalence of about 15% of subclinical obsessive-compulsive symptomatology in 591 subjects . Compulsivity can, among others, influence eating (facilitating obesity) and addictive behavior (e.g., substance use, internet use) even in healthy people, thus leading to distress. Moreover, the healthcare and socioeconomic costs of compulsive disorders are likely transferable to subclinical compulsive behavior.

Nevertheless, there is no clear distinction between "impulsive" and "compulsive" disorders. An overlap can be seen on a neuronal level, as alterations in inhibitory cortical-striatal-thalamic-cortical circuits have been found in both OCD and attention-deficit-hyperactivity disorder , which is mostly associated with impulsivity . In both OCD and various substance and behavioral addictions, compulsivity is associated with dysfunctional reward and punishment processing, learning and flexibility in favor of symptom-related stimuli that can be attributed to altered frontostriatal and limbic activation . Phenontypically, patients often show increases in both impulsivity and compulsivity in different disorders such as OCD and addictions . Furthermore, some etiological models propose e.g. substance use to originate in impulsive behavior, which then turns compulsive as the SUD chronifies . Both characteristics have been found to drive dysfunctional behavior such as compulsive exercise, overeating and alcohol use in parallel , but their interaction has so far not been resolved. Thus, transdiagnostic research is highly warranted .

Both impulsivity and compulsivity can not only be observed in complex behavior, but also on a basic behavioral level, as they involve weakened motor inhibition and are defined by premature or repetitive responses, respectively. Of particular interest here is their relationship to response inhibition, which describes the act of withholding a prepotent response in order to adapt to the present context . As such, response inhibition plays an important role in goal-directed behavior . The most prominent tasks to assess response inhibition are the Go/Nogo and the stop-signal-task (SST). Although the paradigms have been found to measure slightly different aspects of response inhibition (i.e., action selection vs. preparation of stopping) and associated neuronal processes , both are used to examine the underlying inhibitory performance. In Go/Nogo paradigms, participants are asked to respond as fast as possible to a specific stimulus (Go) but withhold their response to a different stimulus of the same modality (Nogo). Similarly, in SST, participants are asked respond quickly to a stimulus (Go) but stop the initiated response if another stimulus is presented subsequently (stop signal). Thereby response inhibition has been found to activate fronto-striatal areas, primarily the dorsal anterior cingulate cortex, right inferior frontal cortex (rIFG), presupplementary motor areas (pre-SMA), the dorsal and ventral prefrontal cortex and basal ganglia . further propose successful motor inhibition to be a top-down process driven by signaling from the rIFG to the pre-SMA. Using electroencephalographic (EEG) methods, several event-related potentials (ERP) have been linked to cognitive processes connected to response inhibition. Firstly, the N2 is a negative deflection peaking 200 ms after stimulus onset and presumably generated frontally in the midcingulate cortex and ventral and dorsolateral prefrontal cortex . In Go/Nogo tasks, it is mostly associated with novelty, or surprise (Wessel 2012), and conflict monitoring . The N2 is followed by a positive peak in the time-window around 300 ms (P3) which can be further disentangled into the frontocentral P3a and the parietal P3b. For the former, some assume the signal to mirror bottom-up attentional orienting to potentially significant or salient events , while others associate it with the pre-SMA and motor response inhibition . The P3b is assumed to reflect top-down processes of attention allocation and updating of working memory to facilitate response selection .

Combining the aforementioned measures, healthy individuals have been found to make more errors in Go/Nogo tasks when scoring higher on impulsivity measures , while other researchers have not found such an

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association . These findings translate to the neuropsychological correlates of response inhibition in respective tasks: Higher impulsivity is often associated with reduced P3 and enlarged N2 amplitudes . As for compulsivity, higher scores are related to more Nogo commission errors in subclinical samples . However, most findings are reported in relation to OCD, where patients are found to show longer reaction times , lower task performance and altered N2 and P3 signals in Go/Nogo paradigms. Similarly, performance on SST appears to be related to OCD and its symptom scores . However, it is unclear whether this is related to compulsivity or to other confounding measures, such as impulsivity, or if alterations rely on clinical impairments.

As stated before, impulsivity and compulsivity are two distinct features, yet share significant overlap in their neurocircuitry, associated neurocognitive abnormalities and influences on dysfunctional behaviors or even clinical impairments . Thus, further investigation of their interaction is needed to understand their impact. Moreover, the effect of impulsivity and compulsivity might have non-linear qualities (see for example ) which could contribute to the partial null findings reported above, as a potential relationship may not be apparent in linear analyses.

Accordingly, this study aimed to determine how impulsivity and compulsivity are dimensionally related to response inhibition and its electrocortical correlates in a visual Go/Nogo task in a non-clinical sample of adults. We explored the questions whether higher self-reported impulsivity is linked to lower task performance, higher amplitude of Nogo N2, and reduced amplitude of Nogo P3. Second, as the association between compulsivity and response inhibition in Go/Nogo tasks has rarely been studied outside of clinical populations, we opted for an exploratory analysis of the effect of self-reported compulsivity on task performance and event-related potentials (Nogo N2 and Nogo P3). Third, we examined how self-reported impulsivity and compulsivity interact in influencing task performance as well as event-related potentials (Nogo N2 and P3). These associations were studied in a linear as well as a non-linear fashion.

## Methods

### Participants

Data was collected from 253 participants from the general Dresden area. Inclusion criteria were: age 18-45 years, normal or corrected-to-normal vision and German as a native language. Participants were excluded if they reported history of neurological disease or head trauma; lifetime diagnosis of bipolar disorder, borderline personality disorder, psychotic episodes or severe alcohol use disorder; acute eating disorder or severe episode of major depression; taking psychotropic substances within the last 3 months; lifetime use of illicit substances of more than twice a year or lifetime use of cannabis of more than twice a month. One participant was excluded from further analyses due to poor task compliance (multiple responses) and two participants due two subsequent detection of exclusion criteria (regular Cannabis use in the past and German as a second language). Thus, the final sample consisted of 250 participants (49% female; age $M = 25.16$, $SD = 5.07$; education level: 94% high school or higher; self-reported lifetime diagnosis of any mental disorder: 12%).

The fit of the models obtained in the subsequent analyses was tested on a set of 43 subjects from the ongoing follow-up project (43% female; age $M = 24.70$, $SD = 5.96$; education level: 89% high school or higher). Participants did not meet diagnostic criteria for any lifetime or current mental disorders; other inclusion and exclusion criteria were identical to the subject group above.

Data analysis for this report was preregistered under https://osf.io/d4ezm/, where data and analysis routines are also accessible. The project has been approved by the ethics committee at the University Hospital Carl Gustav Carus, TUD (EK 372092017) and was conducted in accordance with the ethical guidelines of the Declaration of Helsinki. All participants gave informed consent and received financial compensation or course credit for participation. The study is part of a larger research project which assessed different cognitive control functions in relation to impulsivity and compulsivity.

### Procedure and measures

#### Go/Nogo task

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All participants completed the Go/Nogo task as part of an EEG session in the lab. In addition to the Go/Nogo task, they completed other EEG tasks as well as a neuropsychological test battery at a first lab appointment and ecological momentary assessment, which will not be reported here. The Go/Nogo task consisted of 256 trials and was divided into two blocks. Each trial started with a white circle presented on a grey background for 200-500 ms. At the center of the circle either a green square was presented as go stimulus (75% of all trials) and participants were instructed to respond as quickly as possible with their right index finger, or a red square was shown as nogo stimulus (25% of all trials) and participants were asked to withhold their response. Stimuli were presented for 500 ms and were separated by a variable inter-stimulus interval of 400-1000 ms. Nogo trials could immediately follow each other or were separated with up to five go trials.

Task performance in the Go/Nogo task was measured by nogo accuracy, reaction time (RT), and the inverse efficiency score (IES) as first introduced by : For every participant, the mean RT for correct responses was normalized by the proportion of correct responses (PC), i.e.,$IES = \frac{\mathrm{RT}}{\mathrm{PC}}$. This allows for inclusion of speed (RT) as well as accuracy (PC) in a combined score.

Personality scales

Impulsivity.

Impulsivity was measured with a German translation of the 11th version of the Barratt Impulsiveness Scale (BIS-11; . Its 30 self-report items comprise attentional, motor, and non-planning impulsiveness. We used its sum score as it has shown good internal consistency ($\alpha = .83$; . Further the UPPS Impulsive Behavior Scale was used . Its 59 items yield separate scores for the traits urgency, lack of premeditation, lack of perseverance and sensation seeking. As Cronbachs's $\alpha$ is only reported for the separate subscales, lying between .8 and .85, we refrained from using the UPPS sum score.

Compulsivity.

The Obsessive-Compulsive Inventory-Revised (OCI-R; is a self-report measure of obsessive-compulsive symptoms, namely washing, checking, doubting, ordering, obsessing (i.e., having obsessional thoughts), hoarding, and mental neutralizing. Its 18 items result in sum score with good internal consistency ($\alpha = .85$), which was used here.

EEG recording and data reduction

EEG was recorded with Ag/AgCl electrodes from 61 sites of equidistant electrode montage (Easycap GmbH, Breitbrunn, Germany) as well as from three external positions: approximately 2 cm below each eye to record eye movements and at the lower back to record the electrocardiogram. The EEG was amplified with two 32-channel BrainAmp amplifiers (Brain Products GmbH, Munich, Germany), a sampling rate of 500 Hz, and referenced to an electrode next to FCz. Offline data analysis was performed with MATLAB R2021a and EEGlab (Delorme & Makeig, 2004) using the high performance computing system (HPC) at the TU Dresden. Continuous data was filtered (0.1 – 30 Hz) and subjected to artifact removal in an adaptive fashion, removing between a single and 10 % of trials in epochs of 1.5 s. Data were submitted to an adaptive mixture independent component analysis (AMICA) and components containing eye-movement and cardioballistic artifacts were removed. Behaviorally, trials with reaction times outside the range of 70 – 600 ms were rejected. EEG data was then referenced to average reference. Stimulus-locked event-related potentials (ERP; N2, P3a, and P3b) were analyzed for correct go and nogo trials. The N2 and P3a were determined in a frontocentral electrode cluster (FCz, FC1, FC2, Cz). The N2 was defined as the most negative value in the time window 230 to 270 ms after stimulus onset and P3a as the average amplitude from 300 to 380 ms after stimulus onset. The P3b was obtained as average amplitude in the time window from 320 to 450 ms in a parietal electrode cluster (CPz, CP1, CP2, Pz, P1, P2). Baseline correction was applied in the 200 ms prior to stimulus onset.

Statistical analysis

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Linear analyses

All further analyses were computed with R . The effects of impulsivity and compulsivity on the dependent variables IES, RT and Nogo accuracy as well as amplitudes of the Nogo N2, Nogo P3a and Nogo P3b were analyzed via robust linear regression. For BIS-11 and OCI-R, the respective sum scores were used as regressors in a multivariate model to uncover possible interaction effects. The UPPS scales were analyzed in a backwards stepwise fashion. Subscales were selected on their influence on the robust final prediction errors (RFPE), where those reducing RFPE were then analyzed in a robust linear regression model. The results of all linear correlation and regression analyses were tested for significance with correction of the false discovery rate as recommended by .

Gradient boosting regression trees

Gradient boosting regression trees (GBRT) are a type of decision tree, a machine-learning method used to uncover non-linear relationships between variables . Decision trees perform binary splits in the predictor space in a way that best minimizes residuals. Every split creates new segments (so called nodes), wherein the mean of the response variable serves as the predicted value. GBRT then, rather than computing a single tree, combine an ensemble of various trees based on the same training data set. The individual trees are grown sequentially, each modifying the previous version (boosting). Ultimately, model performance is enhanced by focusing on the observations that proved difficult to predict in the previous iterations. GBRT have several tuning parameters: Firstly, the number of trees $B$ and the number of splits (interaction depth)$d$ , which can lead to overfitting if too high. This risk is controlled by the learning rate or shrinkage parameter $\lambda$. We optimized the tuning parameters for each analysis through a grid search (see the provided R code for the range of values).

Model fit

Both the linear and the non-linear models assess the influence of impulsivity and compulsivity on different ERP and the IES. To test their accuracy, we employed two methods: Firstly, each model was created via nested cross-validation. Here, two cross-validation processes are nested into each other to allow model selection and assessment of prediction performance on the same data set. In order to do this, we divided the data into $k$ subsets or outer loops. Nested into each of these were inner loops, which were used via cross-validation to optimize GBRT tuning parameters. The fit of the optimized model was then assessed in the outer layer. This should prevent an overly optimistic generalization error as model training and validation are separated. Second, the models were run on a test set of 43 completely separate participants. We estimated model performance through the root square mean error ($RMSE$ ) both in the nested cross-validation process as well as for the test set prediction. These were then compared to the $RMSE$ of the linear regression analyses. Lastly, the regression tree analyses were repeated in permutation tests with randomly assigned outcome variables to test whether the models perform above chance level.$P$ values for the permutation test were calculated as the proportion of $RMSE$ resulting from the permutations ($N = 1000$) that were lower than the actual $RMSE$ .

Results

Behavioral results

Participants achieved an average Nogo accuracy of 0.86 ($SD = 0.11$) and a Go reaction time of 282 ms ($SD = 30$), resulting in an IES of 293 ($SD = 28$). See Table 1 for more details and Table 2 for questionnaire data.

Table 1. Behavioral performance

| | | *M* | *SD* | range |
|---|---|---|---|---|
| Accuracy *(in %)* | Go | 0.99 | 0.01 | 0.93 - 1 |
| | Nogo | 0.86 | 0.11 | 0.48 - 1 |

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| | | *M* | *SD* | range |
|---|---|---|---|---|
| RT *(in ms)* | Go | 282 | 30 | 219 - 439 |
| | Nogo | 253 | 60 | 176 - 734 |
| | overall | 282 | 29 | 221- 439 |
| IES | | 293 | 28 | 241- 470 |

*Notes.* Accuracy = proportion of correct responses; RT = reaction time in ms; IES = inverse efficiency score.

Table 2. Questionnaire results

| | | *M* | *SD* | range |
|---|---|---|---|---|
| OCI | sum score | 12.99 | 9.49 | 0 – 46 |
| BIS | sum score | 60.57 | 8.96 | 38 – 96 |
| UPPS | perseverance | 19.44 | 4.54 | 10 – 34 |
| | premeditation | 22.32 | 4.29 | 13 – 38 |
| | sensation seeking | 32.78 | 7.07 | 14 – 46 |
| | urgency | 26.26 | 5.92 | 14 – 44 |

*Notes.* OCI = sum score Obsessive Compulsive Inventory Revised; BIS = sum score Barratt Impulsiveness Scale; UPPS = UPPS Impulsive Behavior Scale.

Linear regression

Linear robust regression analyses yielded no significant effects of BIS-11 or OCI-R scores on the ERP amplitudes or behavioral performance (Table 3). For the UPPS scales, there was no influence on N2 and P3b amplitude, since the models with just the intercept had the smallest robust final prediction error. Models with the surviving regressors for the remaining response variables are reported in Table 4. Only the effect of the premeditation scale on the Nogo accuracy and IES showed significance after correction of the false discovery rate. Linear (pearson) correlations between behavioral and neural measures with questionnaire scores were also not observed (see supplementary table 1). Figure 1 depicts the EEG at frontocentral electrodes for participants with the highest and lowest 25% of OCI-R and BIS-11 scores, respectively .

Table 3. Linear regression models for BIS-11 and OCI-R scores

| | | β αλυε | SE | t | p |
|---|---|---|---|---|---|
| N2 | Intercept | -3.07 | 2.20 | -.002 | .17 |
| | BIS | -.03 | .04 | -.81 | .42 |
| | OCI-R | -.09 | .15 | -.60 | .55 |
| | BIS*OCI | .00 | .002 | .77 | .47 |
| P3a | Intercept | 9.59 | 2.8796 | 3.33 | .001 |
| | BIS | -.05 | .05 | -.99 | .33 |
| | OCI | -.19 | .19 | -1.01 | .32 |
| | BIS*OCI | .00 | .00 | 1.18 | .24 |
| P3b | Intercept | 8.22 | 2.04 | 4.04 | <0.001 |
| | BIS | -.02 | .03 | -.75 | .45 |
| | OCI | -.07 | .13 | -.58 | .57 |
| | BIS*OCI | .00 | .002 | .76 | .45 |
| IES | Intercept | 283.79 | 16.04 | 17.70 | <0.001 |
| | BIS | .13 | .26 | .48 | .63 |
| | OCI | .40 | 1.06 | 1.33 | .18 |

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| | | β αλυε | SE | t | p |
|---|---|---|---|---|---|
| | BIS*OCI | -.02 | .02 | -1.41 | .16 |
| Go RT | Intercept | 270.66 | 18.09 | 14.96 | <.001 |
| | BIS | .16 | .30 | .54 | .58 |
| | OCI | 1.71 | 1.19 | 1.43 | .15 |
| | BIS*OCI | -.02 | .20 | -1.50 | .13 |
| Nogo accuracy | Intercept | .85 | .07 | 12.68 | <.001 |
| | BIS | .00 | .00 | .26 | .79 |
| | OCI | .00 | .00 | .49 | .63 |
| | BIS*OCI | .00 | .00 | -.42 | .67 |

*Notes.* N2 = maximal amplitude for Nogo N2 signal. P3a = mean amplitude of Nogo P3a signal. P3b = mean amplitude of Nogo P3b signal. IES = inverse efficiency score. Go RT = mean reaction time in Go trials in ms. Nogo accuracy = proportion of correct responses in Nogo trials. OCI = sum score Obsessive Compulsive Inventory Revised; BIS = sum score Barratt Impulsiveness Scale.

$p$ = uncorrected $p$ values.

**Table 4. Linear regression models for UPPS scales after stepwise variable selection**

| | | β αλυε | SE | t | p |
|---|---|---|---|---|---|
| P3a | intercept | 7.68 | 1.82 | 4.19 | <.001 |
| | premeditation | -.12 | .07 | -1.62 | .10 |
| | sensation seeking | .06 | .04 | 1.402 | .162 |
| IES | intercept | 322.35 | 9.23 | 24.90 | <.001 |
| | urgency | -.42 | .26 | -1.59 | .11 |
| | premeditation | -1.01 | .37 | -2.75 | .006* |
| Go RT | intercept | 324.59 | 11.71 | 27.71 | <.001 |
| | urgency | -.42 | .29 | -1.39 | .17 |
| | premeditation | -.90 | .43 | -2.08 | .03 |
| | sensation seeking | -.45 | .25 | -1.79 | .07 |
| Nogo accuracy | intercept | .95 | .03 | 27.77 | <.001 |
| | premeditation | -.0025 | .0015 | -1.68 | .02* |

*Notes.* P3a = mean amplitude of Nogo P3a signal. IES = inverse efficiency score. Go RT = mean reaction time in Go trials in ms. Nogo accuracy = proportion of correct responses in Nogo trials. OCI = sum score Obsessive Compulsive Inventory Revised; BIS = sum score Barratt Impulsiveness Scale.

$p$ = uncorrected $p$ values.

* $p$ values remained significant after FDR correction after Benjamini & Hochberg

**Non-linear regression trees**

In addition to linear relations, we also investigated non-linear relationships between measures of inhibitory performance and questionnaires with regression trees. To test the influence of BIS-11 and OCI-R scores, each model was tested via nested cross-validation and on a test set of participants from a different sample. Regression trees from nested cross-validation were also tested in permutation models with shuffled dependent variables to assess their superiority over a random model. Model accuracy is reported via root mean squared error. As is evident from Table 5, the models differ only marginally from one another in terms of accuracy. While the regression tree seems to explain IES better than the linear model on the test set, this does not hold

true for our sample data in nested cross-validation. Therefore, non-linear associations between impulsivity and compulsivity with inhibitory performance were not observed.

**Table 5. Model comparisons via root mean squared error**

| | *Regression trees* | *Regression trees* | *Regression trees* | *Linear regressions* | *Linear regressions* |
|---|---|---|---|---|---|
| | *test set RMSE* | *nested cv RMSE* | *Permutation test p value* | *nested cv RMSE* | *test set RMSE* |
| N2 | 3.61 | 3.32 | .997 | 3.36 | 3.29 |
| P3a | 4.34 | 4.26 | 1 | 4.29 | 4.24 |
| P3b | 3.29 | 2.81 | .89 | 2.83 | 2.80 |
| IES | 21.68 | 27.72 | .82 | 27.86 | 28.01 |

*Notes.* RMSE = Root mean squared error. N2 = maximal amplitude for Nogo N2 signal. P3a = mean amplitude of Nogo P3a signal. P3b = mean amplitude of Nogo P3b signal. IES = inverse efficiency score. Nested cv = nested cross validation. p value = proportion of RMSE from permutation test smaller than RMSE from nested cv.

Discussion

The present study investigated the relationship between self-reported impulsivity and compulsivity with response inhibition in a large non-clinical sample. In order to do this, we obtained behavioral (RT, Nogo accuracy and inverse efficiency score) and electrophysiological (N2, P3a, P3b) data from a standard Go/Nogo task. Data were analyzed with robust linear regression as well as regression trees, which allow non-linear exploration via a machine-learning algorithm. The linear analyses yielded no significant associations of impulsivity or compulsivity with task performance or EEG measures except for the effect of the UPPS subscale premeditation on Nogo accuracy and IES. We did not observe any non-linear relationship either, as the regression trees did not appear to outperform the linear regression or permutated models as measured by *RMSE* . Possible explanations for this will be discussed below.

Task and sample characteristics

Firstly, as pointed out before, literature on the relationship between impulsivity and compulsivity and response inhibition is ambiguous, as said effect is often not found at either the behavioral or psychophysiological level. This could be due to the fact that the Go/Nogo tasks employed are not always entirely comparable. One feature of the tasks seems to be emotion: Accordingly, adapted Go/Nogo tasks show an incline in activation for emotional vs. neutral stimuli, which in turn is modulated by higher trait impulsivity. This could be shown for Nogo P3 amplitude as well as activation in the middle temporal gyrus , an area linked to disorders of cognitive control . Behaviorally, emotional stimuli seem to impair response inhibition , especially when the demand on executive functions is high . Since emotion and physiological arousal are intricately linked, arousal is thought to be related to impulsivity as well . For example, have shown scores on the BIS-11 to be positively correlated to corticolimbic structures such as the ventral amygdala, caudate and dorsal anterior cingulate gyrus, while negatively correlated with control circuits, e.g., the ventral prefrontal cortex. Thus, trait impulsivity might be associated with an imbalance of physiological arousal and inhibitory control. This would likely also reflect on self-report measures, as these items refer to behavior in the individual's everyday life, which is not taking place in an experimental setting, but influenced by states of affect and arousal. Furthermore, both impulsivity and compulsivity are defined by affective elements and the respective behavior is heavily influenced by emotion. However, the Go/Nogo paradigm in the present study was designed to examine response inhibition as a cognitive process, hence emotion or arousal processes were not involved. This may have attenuated the relationship between response inhibition and its neurophysiological correlates and dispositional impulsivity and compulsivity as indexed by the BIS-11, UPPS and OCI-R. Similarly, in studies investigating the role of cognitive load, the negative effect of trait impulsivity on response inhibition became even more pronounced when cognitive demands were high or executive functioning itself was worse .



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Again, research on compulsivity mainly focuses on individuals with OCD, but there decreases in stop-signal task performance as well as activity in the supplementary motor area and inferior parietal lobule have been found with higher cognitive demand as well . As our Go/Nogo paradigm was intentionally low on cognitive demand, possible relationships to impulsive or compulsive behavior in the participant's everyday life, where they are confronted with a myriad of different stimuli and simultaneous processes, may have been dampened.

One could also raise the question how our participants' questionnaire scores relate to other studies, as this might affect comparability. Regarding the literature mentioned above that examined healthy control groups, our scores for impulsivity and compulsivity (see table 2) are quite similar to their distributions or lie between their groups of high- and low-scoring participants . Notably, our impulsivity scores strongly resemble those of the respective general population samples used for validation of the BIS-11 and UPPS , while participants in the original publications of the OCI-R achieved higher scores . In some cases, our participants achieved slightly higher impulsivity scores than in other studies using healthy control participants . In sum, our sample characteristics resemble those of earlier studies.

Yet, our analyses also differ from those with clinical populations, where results appear more stable. As OCD is defined by obsessive and compulsive symptomatology, individuals with OCD score generally score higher on the OCI-R . Further deficits in behavioral response inhibition have been found , as well as alterations in Nogo ERP signals and higher BIS-11 scores predicted by symptom severity . Similar changes have been found for other disorders often associated with impulsivity and/or compulsivity such as substance use , behavioral addictions , borderline personality disorder , and schizophrenia . Again, other studies have found elevated scores on varying impulsivity or compulsivity measures in these clinical groups compared to our sample . A possible explanation for the difference is that high manifestations of trait impulsivity and compulsivity, as often seen in clinical groups, are needed for its effects to unfold. Present psychopathology may also exacerbate the effect of trait impulsivity and compulsivity when typically compensating processes are impaired. This could be due to either to adverse effects of the mental disorder itself or a common predisposition that facilitated its onset.

It is thus not surprising that many of the studies that did find significant differences in behavioral or electrocortical response inhibition did so via group comparisons based on diagnostic status or high vs. low scores on impulsivity or compulsivity measures. A clear effect is probably found more easily in these distinct groups than in our rather homogenous sample. Furthermore, using patient groups as proxies for high impulsivity and compulsivity complicates interpretability, as potential confounding factors increase. For example, impairments in neurocognitive functions such as attention and processing speed that would in turn affect performance in a Go/Nogo task are reported among others for OCD , alcohol use disorder and borderline personality disorder .

Methodological considerations

When analyzed directly, impulsivity as indexed by self-report measures and cognitive tasks are associated with everyday behavior but show little or no correlation with each other . Presumably these measures do not simply reflect one overarching characteristic of impulsivity, but instead tap into different aspects of it. Sharma et al. (2014) proposed self-report scales to depict more long-term, emotionally laden response-patterns, which would also complement the findings on emotional response inhibition tasks described earlier. Cognitive tasks, on the other hand, may represent purer and "microlevel" processes, which would then also apply to the event-related potentials we analyzed. Thus, this problem of construct validity could explain the lacking association between self-report scales and behavioral and psychophysiological variables, as they measure rather independent dimensions.

Limitations and future directions

It should also be noted that the effect of impulsivity or compulsivity could affect the time course of the ERP instead of their amplitude. Later onsets have been found for the N2 with higher impulsivity and for individuals with OCD , while P3 latency was positively associated with impulsivity on a trend level . Thus, alterations in inhibitory control might not be represented in the strength of the conflict or inhibitory

processing, but in the time and resources needed. In an exploratory analysis we did not find the N2 latency to be explained by impulsivity or compulsivity. Unfortunately, as the P3a and P3b were computed through the mean in a specific time window, latencies could not be reported. Future studies investigating the effect on the time course of response inhibition would be desirable. Another problem arises from the use of the gradient-boosting regression trees for the analysis of possible non-linear effects. This method is rather susceptible to the influence of outliers , as it explicitly focuses on the data points that could not be explained in the previous iterations. While this might have a slight effect on the reliability of our analyses, we chose not to exclude outliers to ensure a better depiction of the true population in our sample.

Conclusions

In sum, our non-clinical sample did not show any clear linear or non-linear relationship between self-reported impulsivity and compulsivity on the one hand and response inhibition as indexed by behavioral performance and N2, P3a and P3b signals in a Go/Nogo paradigm on the other hand. Possibly the effect of said personality traits on response inhibition does not hold true in a general population sample, but needs a clinical sample to unfold, or else to be exacerbated by adaptations to the Go/Nogo task. As research on the effect of impulsivity and compulsivity on cognitive control is vital, but as of yet discordant, a discussion on the best ways to uncover possible associations and interactions is highly warranted. With our comparatively large sample and the different statistical methods we applied, we hope this is a first step in elucidating the question at hand.

Figure legends

**Figure 1.** Grand mean EEG at FCz and CPz for participants with the highest and lowest 25% BIS-11 (left) and OCI-R (right) sum scores.

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