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Differences in response inhibition processes between adolescents and adults are modulated by sensory processes

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ABSTRACT

Response inhibition processes undergo strong developmental changes. The same is true for sensory processes, and recent evidence shows that there also within-modality differences in the efficacy to trigger motor response inhibition. Yet, modulatory effects of within-modality differences during age-related changes in response inhibition between adolescence and adulthood are still indeterminate. We investigated this question in a system neurophysiological approach combining analysis of event-related potentials (ERPs) with temporal EEG signal decomposition and source localization processes. We used the somatosensory system to examine possible *within-modality* differences. The study shows that differences in response inhibition processes between adolescents and adults are modulated by sensory processes. Adolescents show deficient response inhibition when stimuli triggering these mechanisms are processed via SI somatosensory areas, compared to SII somatosensory areas. Opposed to this, no differences between adolescents and adults are evident, when response inhibition processes are triggered via SII cortical regions. The EEG data suggests that specific neurophysiological subprocesses are associated with this. Adolescents seem to encounter problems assigning processing resources to integrate motor with tactile information in posterior parietal areas when this information is processed via SI. Thus, basic perceptual and age-related processes interactively modulate response inhibition as an important instance of cognitive control.

1. Introduction

The ability to inhibit prepotent or inappropriate motor responses has been studied widely (Aron et al., 2004; Bari and Robbins, 2013; Diamond, 2013), and is known to undergo strong developmental changes between children and adults (Brandeis et al., 1998; Hämmerer et al., 2010; Johnstone et al., 2007; Jonkman, 2006; Jonkman et al., 2007; Lewis et al., 2006; Liu et al., 2014; Smith et al., 2004; Woltering et al., 2013). However, only recently the importance of lower level sensory processes for motor response inhibition has been considered (Bodmer et al., 2018; Huster et al., 2010; Shedden and Reid, 2001; Stock et al., 2016; Verbruggen et al., 2006).

It has been shown that the somatosensory modality is particularly potent to trigger response inhibition processes (Bodmer and Beste, 2017), which has been explained by the strong structural neuroanatomical connections between the somatosensory cortex and prefrontal areas (Bodmer and Beste, 2017; Friedrich et al., 2017). Regarding developmental effects in response inhibition it is important to consider that especially the somatosensory system is subject to strong developmental effects in children (Taylor et al., 2016). Yet, even within the somatosensory system (i.e. between the SI and SII somatosensory areas) differences exist how efficient response inhibition processes can be accomplished (Friedrich et al., 2017). Recent evidence suggest that response inhibition processes are better when being triggered via stimuli that are processed in area SI, compared to stimuli that are processed in area SII (Friedrich et al., 2017). This is also crucial regarding developmental effects, because functions of SI and SII cortical areas undergo transformations from childhood to adulthood (Uppal et al., 2016; Nevalainen et al., 2014). Several lines of evidence indicate that children are overresponsive to somatosensory inputs that are hardly noticed by adults (Uppal et al., 2016; Royeen and Mu, 2003; Dunn and Westman, 1997). It may therefore be hypothesized that due to the overresponsiveness to somatosensory (tactile) stimuli in children (Uppal et al., 2016; Royeen and Mu, 2003; Dunn and Westman, 1997) response inhibition is better in children than adults when these processes are triggered using somatosensory stimuli. However, it has also been shown that the connections to and the neurons in area SII are sufficiently developed at birth to produce somatosensory evoked potentials in cortical regions at a latency similar to the one in adults (Nevalainen et al., 2014; Hari and Forss, 1999). It therefore seems that already the

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neonatal SII area has some similar neurophysiological characteristics with the SII area in adults (Nevalainen et al., 2014). The SII region has been shown to encode cognitive aspects of tactile processing (Ackerley and Kavounoudias, 2015) that are crucial for behavioral decisions (Romo et al., 2002b, 2002a). Given all these considerations, it is possible that differences between children and adults in the efficacy to exert motor inhibitory control using somatosensory stimuli may be dependent on whether somatosensory stimuli are processed in different parts of the somatosensory system. Since neurophysiological processes in SII are very similar between children and adults, it is possible that no differences in response inhibition exist between children when response inhibition is triggered via SII. However, it is possible that such differences emerge when SI is used to trigger response inhibition processes: i.e., response inhibition processes are subject to stronger modulations when being triggered via SI or SII cortical areas in children than adults. This would suggest that within-modality differences to effectively trigger response inhibition are further subject to ontogenetic (age-related) modulations between adolescents and adults.

To examine above hypothesis in a system neurophysiological approach, we combine high-density EEG recordings with signal decomposition and source localization methods. Previous results show that reliable differences between areas SI and SII to trigger response inhibition processes can best be detected when intermingled stimulus and response selection processes (codes) in the neurophysiological signal are dissociated using a temporal signal decomposition method (Friedrich et al., 2017). It has been shown that stimulus and response selection codes in the neurophysiological signal can be dissociated using residue iteration decomposition (RIDE) (Mückschel et al., 2017; Ouyang et al., 2011a). In the current study, this procedure is also important, because it accounts for intra-individual variability in the data (Mückschel et al., 2017; Ouyang et al., 2011a). This intra-individual variability is well-known to be strongly affected by developmental processes (Bielak et al., 2014; Garrett et al., 2012; Mella et al., 2015, 2016; Störmer et al., 2014; Tamnes et al., 2012) and that intra-individual variability is larger in children than in adults (Mella et al., 2015, 2016). Most important, it has been shown that differences in intra-individual variability can bias comparison between children/adolescents and adults (Bodmer et al., 2018) and lead to non-reliable insights in cognitive-neurophysiological mechanisms associated with age-related differences (Bodmer et al., 2018). Moreover, especially within-subjects modality differences to trigger response inhibition processes have been shown to be reliably detectable using this method (Friedrich et al., 2017). RIDE decomposes event-related potential (ERP) data into several component clusters with dissociable functional relevance (Ouyang et al., 2011b, 2015a): the S-cluster refers to stimulus-related processes (like perception and attention), the R-cluster refers to response-related processes (like motor preparation/execution) and the C-cluster refers to intermediate processes between S and R (like response selection) (Ouyang et al., 2011b). However, an R-cluster cannot reliably be calculated in Go/Nogo tasks (Ouyang et al., 2013), because of a lack of motor responses in correct Nogo trials. Any response-related processes (like motor preparation/execution) are therefore represented by the C-cluster.

Because the S-cluster reflects mechanisms involved in the processing of stimuli, and we expect that there are within-modality differences to effectively trigger response inhibition between adolescents and adults, we hypothesize that especially the S-cluster shows interactive effects between age groups and stimuli that are processed by SI or SII cortical areas. Previous findings in a dual visual and auditory Go/Nogo tasks suggest that especially mechanisms of resource allocation are modulated when variations in sensory input are likely to affect response inhibition (Witold X. Chmielewski et al., 2016a,b;Chmielewski et al., 2016a,b). These modulations in resource allocation processes and in attention resources are reflected by the P2 ERP and could be detected in tasks with auditory stimuli (Campbell and Sharma, 2013), somatosensory stimuli (Sugimoto and Katayama, 2013) and olfactory as well trigeminal stimuli (Geisler and Murphy, 2000). Therefore we hypothesize that the S-cluster in the P2 time window is smaller in SI Nogo trials, than in SII Nogo trials in adolescents. In adults, no modulations are expected. These modulations are expected to be associated with superior parietal structures, as these are known to mediate sensory integration for the sake of behavioral control (Bizley et al., 2016).

However, the C-cluster has already been shown to be modulated by variations of somatosensory stimuli that are processed in SI and SII cortical regions (Friedrich et al., 2017). We therefore hypothesize that above-mentioned differential effects between adolescents and adults are reflected by modulations in the C-cluster amplitude. This is also the case because the C-cluster has been considered to reflect processes similar to the Nogo-P3, which has been attributed to the process of the motor inhibition itself (Beste et al., 2009, 2010, 2011, 2016; Huster et al., 2013; Wessel and Aron, 2015). Therefore, anterior cingulate and/or inferior frontal regions are expected to reflect modulations of the C-cluster.

Traditionally, response inhibition processes are considered to be reflected by two ERP components: the Nogo-N2 and the Nogo-P3. The Nogo-N2 reflects processes like conflict monitoring or updating of the response program during response inhibition (Beste et al., 2009, 2010, 2011, 2016; Huster et al., 2013; Wessel and Aron, 2015, 2015) or the activity of a modality-specific inhibition process at premotor level (Falkenstein et al., 1999). While another hypothesis at premotor level proposes that the N2 component in Go and Nogo conditions represents proactive inhibitory control and reflects activity of late motor-preparation processes in premotor areas. In this areas the activity might be equal between Go and Nogo conditions with smaller and more posterior N2 components in Go conditions than for Nogo conditions. These proactive processes are more affected in Go conditions through overlapped prefrontal positivity characteristic than in Nogo conditions. (Di Russo et al., 2017; Perri et al., 2015) However the Nogo-P3 ERP likely reflects the inhibition itself (Beste et al., 2009, 2010, 2011, 2016; Huster et al., 2013; Wessel and Aron, 2015). Since the C-cluster strongly reflects processes that are considered to be reflected by the Nogo-P3 (Ouyang et al., 2017; Verleger et al., 2014; Wolff et al., 2017), it is possible that also the Nogo-P3 ERP-component reflects age-dependent differential effects in response inhibition processes when being triggered via SII, compared to SI cortical area. However, especially in ERP components with longer latencies (like the P3 ERP component) variations in amplitude are confounded with a latency jitter (Ouyang et al., 2017). This, together with the high intra-individual variability of longer latencies ERP components (Ouyang et al., 2015a,b,b,a; 2017; Verleger et al., 2014) makes it unlikely, that reliable neurophysiological modulations in line with the behavioral data are obtained using standard ERP components.

2. Materials and methods

2.1. Participants

This study includes two groups with N = 30 adults between 20 and 30 years (mean age 23.70 ± 0.83) and N = 30 adolescents between 14 and 15 years (mean age 14.57 ± 0.18). All participants were right-handed, had a normal or corrected-to-normal vision and confirmed that they don't have any psychiatric or neurological disorders. The institutional review board of the Medical faculty of the TU Dresden approved the study and the participants obtained a written informed consent before the experiment started.

2.2. Task

To examine the effects of somatosensory stimuli being processes in the SI versus SII cortical areas on response inhibition processes we used a Go/Nogo task with vibro-tactile stimuli (Friedrich et al., 2017). It is well-known that slow frequencies predominantly activate the SI cortex

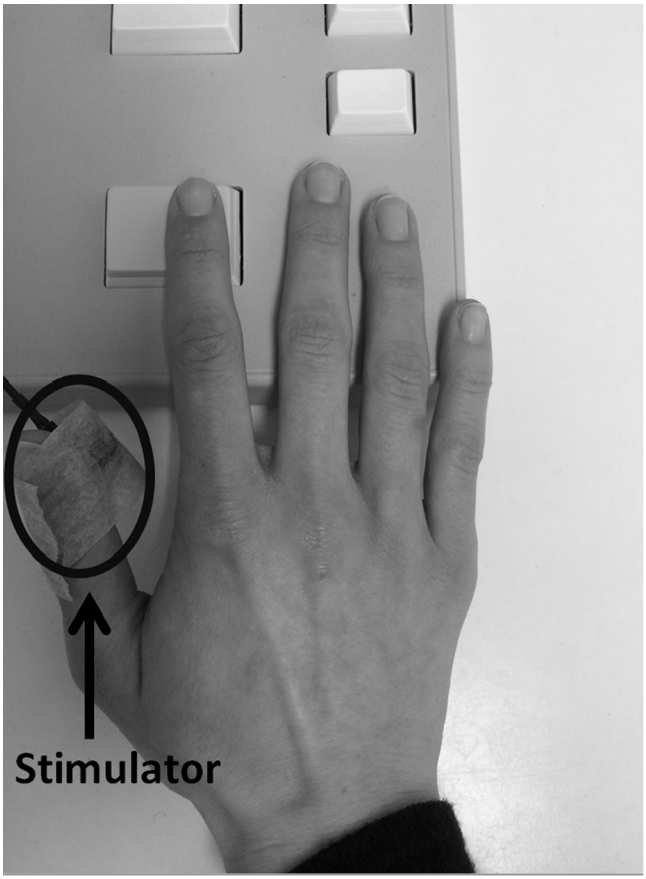

Fig. 1. Illustration of the experimental setup. The vibro-tactile stimuli were delivered via a small electromagnetic stimulator on the right thumb. The participants were instructed to press a button with their right index finger using a customized keyboard.

(Chung et al., 2013; Francis et al., 2000; Harrington and Hunter Downs, 2001) and that high frequent stimuli are processed in the SII cortical area (Chung et al., 2013; Francis et al., 2000; Hämäläinen et al., 1990; Harrington and Hunter Downs, 2001; Kalberlah et al., 2013). The vibro-tactile stimuli were delivered via small electromagnetic stimulators (Dancer Design; for more detailed information see http://www.dancerdesign.co.uk). The stimulators were controlled by the "main module" (Neurocore; http://www.neurocore.de/). The experimental setup is shown in Fig. 1. The thumb of the right hand was stimulated, because it was the only finger not directly touching the table and the response device.

In the SI condition, a slow frequency was used as NOGO stimulus whereas a high frequency served as GO stimulus. In the SII condition, a high frequency stimulus known to be constituted the NOGO stimulus and the slow frequency the GO stimulus. Stimulation was delivered to the right thumb and a right index finger response was required to ensure that stimulus processing and response selection/inhibition is executed by the same hemisphere. In the SI condition, 150 Hz vibration of 100 ms duration served as GO stimulus and the 40 Hz of equal duration as NOGO stimulus. In the SII condition this assignment was reversed; i.e. the 40 Hz stimuli served as GO and the 150 Hz stimuli served as NOGO stimuli. The amplitude of both frequencies was the same. The low frequency of 40 Hz was used due to the dominant role of the SI area in processing "flutter sensations" ranging from 10 to 50 Hz (Chung et al., 2013; Francis et al., 2000; Harrington and Hunter Downs, 2001). 40 Hz was chosen since it is not the upper limit of this approximated range but produces sufficient vibratory sensation in the short 100 ms period. The high 150 Hz frequency is in the range from 100 to 400 Hz which is assumed to predominantly activate the SII area (Chung et al., 2013; Francis et al., 2000; Hämäläinen et al., 1990; Harrington and Hunter Downs, 2001; Kalberlah et al., 2013). The short 100 ms stimulation duration was chosen to produce a strong reaction tendency making it more difficult to withhold responses. Longer stimulus duration is extending the time participants have for stimulus categorization and therefore reduces the probability that a premature response has to actually be inhibited (Friedrich et al., 2017). All participants were familiarized with the different frequencies.

During the experiment, the participants were positioned in front a computer monitor presenting a white fixation cross. The participants were asked look at this fixation cross to reduce eye-movement artifacts in the EEG. Participants were asked to respond as fast as possible to Go stimuli and to refrain from responding on Nogo stimuli. To increase a tendency to respond, 70% of trials were Go trials and 30% of trials were NOGO trials (Witold X. Chmielewski et al., 2016a,b; Dippel et al., 2016; Quetscher et al., 2015). A button press from 100 ms up to 1000 ms after the GO stimulation was classified as correct, whereas no response should occur in the same interval in NOGO trials. Towards the response or after 1000 ms one trial was completed. The time to next trial, which started with the response or after 1000 ms had passed was jittered from 700 to 1100 ms (inter-trial interval (ITI). Therefore, participants were not able to predict Go or Nogo stimulus onset of the following trial. The paced trial presentation further increases a premature response tendency. For adults and adolescents, the experiment encompassed 832 trials divided into 4 blocks. In two blocks (A-blocks) the SI area was stimulated, in the other two blocks (B-blocks), the SII area was stimulated. At the beginning of every block, participants were informed whether they had to respond to the slow or the fast frequency. Participants either received the ABBA or BAAB sequence so that subjects would not expect the occurrence of a certain block. The number of participants receiving either the ABBA or the BAAB sequence was equal in adolescents and adults.

2.3. EEG recording and analysis

The procedure was identical to a previous study (Friedrich et al., 2017): The EEG was recorded from 60 passive Ag/AgCl ring electrodes at equidistant positions connected to a QuickAmp amplifier (Brain-Products Inc.). The ground and reference electrodes were placed at coordinates theta = 58, phi = 78 and theta = 90, phi = 90, respectively. The sampling rate was 500 Hz (impedances $< 5\,k\Omega$) and the recording bandwidth was from 0.5 to 80 Hz. Offline, the data was down-sampled to 256 Hz and band-pass IIR filter (0.5 Hz to 20 Hz, slope of 48db/oct) was applied to the un-epoched data set. After a manual raw data inspection to remove infrequent technical or muscular artifacts, an independent component analysis was applied (ICA; infomax algorithm) to detect eye-related artifacts like blinks or lateral eye movements and pulse artifacts. ICA components showing these artifacts were rejected. Then, the data was segmented into Go and Nogo trials for the SI and SII condition. The segments started 200 ms before target stimulus presentation and ended 1200 ms thereafter. Only trials with correct responses were used for analyses; i.e. only Go trials with responses in the interval from 100 ms up to 1000 ms after the stimulus and Nogo trials without a response in that interval. This was followed by an artifact rejection procedure discarding epochs showing a maximal value difference of 200 μV in a 200 ms, amplitudes below −200 μV and above 200 μV as well as amplitudes below 0.5 μV in a 100 ms interval. Subsequently, current source density (CSD) transformation was used to eliminate the reference potential from the data and to re-reference the data resulting in amplitude values in $\mu V/m^2$. We used 4 splines and 10 polynominals. CSDs work as a spatial filter (Nunez and Pilgreen, 1991; Tenke and Kayser, 2012), which accentuates electrode sites and makes it easier to identify electrode sites that best reflect relevant neuronal activity. Before averaging the data on the single subject level, a baseline correction from −200 to 0 (with 0 marking the time point of stimulus presentation) was conducted.

The ERP data (i.e. mean amplitude) was quantified on the single-subject level for Go and Nogo trials and two stimulation conditions (i.e. SI and SII). The initial choice of electrode sites and time windows used for data quantification was based on a visual inspection of the scalp topography. Importantly, this choice was validated subsequently, using statistical methods. For details regarding this methods see (Mückschel et al., 2014): Accordingly, the P2 mean amplitude was quantified in adolescents in the time range from 210 to 220 ms and for adults in the time windows from 185 to 195 ms in Go and Nogo trials for the SI and SII conditions at electrodes Cz, FCz and FC1. The N2 ERP-component was clearly seen at electrode FCz. The mean N2 amplitude was calculated in the interval between 310 to 320 ms for both groups and SI/SII conditions in Go and Nogo trials. The P3 ERP-component was visible at electrode Cz and the mean amplitude was calculated in the time interval from 400 to 450 ms in Go and Nogo trials for both groups and the SI/SII conditions.

2.4. Residue iteration decomposition (RIDE)

For the residue iteration decomposition (RIDE), the RIDE toolbox (available on http://cns.hkbu.edu.hk/RIDE.htm) was employed to conduct the RIDE analysis using MATLAB (MATLAB 12.0; Mathworks Inc.) using established procedures (Mückschel et al., 2017; Ouyang et al., 2011a; Verleger et al., 2014). Detailed information concerning the mathematical principles of the RIDE decomposition algorithm is given in Ouyang et al. (Ouyang et al., 2015b). Therefore, the conducted CSD transformation does not affect the results. RIDE uses the latency variability of the ERP signal to decompose the data into clusters (Ouyang et al., 2015a) that are either correlated to the stimulus onset (S-cluster or to the response time (R-cluster), as well as a central C-cluster with variable latency, which is estimated initially and iteratively improved. Since no response time measure can be collected in NOGO trials when no button press is required, it is not possible to depict processes related to the response (Ouyang et al., 2013). Therefore, the R-cluster was not computed and any response-related processes (like motor preparation/execution) are therefore represented by the C-cluster. RIDE uses a self-optimized iteration scheme for latency estimation through which the latency estimation of the C-cluster is improved. The initial latency of the C-cluster is estimated using a time window function. In an iterative procedure, the S-cluster is removed, and the latency of the C-cluster is re-estimated based on a template matching approach until convergence of the initial latency estimation and the S and C-cluster. For the current study, the initial time window for the estimation of the C-cluster was set to 100–900 ms after stimulus onset. Additionally, the latest RIDE algorithm uses a time window confinement for each cluster. The time window for the S-cluster was set to -200 to 500 ms around stimulus onset.

The RIDE data (i.e. mean amplitudes of the RIDE clusters) were quantified on the single-subject level for Go and Nogo trials and two stimulation conditions (i.e. SI and SII). The S-cluster was quantified in the time windows similar to the N2 and P2 ERP-components. Electrode FCz revealed the largest S-cluster in the (Nogo)-N2 at time windows from 280 to 295 ms. The mean S-cluster amplitude in the P2 time window was calculated between 215 and 230 ms. For the C-cluster it has already been shown that it reflects processes that are commonly reflected by the (Nogo)-P3 ERP-component (Ouyang et al., 2017; Verleger et al., 2014; Wolff et al., 2017). The mean C-cluster amplitude was calculated in the time window from 400 to 450 ms. The time windows and electrode sites used for the quantification of the S-cluster and C-cluster data were selected by visual inspection of the of the scalp topography. This choice was, again, validated using the same statistical methods as used for the ERP data (Mückschel et al., 2014). This procedure revealed the same electrodes as previously chosen by visual inspection.

2.5. Source localization

For the source localization analysis sLORETA (standardized low resolution brain electromagnetic tomography) (Pascual-Marqui, 2002) was used. As a basis for the source localization analysis we used the estimated RIDE clusters as done in previous studies by our group (Mückschel et al., 2017; Wolff et al., 2017). This was done because the RIDE cluster revealed strongest interactive effects between “Go/Nogo trials”, “age group” and “SI/SII condition” (please see results section for details). sLORETA provides a single solution to the inverse problem (Marco-Pallarés et al., 2005; Pascual-Marqui, 2002; Sekihara et al., 2005). For sLORETA, the intracerebral volume is partitioned into 6239 voxels at 5 mm spatial resolution. Then, the standardized current density at each voxel is calculated in a realistic head model (Fuchs et al., 2002) based on the MNI152 template (Mazziotta et al., 2001). It has been mathematically proven that sLORETA provides reliable results without a localization bias (Sekihara et al., 2005). Moreover, there is evidence from EEG/fMRI and neuronavigated EEG/TMS studies underlining the validity of the sources estimated using sLORETA (Dippel and Beste, 2015; Sekihara et al., 2005). The voxel-based sLORETA images were compared across conditions and groups using the sLORETA-built-in voxel-wise randomization tests with 2000 permutations, based on statistical nonparametric mapping (SnPM). Voxels with significant differences ($p < .01$, corrected for multiple comparisons) between contrasted conditions were located in the MNI-brain.

2.6. Statistics

Mixed effects ANOVAs were calculated for the behavioral data (percentage of hits and hit reaction times in GO trials and percentage of false alarms in NOGO trials). In these models, the factor “condition” (slow NOGO (SI) /fast NOGO (SII) condition) served as within-subject factor and the factor “group” (adolescents vs. adults) served as between-subject factor. For the neurophysiological data (ERP data, RIDE data), “trial type” (Go/Nogo) were set as additional within-subject factor and also the within-subject factor “electrode” was included when necessary. All tests were Greenhouse-Geisser corrected and all post-hoc tests Bonferroni corrected. In the following section, mean values and the standard error of the mean (SEM) are given in brackets for the descriptive statistics.

3. Results

3.1. Behavioral data

A repeated measures ANOVA revealed a main effect of the RTs on Go trials [$F(1,58) = 102.99$, $p < 0.001$; $\eta^2 = .640$] with faster responses in adults (SI condition: 400 ms ± 141; SII condition: 357 ± 143 ms) concerning adolescents (SI condition: 480 ms ± 141; SII condition: 426 ms ± 143). The interaction “SI/SII also failed to reach significance [$F(1,58) = 1.44$, $p = .235$; $\eta^2 = .024$]. There were no significant group differences in the rate of missed Go trials [$F(1,58) = 3.21$, $p = .078$; $\eta^2 = .053$] (Adults in SI condition: 3.7 ± 1.07; SII condition: 1.47 ± 1.36 and adolescents in SI condition: 4.90 ± 1.07; SII condition: 3.77 ± 1.36). Also the interaction “SI/SII x adolescents/adults” [$F(1,58) = 0.277$, $p = 0.607$; $\eta^2 = .005$] didn’t reach the level of significance. RTs on Nogo trials (i.e. false alarm RTs) didn’t differ between the groups [$F(1,58) = 0.85$, $p = .360$; $\eta^2 = .014$] (Adults in SI condition: 381 ± 193; SII condition: 368 ± 194 and adolescents in SI condition: 456 ± 193, SII condition: 435 ± 194) and the interaction “SI/SII x adolescents/adolescents” was not significant [$F(1,58) = 0.13$, $p = 0.911$; $\eta^2 < .001$].

However, the rate of false alarms is the most important parameter in Go/Nogo tasks because it reflects erroneous response on Nogo trials. For this parameter, there was no significant main effect concerning the absolute frequency (number) of false alarms (FA) [all $F < 1.91$,

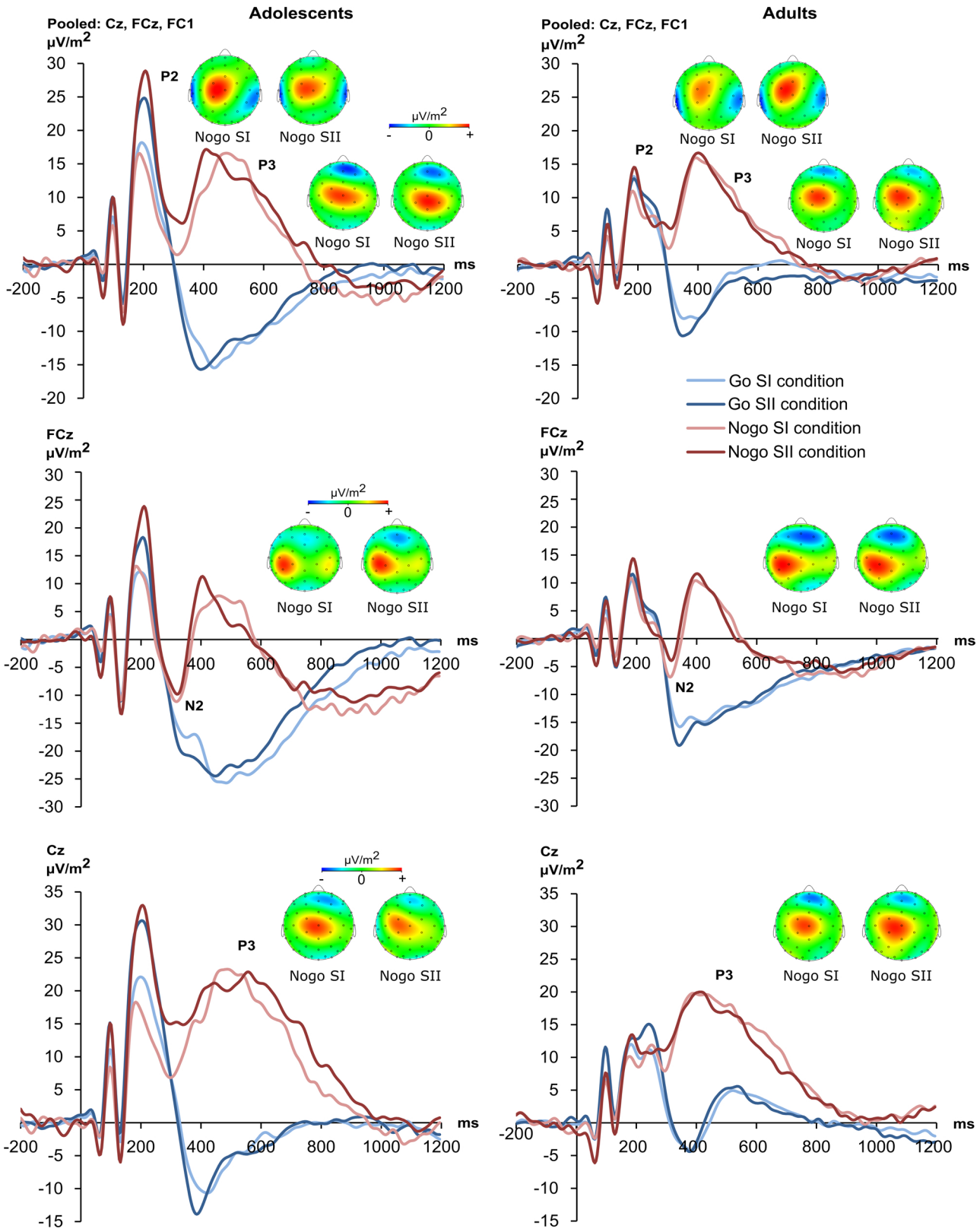

Fig. 2. Event-related potential (ERP) (CSD transformed data) for the adolescent (left side) and adult group (right side). The top row shows the P2 and P3 ERP components pooled across electrode Cz, FCz and FC1. The colours of the different trace denote the different experimental conditions. The scalp topography plots are shown for the peak of the respective ERP-component. Red colours denote positive values, blue colours denote negative values. The middle row shows the N2 at electrode FCz. The bottom row shows the P3 at electrode Cz. In all plots the *y*-axis denotes μV/m^2 and the *x*-axis denotes time in ms. The time point 0 represents stimulus presentation (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article).

$p > 0.193$]. However, there was an interaction "Condition x group" [$F(1,58) = 4.53$, $p = .038$; $\eta^2 = .072$]. Paired post hoc *t*-Tests revealed no FA differences between SI and SII condition in adults [$t(29) = -4.13$, $p = 0.683$] (SI condition 9.13 ± 1.13 and SII condition 9.53 ± 1.10). However, in adolescents the false alarms were higher in the SI condition (19.13 ± 2.78), than in the SII condition (15.50 ± 2.22) [$t(29) = 2.23$, $p = 0.033$]. No other interaction effects reached the level of significance (all $F < 0.13$, $p > .911$).

3.2. Standard event-related potentials (ERPs)

The standard event-related potentials component (P2, N2 and P3) are shown in Fig. 2 (This data, using no CSD transformation is shown in Supplementary Fig. 1).

For the P2 amplitudes (pooled across electrodes Cz, FCz and FC1), a main effect of "SI/SII" [$F(58) = 70.72$, $p < 0.001$; $\eta^2 = .548$] with more positive amplitudes in the SII condition (17.00 μV/m² ± 1.58) than the SI condition (10.90 μV/m² ± 1.11) was detectable. The interactions "SI/SII x adolescents/adults" [$F(1,58) = 32.01$, $p < 0.001$; $\eta^2 = .356$] and "Go/Nogo x SI/SII" [$F(1,58) = 7.39$, $p = 0.009$; $\eta^2 = .113$] reached the level of significance, but also the interaction "Go/Nogo x SI/SII x Adolescents/adults" was significant [$F(1,58) = 10.56$, $p = 0.002$; $\eta^2 = .154$]. Post hoc *t*-tests showed significant differences between Nogo trials in SI and Nogo trials in SII condition in adolescents (13.86 μV/m² ± 1.83) [$t(29) = 7{,}56$, $p < .001$], but not in adults (1.66 μV/m² ± 1.08) [$t(29) = 1.54$, $p = 0.133$]. There were no differences in Go trials between the SI and SII condition in adolescents and adults [all $t < 0.53$; $p > .2$]. No other main or interaction effects reached the level of significance (all $F < 0.63$, $p > .431$).

The analysis of the N2 at electrode FCz revealed a main effect of "Go/Nogo" [$F(1,58) = 18.39$, $p < 0.001$; $\eta^2 = .241$] with more negative amplitudes in the Go condition (−13.61 μV/m² ± 1.79) than in the Nogo condition (−7.71 μV/m² ± 1.92). Furthermore, there was an interaction "Go/Nogo x SI/SII" [$F(1,58) = 6.26$, $p = 0.015$; $\eta^2 = .097$]. Post hoc *t*-tests showed that this interaction was based on significantly larger SI and SII amplitude differences in Go trials than Nogo trials (-4.42 μV/m² ± 1.75) [$t(59) = -2.52$, $p = 0.014$]. There were no amplitude differences between Nogo in SI condition and Nogo in SII condition (−2.32 μV/m² ± 1.56 [$t(29) = -1.483$, $p = .143$]. No other main or interaction effects reached the level of significance (all $F < 0.01$, $p > .906$).

Concerning the P3 amplitudes at electrode Cz a main effect "Go/Nogo" [$F(1,58) = 152.70$, $p < 0.001$; $\eta^2 = .725$] was evident with stronger amplitudes in the Nogo condition (19.39 μV/m² 1.97) than in the Go condition (-5.90 μV/m² 1.69). Furthermore, there was a interaction "Go/Nogo x adolescents/adults" [$F(1,58) = 4.64$, $p = 0.035$; $\eta^2 = .074$]. However, post-hoc tests revealed that there were no amplitude differences between Nogo trials in the SI condition and the SII condition in adolescents [$t(29) = -0.895$, $p = 0.378$] and adults [$t(29) = -0.001$, $p = 0.999$]. No other main or interaction effects reached the level of significance (all $F < 0.04$, $p > .834$).

3.3. Residue iteration decomposition (RIDE)

3.3.1. S-cluster

The S-cluster pooled across electrodes Cz, FCz and FC1 is shown in Fig. 3.

In the P2 time window, a main effect "Go/Nogo" [$F(1,58) = 22.76$, $p < 0.001$; $\eta^2 = .282$] with more positive amplitudes in Go (15.60 μV/m² 1.34) than in Nogo (12.56 μV/m² 1.33) conditions was detected. Furthermore, there was a main effect "SI/SII" [$F(1,58) = 75.27$, $p < 0.001$; $\eta^2 = .565$] with more positive amplitudes in SII condition (17.21 μV/m² 1.55) than in the SI condition (10.94 μV/m² 1.10). The interactions "SI/SII x adolescents/adults" [$F(1,58) = 27.96$, $p < 0.001$; $\eta^2 = .325$] and "Go/Nogo x SI/SII" [$F(1,58) = 5.91$, $p = 0.018$; $\eta^2 = .0.93$] reached the level of significance, however, also the interaction "Go/Nogo x SI/SII x adolescents/adults" was significant [$F(1,58) = 7.18$, $p = 0.010$; $\eta^2 = .110$]. Post-hoc tests revealed that amplitude differences between Nogo SI condition and Nogo SII condition were larger in adolescents (−13.04 μV/m² ± 1.71) than adults (-2.30 μV/m² ± 1.11) [$t(58) = -5.27$, $p < .001$]. The same was, however, the case in Go trials between adolescents (−7.13 μV/m² ± 1.39) and adults (-2.59 μV/m² ± 0.87) [$t(58) = -2.76$, $p = .008$]. However, within the adolescent group, the difference between the SI and the SII condition was larger in Nogo trials than Go trials [$t(29) = 2.86$, $p = .008$]. This was not the case in the adult group [$t(29) = -0.28$, $p = .785$]. The sLORETA analysis revealed that interactive effects "SI/SII x adolescents/adults" in Nogo trials were associated with activation differences in the post-central gyrus (BA7) and the paracentral lobe (BA5).

For the S-cluster in the N2 time window at electrode FCz, a main effect "Go/Nogo" [$F(1,58) = 64.22$, $p < 0.001$; $\eta^2 = .525$] showing more negative amplitudes in Nogo (-4.93 μV/m 1.74) than in Go trials (2.57 μV/m² 1.49). No other main or interaction effects reached the level of significance (all $F < 0.177$, $p > .675$).

3.3.2. C-cluster

The C-cluster at electrode Cz is shown in Fig. 4.

Concerning the P3 time window for electrode Cz a main effect "Go/Nogo" [$F(58) = 106.01$, $p < 0.001$; $\eta^2 = .646$] with larger amplitudes in Nogo trials (21.49 μV/m² 2.09) than in Go trials (−4.58 μV/m² 1.93) was significant. Furthermore, significant interactions "Go/Nogo x adolescents/adults" [$F(1,58) = 18.32$, $p < 0.001$; $\eta^2 = .240$] and "Go/Nogo x SI/SII" [$F(1,58) = 11.49$, $p = 0.001$; $\eta^2 = .165$] were evident but also the interaction "Go/Nogo x SI/SII x adolescents/adults" was significant [$F(1,58) = 4.16$, $p = 0.046$; $\eta^2 = .067$]. Subsequent ANOVAs were calculated for Go trials and Nogo trials separately. It was shown that the interaction "Go/Nogo x SI/SII" was evident in adolescents [$F(1,29) = 9.60$, $p = 0.004$; $\eta^2 = .249$], but not in adults [$F(1,29) = 1.96$, $p = 0.172$; $\eta^2 = .0.63$]. Further post hoc *t*-tests comparing Nogo SI trials and Nogo SII trials revealed significant differences in adolescents (8.76 μV/m² ± 2.93) [$t(29) = 2.995$, $p = .006$], but not in adults (1.49 μV/m² ± 1.58) [$t(29) = 0.939$, $p = 0.356$]. In Go trials, there were no differences between the SI and the SII condition in adolescents and adults [all $t < 0.64$; $p > .2$]. The sLORETA analysis revealed that interactive effects "SI/SII x adolescents/adults" in Nogo trials were associated with activation differences in the anterior cingulate cortex (ACC) (BA24).

4. Discussion

In the current study we examined whether within-modality differences to trigger response inhibition processes are subject to ontogenetic (age-related) modulations between adolescents and adults. We used the somatosensory modality as a 'model system' because existing research implying that SI and SII areas differ in their maturity in children (Nevalainen et al., 2014; Hari and Forss, 1999) and previous findings suggest that these areas differ in their efficacy to trigger response inhibition processes (Friedrich et al., 2017).

In line with the hypotheses, the results show that variations of somatosensory stimuli that were either optimized for processing via SI or optimized for processing via SII cortical regions induced variations in response inhibition performance in adolescents but not in adults. For the adolescent group, response inhibition performance, as evidenced by the rate of false alarms, was better when somatosensory stimuli were used that are optimized for processing via area SII than via area SI. It has been shown that the connections to and the neurons in area SII are well developed at birth and neurophysiological properties of basic sensory processing are similar to adults (Nevalainen et al., 2014; Hari and Forss, 1999). This well explains the pattern of behavioral results. Given that neural properties of area SII are already well developed

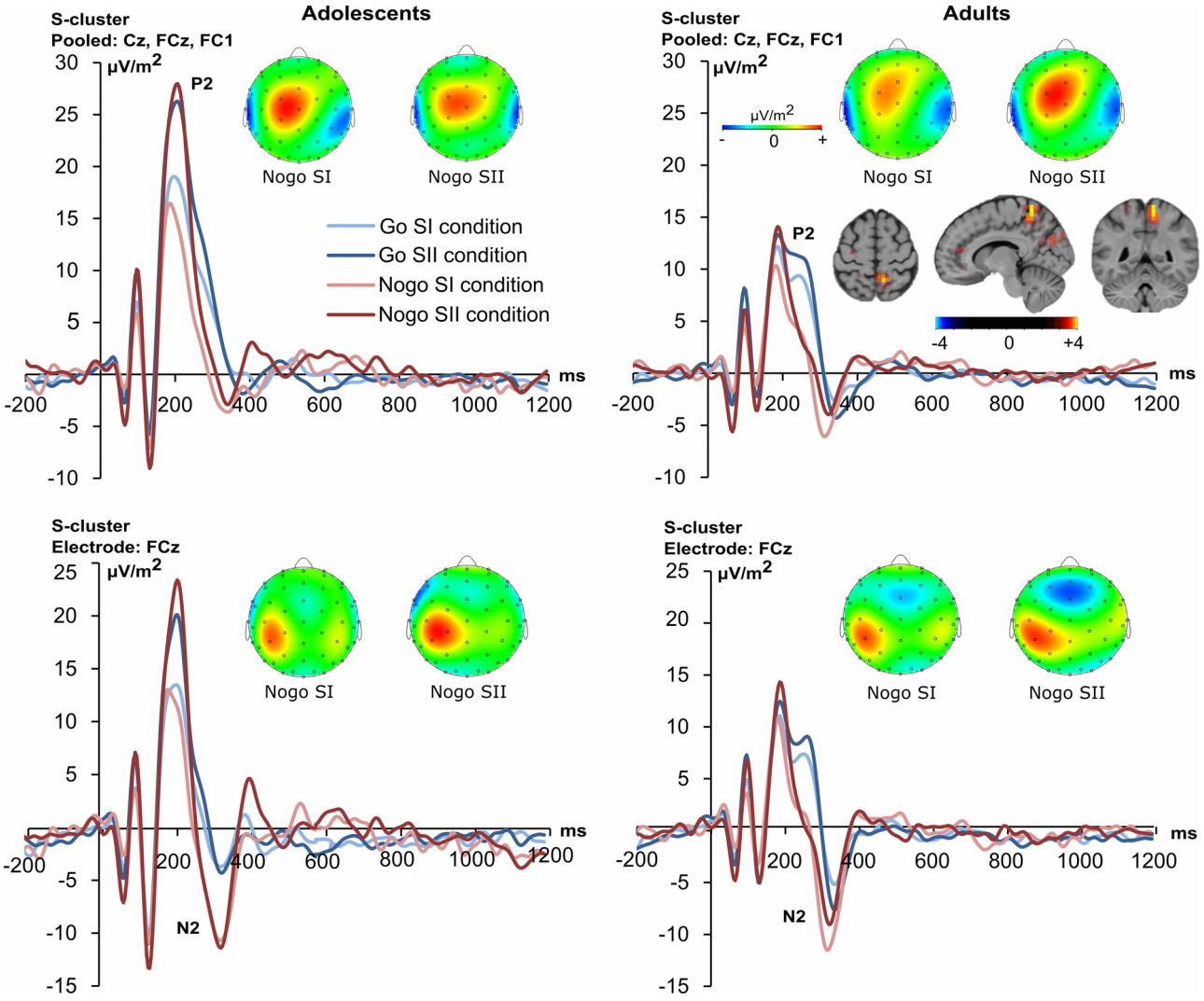

Fig. 3. The RIDE S-cluster pooled across electrodes Cz, FCz and FC1 (plot at the top) and electrode FCz (plot at the bottom) is shown for Go and Nogo trials for adolescents (left side) and for adults (right side). In all plots the *y*-axis denotes μV/m^2 and the *x*-axis denotes time in ms. The time point 0 represents stimulus presentation. At the top, the P2 ERP component as reflected in the S-cluster is shown including scalp topography at the peak of the P2 ERP component in Nogo trials. On the bottom, the N2 ERP component reflected in the S-cluster at electrode FCz is shown including scalp topography at the peak of the N2 in Nogo trials. Positive values are coded in red, negative in blue. The sLORETA plot shows the source of the difference in the P2 modulation in Nogo SI and SII trials between children and adults. The sLORETA colour scales shows the critical t-values (corrected for multiple comparisons) (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article).

(Nevalainen et al., 2014; Hari and Forss, 1999), cognitive processes triggered via this cortical region may be more efficient which ultimately leads to a better behavior in response inhibition. What may also contribute to this is that especially the SII area has been shown to encode cognitive aspects of tactile processing (Ackerley and Kavounoudias, 2015) that are crucial for behavioral decisions (Romo et al., 2002b, 2002a). In line with recent results (Friedrich et al., 2017) no differences in false alarm rates between SI and SII experimental conditions were evident in adults. Thus, the behavioral data shows that there are within-modality differences to trigger response inhibition that are subject to ontogenetic (age-related) modulations between adolescents and adults. The neurophysiological data provides insights what cognitive sub-processes are associated with these modulations.

Since the somatosensory (stimulus) inputs were modulated in this experiment, we hypothesized that especially stimulus-related processes underlie within-modality differences to trigger response inhibition that are subject to ontogenetic (age-related) modulations between adolescents and adults. In line with this hypothesis, the P2 ERP-component amplitude as well as the RIDE S-cluster amplitude in the P2 time window revealed an interaction "Go/Nogo x SI/SII x Adolescents/adults". For both, the ERP and the S-cluster, it was shown that the P2 was smaller in SI Nogo trials, than in SII Nogo trials in adolescents. In adults, no modulations were observed. The P2 amplitude in adolescents (means in μV/m^2: Go SI: 15.81, Go SII: 22.41, Nogo SI: 22.41, Nogo SII: 12.59) seemed to be larger in all conditions than the P2 amplitude in adults (means in μV/m^2: Go SI: 8.11, Go SII: 10.41, Nogo SI: 7.06, Nogo SII: 8.73). However, this was not significant due to the larger variance in the adolescent group (12.64 μV/m^2) than in the adult group (8.74 μV/m^2). As expected, effect sizes were stronger for the S-cluster data than for the original ERP data. Furthermore, unusual effects of larger Go amplitudes compared to Nogo amplitudes in N2 ERP could only detected in standard ERP data, whereas the S-cluster revealed larger Nogo amplitudes than Go amplitudes in the N2 time window. This result can be explained by the fact that RIDE accounts and reduces intra-individual variability in the data (Mückschel et al., 2017; Ouyang et al., 2011a). Processes in the P2 time window have been suggested to reflect resource allocation processes deployed for information processing (Campbell and Sharma, 2013; Geisler and Murphy, 2000; Sugimoto

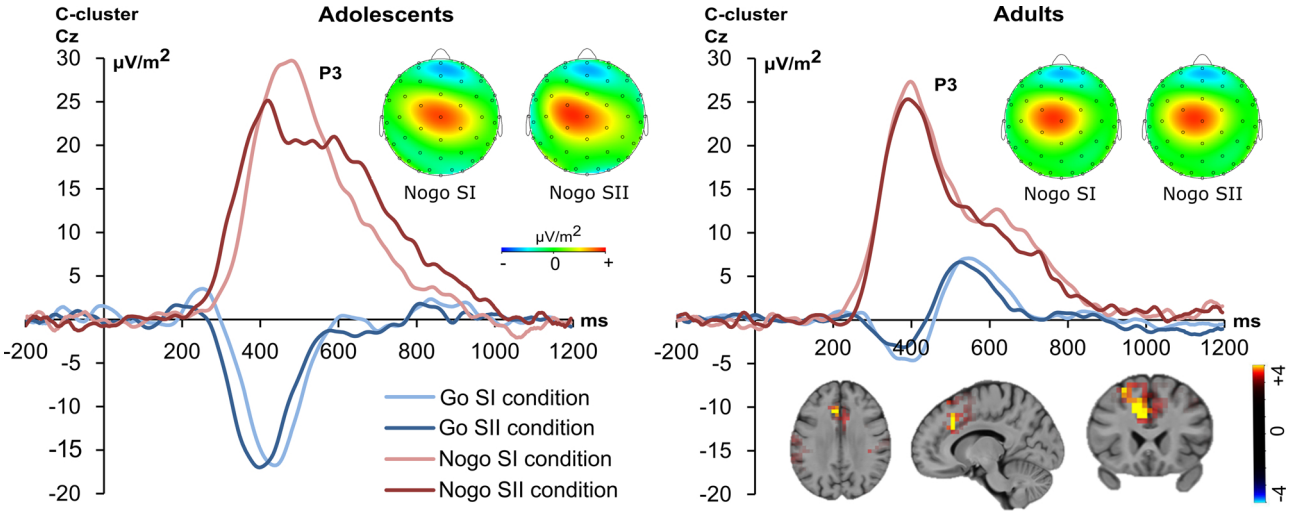

Fig. 4. The RIDE C-cluster at electrode Cz is shown for adolescents (left side) and adults (right side) in Go and Nogo trials. In all plots the *y*-axis denotes μV/m^2 and the *x*-axis denotes time in ms. Time point 0 represents stimulus presentation. The P3 ERP component as reflected in the C-cluster is shown in the plot including the scalp topography at the peak of the P3 in Nogo trials. Positive values are coded in red, negative in blue. The sLORETA plot shows the source of the difference in the P3 modulation on Nogo SI and SII trials between adolescents and adults. The sLORETA colour scales show the critical t-values (corrected for multiple comparisons) (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article).

and Katayama, 2013) and it has been shown that the P2 is modulated by the content or nature of sensory stimulation that needs to be processed to trigger response inhibition processes (Chmielewski et al., 2016a,b). Resource allocation processes therefore seem to be diminished in SI Nogo trials, compared to the SII Nogo trials in adolescents. The smaller processing resources which were allocated to Nogo stimuli in the SI condition than the SII condition in adolescents likely explain why also response inhibition was worse in adolescents in the SI condition, compared to the SII condition. It is possible that response inhibition becomes worse in the SI condition, because smaller processing resources are assigned to Nogo stimuli that are processed via area SII. Interestingly, the source localization (sLORETA) analysis suggests that regions in the post-central gyrus (BA7) and the paracentral lobe (BA5) reflect differences in the modulation of the S-cluster in the P2 time window in SI and SII Nogo trials between adolescents and adults. The results show that activation differences in these areas between the SI and SII Nogo condition were larger in adolescents than adults. Even though an involvement of BA7 during response inhibition processes has only occasionally been reported (Barber et al., 2013; Dippel et al., 2016; Fan et al., 2014; Ocklenburg et al., 2011), it has been suggested that BA7 is involved in response inhibition whenever sensory information is complex and probably difficult to categorize but essential for behavioral control (Fokin et al., 2008; Ocklenburg et al., 2011; Takeichi et al., 2010) (Bodmer and Beste, 2017). Moreover, these regions have been shown to integrate motor signals with touch information (Ackerley et al., 2012; Andersen and Buneo, 2002; Azañón et al., 2010; Gottlieb, 2007; Padberg et al., 2010). It is therefore possible that adolescents seem to have problems assigning processing resources to integrate motor with touch information whenever sensory information triggering response inhibition mechanisms is processed via SI cortical areas. Interestingly, previous results suggest that especially processing via the SI regions seems to be particularly effective to trigger response inhibition processes (Friedrich et al., 2017), because SI area shows more and straighter connections to prefrontal areas than area SII (Kaas, 1993). The current data suggest that this 'SI advantage' is not evident until adolescence and therefore seems to reflect a late-emerging developmental property affecting the interrelation of lower level sensory processes and motor response inhibition.

However, also the C-cluster revealed an interaction "Go/Nogo x SI/SII x Adolescents/adults". No effects were obtained for the P3 ERP-component. This dissociation between C-cluster and the P3 ERP-component has been reported previously (Bodmer et al., 2018; Friedrich et al., 2017) and reflects the fact that in ERP components with longer latencies (like the P3 ERP component) variations in amplitude are confounded with a latency jitter (Ouyang et al., 2017). This, together with (i) high intra-individual variability of longer latencies ERP components (Ouyang et al., 2015b, 2017; Verleger et al., 2014) and (ii) strong variations of intra-individual variability across age (Bielak et al., 2014; Garrett et al., 2012; Mella et al., 2015, 2016; Störmer et al., 2014; Tamnes et al., 2012) increases unexplainable variance in the data. The pattern of modulations observed in the C-cluster was different to pattern of modulations in the S-cluster. Furthermore the effect sizes for the interaction "Go/Nogo x SI/SII x adolescents/adults" were stronger for the S-cluster than for the C-cluster. The effects of these within-modality differences are related to processing resources concerning the SI and SII somatosensory areas for integrating motor with tactile information. These processes are stimulus related (S-cluster) and do less affect the response selection level (C-cluster). This likely leads to the higher effect sizes of the interaction in the S-cluster than in the C-cluster. For the C-cluster data, smaller amplitudes were observed in the Nogo SII condition than in the Nogo SI condition. The opposite was the case in the S-cluster in the P2 time window. The C-cluster has been considered to reflect processes similar to the Nogo-P3 (Ouyang et al., 2017; Verleger et al., 2014; Wolff et al., 2017), which has been attributed to the process of the motor inhibition itself (Beste et al., 2009, 2010, 2011, 2016; Huster et al., 2013; Wessel and Aron, 2015). From this it seems that these processes are intensified in adolescents in the Nogo SI conditions, compared to the Nogo SII condition. The source localization analyses revealed that this was associated with activation differences in the anterior cingulate cortex (ACC), which has previously been shown to be associated with modulations in the P3 time window during Nogo trials (Beste et al., 2016; Huster et al., 2013; Mückschel et al., 2016; Wessel and Aron, 2015). The finding that motor inhibition processes were enhanced in Nogo SI trials in adolescents, may be interpreted to reflect "compensatory" mechanisms. The S-cluster data in the P2 time window suggest that resource allocation processes are diminished. From this it seems that anterior cingulate structures and intensified motor inhibition processes may be deployed to compensate dysfunctional resource allocation processes. Yet, since the behavioral results in adolescents clearly show response inhibition deficits in the Nogo SI condition, this "compensation" is not effective. It may be speculated that these mechanisms are simply too slow to become effective.

The results of this study have clinical implications for neurological disorders like the Tourette's Syndrome with a hypersensitivity of the somatosensory system. These somatosensory areas with medial frontal regions and the ACC are involved in Tic generation and premonitory perceptions (Bohlhalter et al., 2006). Simultaneous the data of the current study revealed a connection between ACC and C-Cluster activity with associations to inhibitory control processes. Therefore, the dependence of inhibitory control processes by sensory modalities in adolescents seems to be useful in understanding specifies in sensory and executive functions in Tourette's Syndrome.

This current study is limited to investigate proactive control processes in somatosensory processing and its relations to inhibition processes (Di Russo et al., 2017). Proactive control plays an important role in response inhibition especially in young age (Berchicci et al., 2015). However, the current study did not employ a cued Go/Nogo paradigm. Moreover, the inter trial interval was jittered between 700 and 1100 ms so it is not possible to detect proactive control processes. Therefore, prefrontal ERP components (like the pN1, Pp1 and pP2) could not be analyzed. In some topographical maps of the current study negative prefrontal focus are shown. Some studies (see Di Russo et al., 2017) described the presence of prefrontal ERP components also in Go/Nogo tasks and linked them to proactive control processes. This may be a question for future studies.

5. Conclusions

The study shows that age-related differences in response inhibition processes between adolescents and adults are modulated by sensory processes. The results show that this refers to *within-modality* differences. Adolescents show deficient response inhibition when stimuli triggering these mechanisms are processed via SI cortical areas. Opposed to this, no differences between adolescents and adults are evident, when response inhibition processes are triggered via SII cortical regions. The data show that specific neurophysiological subprocesses are associated with this. Adolescents seem to encounter problems assigning processing resources to integrate motor with touch information in posterior parietal areas whenever sensory information triggering response inhibition mechanisms is processed via SI cortical areas. Thus, basic perceptual and age-related processes interactively modulate response inhibition as an important instance of cognitive control.

Conflict of Interest

None.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.dcn.2018.04.008.

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