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PAPER

Development of action monitoring through adolescence into adulthood: ERP and source localization

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Abstract

In this study we examined the development of three action monitoring event-related potentials (ERPs) – the error-related negativity (ERN/Ne), error positivity (P_E) and the N2 – and estimated their neural sources. These ERPs were recorded during a flanker task in the following groups: early adolescents (mean age = 12 years), late adolescents (mean age = 16 years), and adults (mean age = 29 years). The amplitudes of the ERN/Ne and N2 were greater in the adult and late adolescent groups than in the early adolescent group. Both of these components had neural sources in the anterior cingulate cortex (ACC). Although P_E was present across groups, P_E amplitude was greater in the late adolescent group compared to the adult group and also had neural sources in the ACC. ERN/Ne amplitude was related to post-error slowing across age groups; it was related to task performance only in the adult group. These findings are discussed in light of the role of the maturation of the ACC in the development of action monitoring processes.

Introduction

Adolescence is a developmental period during which there are significant changes in cognitive control processes. These changes are accompanied by important maturational changes in brain structures that connect with the prefrontal frontal cortex (PFC) (Lewis, Cruz, Eggan & Erikson, 2004; Luna & Sweeney, 2004; Luna, Thulborn, Munoz, Merriam, Garver, Minshew, Keshavan, Genovese, Eddy & Sweeney, 2001; Spear, 2000). One of these brain structures is the anterior cingulate cortex (ACC) (Casey, Trainor, Giedd, Vauss, Vaituzis, Hamburger, Kozuch & Rapoport, 1997), a neural structure in the medial wall of the PFC that plays an important role in conflict monitoring and error detection as well as signalling the need to recruit or reallocate attention. Several electrophysiological studies conducted in adults have identified event-related potentials (ERP) components that have neural generators in the vicinity of the ACC and that appear to index cognitive control sub-processes such as conflict monitoring or error detection (Dehaene, Posner & Tucker, 1994; Falkenstein, Hohnsbein, Hoormann & Blanke, 1991; Gehring, Coles, Meyer & Donchin, 1995; Gehring, Goss, Coles, Meyer & Donchin, 1993; van Veen & Carter, 2002b). Examining the development of these ERP components can help elucidate some of the brain mechanisms underlying cognitive control processes in adolescence.

One of these ERP components is the error-related negativity (ERN) or error negativity (Ne). The ERN/Ne is a response-locked sharp negative deflection occurring between 50 and 150 ms after the execution of an incorrect response in a simple choice reaction time task such as the Eriksen flanker task (Eriksen & Eriksen, 1974). The amplitude of this negativity is maximally distributed at centro-medial sites and is larger when response accuracy is emphasized over speed (Gehring *et al.*, 1993). The ERN/Ne seems to be evoked regardless of stimulus or response modality (Holroyd, Dien & Coles, 1998). Source localization studies, which are mathematical computations estimating the location of the ERP neural generators, point to the medial frontal cortex, in the vicinity of the ACC, as the neural generator of the ERN/Ne (Dehaene *et al.*, 1994). Involvement of the ACC in the monitoring of conflict and errors has also been confirmed by functional neuroimaging studies (Carter, Braver, Barch, Botvinick, Noll & Cohen, 1998; Kiehl, Liddle & Hopfinger, 2000).

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The ERN/Ne is generally followed by the error positivity (P_E). Relatively little is known about the functional significance of the P_E. The P_E is a slow positive deflection in the EEG that reaches its maximum between 200 and 400 ms after the subject makes an error (Falkenstein, Hoormann, Christ & Hohnsbein, 2000; Nieuwenhuis, Ridderinkhof, Blom, Band & Kok, 2001). The topography of the P_E is quite diffuse, but appears to be concentrated mostly at centro-parietal sites (Falkenstein *et al.*, 2000). It is currently thought to reflect subjective/emotional aspects of error assessment processes, which seem to be modulated by the meaning of having committed an error (Falkenstein *et al.*, 2000; Overbeek, Nieuwenhuis & Ridderinkhof, 2005). This conceptualization of the P_E is consistent with source localization analyses suggesting that the neural generators of the P_E are located in the vicinity of the rostral ACC, which is thought to be linked to affective processes (Falkenstein *et al.*, 2000). Thus, although both the ERN/Ne and the P_E are related to error responses, the P_E is considered to be independent of the ERN/Ne (Falkenstein *et al.*, 2000) and may be related to some affective appraisal of the error.

Theories of the functional role of the ERN/Ne have centered around the notion that the ERN/Ne reflects cognitive control mechanisms involved in monitoring processes (Bush, Luu & Posner, 2000; Falkenstein *et al.*, 2000). One theory proposes that the ERN/Ne reflects error monitoring (for a review see, Falkenstein, 2004). It is based on the premise that individuals tend to activate and execute a response before stimulus processing is complete (Coles, Gratton, Bashore, Eriksen & Donchin, 1985). This theory has been extended by combining the mismatch between correct and erroneous responses with reinforcement learning principles whereby the ERN/Ne would reflect a learning signal that is transmitted to the ACC from the basal ganglia via the mesencephalic dopamine system to modify ongoing task performance (Holroyd & Coles, 2002). These theoretical views attribute an 'error detection' role to the ERN/Ne. However, they do not elaborate on the role of the ERN/Ne with regard to the mechanisms of cognitive control processes.

An alternative theoretical perspective that attempts to characterize the functional role of the ERN/Ne in terms of mechanisms of cognitive control processes is the 'conflict monitoring' theory (Botvinick, Cohen & Carter, 2004; Braver, Barch & Carter, 2001; Carter *et al.*, 1998; Yeung, Botvinick & Cohen, 2004). According to this theory, the ERN/Ne indexes action monitoring processes, which are evaluative cognitive control sub-processes that serve to detect unwilled, unintentional behavioral responses such as errors or the occurrence of response conflict or 'crosstalk' between incompatible responses (Braver *et al.*, 2001). From this perspective, errors are considered to reflect a high level of response conflict (for a review see, Yeung *et al.*, 2004). The detection of conflict between incompatible responses (such as between correct and incorrect responses when errors occur) is thought to serve to alert higher-order and more specialized cognitive control processes to come on-line to adjust performance (Botvinick *et al.*, 2004; Braver *et al.*, 2001; van Veen & Carter, 2002a; Yeung *et al.*, 2004). This 'conflict monitoring' view of the ERN/Ne has been supported by functional neuroimaging studies showing co-activation of the ACC during errors and during response conflict (Carter *et al.*, 1998; Kerns, Cohen, MacDonald, Cho, Stenger & Carter, 2004; Ursu, Stenger, Shear, Jones & Carter, 2003; van Veen & Carter, 2002b).

Some researchers have identified a stimulus-locked ERP-N2 in high-conflict correct trials that may also reflect response conflict monitoring. Van Veen and Carter (2002) recorded high-density ERP components in a version of the Eriksen flanker task (Eriksen & Eriksen, 1974). Through source localization analyses, they found that the ACC was active following errors on the task, but also preceding correct responses on incongruent trials, which is what would be expected according to the conflict monitoring model. In this study, the N2 was described as a negative deflection in the EEG occurring approximately 250 ms following correct responses on incongruent trials and having frontocentral scalp topography, similar to the ERN/Ne (Liotti, Woldorff, Perez & Mayberg, 2000; Smid, Jakob & Heinze, 1999). Van Veen *et al.* (2002b) interpreted their findings as indicating that both the ERN/Ne and the N2 reflect conflict monitoring processes, with the ERN/Ne reflecting conflict monitoring occurring after an error has occurred and the N2 reflecting conflict monitoring before correct responses on high conflict trials such as incongruent flanker trials. Taken together these results suggest that the ERN/Ne and N2 might reflect similar cortical mechanisms (Kopp *et al.*, 1996) or more general conflict monitoring processes (Donkers & van Boxtel, 2004).

Traditionally, the N2 has not been associated with conflict monitoring but rather with response inhibition (Falkenstein, Hoormann & Hohnsbein, 1999; Garavan, Ross, Murphy, Roche & Stein, 2002; Kok, 1986; Pfefferbaum, Ford, Weller & Kopell, 1985). The interpretation of the N2 as reflecting response inhibition stems from results from multiple studies that have identified an N2 using the go/no-go task, frequently used to investigate inhibitory control. The go/no-go task consists of having subjects produce a speeded response to one type of stimulus (go trials) and withold the response to another type of stimulus (no-go trials) (Pfefferbaum *et al.*, 1985). On the go/no-go task, the N2 can be detected approximately 200–300 ms following stimulus onset and is usually greater

in no-go compared to go trials (Eimer, 1993; Fallgatter & Strik, 1999; Kok, 1986; Pfefferbaum *et al.*, 1985). Most of the localization studies conducted with the go/no-go task have localized the neural generators of the N2 to the inferior and lateral frontal regions (Kiefer, Marzinzik, Weisbrod, Scherg & Spitzer, 1998; Lavric, Pizzagalli & Forstmeier, 2004). In another localization study, Bokura, Yamaguchi and Kabayashi (2001) found both ACC and ventral PFC as plausible loci of the N2, suggesting that both the ACC (conflict monitoring) and PFC (response inhibition) systems may be involved in the generation of the N2. These results support findings from Lavric *et al.* (2004) suggesting that the N2 may reflect both conflict monitoring and inhibition. The focus of the present study does not aim at settling the controversy about the functional role of the ERN/Ne or the N2, but rather tries to understand how these action monitoring ERP components develop in adolescence and to what extent developmental changes in these ERP components may be related to the maturation of the ACC and changes in cognitive control processes.

Overall, the studies reviewed above indicate that the ACC is a key structure in monitoring behavioral responses in cognitive tasks. The ACC has rich connections to prefrontal regions and to limbic subcortical areas, which is why it is considered a transitional cortex interfacing motivation, cognition, and action (Devinsky & Luciano, 1993; Devinsky, Morrell & Vogt, 1995; Vogt & Pandya, 1987). Recent physiological and anatomical research suggests that, like the PFC, the ACC undergoes considerable maturational changes from childhood into early adulthood (Casey *et al.*, 1997; Cunningham, Bhattacharyya & Benes, 2002). For instance, neuroimaging studies show that differences between children and adults in the size of the ACC may correspond to more efficient controlled attention processes (Casey *et al.*, 1997). In addition to changes in the size of the ACC between childhood and adulthood, there is evidence suggesting changes in the level of activation of the ACC from childhood to early adulthood (Adleman, Menon, Blasey, White, Warsofsky, Glover & Reiss, 2002). Given evidence from electrophysiological studies conducted with adults regarding the role of the ACC in action monitoring processes, it is possible that the ERN/Ne, N2 and P_E may index maturational changes of these processes through adolescence into adulthood. To our knowledge, very few developmental studies have been conducted to investigate this issue.

Recent studies have started to examine the development of the ERN/Ne, N2 and the P_E (Davies, Segalowitz & Gavin, 2004; Ladouceur, Dahl & Carter, 2004). Davies *et al.* (2004) examined the development of the ERN/Ne and the P_E in a large normative sample of children, adolescents, and adults between 7 and 25 years of age. The ERP components were measured during a 480-trial letter flanker task (Eriksen & Eriksen, 1974). The results of this study showed that although there were no significant changes in P_E amplitude related to age, there were, however, significant linear and quadratic age effects in ERN/Ne amplitude. These age changes interacted with gender. The amplitude of the ERN/Ne was lowest at age 10 for girls and at age 13 for boys and gradually increased through adolescence for both groups. Overall, results indicated that ERN/Ne amplitude did not reach adult levels until mid to late adolescence. These results were consistent with previous preliminary findings (Ladouceur *et al.*, 2004). In a preliminary study, we used a cross-sectional design to compare the ERN/Ne and the P_E across a group of younger and older adolescents and showed that although the P_E was present early in adolescence the ERN/Ne did not seem to develop until late adolescence. We also measured the N2 component and showed that like the ERN/Ne, the N2 did not seem to develop until late adolescence (Ladouceur *et al.*, 2004). More recently, in addition to investigating the normal development of the ERN/Ne and P_E from adolescence into adulthood, Hogan, Vargha-Khadem, Kirkham and Baldeweg (2005) also examined the influence of increasing task complexity on these components using two visual forced-choice response tasks (Hogan *et al.*, 2005). Their findings showed that there were no significant group differences in ERN/Ne amplitude in the simple condition but that ERN/Ne amplitude was increased in adults compared to adolescents in the more difficult task condition (i.e. 4-CR). The authors interpreted these findings as an indication that the neural structures eliciting the ERN/Ne are present and 'adult-like' in adolescents but appear less mature when challenged by increasing task difficulty.

Although developmental studies interpreted their results in terms of maturational changes in the ACC, none of these included source localization analyses. Therefore, the primary goal of the current study was to investigate the location of the neural generators of the ERN/Ne, P_E and the N2 by conducting source localization analyses and to examine if there were any developmental differences. Furthermore, few studies have examined the relationship between the ERN/Ne and the N2 from a developmental perspective. Therefore, the secondary goal of this study was to examine the development of the N2 in parallel with the ERN/Ne. Thus, in this study the amplitudes of the ERN/Ne, P_E, and N2 were measured while participants performed an arrow version of the Eriksen flanker task (Eriksen & Eriksen, 1974). In this task, participants were required to be fast and accurate while making a discriminative response to the orientation of a target arrow stimulus flanked by distracting arrows

pointing either in the same direction (congruent condition, e.g. →→→→→) or in the opposite direction (incongruent condition, e.g. ←←→←←). Given Van Veen *et al.*'s (2002) findings localizing the N2 on the flanker task to the ACC, we expected that maturation of the ACC during adolescence would translate into an increase in the amplitude of the ERN/Ne and N2 and that neural generators of these components would be localized in the vicinity of the caudal ACC. Based on the results from previous studies (Davies *et al.*, 2004; Ladouceur *et al.*, 2004), we did not expect any group differences in the amplitude of the P_E.

Another goal of this study was to examine the extent to which development of the ERN/Ne plays a role in modulating cognitive control processes. Slowing down immediately after an incorrect response (also referred to as post-error slowing) is a well-known indicator of behavioral adjustment on a particular task (Rabbitt, 1966). Such behavioral adjustment can also be considered to reflect the recruitment of cognitive control processes, which are supported by the PFC (Braver *et al.*, 2001). Thus, if the ERN/Ne does trigger cognitive control processes, as predicted by the conflict monitoring theory, we should observe greater ERN/Ne amplitude on error trials that are immediately followed by correct trials with slower reaction times (slow post-error slowing). Therefore, we hypothesized that greater ERN/Ne amplitude would be associated with slow post-error slowing and that this effect would be stronger with age. Another approach to investigating the modulating role of the ERN/Ne in cognitive control processes is to examine whether developmental changes in ERN/Ne amplitude are related to performance. Thus, we examined whether ERN/Ne amplitude varied according to performance and whether this effect was influenced by age. We predicted that ERN/Ne amplitude would increase in subjects with better performance and that this effect would be stronger as a function of age.

Method

Participants

The sample comprised a total of 46 participants between 9 and 50 years old, who were sub-divided into three groups: early adolescents ($n = 15$, mean age = 12.36, SD = 1.59; age range: 8.66–13.83; eight girls), late adolescents: ($n = 15$, mean age = 16.53, SD = 1.70; age range: 14.08–18.95; 10 girls), and adults ($n = 16$, mean age = 28.74, SD = 10.27; age range: 19.08–49.83; 10 women). The adolescent groups were formed based on an age median-split. The median age also corresponds with the age at which there are significant changes in the development of cognitive control processes (see Luna & Sweeney, 2004). The majority of the participants (91%) were right-handed, as assessed by the Edinburgh Handedness Inventory (Oldfield, 1971). Participants were screened for neurological and psychiatric disorders, and for visual acuity. The University of Pittsburgh Institutional Review Board approved the study. To participate, participants under 18 years of age and their parents were required to sign assent and informed consent forms, respectively.

Task

EEG measures were taken while participants performed an arrow version of the Eriksen flanker task with congruent (i.e. →→→→→ and ←←←←←) and incongruent (i.e. ←←→←← and →→←→→) conditions (Eriksen & Eriksen, 1974). The five-arrow stimulus arrays were presented using E-prime (Psychological Software Tools, Pittsburgh, PA). Each trial started with the presentation of a central fixation cross '+'. Following the fixation cross, one of the four stimulus arrays (→→→→→, ←←←←←, ←←→←←, →→←→→) appeared on the computer screen. The probability of occurrence of each stimulus array was .25. Participants were asked to respond on a button box using their left index finger if the central arrow pointed to the left and their right index finger if the central arrow pointed to the right. In order to enhance the effect of the flankers with regard to priming incorrect responses in the incongruent condition, the flanker stimuli appeared 100 ms prior to the target stimulus, which appeared in the same location as the fixation cross. Flankers and the central arrow remained on the screen until a response was made after which they disappeared simultaneously and a fixation cross appeared, which indicated the intertrial interval. The intertrial interval was randomized between 500 and 1500 ms. Each block of trials began with a fixation cross having a duration of 3000 ms.

Procedure

Each participant was seated 0.5 m directly in front of a computer monitor. After a description of the experiment, participants were given detailed task instructions. They were told that they were going to see a series of trials with five arrows appearing on the computer screen and that they had to press the left button with their left index finger if the arrow in the middle pointed to the left and press the right button with their right index finger if the arrow in the middle pointed to the right. They were also told to respond as quickly and as accurately as possible. To ensure that participants remained motivated throughout the task, they were told at the beginning of the task that they could win a 'cash bonus' if they performed

extremely well on this task (all were told that they did very well and received an extra $5). After hearing the instructions read by the experimenter, participants were asked to repeat them back to the experimenter to ensure that they understood the instructions. They then proceeded to complete one block of 60 practice trials. The experimenter monitored participants' performance during the practice block and provided appropriate feedback.

Following the electrode net application, the experimenter asked participants to try not to move, grind their teeth, or blink too much. If they felt the need to blink, they could do so while the fixation cross was on the screen. Before starting the task, the experimenter reminded participants of the instructions and answered any questions. Participants received seven blocks of 120 trials (840 total trials) with each block initiated by the participant. Participants were permitted to rest between blocks. The percentage of correct responses was computed between blocks informing the experimenter of the participants' performance; this information was used to guide the experimenter in providing feedback to participants. It was not made available to participants. If the percentage of correct responses was above .95, the experimenter reminded participants to be accurate but to go faster. If the percentage of errors was below .80, the experimenter reminded participants to go fast but to be more accurate. Once testing was complete, participants were briefed on the purpose of the experiment.

EEG data acquisition and processing

The EEG data were recorded with a 128-channel Geodesic Sensor Net and analyzed using EGI software (EGI, Eugene, OR). The EEG was recorded with a 0.1–100 Hz bandpass filter (3dB attenuation). The EEG signal was digitized at 250 Hz with a 16 bit A/D converter. During data acquisition, channels were referenced to channel 129 (Cz) and re-referenced, prior to data analyses, to the average of all of the sensors at each time point. Impedances were measured prior to and following EEG recording. Before recording, impedances were below 50 kΩ. Channels that had impedances above 100 kΩ at the end of the recording were noted and visually inspected during the data processing phase (i.e. bad channel replacement).

EEG data processing involved the following steps. EEG data were digitally filtered using a 0.3–30 Hz bandpass filter. The EEG was segmented −400 to +800 ms and divided according to correct and error trials. Artifacts were screened with automatic detection methods (Net Station, Electrical Geodesics, Inc.). In particular, segments containing eye blink and eye movement artifacts (70 μV threshold), signals exceeding 200 μV, and fast transits exceeding 100 μV were excluded from the averaging. Bad channel data were replaced using spherical spline interpolation of neighboring channel values (Perrin, Bertrand & Pernier, 1987). Analyses conducted to examine whether there were any age-related differences in the number of EEG segments excluded due to artifact (i.e. eye blinks, eye movements, or number of bad channels) did not yield any significant group differences.

EEG data reduction and analyses

To quantify the response-locked ERN/Ne and P_E, averages of peak amplitudes were computed separately for correct and erroneous trials for each age group. To quantify the stimulus-locked N2, averages were computed separately for congruent and incongruent correct trials for each age group. The data were then baseline corrected, which involved subtracting from each data point after response onset the average activity in the 150-ms window (−150 to −50 ms) prior to response onset. For the N2, baseline correction involved subtracting from each data point after stimulus onset the average activity in the 150-ms window prior to stimulus onset. The ERN/Ne was defined as the maximal peak negative amplitude in the window 0–120 ms following response onset. The P_E was defined as the maximal peak positive amplitude in the window 120–420 ms following response onset. The N2 was defined as the maximal peak negative amplitude in the window 250–370 ms following the onset of the central arrow.

In order to examine whether ERN/Ne amplitude was related to post-error slowing, error trials were binned according to fast or slow post-error slowing. This was accomplished by first calculating for each participant the median RT on correct-response trials that followed error trials and then binning error trials according to below-median RT (fast post-error slowing) or above-median RT (slow post-error slowing) on those correct-response trials.

Statistical analyses

Statistical analyses were performed on the behavioral and ERP measures using SPSS (version 12.0.1). Specifically, behavioral measures were analyzed using a mixed MANOVA model with age group (early adolescents, late adolescents, adults) as between-subject variable and trial type (congruent, incongruent) and response type (correct, error) as within-subject variables. ERP measures were analyzed using a mixed MANOVA model with age group (early adolescents, late adolescents, adults) and electrode site (Fz, Cz, Pz), response type (correct, error), or post-error slowing (fast, slow) as within-subject variables, as appropriate. The multivariate test statistic reported is Wilks' lambda. Greenhouse-Geisser correction

was applied upon any violations of the assumption of sphericity. Follow-up analyses included univariate analyses of variance and *t*-tests with Bonferonni correction.

Source localization analyses

The locations of the neural generators for the ERN/Ne, P_E and N2 were modeled using Brain Electrical Source Analysis software (BESA 5.0.8; MEGIS Software, Munich, Germany), a software program that allows iterative estimation of the dipole location and orientation in a four-shell, spherical head model with ellipsoidal correction until difference between the recorded surface data and the calculated surface data of the dipole-model is minimized (least square fit) (Berg & Scherg, 1994; Scherg, 2003). The mean residual variance (RV) provides an index of the goodness of fit of the model. Because bone conductivities are age dependent and not equal for children between 5 and 15 years of age, we included in the model estimates (obtained from MEGIS software) for bone thickness and conductivity according to age. Bone thickness of 6 mm and 6.7 mm and bone conductivity of 0.01 and .006 were used for the early and late adolescent groups, respectively. Default bone conductivities were used for the adult group (i.e. bone thickness: 7 mm and bone conductivity: 0.0042). For the early adolescent group, source localization was performed on the onset to the peak maximum of the difference waveforms for the P_E only as they did not show any ERN/Ne peak to include in the model.

The BESA procedure (Scherg & Berg, 1996) was applied to the difference waveforms grand averages of the ERN/Ne, P_E and N2. Difference waveforms were created by subtracting the signal elicited on correct trials from the signal elicited on error trials for the ERN/Ne and the P_E and by subtracting the signal elicited on congruent trials from the signal elicited on incongruent trials for the N2. The difference waveforms were fitted from the onset to the maximal peak using a sequential strategy. The appropriateness of the solutions was tested with the techniques outlined by Scherg and Berg (1996). These include, for example, verifying the consistency of the results by repeating the procedure with randomly seeded initial dipole configurations, by comparing the solution with plausible alternatives, and by assessing the stability of the solution by including additional dipoles.

Results

Behavioral measures: RTs and accuracy

Analyses conducted on reaction times (RTs) indicated a main effect of age group, $F(2, 40) = 4.75$, $p < .05$, and response type, $F(1, 40) = 84.20$, $p < .001$, and a significant interaction of trial type by response type, $F(1, 40) = 88.09$, $p < .001$. Subsequent independent *t*-tests were carried out between the early and late adolescent groups, $t(28) = 2.94$, $p < .01$, between the early adolescent and adult groups, $t(29) = 2.41$, $p < .05$, and between the late adolescent and adult groups, $t(29) = -.90$, $p = .36$, indicating that the late adolescent and adult groups had significantly faster RTs compared to the early adolescent group. Paired *t*-test results between correct and error trials indicated as expected that error RTs were significantly faster than correct RTs, $t(45) = 15.25$, $p < .001$ (see Table 1).

Table 1 *Means and standard deviations for the early adolescent, late adolescent, and adult age groups on various behavioral performance measures*

	Age groups		
Variables	Early adolescents ($n = 15$)	Late adolescents ($n = 15$)	Adults ($n = 16$)
Reaction time			
Overall*	485.86[a]	392.34[b]	406.08[b]
	(118.96)	(32.22)	(50.17)
Correct	500.07	402.04	415.26
	(119.12)	(32.88)	(48.62)
Incorrect	408.15	314.52	334.16
	(126.86)	(44.06)	(71.06)
Congruent	456.02	365.32	373.82
	(108.95)	(31.59)	(44.27)
Incongruent	515.70	419.37	438.34
	(130.23)	(34.28)	(57.05)
Percentage of errors			
Overall*	11.31[a]	7.21[b]	7.65[c]
	(4.43)	(3.07)	(4.52)
Congruent	4.46	1.25	1.97
	(3.12)	(1.88)	(1.99)
Incongruent	18.16	13.17	13.32
	(6.84)	(4.87)	(7.53)
Post-error slowing	24.01	18.09	26.92
	(50.33)	(23.56)	(43.66)

Note: * $p < .05$; [a,b,c] Means with different letters in their superscripts differ at .05 or better. Post-error slowing: post-error minus post-correct trial reaction time.

Accuracy was determined by the percentage of errors. As shown in Table 1, there were age, $F(2, 43) = 4.60$, $p < .05$, and trial type main effects, $F(1, 43) = 228.97$, $p < .001$. Post-hoc independent *t*-tests that were carried out between the early and late adolescent groups, $t(28) = 2.94$, $p < .01$, between the early adolescent and adult groups, $t(29) = 2.27$, $p < .05$, and between the late adolescent and adult groups, $t(29) = -.31$, $p = .76$, indicated that the late adolescent and adult groups had significantly fewer errors compared to the early adolescent group. Paired *t*-tests between congruent and incongruent trials showed a greater percentage of errors in the incongruent trials, $t(45) = -15.19$, $p < .001$.

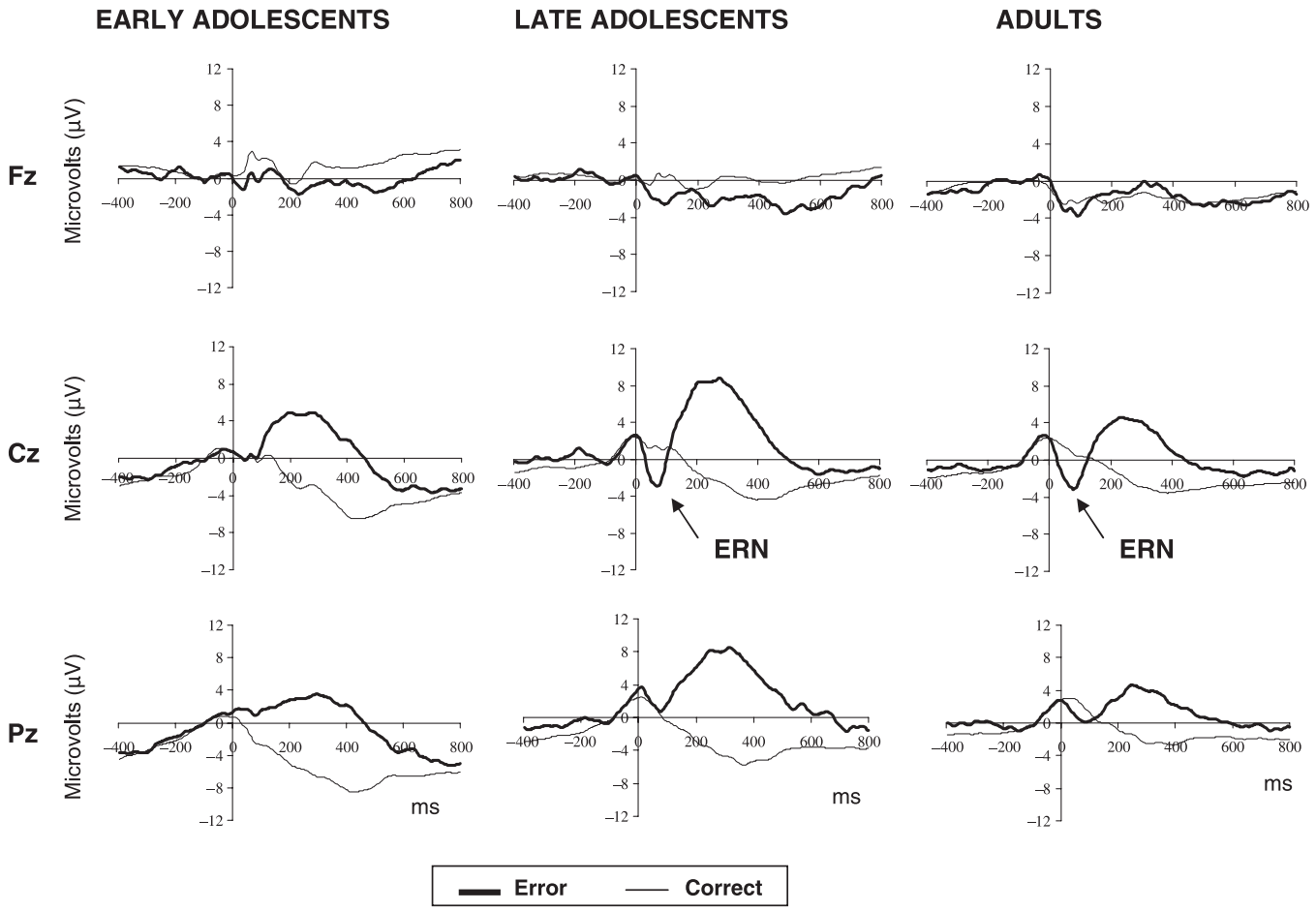

Figure 1 *Grand averaged response-locked waveforms for the early adolescent, late adolescent, and adult groups.*

With regard to post-error slowing, across age groups the average RTs for correct trials following error trials were significantly longer than the average RTs for correct trials following correct trials, $F(1, 43) = 14.56$, $p < .001$. The age group by response type interaction was not significant, suggesting that post-error slowing was similar across groups.

Additional correlational analyses were conducted to examine whether there were any speed–accuracy trade-off effects. Partial correlations (controlling for age) were conducted between the overall percentage of errors and correct RTs ($r(43) = .18$), and the percentage of errors and incorrect RTs ($r(43) = .13$). Results are consistent with the absence of overall speed–accuracy trade-offs.

Response-locked ERP components: error-related negativity (ERN/Ne) and error positivity (P_E)

Figure 1 shows the response-locked ERP components at Fz, Cz, and Pz for correct and error trials for the three age groups. As clearly shown, the late adolescent and adult groups showed a negative deflection – the ERN/Ne – that began immediately following incorrect responses and that peaked approximately 50–70 ms later. The age group by response type by electrode site interaction was significant, $F(4, 84) = 6.78$, $p < .01$. Follow-up age group by response type mixed ANOVA models conducted at each of the electrode sites indicated that ERN/Ne amplitude was significantly maximal at the Cz electrode site, $F(2, 43) = 5.69$, $p < .01$, and not at the other sites. Post-hoc paired *t*-tests comparing amplitude measures in correct and error trials at the Cz electrode site for each of the age groups revealed that ERN amplitude was significantly greater on error trials for the late adolescent and adult groups, $t(14) = 4.4$, $p < .01$, and $t(15) = 3.9$, $p < .01$, respectively, but not for the early adolescent group, $t(14) = 0.39$, $p = .70$.

As shown in Figure 1, all three age groups showed a positive deflection – the P_E – that peaked approximately 230–290 ms following incorrect responses. A separate mixed MANOVA model was performed to examine group differences in P_E amplitude. We found a significant

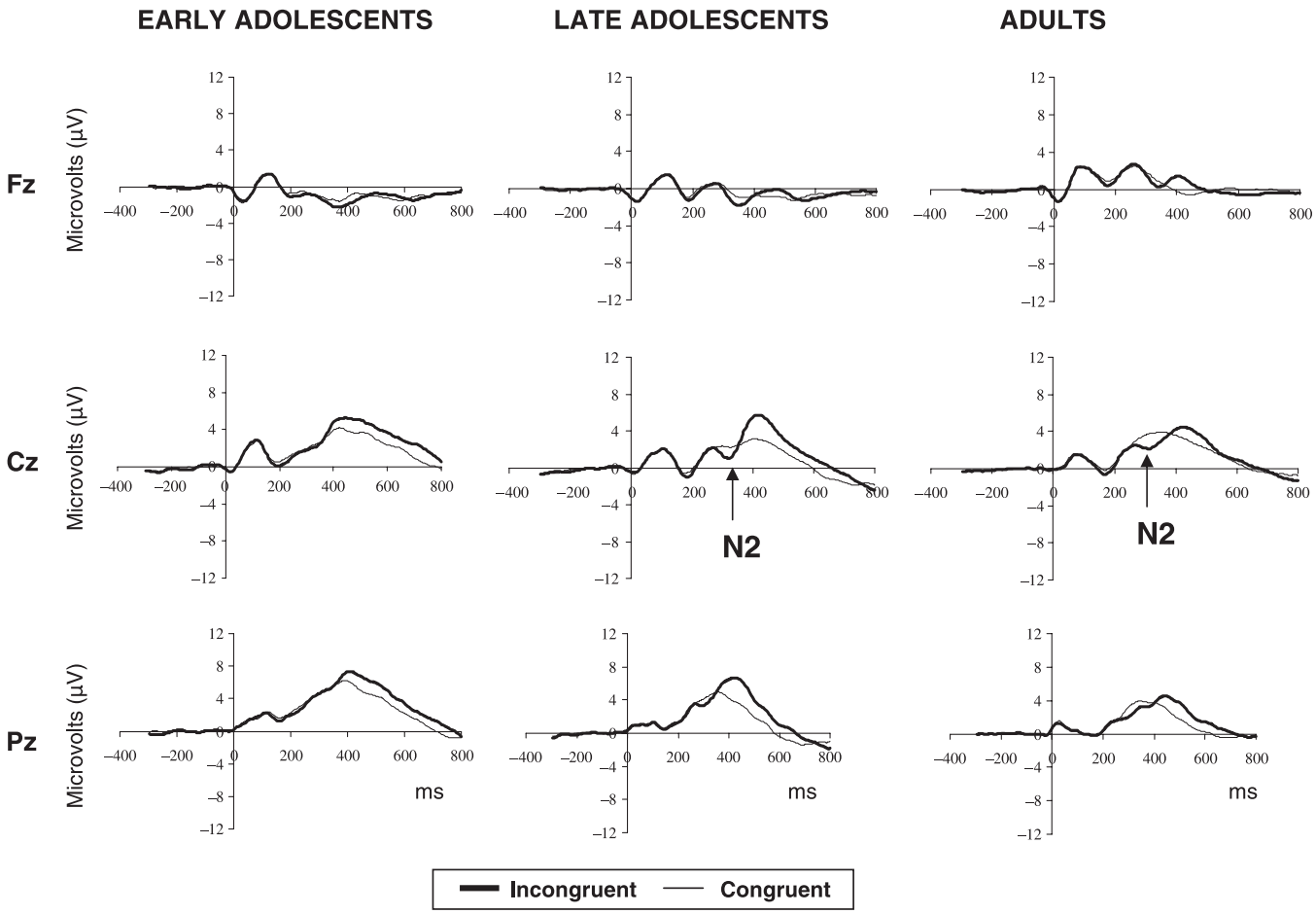

Figure 2 *Grand averaged stimulus-locked waveforms for the early adolescent, late adolescent, and adult groups.*

main effect for electrode site, $F(2, 42) = 33.91$, $p < .001$. Post-hoc comparisons indicated that P_E amplitude was significantly maximal at Cz and Pz sites followed by Fz, $p < .001$, indicating that the P_E had a central-parietal distribution. The age group by electrode site interaction was also significant, $F(4, 84) = 3.37$, $p < .05$. Post-hoc independent *t*-tests indicated that the late adolescent group had a significantly greater P_E than the adult group at both the Cz and Pz electrode sites (Cz: $t(29) = 2.63$, $p < .05$; Pz: $t(29) = 2.45$, $p < .05$). They also had significantly greater P_E than the early adolescent group at the Pz site, $t(29) = -2.76$, $p < .05$.

Stimulus-locked ERP: N2

Figure 2 shows stimulus-locked ERP components at Fz, Cz, and Pz for congruent and incongruent trials for the three age groups. As is clearly visible, the late adolescent and adult groups showed a negative deflection – the N2 – in incongruent trials that peaked approximately 290–320 ms following stimulus onset. Like the ERN/Ne, the age group by response type by electrode site interaction was significant, $F(4, 84) = 2.51$, $p < .05$. Follow-up age group by response type mixed ANOVA models conducted at each of the electrode sites indicated that ERN/Ne amplitude was significantly maximal at the Cz electrode site, $F(2, 43) = 3.9$, $p < .05$, and not at the other sites. Post-hoc paired *t*-tests comparing amplitude measures in correct and error trials at the Cz electrode site for each of the age groups revealed that ERN amplitude was significantly greater on error trials for the late adolescent and adult groups, $t(14) = 3.2$, $p < .01$ and $t(15) = 3.7$, $p < .01$, respectively, but not for the early adolescent group, $t(14) = 0.93$, $p = .37$.

Source localization

ERN/Ne and P_E

For both the adult and late adolescent groups, a two-dipole model was obtained. Consistent with previous studies (Dehaene *et al.*, 1994; van Veen & Carter, 2002b),

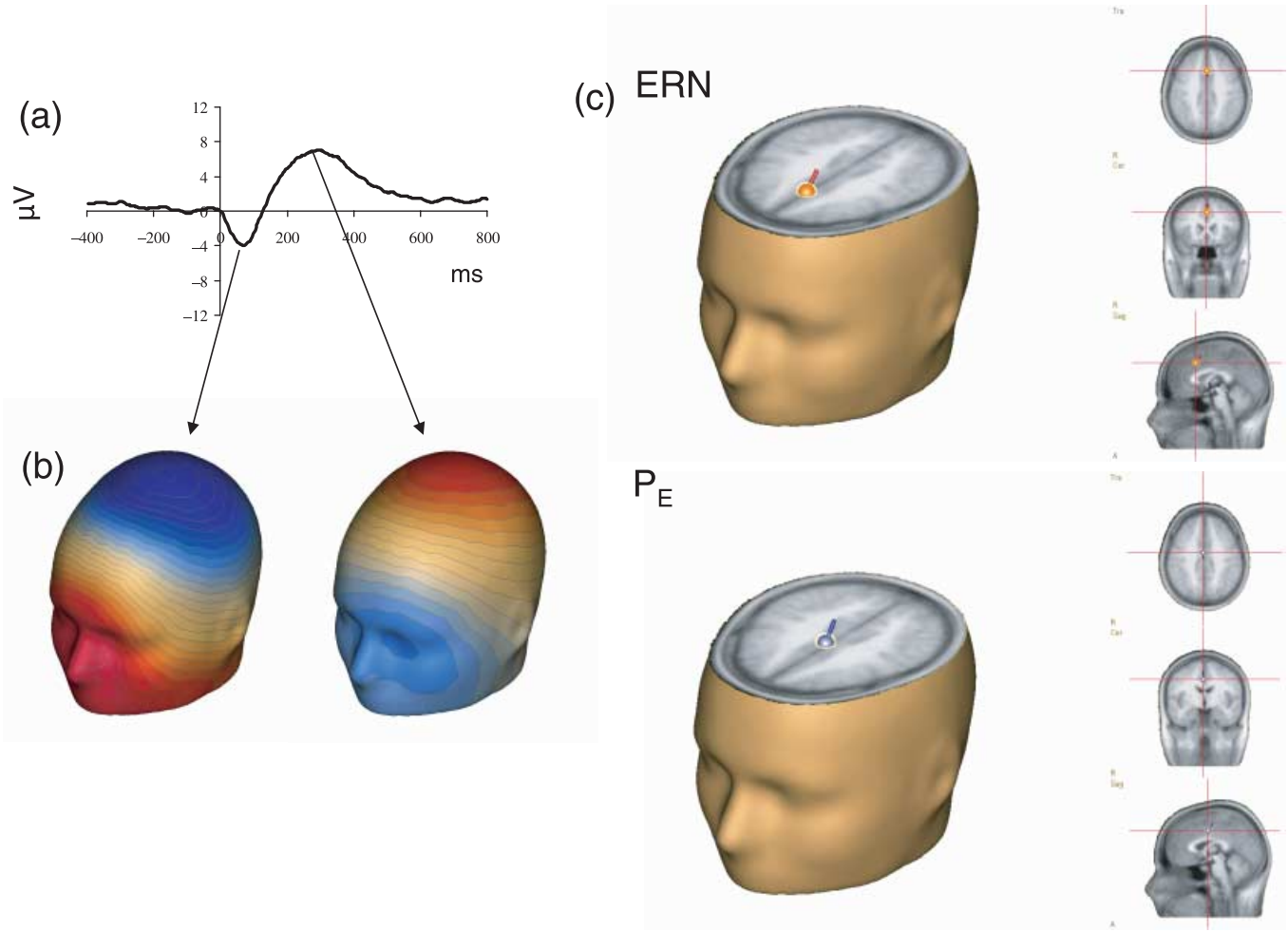

Figure 3 *Adult ERN and P_E: a) difference waveform voltage plots, b) three-dimensional maps illustrating the scalp distribution of the difference waveform at the maximal peaks, c) dipole source models (large) with transversal, coronal, and sagittal views of dipole (small) superimposed on a standard realistic MR-based head model. Note: blue = more negative; red = more positive.*

one dipole located in the vicinity of the ACC was able to account for a large part of the variance in the observed data for the ERN/Ne in the adult group (ERN/Ne location: $x = -0.02$, $y = 0.39$, $z = 0.40$, Cartesian US; residual variance (RV): 5.9%) (see Figure 3) and the late adolescent group (ERN/Ne location: $x = 0.01$, $y = 0.39$, $z = 0.38$; RV: 12.4%) (see Figure 4). While holding constant the location and the orientation of the ERN/Ne dipole, a second dipole located in the vicinity of the ACC but posterior to the ERN/Ne was obtained for the adult group (P_E location: $x = 0.03$, $y = 0.15$, $z = 0.43$; RV: 4.9%) (see Figure 3) and the late adolescent group (P_E location: $x = -0.03$, $y = 0.15$, $z = 0.53$; RV: 2.97%) (see Figure 4). For the early adolescent group, a single dipole located in the posterior region of the ACC was able to account for a large part of the variance in the observed data for the P_E (location: $x = -0.05$, $y = 0.13$, $z = 0.36$; RV: 3%) (see Figure 5). Other dipoles were added to the models but these localized either to the eye regions or outside of the brain, suggesting that the residual activity was due to noise and/or activity not related to the ERN/Ne or the P_E.

N2

For both the adult and late adolescent groups, a single dipole located in the vicinity of the ACC (similar region to the ERN/Ne) was able to account for most of the variance in the observed data for the N2 in the adult group (N2 location: $x = -0.02$, $y = 0.32$, $z = 0.36$; RV: 9.9%) (see Figure 6) and the late adolescent group (N2 location: $x = 0.08$, $y = 0.37$, $z = 0.31$; RV: 14.3%) (see Figure 7).

ERN/Ne and post-error slowing

Figure 8 shows the response-locked ERP components at the Fz, Cz, and Pz for the ERN/Nes that are followed

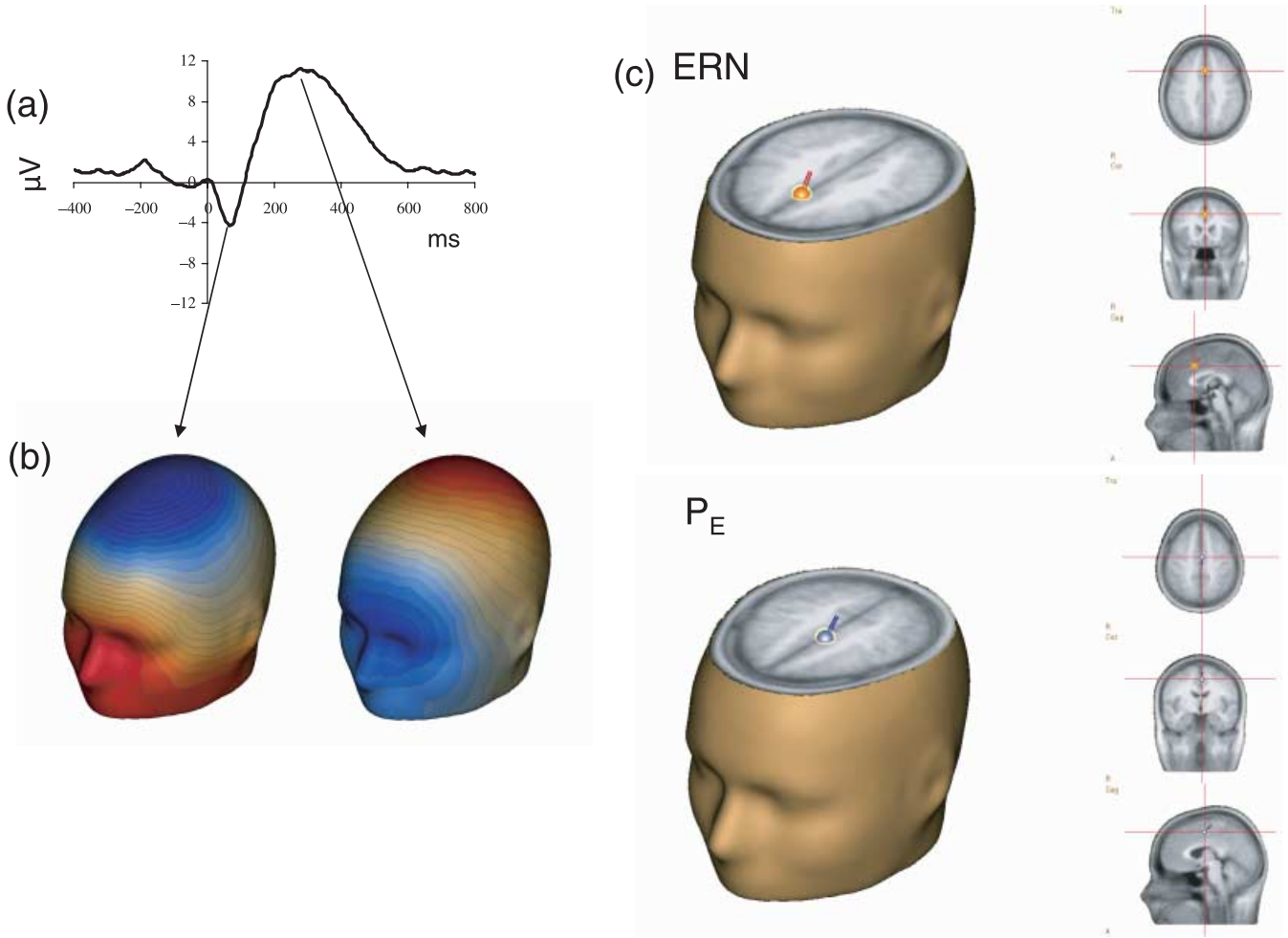

Figure 4 *Late Adolescent ERN and P_E: a) difference waveform voltage plots, b) three-dimensional maps illustrating the scalp distribution of the difference waveform at the maximal peaks, c) dipole source models (large) with transversal, coronal, and sagittal views of dipole (small) superimposed on a standard realistic MR-based head model. Note: blue = more negative; red = more positive.*

by fast post-error slowing and slow post-error slowing for the three age groups. As shown, ERN/Ne amplitude was more negative when followed by slow post-error slowing for all three groups. That is, ERN/Ne amplitude was more negative when RTs on correct trials following error trials were slow (slow post-error slowing) than when RTs on correct trials following error trials were fast (fast post-error slowing), which was supported by a significant main effect for response type, $F(1, 43) = 12.39$, $p < .01$. No other main effects or interactions were significant.

Role of the ERN/Ne in task performance

In order to examine whether developmental changes in ERN/Ne amplitude were related to task performance, we conducted regression analyses examining the extent to which ERN/Ne amplitude is related to performance in the late adolescent and adult groups. Results showed that, in adults, ERN/Ne amplitude significantly predicted performance, $\beta = .69$, $t(14) = 3.59$, $p < .01$, and also explained a significant portion of the variance, $R^2 = .48$, $F(1, 15) = 12.87$, $p < .01$. However, no such relationship was found in the late adolescent group, $\beta = .13$, $t(13) = .48$, $p = .64$.

Discussion

The goal of this study was to examine the development and neural sources of action monitoring ERP components as a way to elucidate some of the possible brain mechanisms underlying cognitive control processes in adolescence. As predicted, ERN/Ne and N2 amplitudes were significantly more negative in the late adolescent

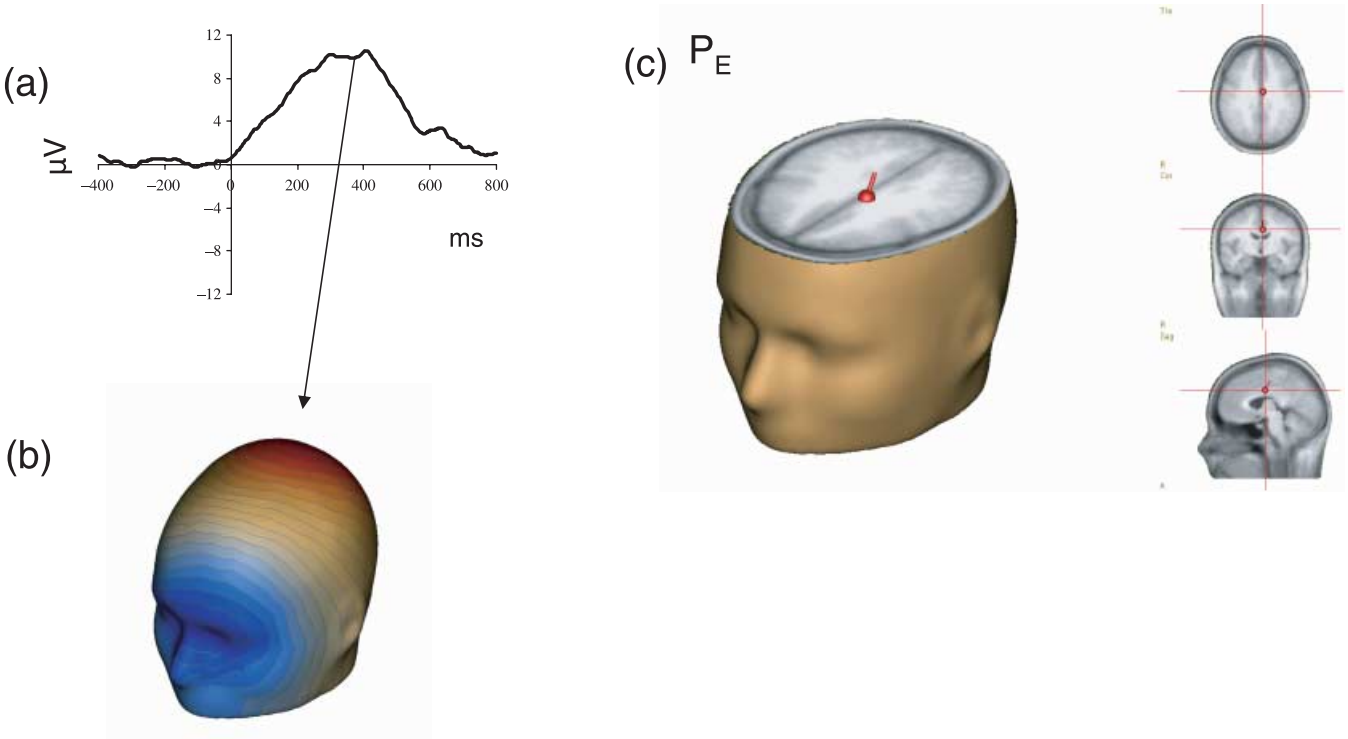

Figure 5 *Early Adolescent P_E: a) difference waveform voltage plots, b) three-dimensional maps illustrating the scalp distribution of the difference waveform at the maximal peaks, c) dipole source models (large) with transversal, coronal, and sagittal views of dipole (small) superimposed on a standard realistic MR-based head model. Note: blue = more negative; red = more positive.*

and adult groups compared to the early adolescent group. In addition, source localization results indicated that the neural generators of the ERN/Ne and the N2 for these groups were located in the vicinity of the ACC, suggesting that changes in these components during adolescence may be related to the maturation of the ACC. Although P_E was present across all age groups, P_E amplitude was more positive in the late adolescent group compared to the adult group at the Pz site. Source localization results showed that the neural generators of the P_E were located in the ACC for all three age groups but appeared somewhat more superior in the late adolescent group, which could possibly explain the differences in P_E for that group. With regard to the role of the ERN/Ne in cognitive control processes, results showed that ERN/Ne amplitude was more negative when RTs on correct trials following error trials were slow (slow post-error slowing) than when RTs on correct trials following error trials were fast (fast post-error slowing). However, contrary to our predictions, this effect did not vary with age. Moreover, results showed that ERN/Ne amplitude significantly predicted performance in the adult group, but not in the late adolescent group, suggesting that the influence of maturational changes in the ACC continue to develop into adulthood.

The significant increase in ERN/Ne amplitude in the late adolescent and adult groups compared to the early adolescent group is not surprising in light of consistent findings from previous studies showing increased ERN/Ne amplitude in mid to late adolescence using similar flanker tasks (Davies *et al.*, 2004; Ladouceur *et al.*, 2004; Santesso, Segalowitz & Schmidt, 2006). Nevertheless, these results serve to replicate previous findings and provide additional evidence indicating that the ERN/Ne is late to mature. The developmental findings for the N2 are novel but harder to reconcile because of the few studies that have examined the N2 on a flanker task from a developmental perspective. The fact that, like the ERN/Ne, N2 amplitude also increased across age groups is consistent with the view that the N2 may reflect pre-response conflict monitoring processes. However, this increase in N2 amplitude with age is inconsistent with findings from a recent developmental study of the N2 on an auditory go/no-go task which showed decreases in N2 amplitude with age (Johnstone, Pleffer, Barry, Clarke & Smith, 2005). Given the significant discrepancy between

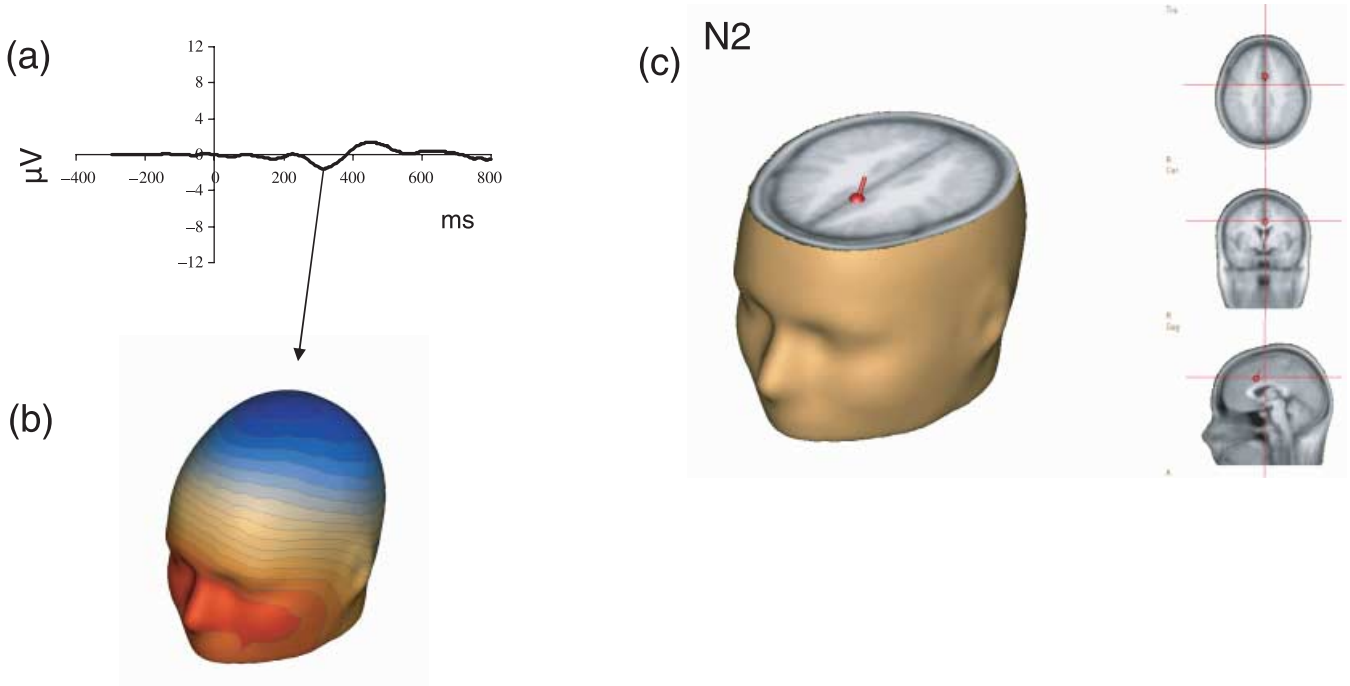

Figure 6 *Adult N2: a) difference waveform voltage plots, b) three-dimensional maps illustrating the scalp distribution of the difference waveform at the maximal peaks, c) dipole source models (large) with transversal, coronal, and sagittal views of dipole (small) superimposed on a standard realistic MR-based head model. Note: blue = more negative; red = more positive.*

the task used in the Johnstone *et al.* (2005) study and the current study (i.e. auditory go/no-go vs. visual flanker), it is difficult to interpret what these differences in N2 across studies actually mean. Moreover, the Johnstone *et al.* (2005) study compared children and adults and did not include adolescents. Our results are also in contrast to Jonkman *et al.* (2003) who found no effect of age on their N2 in a visual go/no-go task. While these results might suggest task-related differences regarding the development of N2, a developmental within-subject design comparing N2 (and other action monitoring ERP components) across the flanker and go/no-go tasks should be considered in the future in order to better understand the functional role of the N2 and its development.

To our knowledge, this study is the first to demonstrate that both the ERN/Ne and N2 appear to have neural generators in the ACC in adolescents, suggesting that changes in ERN/Ne and N2 amplitudes during adolescence may be related to the maturation of the ACC. Localizing the ERN/Ne to the ACC supports previous findings from studies conducted in adults (Dehaene *et al.*, 1994; Herrmann, Rommler, Ehlis, Heidrich & Fallgatter, 2004; Mathewson, Dywan & Segalowitz, 2005; Nieuwenhuis, Yeung, Wildenberg & Ridderinkhof, 2003; van Veen & Carter, 2002b). Localizing the N2 to the same region of the ACC as the ERN/Ne is consistent also with the results from previous studies in adults indicating that the N2 and the ERN share similar topography and neural source (Nieuwenhuis *et al.*, 2003; van Veen & Carter, 2002b; Yeung *et al.*, 2004). These results are inconsistent, however, with results from ERP and source localization studies of the N2 elicited in the go/no-go task (Bokura *et al.*, 2001; Kiefer *et al.*, 1998; Lavric *et al.*, 2004; Nakata, Inui, Wasaka, Akatsuka & Kakigi, 2005). The few studies that have examined the neural generators of the N2 on the go/no-go task reported source models mainly in the inferior and lateral regions of the PFC. For instance, Kiefer *et al.* (1998) reported bilateral sources in the inferior prefrontal areas and in the left premotor cortex or motor cortex. Bokura *et al.* (2001) found sources in the right lateral orbitofrontal cortex and using MEG, Nakata *et al.* (2005) recently localized MEG signals co-registered to N2 ERP activity to the posterior region of the inferior frontal sulci of the PFC. A recent study pointed out, however, that most of the source models of the N2 on the go/no-go task were derived from difference waveforms (i.e. ERP no-go trial minus ERP go trials) and as such, there is a possibility that these findings might have been confounded by motor- and attention-related activity from the go trials (Bekker, Kenemans & Verbaten, 2005). After controlling for these possible confounding factors, Bekker *et al.* (2005) derived an alternative source

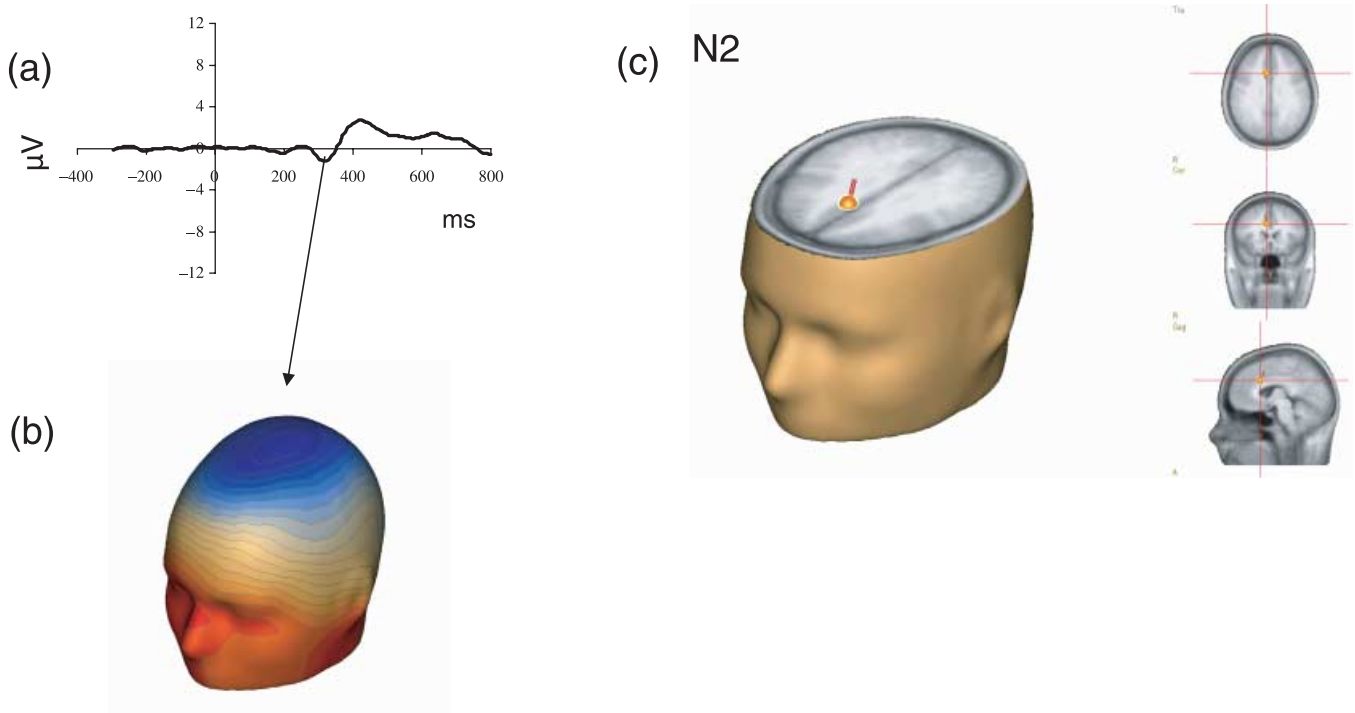

Figure 7 *Late Adolescent N2: a) difference waveform voltage plots, b) three-dimensional maps illustrating the scalp distribution of the difference waveform at the maximal peaks, c) dipole source models (large) with transversal, coronal, and sagittal views of dipole (small) superimposed on a standard realistic MR-based head model. Note: blue = more negative; red = more positive.*

model of the now 'pure' no-go-N2 and found a neural generator in the ACC, suggesting that ACC plays an important role in generating the N2 and that results from other source localizing studies may be contaminated by go-related activity (Bekker *et al.*, 2005). These findings would be in line with the conflict monitoring view that the N2 may reflect the detection of response conflict generated from competition between the execution and the inhibition of a single response. It is important to note, however, that given the low spatial resolution of the source analysis procedures used in ERP studies, it is possible that the ACC neural generators for the N2 elicited on flankers tasks and for the N2 elicitied on the go/no-go task may be localized in nearby but functionally different sub-regions of the ACC (Mathalon, Bennett, Askari, Gray, Rosenbloom & Ford, 2003). However, we conducted follow-up correlational analyses on ERN/Ne and the N2 amplitudes within the late adolescent and adult groups and found significant linear correlations between the ERN/Ne and N2 (late adolescents: Pearson $r(15) = .52$, $p < .05$; adults: Pearson $r(16) = .52$, $p < .05$)), suggesting that these components may indeed share similar neural generators.

Studies using fMRI have shown increases in the level of activation of the ACC from childhood to early adulthood (Adleman *et al.*, 2002). This increase in the level of activation may be related to stronger or more synchronous firing of neurons in the ACC, which could also explain the increase in ERN/Ne amplitude with age. Another possibility is that with age, the ACC becomes more sensitive to errors or response conflict. One way in which increased ACC sensitivity could influence efficiency of cognitive control processes is from greater connectivity between the ACC and the dorsolateral prefrontal region (DLPFC) given that activity between these two regions is highly correlated (Badre & Wagner, 2004; Carter *et al.*, 1998; Kerns *et al.*, 2004; Kiehl *et al.*, 2000). Increased ERN/Ne and N2 amplitude could also be the consequence of the size of the ACC, which has been observed to increase with age and to be related to more efficient attention control processes (Casey *et al.*, 1997). Future studies using concurrent ERP and fMRI technique and computational modelling would provide more insight into the functional significance of the increased ERN/Ne and N2 amplitude in adolescence as a function of ACC maturation.

In this study, we also attempted to address the question of the functional role of the ERN/Ne with respect to the development of cognitive control processes in two ways. The first was to examine the extent to which ERN/Ne

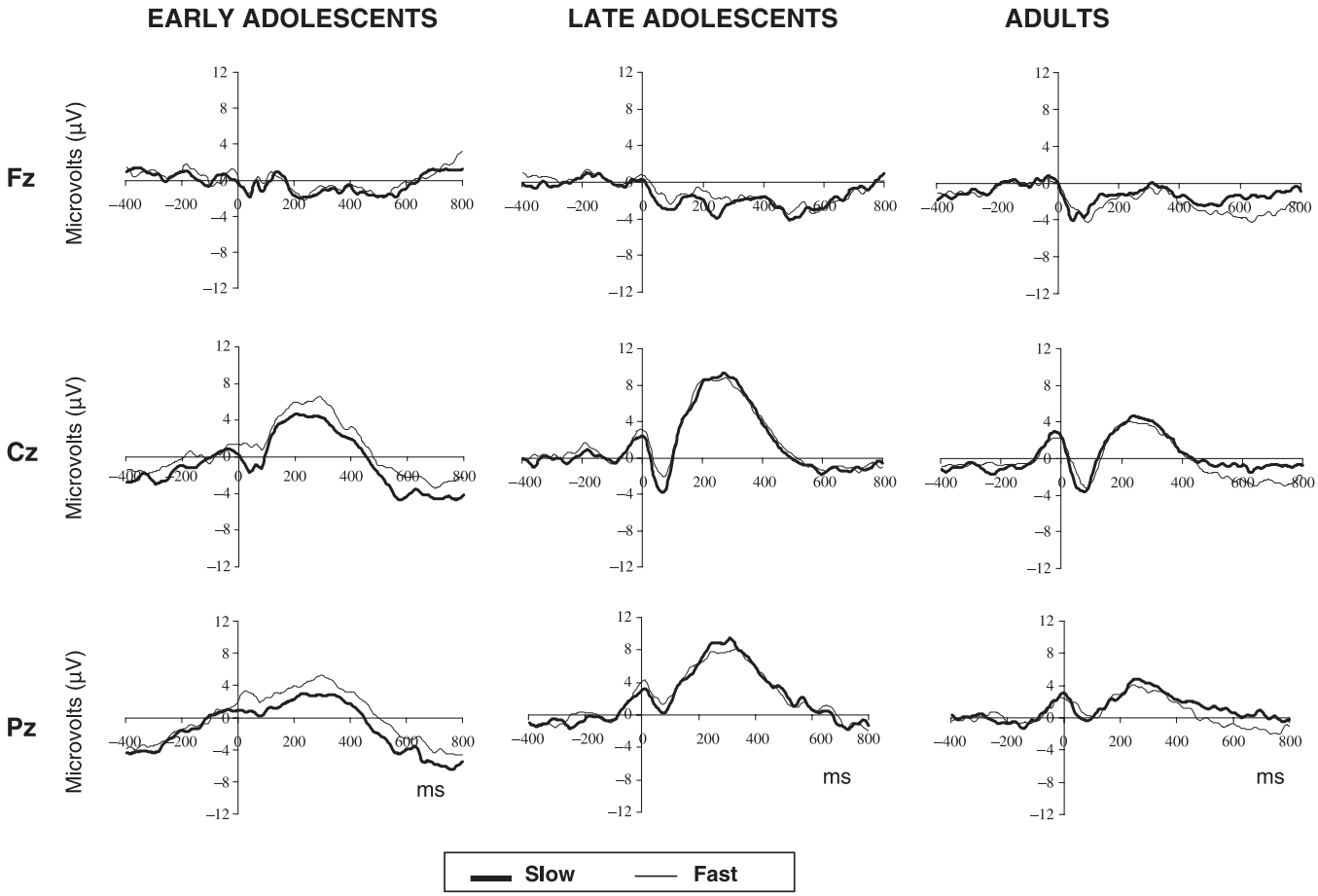

Figure 8 *Grand averaged ERN as a function of slow and fast post-error slowing for the early adolescent, late adolescent, and adult groups.*

amplitude varied with post-error slowing. Post-error slowing is a well-known indicator of behavioral adjustment on a particular task (Rabbitt, 1966) and can also be considered to reflect the recruitment of cognitive control processes, which are supported by the PFC (Braver *et al.*, 2001). Thus, we conducted secondary analyses comparing the amplitude of the ERN/Ne on error trials that were immediately followed by correct trials with fast and slow reaction times based on each participant's own median reaction time on those trials. We predicted that ERN/Ne amplitude would be greater on error trials that were immediately followed by correct trials with slow reaction times and expected that this effect would be more prominent with age. Results partially supported our hypothesis. ERN/Ne amplitude was more negative on error trials that were immediately followed by correct trials with slow reaction times. However, age did not seem to influence this effect.

The increased ERN/Ne amplitude on error trials immediately followed by slow correct trials suggests that activity in the ACC (reflected by the magnitude of ERN/Ne amplitude) is related to performance adjustment during the completion of a task. These results are consistent with recent fMRI findings showing increased ACC activity in error trials followed by subsequent post-error slowing (Kerns *et al.*, 2004). They also support the idea put forward by the conflict monitoring theory that the detection of error-related response conflict, indexed by the ERN/Ne, may play a role in triggering higher-order cognitive control processes. In the case of the flanker task used in this study, the slower reaction times on correct trials that immediately follow errors may reflect the mobilizing of increased attentional resources allowing the participant to process more efficiently stimuli on subsequent trials, thereby adjusting performance in order to minimize the possibility of future error. The fact that age did not significantly influence this effect may be interpreted as an indication that detecting response conflict and engaging cognitive control processes go hand in hand. In fact, Kerns *et al.* (2004) showed that post-error slowing was

associated with increased activity in the dorsolateral prefrontal cortex (DLPFC), a brain region associated with cognitive control, and that this activity was related to the level of ACC activity on the preceding error trial. Thus, although between-group analyses indicated that ERN/Ne amplitude was significantly smaller in the early adolescent group compared to the late adolescent and adult groups, it is possible that an ERN/Ne was evoked for a sub-group of younger adolescents – those who tended to adjust their performance following an error. However, until we have more data on the role of the ERN/Ne in recruiting cognitive control processes in adolescence, these interpretations remain speculative. Furthermore, we also investigated whether developmental changes in the ERN/Ne were related to overall task performance. Results indicated that ERN/Ne amplitude predicted performance in adults but not in the late adolescent group, suggesting that, although adolescents elicited an ERN/Ne indexing response monitoring processes, the influence of such processes on overall task performance does not appear until adulthood. More research on the impact of ACC maturation in the development of cognitive control processes in adolescence is required to better understand its functional role in response monitoring and the recruitment of regulatory abilities.

With regard to the P_E, results showed that although P_E was present across all three age groups, P_E amplitude was significantly more positive in the late adolescent group compared to the adult group at the Pz electrode site (see Figure 1). The increase in P_E amplitude in late adolescents does not support previous developmental findings (Davies *et al.*, 2004; Ladouceur *et al.*, 2004) and is inconsistent with our predictions. Results from the source localization analyses indicated that the neural generators of the P_E appeared to be located in the vicinity of the ACC (Brodman's area 24) across all three age groups, which is consistent with recent findings (Herrmann *et al.*, 2004; Mathewson *et al.*, 2005). However, the neural generators of the P_E were somewhat more superior in the late adolescent group, which may explain the increase in P_E amplitude observed in that group.

Another possible explanation related to the increase in P_E amplitude in the late adolescent group may be related to how some participants felt about having made errors on the task. We examined the responses to questions asked in a post-task questionnaire administered following the experiment for participants who had the largest P_E in that age group ($n = 5$). The data revealed high scores on items assessing 'how bad' they felt about making an error and 'how certain' they were about having made an error. Such reports are consistent with the idea that the P_E may be related to emotional reactions to errors (Falkenstein *et al.*, 2000; van Veen & Carter, 2002b) and awareness of having made an error (Nieuwenhuis *et al.*, 2001). More research is needed, however, to further elucidate the functional role of the P_E and the extent to which the development of the P_E may be modulated by individual differences in adolescence.

There are certain methodological issues that merit discussion. First, our ERN/Ne developmental findings do not take into consideration the role of task difficulty. Hogan *et al.* (2005) recently showed that with increasing task difficulty, ERN/Ne amplitude in adolescents is smaller than adults. As such, these findings can only be generalized to the performance of a flanker task, which does not vary in the level of task difficulty. Future studies examining the ERN/Ne on tasks varying in complexity will be helpful to better understand how the ERN/Ne changes across development, possibly allowing for the inclusion of even younger subjects. Second, although we took into consideration age-related differences in bone thickness and bone conductivity when conducting source localization analyses, these models remain approximations and would definitively need to be replicated in a different sample. Third, source localization analyses are based on the computation of inverse solutions and provide a best estimate model for the location of the neural generators. Therefore, it is not clear whether changes in ERN/Ne amplitudes are due to structural or functional changes of the ACC. According to Casey *et al.* (1997), differences in the size of the ACC in children compared to adults correspond to increased efficiency in attentional control. However, a recent study suggests that ERN/Ne amplitude may be related to changes in ACC white matter organization that take place during adolescence (Sullwold, Luciana & Collins, 2005). Future research co-registering ERP and fMRI measures related to performance on this task along with structural and dense tensor imaging techniques would allow us to address this question.

In summary, the results of this study replicate previous findings showing that the ERN/Ne undergoes developmental changes in adolescence. They also provide novel findings showing that the N2 seems to develop in parallel with the ERN/Ne and that the neural generators for both the N2 and the ERN/Ne are localized in the ACC, suggesting that developmental changes in these components may be related to the maturation of the ACC. It remains unclear, however, which aspect of ACC maturation is related to the functional role of the ERN/Ne and N2. Behavioral results showed that, overall, subjects slowed down after errors and that ERN/Ne amplitude was significantly greater on error trials immediately preceding slow post-error slowing, suggesting that ERN/Ne plays some role in performance adjustment but that this role does not seem to be influenced by development. Moreover, results indicate that although ERN/Ne amplitude increases

to adult-like levels later in adolescence, ERN/Ne amplitude is not related to task performance until adulthood. More research is need to understand whether maturational changes in the ERN/Ne are attributable to structural or functional changes in the ACC and the extent to which the ERN/Ne is related to developmental changes in cognitive control processes. As for the P_E, source localization analyses indicated that it also had neural generators in the ACC. Future research is needed to examine how P_E fluctuates with individual differences about subjective reactions to making errors in adolescence. Given the high rates of risk-taking, reckless behavior, and evidence of vulnerability for poor judgment in many aspects of adolescent behavior (Dahl, 2004), future studies examining the possible influence of puberty, affect and individual differences will help us achieve a deeper understanding of the development of these regulatory processes in adolescence.

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