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The Neural Development of Response Inhibition in 5- and 6-Year-Old Preschoolers: An ERP and EEG Study

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Many studies have used event-related potential and neural oscillations to probe the underlying neural mechanisms of inhibitory control in adults, but little has been done in typically developing preschoolers. In this study we tested healthy preschool children between the ages of 5 and 6, and observed better

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response inhibition in 6-year-olds compared to 5-year-olds. Importantly, this age-related difference could not be explained by the N2 component from event-related potential, but was reflected in an increase in right frontal beta power from electroencephalogram. These results suggest that frontal beta power during the preschool period may reflect neural development of inhibitory control.

Many real-life situations require response inhibition in which people's ongoing actions are suddenly rendered inappropriate by unanticipated events in the immediate environment. This ability to inhibit a response is important throughout one's lifespan, and has been demonstrated to be highly correlated with academic achievement in subjects such as mathematical and reading ability (Blair & Razza, 2007; Bull & Scerif, 2001; Espy et al., 2004; Hillman et al., 2012) even at a young age (e.g., 3–5 yrs). Inhibitory control may therefore represent an important index at preschool age as it may contribute to the acquisition of pre-academic skills in school, where children will spend most of their time in the following decade. Indeed, the preschool stage from 4 to 6 years of age is a period of rapid development in response inhibition (Schachar & Logan, 1990; Tillman, Thorell, Brocki, & Bohlin, 2008; Williams, Ponesse, Schachar, Logan & Tannock, 1999). At the behavioral level, preschoolers are starting to be more capable of stopping an inappropriate action (Tillman et al., 2008). At the neuronal level, the rapid development of response inhibition may be a result of the maturation of the prefrontal cortex, which plays an important role in response inhibition and matures last during childhood and early adolescence (Casey, Tottenham, Liston, & Durston, 2005; Sowell et al., 2004).

The ability to inhibit one's responses is often measured with the stop signal paradigm. The stop-signal paradigm, in short, displays a "go" signal that requires a motor response from the participants, with an irregularly intervening post-go "stop" signal that requires sudden withdrawal (Logan & Cowan, 1984). Previous electrophysiological studies have used specific event-related potential (ERP) components to probe the relationship between response inhibition and its neural correlates, such as the prefrontal cortex. In particular, the N2 component has been suggested to be an electrophysiological index of response inhibition in the context of a stop signal task, showing a larger negative deflection in unsuccessful stop trials than in successful stop trials around 200–250 msec after the presentation of the stop signal (Kok, Ramautar, De Ruiter, Band, & Ridderinkhof, 2004; Ramautar, Kok, & Ridderinkhof, 2004, 2006). In one notable study by van Boxtel, van der Molen, Jennings, and Brunia (2001), the authors used a stop signal task and its behavioral measure (i.e., the stop-signal reaction time (SSRT)) to separate their participants into a fast group and a slow group. These authors found larger N2 amplitudes for participants with fast SSRTs in comparison to those with slow SSRTs, which is consistent with the idea that the N2 amplitude can reliably reflect inhibitory processing (Cragg, Fox, Nation, Reid, & Anderson, 2009; Lamm, Zelazo, & Lewis, 2006). Recently, a study by Johnstone and Barry (2007) using a cued Go/No-Go task found that P3, but not N2, reflects inhibition of a planned response and/or conflict. Another study by Ramautar et al. (2004), however, found that the stop-signal N2 showed a less pronounced anterior topography than the typical No-Go-N2, which possibly implies that the N2 component from the stop signal and Go/No-Go paradigms may not be identical. Indeed, Kok et al. (2004) also found that P3 latency ($\sim$275 msec after stop signal onset) usually outlasts the SSRT, which indicates that it cannot entirely account for the inhibitory process in terms of temporal precedence. As such, Kok et al.'s findings suggest that P3 latency may be too late in time to be involved in the inhibitory processes that precede stop-signal motor suppression. To this

end, Lamm et al. (2006) also used N2 amplitude as an electrophysiological index to show that better response inhibition performance was associated with higher N2 amplitude in 7- to 16-year-olds. Again, this not only shows that older children have better inhibitory processing, but that the N2 component can be an appropriate index of such cognitive ability.

Recently, researchers have found that electroencephalography (EEG) time-frequency analysis, including power and phase information, can be more sensitive than traditional ERP approaches in probing certain cognitive processes (Roach & Mathalon, 2008). Time-frequency analysis, in short, is the decomposition of EEG signals into magnitude and phase information for each frequency band. Event-related changes in the magnitude of EEG oscillations contain phase resetting information presumably elicited by the experimental stimuli, as well as non-phase-locked magnitude information in specific frequencies. As a result, this approach may provide more refined and detailed information about neuro-oscillatory activity in the brain (e.g., Uhlhaas, Roux, Rodriguez, Rotarska-Jagiela, & Singer, 2010). For example, synchronization information can reflect the maturity and efficiency of cortical networks (Buzsaki & Draguhn, 2004; Engel, Konig, Kreiter, & Singer, 1991). In healthy children, Benasich, Gou, Choudhury, and Harris (2008) measured the correlation of 2-year-old children's resting EEG signals with seven measures of temperament (assessed by the Toddler Behavior Assessment Questionnaire) and found that children with higher frontal gamma power (31–50 Hz) in resting EEG had better inhibitory control and attentional shifting abilities than their age-matched counterparts. Besides gamma band, Swann et al. (2009, 2012) also found that, in adults, higher beta power near the right frontal regions such as the rIFG and preSMA was associated with successful response inhibition. Indeed, many studies have demonstrated a functional role for beta rhythm in motor preparation and inhibition (Salenius & Hari, 2003; Jensen et al., 2005). For example, Kühn et al. (2004) found a reduction in beta power when people performed a voluntary movement. Zhang, Chen, Bressler, and Ding (2008) also observed increased beta oscillation above baseline when monkeys make an inhibitory decision ($\sim$190 msec). These findings suggest that mature neural synchrony, as revealed by beta and gamma power, may be a critical indicator for the cognitive development of response inhibition.

The studies described above suggest that EEG may be a suitable measure to complement ERP in assessing children's development in inhibitory control. However, note that most EEG studies done in children have only used resting-state neural synchrony as a correlate of the development of behavioral performance. Therefore, studies with task-related neural synchrony (instead of resting EEG signals) during the processes of inhibitory control in children are still much needed. To this end, the present study sought to investigate the neural indexes, as well as the developmental trajectory, of response inhibition in normal preschool children. Here we collected online EEG data while children performed a stop signal task. Behaviorally, although the age gap is only one year, we expected the 6-year-olds to nonetheless outperform their 5-year-old counterparts. Neurophysiologically, we hypothesized that if N2 amplitude and beta and gamma power are important indicators of ability in inhibitory control, even in typically developing preschoolers between the ages of 5 and 6, then we should observe an increase in the electrophysiological components that correlate with behavioral improvement in inhibitory control for this age range. Such a result would confirm the N2 component and beta and gamma band powers as important measures in preschoolers, and also provide electrophysiological evidence of response inhibition in preschoolers. In addition, because previous studies observed that beta amplitude increased shortly after the presentation of the stop signal in right frontal areas (Swann et al., 2009, 2012), we examined ERP and EEG data in the right frontal regions as well. If beta power is important

to successfully inhibit a motor response (Swann et al., 2012), it should be stronger in successful stop trials and related to behavioral performance (i.e., SSRT).

METHODS

Participants

Fifty preschoolers (24 males, 26 females) between the ages of 5 and 6 (range 5.1 to 6.9 years; mean age = 5.89, *SD* = 0.48) were recruited from three kindergartens. Participants were screened for neurological and psychiatric disorders. Participants were divided into two groups by their age, with 26 children in the 5-year-old group (14 males, 12 females; range 5.1 to 5.9 years; mean age = 5.49, *SD* = 0.23) and 24 children in the 6-year-old group (10 males, 14 females; range 6.0 to 6.9 years; mean age = 6.33, *SD* = 0.25).[1] Informed consent was obtained from all participants and their parents prior to the study. Candy was given to the participants as reward upon completion of each experimental block. Their parents received reimbursement for their transportation at the end of the experiment.

Procedure

In the stop signal task there were two kinds of trials: go trials and stop trials, which were signalled in the form of images of a sheep and a red X, respectively (Figure 1). Each trial began with a 500 msec central fixation cross, followed by a 200 msec blank screen. After the blank screen, a sheep facing either right or left was displayed. Participants were asked to press a button with their left or right index finger to indicate which direction the sheep was facing as soon as it appeared (go signal). For example, when the sheep was facing right, participants were asked to press the right button with their right index finger, and vice versa. They were also told that sometimes a stop signal (red X) would follow the go signal, and be presented in the middle of the display, and that they should not press the button when it occurred.

In the first session, 50 go trials were presented to record each participant's choice reaction time (RT) and standard deviation. This allowed individually tailored timeframes for the go signal in the following sessions to be set such that the task was neither too easy nor too difficult for each participant. If a participant's RT in any subsequent session was two standard deviations longer than his/her choice RT in the initial block, they would receive a visual feedback warning (English translation: "too slow to press the button") on the screen, which served as a reminder for them to press the button as soon as the sheep appeared.

In the second session, a tracking method was used for the stop signal task. This allowed overall inhibition probability to be maintained near 50%. The 50% of inhibition probability is specifically useful when recording ERPs because it results in roughly equal numbers of successful and unsuccessful stop trials, allowing a valid comparison of the potentials between the two. In our stop signal task, each participant first completed 20 practice trials (6 of which were stop trials),

[1] In these children, 30 out of 50 participants volunteered to take the short form WPPSI-R (Age 5: $n = 15$; Age 6: $n = 15$). The average IQ was 105.47 ($SD = 15.23$) in 5-year-olds and 110.80 ($SD = 10.84$) in 6-year-olds. These IQ scores were not statistically different between the two age groups ($t(28) = 1.105$, $p = .279$).

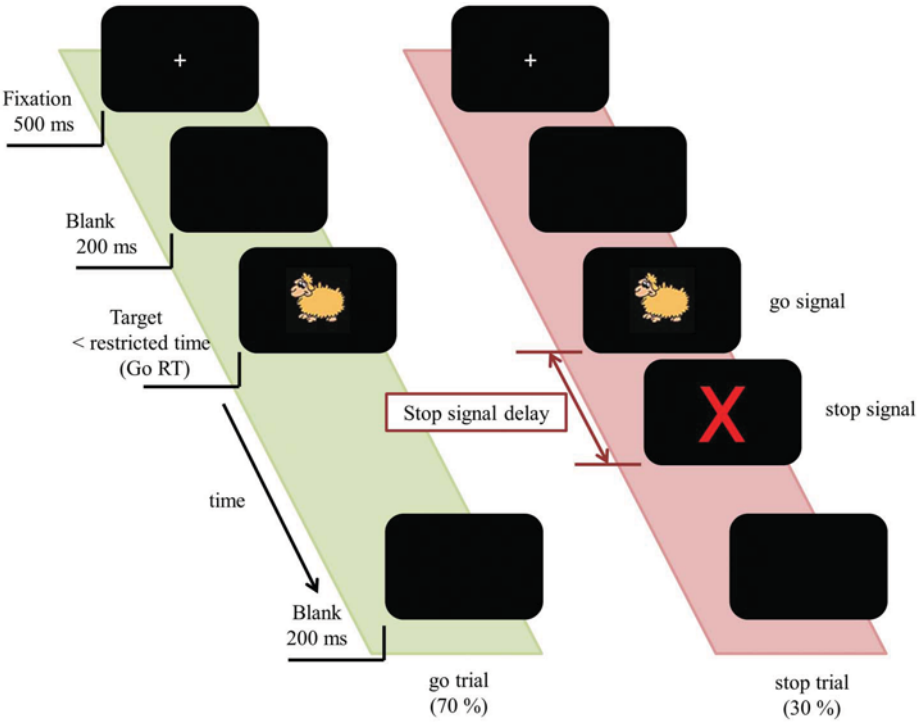

FIGURE 1 Stimulus timeline for the stop signal task. (color figure available online)

and then a total of four blocks of 60 experimental trials, with 30% being stop trials, resulting in a total of 18 stop trials and 42 go trials in each block. The stop trials would appear randomly in each block. The initial stop signal delay (SSD) was set at 250 msec, meaning that the go signal in the first stop trial was displayed for 250 msec before a stop signal appeared. If a response in a stop trial was correct, the next stop trial was made more difficult by adding 40 msec to the SSD. If the response was incorrect, the SSD in the next stop trial was reduced by 40 msec. At the end of the experiment, a critical SSD was calculated and subtracted from the mean reaction time of the correct go trials to obtain the SSRT (Logan & Cowan, 1984).

Electroencephalography Recording

EEG was continuously recorded from 32 Ag/AgCl electrodes (NeuroScan Synamp2) using standard positions according to the extended 10/20 system (Pivik et al., 1993). Electrodes were mounted on a plastic cap (Quick-Cap). The sampling rate was 500 Hz, with an analog 0.05–70 Hz bandpass filter. The reference was the average of electrodes at the left and right mastoids (M1 and M2), and the ground electrode was placed between FPz and Fz. Additionally, two sets of bipolar electrodes were placed on the upper and lower sides of the left eye, and on the canthi of both eyes to measure vertical (VEOG) and horizontal (HEOG) eye-movements. Impedances

for all electrodes were below 10 kΩ. A correction for eye-blinks was first applied to the EEG data acquired, with eye-blink peaks derived from VEOG by means of regression and correlation. These data were used to perform a correction for all channels.

Event-Related Potential Data Analysis and Averaging

The continuous ocular-corrected EEG data were first segmented into epochs starting from 100 msec before a stop stimulus onset and ending at 800 msec after a stop stimulus onset. A digital low-pass filter of 30 Hz (12 dB/octave) was applied to filter out high frequency noise. Baseline correction was executed using a pre-stimulus interval. Epochs with artifacts fluctuating over ±100 μV on the HEOG channel and the rest of the channels were rejected (Spronk, Jonkman, & Kemner, 2008). Three conditions, correct go, successful stop (SST), and unsuccessful stop trials (USST), were averaged according to each condition and included in the ERP analysis. Because the time between the go signal and stop signal is too close in the stop signal task, Kok et al. (2004) proposed an analytical procedure to isolate the pure stop-signal ERP waveforms from the averaged waveforms in stop trials. The majority of SSTs are generated by stop signals with short delays, while the majority of USSTs are generated by stop signals with long delays. In the stop-locked ERPs, this leads to different patterns of overlap between the residual ERP activity elicited by go signal and stop-signal locked components. To address this, Kok et al. (2004) suggested that go signal trials should be divided into Go_{Fast} and Go_{Slow} trials based on the mean RT for correct go trials, where trials of Go_{Fast} and Go_{Slow} are time-locked with the onset of the stop signal (i.e., critical SSD). After aligning the ERPs of the Go_{Fast} with the ERPs of USSTs, and the ERPs of Go_{Slow} with the ERPs of SSTs, the difference waveforms between the ERPs of stop signal trials and its corresponding go signal ERPs (i.e., USST-Go_{Fast}; SST-Go_{Slow}) should provide pure ERPs induced by the stop signals. In this study we compared these two difference waveforms (i.e., USST-Go_{Fast}; SST-Go_{Slow}) to measure the inhibitory processes of motor responses.

A repeated-measures ANOVA was conducted for N2 amplitude from 250–450 msec after the onset of stop signals, where a negative wave was centered. Previous studies observed a more obvious N2 effect at electrodes on the midline (Dimoska, Johnstone, & Barry, 2006; Kok et al., 2004; Ramautar et al., 2004, 2006); therefore, we chose the Fz, Cz, and Pz electrodes for analysis. Two within-subjects factors of trial type (SST and USST) and electrode sites (Fz, Cz, and Pz) were used, and age group was used as a between-subjects factor (5 and 6 years old).

EEG Data With Time-Frequency Analysis

All data analysis was performed offline using SPM8 for MEG/EEG (Wellcome Department of Cognitive Neurology, London, UK; www.fil.ion.ucl.ac.uk/spm/) and custom MATLAB (MathWorks) scripts. The continuous EEG data were stimulus-locked to the stop signal, from 1,000 msec prior to and 1,000 msec following the stop signal. Unlike the ERP analysis, trials containing artifacts exceeding ±150 μV were excluded from the time-frequency analysis. This slight change in criterion was done because the time window of each epoch in the time-frequency analysis was much longer than those in the ERP analysis. Thus, adopting the same artifact rejection criteria from ERP in the context of a longer time window would lead to rejection of more

trials in the EEG analysis. Each epoch was analyzed with a Morlet wavelet transform (i.e., $mf_0\sigma_t = 7$) from 2 to 65 Hz (Roach & Mathalon, 2008). Oscillatory power, defined as the square of the modulus of the resulting complex number, was then averaged across trials. The averaged oscillatory power for each condition for each participant was rescaled by dividing it by the baseline value from 100 to 0 msec before the stop signal onset, and the log10 transform of this quotient was taken. Both trial types (SST and USST) were subjected to statistical analysis with all trials averaged according to their trial type. A mixed ANOVA was conducted to test if power changes between SST and USST occurred between the ages of 5 and 6. There was one within-subjects factor of trial type and one between-subjects factor of age group. The time window for statistical analyses ranged from 100 msec before stop signal onset to 700 msec afterward. Electrode sites (frontal [F3, Fz, and F4], central [C3, Cz, and C4] and posterior regions [P3, Pz, and P4]) were tested in the time frequency analysis. For a given channel, values for all time-points, frequencies, and conditions were considered for ANOVA analyses, with either the criterion of $p < .01$ (uncorrected) or a false discovery rate (FDR) correction for multiple comparisons (corrected).

RESULTS

Behavioral Performance

Seven out of 50 participants did not meet the inclusion criteria, which demanded over 85% correct responses for go trials. Data from the remaining 43 participants were included in the behavioral and ERP analyses (22 participants in the 5-year-old group and 21 participants in the 6-year-old group). The mean SSRT for the 5-year-old children was 307.62 msec (SE = 6.86), while the mean SSRT for 6-year-old children was 272.11 msec (SE = 12.68). To test the hypothesis that SSRT would improve with age between 5- and 6-year-old children, an independent samples *t*-test was conducted, with the independent variable of age and dependent variable of SSRT. As shown in Table 1, SSRT and Go RT were significantly different between 5- and 6-year-old children, SSRT: $t(41) = 2.493$, $p = .017$; Go RT: $t(41) = 2.186$, $p = .035$. Children of 6 years of age had significantly shorter go-RTs and shorter SSRTs than the 5-year-olds.

TABLE 1
Mean RT and Accuracy for the Stop Signal Task in 5- and 6-Year-Old Children

	5-Year-Old Children (n = 22)	*6-Year-Old Children (n = 21)*	*t*	*P*
Accuracy on go trials	91.88%	93.68%	1.329	0.191
Accuracy on stop trials	51.96%	51.98%	0.035	0.972
Mean RT on go trials	670.21 (16.82)	606.42 (24.12)	2.186	0.035*
Mean RT of USST	563.87 (9.99)	511.67 (18.45)	2.519	0.016*
SSRT	307.62 (6.86)	272.11 (12.68)	2.493	0.017*

Note. Children of 6 years of age had significantly shorter reaction times in go trials and shorter SSRTs than the 5-year-olds. Standard errors are shown in parentheses. RT = reaction time; USST = unsuccessful stop trials; SSRT = stop signal reaction time.
*$p < .05$.

ERP Results

Twenty participants (10 in each age group) reached our ERP analysis inclusion criterion, which demanded at least 12 valid trials in each condition (successful and unsuccessful). The average number of valid trials in 5-year-olds was 21.5 SSTs and 21.9 USSTs while the average number of valid trials in 6-year-olds was 24.7 and 24.2 in SSTs and USSTs, respectively. Behavioral data for the two age groups showed the same pattern for all participants ($n = 43$), SSRT: $t(18) = 4.525$, $p < .001$.

The time window of the N2 component was between 250 and 450 msec. A repeated-measures ANOVA was conducted to test the N2 difference between SSTs and USSTs in 5- and 6-year-olds. Figure 2 shows the stimulus-locked grand average waveforms for SSTs and USSTs at Fz, Cz, and Pz for the two age groups. There was no significant interaction between age group, trial type, and electrode site, $F(2,36) = 1.545$, $p = .227$. Only the interaction between trial type and electrode site was significant, $F(2,36) = 3.348$, $p = .046$. Therefore, electrode sites were separated into frontal, central, and posterior regions, along with the within-subject factor of trial type (SST and USST) for separate paired-sample t-test comparisons. These comparisons showed that the

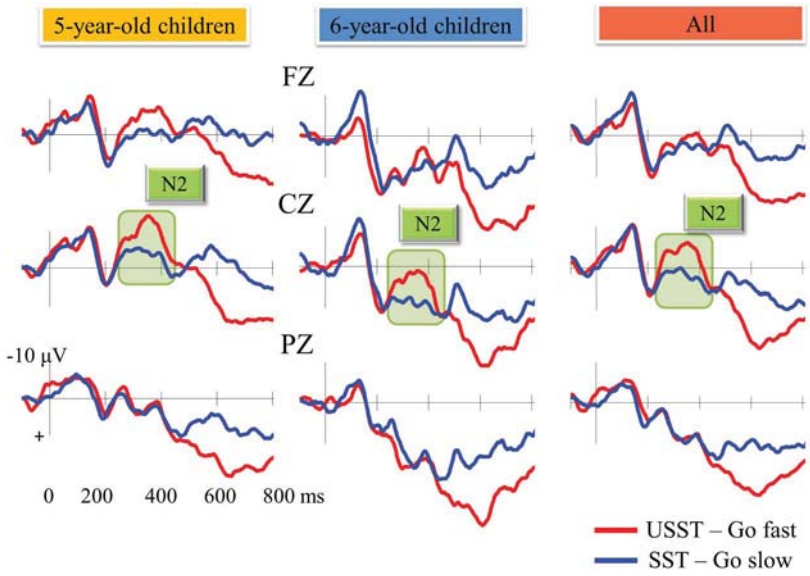

FIGURE 2 The grand average difference waveforms of successful and unsuccessful stop conditions in 5- and 6-year-old children. The zero point on the x-axis represents the stop signal onset time. The highlighted region is the time window of the N2 component between 250 and 450 msec after the stop signal onset. Red lines represent the difference waveforms between the event-related potentials (ERPs) of unsuccessful stop signal trials and fast go signal ERPs (USST-Go_{Fast}); blue lines represent the difference waveforms between the ERPs of successful stop signal trials and slow go signal ERPs (SST-Go_{Slow}). USST = unsuccessful stop trials; SSRT = stop signal reaction time. (color figure available online)

N2 amplitude of USSTs in the central region (Cz electrode) was larger than that of SSTs, $F(1,19) = 9.212$, $p = .007$. In summary, both 5- and 6-year-old children had larger N2 amplitudes in USSTs, compared with SSTs, in the Cz electrode site.

We also conducted a three-way ANOVA to test the right lateralization of the N2 difference between successful and unsuccessful stop trials in 5- and 6-year-olds, with trial type (SST and USST) and hemisphere (left [F3], center [Fz], and right [F4] area) as within-subject factors and age (5- and 6-year-olds) as between-subject factor. This was done because previous electrocorticography (ECoG) studies have shown a right lateralization of frontal beta power increase when a stop trial is successful (Swann et al., 2009, 2012). Here the interaction between these three factors, however, did not reach statistical significance ($F(2,36) = 0.086$, $p = .918$), neither did the interaction between trial type and hemisphere ($F(2,36) = 1.115$, $p = .339$). This result suggests that right lateralization in frontal regions was not observed in our ERP analysis.

Time-Frequency Decompositions With Morlet Wavelets

EEG data from the same 20 participants were also analyzed using time-frequency analysis. After excluding trials containing artifacts exceeding ± 150 μV, the average number of valid trials in 5-year-olds was 30.1 in SSTs and 29.1 in USSTs, and the average number of valid trials in 6-year-olds was 33.0 and 30.5 in SSTs and USSTs. In order to test our hypothesis that motor inhibition is right lateralized, left and right electrodes in frontal, central and posterior regions were included in the analysis. A mixed-effects ANOVA was conducted to test for any power difference in frequency bands as a function of age or trial type. Electrode sites (frontal [F3, Fz, and F4], central [C3, Cz, and C4] and posterior regions [P3, Pz, and P4]) were tested separately because the fronto-posterior region showed different patterns in the ERP analysis. The main effect of trial type was significant in the gamma band (35–50 Hz) from 200 to 300 msec after the stop signal onset in the frontal and central regions, and in the alpha band (9–12 Hz) from 300 msec to 550 msec following the stop signal in the central region ($p < .01$, uncorrected). There was also an interaction between trial type and age group in the beta and lower gamma bands (24–36 Hz) from 50 msec to 100 msec following stop signal onset in the frontal region ($p < .01$, uncorrected, Figure 3a). These data were all submitted to Lilliefors goodness-of-fit test to ensure that the assumption of a normal distribution (during these windows where significant statistics were found) was not violated (200–300 msec in gamma: $p = .2431$; 300–550 msec in alpha: $p = .2828$; 50–100 msec in higher beta and lower gamma: $p = .3752$). Other interactions between trial type and age group in the central and posterior regions were not statistically significant.

Swann et al.'s (2012) findings suggested that the motor inhibitory mechanism is lateralized to right frontal regions in the beta band, and we also observed stronger beta power in successful stop trials from 24 to 78 msec after the stop signal onset in 6-year-olds ($p < .01$, uncorrected, right panel in Figure 3a). Based on these findings, we expected to test whether this right-lateralization pattern was different in the two age groups. A mixed ANOVA was conducted to test the 5- and 6-year-olds' beta power for successful and unsuccessful stop trials in the right frontal site (F4). There was an interaction between trial type and age group in the beta band (23–26 Hz) from 30 msec to 80 msec after stop signal onset for this region ($p < .05$, FDR corrected, left panel in Figure 3b). A paired t-test was conducted to test the beta power difference between successful and unsuccessful stop trials in the right frontal site (F4) in 5- and 6-year-olds, respectively. We observed a stronger beta power (23–26 Hz) in successful stop trials from 30 to 65 msec after

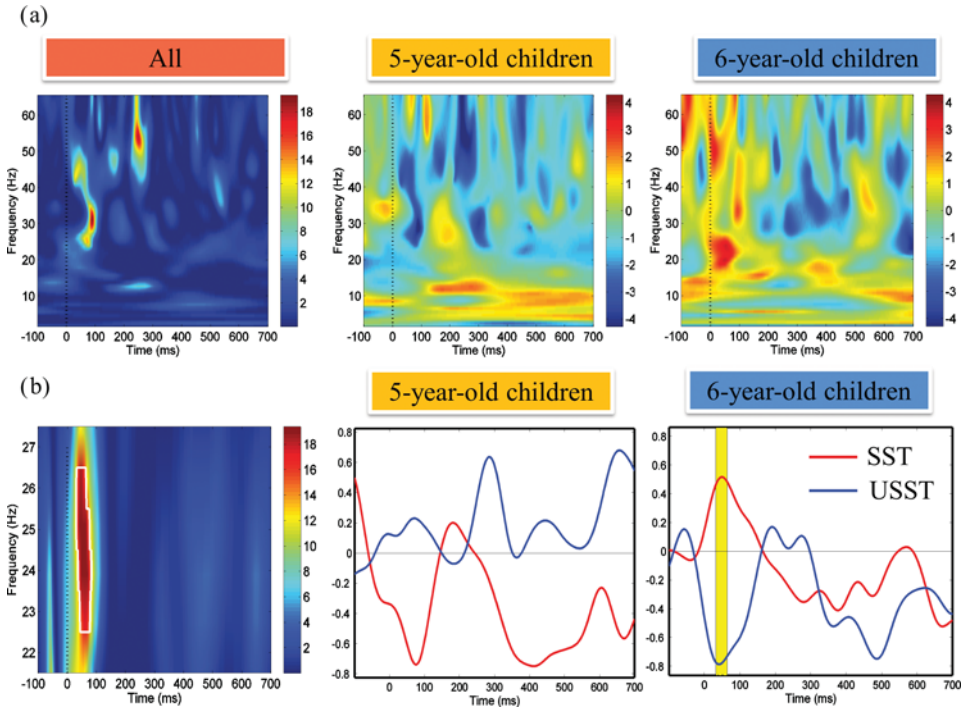

FIGURE 3 (a) Time-frequency decompositions with Morlet wavelets in the frontal regions (F3, Fz, and F4). The left panel shows the significant interaction between age group and trial type (successful versus unsuccessful stop trials), which was driven by a stronger beta and lower gamma power in successful stop trials in 6-year-olds but not 5-year-olds. The middle panel shows weaker power in the beta and lower gamma bands for successful versus unsuccessful stop trials in 5-year-olds. The right panel shows stronger power in the beta (18–26 Hz) and lower gamma bands for successful versus unsuccessful stop trials in 6-year-olds. The zero time point marks the onset of the stop signal. The color of the graph specifies the direction of the *t*-values in the right and middle panels, and *F*-values in the left panel. (b) The left panel shows the significant interaction between age group and trial type in beta power from 30 to 80 msec following stop signal onset ($p < .05$, false discovery rate [FDR] corrected). The middle and right panels show the power in the beta band (22–27 Hz) in successful (red) and unsuccessful (blue) stop trials. In 5-year-olds, the beta power was no different between successful and successful stop trials (middle panel), while a stronger beta power was observed in successful stop trials over unsuccessful stop trials in 6-year-olds between 30 to 65 msec after the stop signal onset (right panel, $p < .05$, FDR corrected). (color figure available online)

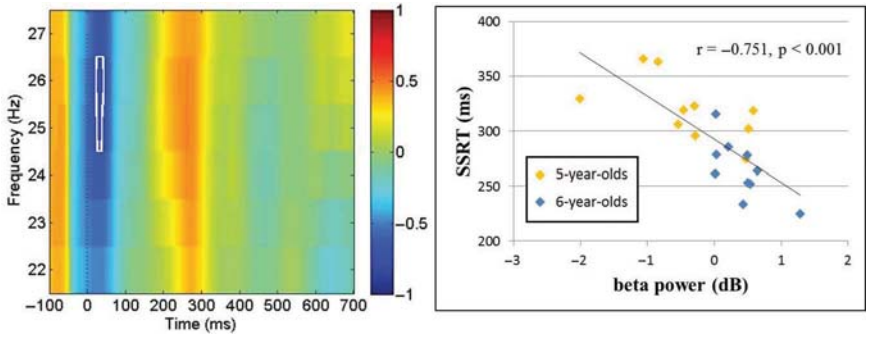

FIGURE 4 The left panel shows correlation between stop signal reaction times (SSRTs) and beta power in successful stop trials in 5- and 6-year-olds. The area enclosed by the white line denotes the area that reached statistical significance ($p < .05$, false discovery rate [FDR] corrected) and is shown in the correlation coefficient plot on the right. There is a negative correlation between SSRT and beta power (25–26 Hz) from 20 to 40 msec following stop signal onset; thus, stronger beta power is associated with more efficient inhibition of an action. Orange dots represent the values for 5-year-olds while blue dots represent values for 6-year-olds. (color figure available online)

stop signal onset in 6-year-olds ($p < .05$, FDR corrected, Figure 3b), but the opposite pattern was seen in 5-year-old children.

To investigate the relationship between behavioral performance and beta band activity, Pearson correlation was computed between SSRT and beta power in successful stop trials after stop signal onset in 5- and 6-year-olds. A negative correlation between SSRT and beta power (25–26 Hz) was observed ($p < .05$, FDR corrected). Figure 4 shows the negative correlation between beta power (25–26 Hz) from 20 to 40 msec following stop signal onset and SSRT ($r = –0.751$, $p < .001$).

DISCUSSION

The aim of the current study was to investigate age-related changes in response inhibition in preschool children via electrophysiological indexes (i.e., the N2 component and specific frequency band activities). In our behavioral results, we observed a significant improvement in response inhibition in 6-year-olds over 5-year-olds. That is, the SSRT in 6-year-old children decreased about 35 msec in comparison to the SSRT of 5-year-old children. Thus, motor response inhibition seems to have dramatically improved during this one-year period. Furthermore, in both age groups, we observed larger N2 amplitudes in unsuccessful trials than successful trials (the N2 effect), as well as increased alpha power in successful trials from a similar time window. The N2 effect and alpha power change were not significantly different between 5- and 6-year-olds, but time-frequency analysis of the EEG data showed that power change in the beta and lower gamma bands (between successful and unsuccessful stop trials), especially in the right frontal regions,

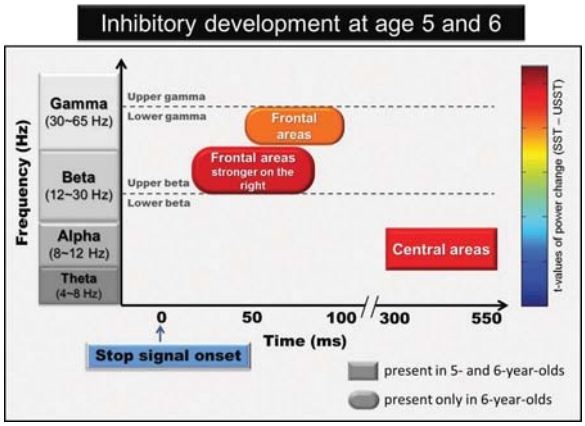

FIGURE 5 Summary of power changes in neural synchrony in 5- and 6-year-olds. The increments of both beta and lower gamma power in successful stop trials were observed in 6-year-olds (represented by rounded boxes); however, only frontal beta power of successful stop trials was correlated with behavioral performance. The stronger alpha power in successful stop trials was observed in 5- and 6-year-olds (represented by a rectangular box). (color figure available online)

was a critical factor that contributed to the behavioral improvement from 5 to 6 years of age (as summarized in Figure 5).

The N2 Effect

The demonstration of the N2 effect in both 5- and 6-year-olds here presents a somewhat different finding from a study by Johnstone et al. (2007), who did not observe the N2 effect in children aged from 7 to 12 years. This inconsistency cannot be explained by the age differences between the two studies (5–6 vs. 7–12 years of age), because several studies have documented the N2 effect in adults (Kok et al., 2004; Ramautar et al., 2004, 2006). We think perhaps this inconsistency may be due to the fact that our ERP analyses adopted Kok et al.'s (2004) analytical procedures. Due to the short time interval between the go signal and the stop signal, ERP data of these two signals often overlap and need to be teased apart. In order to isolate processing specifically related to a successful or unsuccessful stop, we used the analytical procedure proposed by Kok et al. (2004) to obtain uncontaminated ERP waveforms from the stop signal trials, which may explain why we were able to observe the N2 effect in 5- and 6-year-old children.

Inconsistent with our prediction, the N2 effect was not significantly different between the two age groups, and therefore was unable to account for the behavioral improvement over age. One possibility is that ERP components cannot effectively differentiate the development of response inhibition between 5- and 6-year-olds, presumably because the nature of ERP components represent transient phase resetting of EEG data (in response to the experimental stimuli), which neglects the equally important non-phase-locked magnitudinal information about amplitude power change in the EEG data. To address this possibility, we performed Morlet wavelet

transform to analyze EEG data (Roach & Mathalon, 2008) with time-frequency analysis to clarify the neural changes of response inhibition in 5- and 6-year-old children.

Power Changes in Neural Synchrony

Time-frequency analysis showed that power changes in beta and lower gamma band between successful and unsuccessful stop trials increased with age from 5 to 6 years old. The beta power in successful stop trials negatively correlated with SSRT, which better explains the observed improvement in behavioral performance across age, in comparison to N2 from ERP analysis. Similarly, higher gamma band oscillation during resting state has been shown to correlate with better cognitive skills in children (Benasich et al., 2008). In Benasich et al.'s (2008) study, the authors found that lower resting gamma power was associated with poor language/cognitive skills, attention, and inhibitory control in young children, suggesting that developmental lag may be reflected in reduced gamma. A study by Barry et al. (2010) also found a negative correlation between absolute gamma and scores on the DSM Inattentive scale, which indicated that inattention was negatively correlated with resting gamma power. Consistent with this finding, the current study found evidence of a relationship between lower gamma band oscillation and the ability to inhibit motor responses in preschool children. In 6-year-old children, an increase in lower gamma band activity in successful stop trials, coupled with quicker SSRTs, was observed in the frontal region. Furthermore, in comparison with unsuccessful stop trials, stronger frontal beta power in successful stop trials was observed in 6-year-olds. The location of these age-related neural activities related to response inhibition are consistent with neuroimaging studies showing that areas in the medial prefrontal and right inferior frontal areas, such as preSMA and rIFG, are involved in response inhibition and mediate response execution or inhibition (Aron & Poldrack, 2006; Aron, Behrens, Smith, Frank, & Poldrack, 2007; Aron, Fletcher, Bullmore, Sahakian, & Robbins, 2003; Chao, Luo, Chang, & Li, 2009; Chen, Muggleton, Tzeng, Hung, & Juan, 2009; Duann, Ide, Luo, & Li, 2009; Hsu et al., 2011; Li, Huang, Constable, & Sinha, 2006). Similarly, a study using ECoG recordings in patients' preSMA and rIFG showed that beta amplitude ($\sim$16 Hz) increased in both preSMA and rIFG shortly after the presentation of a stop signal, reflecting stronger beta power in successful stop trials lateralized to the right frontal regions (Swann et al., 2012). In our findings, right frontal beta power in 5- and 6-year-olds was correlated with behavioral performance. These results suggest that maturational change in frontal beta band activity may play a role in the developmental course of response inhibition. It is important to note, however, that although power change in both beta and lower gamma band increased with age from 5 to 6 years old, only right frontal beta power in successful stop trials correlated with behavioral performance. Therefore, it seems that beta activity is more linked to the maturation of response inhibition. It would be fruitful for future studies to disambiguate the relationship between beta and gamma power in the frontal region, as well as their precise relationship to the mediation of inhibitory control.

The time-frequency decomposition of the data showed an effect within a time window similar to the N2 effect observed in our ERP results (from 250 to 450 msec). There was an alpha power increase in successful stop trials from 300 msec to 550 msec following the stop signal in the central regions. This alpha power increase was not statistically different between 5- and 6-year-olds, which is consistent with our ERP findings. However, alpha activity may still reflect a

functional component within the inhibitory control network. Evidence from the working memory literature suggests that increased alpha power is associated with better inhibition of distractors, indicating increased attentional control (Sauseng et al., 2009; for a review, see Klimesch, 2012). Thus, greater activation of alpha power in successful stop trials from the present study may play an attentional and/or inhibitory role in the stop signal task during the 300 msec to 550 msec time window after the stop signal onset.

CONCLUSION

The behavioral development of 5- and 6-year-old children's motor response inhibition is reflective of their brain/neural development. In this study we have identified an increment in right frontal beta power during successful stop trials. This suggests that age-related changes in response inhibition may reflect the relatively immature development of the frontal cortex in preschool children. Moreover, the results here suggest that beta band activity may serve as a potential electrophysiological index of inhibitory control in preschoolers.

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