Event-related potential measures of executive functioning from preschool to adolescence

MICHELLE DOWNES[1]* | JOE BATHELT[2]* | MICHELLE DE HAAN[3]

1 School of Psychology, University College Dublin, Dublin, Ireland. **2** MRC Cognition and Brain Sciences Unit, Cambridge; **3** Developmental Neurosciences Programme, University College London, London, UK

Correspondence to Michelle de Haan, Developmental Neurosciences Programme, UCL, Great Ormond Street Institute of Child Health, 30 Guilford Street, London WC1N 1EH, UK.
E-mail: m.de-haan@ucl.ac.uk

***These authors contributed equally to this work.**

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ABBREVIATIONS

CDA	Contralateral delay activity
ERN	Error-related negativity
ERP	Event-related potential
FRN	Feedback-related negativity
Pe	Error-related positivity

Executive functions are a collection of cognitive abilities necessary for behavioural control and regulation, and are important for school success. Executive deficits are common across acquired and developmental disorders in childhood and beyond. This review aims to summarize how studies using event-related potential (ERP) can provide insight into mechanisms underpinning how executive functions develop in children from preschool to adolescence. We specifically focus on ERP components that are considered to be well-established markers of executive functioning, including the ability to resist distraction (inhibition, N200), hold scenes in mind (visuospatial working memory, contralateral delay activity), attend to specific stimuli (information processing, P300), follow rules (response monitoring, error-related negativity [ERN], and error-related positivity [Pe]), and adjust to feedback (outcome monitoring, feedback-related negativity). All of these components show developmental changes from preschool to adolescence, in line with behavioural and neuroimaging findings. These ERP markers also show altered developmental trajectories in the context of atypical executive functions. As an example, deficits in executive function are prominently implicated in attention-deficit–hyperactivity disorder. Therefore, this review highlights ERP studies that have investigated the above ERP components in this population. Overall, ERPs provide a useful marker for the development and dysfunction of executive skills, and provide insight into their neurophysiological basis.

Executive functions are a collection of cognitive processes that help us to regulate our thoughts and behaviours to make plans, solve problems, and attain goals.[1,2] These skills are important throughout the lifespan, contributing to school readiness and academic achievement[3] and to later career success. Major subcomponents of executive functioning have been described as attention, inhibition, self-regulation, working memory, cognitive flexibility, planning, organization, problem-solving, and performance-monitoring.[4] Basic executive functions, including the inhibition of an inappropriate motor response, emerge early in life and subsequently lay down the foundations for later development of higher-order executive functions, including planning and problem-solving. Whether the subcomponents of executive functioning are already differentiated in the first few years of life or emerge from a more undifferentiated system with development is still debated. Although it is widely agreed that, from around 7 years of age, the overarching structure of executive functions is relatively stable,[5,6] the structure of executive functions may be more unitary and less differentiated earlier in life.[7,8]

Executive functions are compromised in different ways across a range of developmental disorders and in acquired brain injury,[9,10] and are susceptible to disease and poorer environments.[11,12] While the prolonged period of development makes executive functions particularly vulnerable, their higher malleability may also provide a window of opportunity to improve executive functions through interventions.[13]

Neuroimaging techniques have shed some light onto the development of the neural systems underlying executive functions.[14,15] A parallel has been drawn between the gradual integration of executive functions and the prolonged development of the prefrontal cortex,[16–18] but it is also clear that executive functions depend upon a wider neural system.[19] In addition to magnetic resonance imaging (MRI) studies, event-related potential (ERP) measures have contributed to our increasing comprehension of the developing neural substrates underlying this cognitive domain.[20–25] This method has several advantages over MRI, including being relatively easy, practical, and cost-effective to use with younger children, and providing more

precise information about the timing of brain events underlying behavioural performance.[26,27] Its high temporal resolution affords a closer look into various processing stages that lead to a single behavioural response, and provides another source of information in the investigation of the developmental differentiation of executive functions.

Here, we provide an overview of ERP studies relevant to executive function development from preschool to adolescence. ERP research from this period of development has not previously been reviewed, despite the growing number of studies and the substantial changes observed in ERP components related to executive functions during this developmental stage. This review aims to provide a summary of developmental changes observed in key ERP components throughout this period, collating studies that look at different domains of executive functioning, and providing a useful reference and overview for researchers and clinicians new to the area of ERP research in developmental populations, as well as an overview of the field for those currently engaged in work in this field. We will focus on four of the most extensively studied areas of executive functioning in neurophysiological research: inhibitory control, working memory, information processing, and performance monitoring and their associated ERP components (as listed in Table SI, online supporting information). As previously described, executive functions are often compromised by acquired brain injury and in various developmental disorders. Thus, following our overview of typical development, we will discuss one application of ERP methods in a neurodevelopmental disorder that has been the most widely investigated disorder using these methods: attention-deficit–hyperactivity disorder (ADHD).[28]

What this review adds

- Event-related components show maturational changes from preschool to adolescence.
- Altered developmental trajectories are associated with atypical executive functioning.
- Event-related potentials can serve as biological markers for the development and dysfunction of executive skills.

THE N200: INHIBITORY CONTROL AND INFERENCE SUPPRESSION

The N200 component of ERP is believed to reflect the cognitive control necessary for successful inhibitory control and interference suppression.[29] Inhibitory control is the ability to control a dominant, pre-potent motor or cognitive response, but it also involves processes such as interference suppression, emotional control, and directed forgetting, where a participant is explicitly told to remember and forget specific stimuli.[30] The ability to control interference from irrelevant stimuli and to inhibit a prepotent response to selectively attend to task-related events is important in the development of behavioural–emotional control and for academic attainment.[31] Inhibition grows increasingly pertinent in the transition from early childhood into adolescence as young people gain more independence.

The N200 is a negative wave produced after successful inhibition with a peak latency of approximately 200 to 300 milliseconds after stimulus onset. Its neural generators include the frontal and superior temporal cortex, and the anterior cingulate cortex.[32] The N200 can sometimes be referred to as the N2a, the N2b, or the N2c, depending on the particular paradigm used and thus the brain areas that are recruited. The degree to which specific brain areas are recruited can vary according to factors such as the demand for other executive skills, such as working memory, in a specific paradigm, the response modality used, and the history of previous responses.[32]

A larger peak in overt response inhibition tasks supports the association between the N200 and inhibitory control. For example, in the Go/No-go paradigm where the participant responds to a 'Go' stimulus but ignores the 'No-go' stimulus, a larger peak is seen when 'No-go' stimuli have some similar dimensions as the 'Go' stimuli, or when there is increased pressure to respond faster.[33,34] The N200 can also be observed in other paradigms, the most common of which are the Go/No-go task, the Stop-signal task, the Stroop task, and the Flanker task (see Table SII, online supporting information, for a detailed description of these tasks).

The N200 response may also vary according to the type of inhibitory control required, with some evidence suggesting a dissociation of interference suppression and response inhibition.[35] In a combined Go/No-go–Flanker task with 14 young adults, the incongruous flanker, which requires the suppression of distracting information, elicited a more central topography and a more delayed N200 peak than the No-go condition, which requires inhibition of a prepotent response.[36] This later peak is seen in tasks with distractors that need to be suppressed for successful task completion and is often referred to as the N2pc. However, some recent findings in three studies of children and adults (n=10–37) do not support the idea that the N2pc exclusively reflects distractor-suppression processes. It has been proposed that the N2pc may instead reflect a combination of attention selection and distractor suppression.[37,38] No firm conclusion about the precise relationship between N2pc and behaviour can be drawn, owing to the limited sample sizes and varying age ranges across the available literature.

There has been some debate in the literature about the interpretation of the N200. Some studies have suggested that it may reflect the monitoring of conflict instead of response inhibition.[29,39] The N200 has also been compared with the error-related negativity (ERN), which is elicited in trials where commissions are made. Some research suggests that the ERN may reflect error detection or inhibition. The ERN and the N200 were shown to have different scalp topographies in a Go/No-go task, which implies that different mechanisms and generators subsume these two components.[40] One study showed that distinct cortical areas were associated with response inhibition, commission errors, and behavioural correction using

electroencephalography (EEG) and functional MRI during a Go/No-go task.[41] Error detection was correlated with activation of the anterior cingulate and pre-supplementary motor area, whereas behavioural correction was related to the anterior cingulate as well as the left prefrontal cortex.

Development of the N200

Developmental research on the N200 typically reports a decrease in amplitude and latency with increasing age.[42–45] However, some studies have observed no age effect on N200 responses.[46] A potential reason for discrepancies in developmental studies is that the N200 may originate from different sources, depending on age and aptitude of the participants. The location of cingulate generators is more anterior for older children and for participants who perform better on inhibitory control tasks.[45] Lamm et al.[45] report in their study of 7- to 16-year-olds (n=33) that differences in N200 amplitudes are more closely associated with task performance rather than age. In contrast to their findings for developmental differences in amplitude, N200 latencies diminished with age but were not related to task performance.

THE P300: INFORMATION PROCESSING

The P300 is a positive waveform that appears at approximately 300 milliseconds in auditory ERP paradigms that involve attending to a target as well as discriminating between a target and a non-target. The P300 is most commonly referred to in the context of attention, working memory, and problem-solving.[47] Although there remains debate surrounding the precise cognitive function that the P300 is most closely associated with, there seems to be a general consensus on its description as a neurophysiological index of information processing and updating in working memory.[48] The latency and amplitude of the P300 have both been connected to behavioural success on executive tasks, including attention and memory, in healthy adult and patient populations, but this association has not been as widely researched in children.

The P300 is typically further subdivided into the P3a component and the P3b component. The P3a or 'novelty P300' activates in passive oddball tasks as a reaction to novel stimuli that do not call for an active response from the participant, whereas the P3b is engaged in active oddball tasks that involve intentional conscious discrimination as the participant responds to the novel stimulus, often by button press.[49] The P3a is observed when a task requires orienting or novelty detection and has a frontocentral topography. The P3a is likely to originate in the frontal cortex and the hippocampus.[50] The P3b is typically observed during active tasks that engage attention and working memory and shows a more parietal topography with sources in the temporal and parietal lobes, and cingulate cortex.[32] Polich[51] has proposed that the P300 is a result of the P3a, which responds to early attention-related processes and further drives the P3b, produced when enhanced attention drives the stimulus signal to temporal and parietal regions. In contrast, the No-go P3 is thought to reflect inhibitory control as it is observed in response to distractor items[40,52] and shows a different topography to P3a and P3b with maximum peaks in centro-parietal channels.

Development of the P300

It has been proposed that the latency and amplitude of the P300 reflect different developmental processes in the brain. Latency is thought to index neural speed and efficiency and amplitude reflects growing cognitive resources, that increases with brain maturation.[53,54]

P300 latency has been reported to decrease as children grow older, with studies showing further decreases in P300 latency up to adolescence.[55,56] Changes in P3a latency usually stabilize at around 12 years of age, while P3b latency continues to shorten until around 17 years of age.[57]

Findings on the developmental trajectory of P300 amplitude are more ambiguous.[58,59] A recent systematic review by van Dinteren et al.[60] suggested a steady increase until a maximum is reached in late adolescence or early in the third decade. Studies examining the P3a and P3b suggest that, similarly to findings for latency, the P3a amplitude matures earlier than the P3b. A reason for the mixed findings of age effects may be explained by other factors such as variation in pubertal stage. Brumback et al.[58] reported an association between P300 amplitude and latency and pubertal stage in their large cohort of 99 children aged between 8 years and 13 years. An advantage of their study was that a larger cohort allowed analysis of the influence of factors other than age.

CONTRALATERAL DELAY ACTIVITY: VISUOSPATIAL WORKING MEMORY

The contralateral delay activity (CDA) is a lateralized ERP over the parietal cortex that reflects the amount of target and distractor stimuli that are encoded or maintained from one hemi-field during the memory display. The CDA increases in amplitude with the number of target and/or distractor items maintained in working memory and is correlated with working memory capacity.[61] Working memory is the ability to temporarily mentally store and manipulate information. Classically, working memory has been divided into 'slave systems', which are separate for visuospatial and phonological information, and a supervisory system called the central executive. The capacity, or number of units of information that can be kept in working memory, is important in the development of academic skills and for general learning.[62]

Development of the CDA

It is typically reported that mature working memory capacity is achieved by adolescence. Although some research has provided evidence for mature visuospatial working memory capacity by 10 to 12 years,[63,64] other reports suggest that adult-like capacity is not reached before 16 years.[65] These differences in findings are though to reflect the level of

executive control that is required to perform the task at hand. There seems to be a later development of working memory capacity in tasks that require higher levels of attentional control.

One study found that the distractor-related CDA responses indicated higher distractor encoding and maintenance by adolescents (n=21) than adults, and that CDA amplitudes were positively associated with successful interference.[66] On higher load conditions, adolescents performed worse than adults, and showed higher CDA amplitudes, whereas amplitudes were comparable between the two ages for low-load groups. This suggests that, at higher loads, the poorer performance of adolescents was caused by greater difficulty in blocking distractors from processing and maintenance in working memory, possibly reflecting continued immaturity of frontoparietal networks. However, the small sample size in the adolescent age range does not allow for investigation of other potentially influential factors such as age, puberty, and sex. Another study using a cued change detection paradigm found that CDA amplitude was modulated by task load in 10- to 12-year-olds (n=22) but not in adults.[67]

OTHER ATTENTION AND WORKING-MEMORY-RELATED ACTIVITIES

ERPs have also been used to investigate other preparatory and inhibitory processes during cued attention and working memory tasks. In tasks where children are required to look towards a cued location and to ignore a distractor location, a series of ERP responses are observed. Early directing attention negativity, possibly reflecting early parietal activation within the frontoparietal network, precedes frontal activity reflected by the anterior directing-attention negativity. After these responses, a late widespread contralateral positivity is observed, which is thought to represent the oculomotor programming of the planned eye movement as well as the orienting of attention.[68] Studies show that these early attentional responses are related to working memory abilities. Shimi et al.[69] report that age-related differences in attention orienting processes before and after encoding stimuli in visual working memory (VWM) can explain differences in VWM performance between the developing brain and the adult brain. Differences on an individual level in the attention orienting processes before encoding can be biased so that relevant items are more efficiently encoded into VWM in children with high VWM capacity.[69] A further study showed that children with large cueing benefits in VWM capacity elicited adult-like responses after selection of the stimuli, whereas children with low capacity did not elicit a contralateral negativity.[38]

ERROR-RELATED NEGATIVITY: RESPONSE MONITORING

The ability to monitor responses and adjust behavioural output according to set goals is another important executive function domain. Responses on tasks used to index monitoring (Go/No-go paradigm, Eriksen Flanker task, and the Simon task) are marked by specific ERP components following error. The ERN is a negative deflection between 80 milliseconds and 150 milliseconds with maximal amplitudes over frontocentral channels[70] thought to be produced in the anterior cingulate cortex.[71] The ERN response does not depend on the conscious awareness of the participant that an error was made.[72] There is also a related response in correct trials with a similar time course and topography but with lower amplitudes, called the correct-related negativity. The ERN appears before a positive deflection (error-related positivity, Pe) with a maximum response over centro-parietal channels with a peak between 200 milliseconds and 500 milliseconds.[73] In contrast to ERN, the Pe depends on conscious error awareness and is not present in all error-trials.[74] On a behavioural level, increases in response accuracy, reaction time, and a reduction in response variability have been found using the Go/No-go paradigm[75] and Eriksen Flanker task.[76] These improvements have been found throughout childhood and adolescence[77,78] until adult-level performance is reached.[75]

Development of the ERN

In parallel with improvements in task performance, increases in ERN amplitude have been documented. ERN can be detected as young as 4 years if age-appropriate tasks are used.[77] Development from mid-childhood to early adulthood shows continuing increases in ERN amplitude[75,79] following a logarithmic developmental profile.[76] The steepest changes in ERN amplitude are found in adolescence, from around 11 years for females and about 15 years for males.[80]

Several factors are thought to influence the prolonged maturation of the ERN. The maturation of ERN amplitude may reflect the maturational profile of the frontal cortex.[18,81] Source reconstruction indicates that the ERN is produced by the same generators in the anterior cingulate in children and adults,[75] consistent with the idea that anatomical changes within this substrate may explain differences in ERN amplitude with age. Another factor influencing developmental trends in ERN amplitude is task difficulty. For instance, Hogan et al.[79] found that differences in ERN amplitude between adolescents and adults (n=23; aged 12–22y) could only be observed in a more difficult task condition. Therefore, changes in ERN amplitude may be more closely linked to improvements in task performance rather than chronological age.[78] Psychological factors such as motivation and character traits have also been found to significantly influences error processing. A study by Kim et al.[82] found larger ERN amplitudes when 7- to 11-year-olds (n=20) were observed by their peers as they were performing a Go/No-go task. A larger-scale study in a cohort of 6-year-olds (n=413) found that maternal anxiety and children's emotional negativity was found to be predictive of smaller ERN amplitudes on a Go/No-go task.[83] The association was in the opposite direction to what is generally reported for older children and adults in

other studies investigating the ERN and anxiety, which report larger ERN with greater anxiety. This illustrates the importance of focusing on larger samples in tighter age ranges to elucidate the impact of increasing age on the elicited ERP component.

In contrast to the ERN, the Pe shows a profile of early maturation. Studies on error monitoring in preschool children found significant correlations between Pe amplitude and response accuracy and reaction time.[84,85] Studies comparing age groups from mid-childhood to adolescence do not find differences in Pe amplitude or a statistically significant relationship between Pe amplitude and behavioural performance.[78–80] The absence of developmental changes in Pe amplitude may be due to the superposition of different components during the Pe time window,[86] and may also be due to low signal as the Pe is not observed in all trials.

FEEDBACK-RELATED NEGATIVITY: FEEDBACK MONITORING

In addition to being able to detect errors in self-generated responses, children must also be able to respond to external feedback to reach optimal performance. Feedback monitoring is mostly elicited in tasks with either probabilistic or random outcome. In probabilistic learning tasks, participants learn to associate stimuli with certain risks for gains or losses. Other tasks look at the effects of positive or negative feedback presented randomly. Feedback typically elicits a negative deflection with a maximum over mediofrontal electrodes with a peak around 270 milliseconds after feedback onset.[87] This component is described as the feedback-related negativity (FRN). The amplitude of this component is consistently larger in response to negative feedback than to positive feedback.[87] The dorsal anterior cingulate cortex is the proposed source of the FRN.[88] Genetic studies suggest that variation in the FRN may be linked to individual-level differences in the dopamine and serotonin systems, which have been previously associated with reward processing and decision-making.[89]

Development of the FRN

Developmental studies report that the FRN response can be reliably detected from 4 years of age using age-appropriate tasks.[90] The FRN amplitude increases linearly between childhood and adulthood.[88] Source reconstruction studies indicate that the FRN originates in the anterior cingulate cortex across different age ranges.[88] In addition to age, the FRN amplitude may be influenced by sex. Adolescent females have been found to showed higher FRN amplitude in response to wins[91] and smaller amplitudes for losses,[92] whereas young males displayed indiscriminately larger amplitudes irrespective of feedback type.[88] The FRN has been widely used as a marker of risk-taking and impulsiveness. Differences in FRN have been linked to an increased likelihood of conduct problems[93] and antisocial behaviour[94] in adolescence.

ERP MARKERS OF ATYPICAL EXECUTIVE FUNCTION DEVELOPMENT

A large part of the available ERP literature that investigates differences in executive function development in childhood disorders focuses on children with ADHD.[95] ADHD is characterized by deficits in attention, hyperactivity, and impulsivity.[28] However, considerable heterogeneity in symptoms and a higher prevalence of co-occurring disorders pose significant challenges to the diagnosis, treatment and investigation of ADHD.[96] Neurophysiological methods are one avenue to identify endophenotypes that could serve as biomarkers and help to distinguish between ADHD subtypes. Indeed, differences in N200 amplitude and latency have been described as a potential endophenotype for ADHD.[97] The following section will review the literature on ERP investigations of ADHD spanning all previously discussed aspects of executive function.

ERP MARKERS OF ATTENTION AND INHIBITION IN ADHD

Differences in ERP markers of attention and inhibition have been identified in children with ADHD. For instance, attenuated P300 amplitude and decreased latency in Go/No-go tasks in children with ADHD are thought to reflect early signs of atypical attention development.[98,99] Further, reduced P200 and N200 effects during Go/No-go and Stop Signal tasks have been attributed to poorer recruitment of neural resources.[95,100] One study reported a more anterior P300 for children with ADHD, which may indicate a greater requirement for frontal inhibitory processes.[101] Differences are not consistently found across studies with either increased, reduced, or absent amplitude and latency effects in different studies.[102] The mixed results may be due to the varying age groups used, differences in task design or analysis techniques, and the heterogeneity within ADHD groups.[103] Nonetheless, ERP components can be used as sensitive markers of executive function as evidenced by a recent randomized control trial that looked at the impact of treatment in 112 school-age children with ADHD and found increases in the P300 amplitude after intervention alongside improvements in response inhibition.[100]

ERP MARKERS OF WORKING MEMORY IN ADHD

The CDA has been used to study working memory in adolescents and adults with ADHD. One study administered a change detection task both to adolescents aged 12 to 16 years, and to adults with and without ADHD and found that performance deteriorated more for the adolescents with (n=15) and without (n=19) ADHD than either adult group in the presence of distracters and when there was a higher working memory load.[104] The CDA showed that adults were able to more efficiently remove distracting details from memory later in the retention interval, resulting in better working memory. ADHD diagnosis was related to smaller CDA amplitude in adolescents and

adults with ADHD than in the comparison group when maintaining a low load, which could reflect an inability to maintain focused attention to cued stimuli when there are low task demands. Thus, overall, the ERP results discussed here suggest no differences in filtering efficiency and visuospatial working memory storage capacity in adolescents and adults with ADHD.

ERP MARKERS OF REWARD PROCESSING AND RESPONSE MONITORING IN ADHD

ERP markers have been used to investigate potential differences in reward processing and response monitoring in ADHD. One study reported smaller ERN amplitudes in 68 children aged 8 to 15 years with ADHD as well as intermediate amplitudes in unaffected siblings than in a matched comparison group.[105] Similarly, Pe amplitude was found to be reduced in 7- to 11-year-old (*n*=16) children with ADHD but not for adults with ADHD.[106] Only children with ADHD who had additional learning difficulties showed reduced Pe amplitude in a larger-scale study.[107]

Van Meel et al.[108] found no significant differences in feedback processing when investigating the FRN in 8- to 12-year-old (*n*=21) children with ADHD, but observed reduced amplitudes in later time windows. Similarly, another study found that while FRN amplitude decreased after the first reward in 8- to 12-year-old typically developing children, it increased in children with ADHD (*n*=14),[109] which may indicate differences in motivation. In summary, studies indicate differences in ERP markers of reward processing and response monitoring in children with ADHD, but the specificity of this association will need to be further elucidated in future research.

CONCLUSION

ERP paradigms provide us with a direct means of analysing the brain basis of typically and atypically developing executive skills in children and adolescents. They also offer valuable insights that cannot be gleaned from behavioural research alone. ERPs can inform cognitive interpretations by indexing constituent processes that contribute to behavioural performance on a particular task. For example, a study of 8- to 10-year-olds with a history of concussion on a Go/No-go task found that children who had experienced recent concussion (*n*=15) made more commission errors behaviourally than those who did not (*n*=15).[110] These behavioural differences were accompanied by longer N200 latencies and more diminished P300 amplitudes on a neural level. Similarly, an ERP study of adolescents with unilateral and bilateral frontal stroke (*n*=11) due to sickle cell disease on a fast-response task found that these patients showed evidence of a diminished ERN response compared with patients with sickle cell disease only (*n*=11) and comparison siblings (*n*=11), despite no differences on a behavioural level. However, the N200 and P300 were not impacted by the lesions, which suggests that although these executive processes were still relatively intact, performance monitoring was not.[111] These studies demonstrate how ERPs can help in the assessment of acquired brain injury and other disorders by contributing to the development of executive profiles that highlight specific strengths and weaknesses, bringing us closer to an 'executive fingerprint'.[112] As described by Ozonoff and Jensen[112] in their report almost two decades ago, a better understanding of an executive dysfunction can lead to a more successful diagnosis and intervention.

Developmental studies show changes in all of the discussed ERP components with increasing age from preschool to adolescence. These changes are likely to reflect the structural and functional maturation of the neural substrates underlying executive skills and help inform theories of executive development.[7,8] The prolonged developmental changes in the frontal lobe and its related systems mean that the timing of brain injury onset can have differential effects on the executive system, depending on its developmental stage, with earlier insult often resulting in wider-reaching dysfunction across executive domains.[113] It can be more difficult to assess the impact of frontal brain injury early in development on later emerging executive skills. Promising new research suggests that neurophysiological indices of executive functions can be identified before they are behaviourally assessable and may even be predictive of future executive performance.[114] For example, Brydges et al.[114] recently showed that the N200 difference

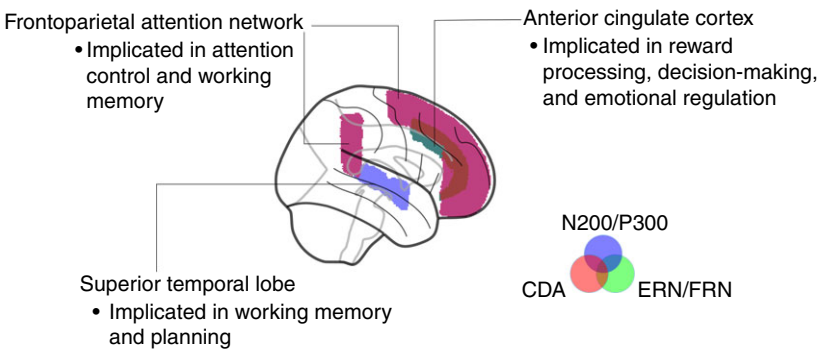

Figure 1: Frontoparietal or executive network (highlighted) and associated event-related potential components depicting the proposed underlying regions. CDA, contralateral delay activity; ERN, error-related negativity; FRN, feedback-related negativity. [Colour figure can be viewed at wileyonlinelibrary.com].

waveform and the P3b amplitude in a group of 7- to 9-year-olds were predictive of a unitary executive factor, showing observable indices of executive functioning before the specific associated behaviours could be distinguished from one another using psychometric assessment. We are yet to fully understand the interpretation of the individual neural correlates that underlie specific executive functions and to grasp how these relate to one another in the context of the developing brain. The potential contribution of factors such as sex, environment, disease, and hormones require further investigation to better understand the significance of sometimes subtle differences in ERP responses.

ERP methods are being used more frequently to assess the efficacy of interventions designed to improve cognition and behaviour.[111,115,116] For instance, one intervention study found specific changes in the N200 response, which implied that emotional regulation training successfully worked by increasing inhibition rather than decreasing emotional arousal.[117] However, ERP techniques also have their own unique design and interpretation issues. For instance, EEG data are often 'noisier' in younger populations because of differences in compliance. This problem is aggravated by arbitrary age groupings, variation in task implementation, and small sample sizes in the available literature. There is currently no general consensus on best practices in paediatric ERP research that would aid interpretation and cross-study comparison.[118]

There are some specific limitations to the current ERP literature on executive function development that should be considered in the development of future studies. For one, the association between specific components and behaviourally defined executive function constructs is often unclear. Irreconcilable conflicts between neurophysiological findings and cognitive theory may necessitate the development of new models. Second, certain domains of executive function such as switching are well established on a behavioural level, but few studies have investigated them with ERP methods so there are insufficient studies for appropriate review.[119] Third, while there is a substantial body of ERP research investigating executive functions in ADHD, other disorders with well-known executive deficits are less well studied. Based on these limitations, we suggest that future studies aim to investigate the relation between the development of ERP components and behavioural executive performance longitudinally in developmental populations, as our current knowledge is limited by a lack of longitudinal focus. Greater sample sizes are also required to account for the substantial ERP changes in this period to enhance power and to better establish ERP correlates of developing executive functions. In this way, we can better understand the influence of some factors investigated in studies discussed in this review such as temperament, puberty, and sex, as well as the influence of age and behavioural ability. We also suggest that more focus should be placed on using the ERP method to focus on less well-established components, such as the neural response associated with switching. Finally, we suggest that researchers use the ERP as a methodology to better understand executive development and dysfunction in less well-studied patient populations such as children with developmental disorders like Tourette syndrome, obsessive–compulsive disorder, and children with acquired brain injury. By taking ERP research further in these directions, we will be better equipped to interpret the significance of individual differences in ERP components and be better able to utilize this method for more informed diagnosis and treatment.

In summary, all of the components reviewed show developmental changes through adolescence and have been linked to specific regions of the brain networks underlying executive skills (Fig. 1). Future research may take advantage of using these components as markers of functional development or dysfunction of these brain regions and as an index of developmental differentiation of the executive system.

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SUPPORTING INFORMATION

The following additional material may be found online:

Table SI: Event-related potential components of executive functioning

Table SII: Commonly used classical experimental paradigms

REFERENCES

1. Lezak MD. The problem of assessing executive functions. *Int J Psychol* 1982; **17**: 281–97.
2. Welsh MC, Pennington BF. Assessing frontal lobe functioning in children: views from developmental psychology. *Dev Neuropsychol* 1988; **4**: 199–230.
3. Verdine BN, Irwin CM, Golinkoff RM, Hirsh-Pasek K. Contributions of executive function and spatial skills to preschool mathematics achievement. *J Exp Child Psychol* 2014; **126**: 37–51.
4. Anderson V, Anderson PJ, Jacobs R, Smith MS. Development and assessment of executive function: from preschool to adolescence. In: Anderson V, Jacobs R, Anderson PJ, editors. Executive Functions and the Frontal Lobes: A Lifespan Perspective. New York, NY: Taylor and Francis, 2008: 123–54.
5. Huizinga M, Dolan CV, van der Molen MW. Age-related change in executive function: developmental trends and a latent variable analysis. *Neuropsychologia* 2006; **44**: 2017–36.
6. Best JR, Miller PH. A developmental perspective on executive function. *Child Dev* 2010; **81**: 1641–60.
7. Senn TE, Espy KA, Kaufmann PM. Using path analysis to understand executive function organization in preschool children. *Dev Neuropsychol* 2004; **26**: 445–64.
8. Wiebe SA, Sheffield T, Nelson JM, Clark CA, Chevalier N, Espy KA. The structure of executive function in 3-year-olds. *J Exp Child Psychol* 2011; **108**: 436–52.
9. Johnson MH. Executive function and developmental disorders: the flip side of the coin. *Trends Cogn Sci* 2012; **16**: 454–57.
10. Gioia GA, Isquith PK, Kenworthy L, Barton RM. Profiles of everyday executive function in acquired and

developmental disorders. *Child Neuropsychol* 2002; **8**: 121–37.

11. Hughes CH, Ensor RA. How do families help or hinder the emergence of early executive function? *New Dir Child Adolesc Dev* 2009; **2009**: 35–50.

12. Muscara F, Catroppa C, Anderson V. The impact of injury severity on executive function 7-10 years following pediatric traumatic brain injury. *Dev Neuropsychol* 2008; **33**: 623–36.

13. Diamond A, Lee K. Interventions shown to aid executive function development in children 4 to 12 years old. *Science* 2011; **333**: 959–64.

14. Asato M, Terwilliger R, Woo J, Luna B. White matter development in adolescence: a DTI study. *Cereb Cortex* 2010; **20**: 2122–31.

15. Morton JB, Bosma R, Ansari D. Age-related changes in brain activation associated with dimensional shifts of attention: an fMRI study. *NeuroImage* 2009; **46**: 249–56.

16. Steinberg L. Cognitive and affective development in adolescence. *Trends Cogn Sci* 2005; **9**: 69–74.

17. Casey B, Tottenham N, Liston C, Durston S. Imaging the developing brain: what have we learned about cognitive development? *Trends Cogn Sci* 2005; **9**: 104–10.

18. Lenroot RK, Giedd JN. Brain development in children and adolescents: insights from anatomical magnetic resonance imaging. *Neurosci Biobehav Rev* 2006; **30**: 718–29.

19. Miller EK, Cohen JD. An integrative theory of prefrontal cortex function. *Ann Rev Neurosci* 2001; **24**: 167–02.

20. Mikkola K, Kushnerenko E, Partanen E, et al. Auditory event-related potentials and cognitive function of preterm children at five years of age. *Clin Neurophysiol* 2007; **118**: 1494–02.

21. Myatchin I, Mennes M, Wouters H, Stiers P, Lagae L. Working memory in children with epilepsy: an event-related potentials study. *Epilepsy Res* 2009; **86**: 183–90.

22. Overtoom CC, Verbaten MN, Kemner C, et al. Associations between event-related potentials and measures of attention and inhibition in the Continuous Performance Task in children with ADHD and normal controls. *J Am Acad Child Adolesc Psychiatry* 1998; **37**: 977–85.

23. Salmond CH, Vargha-Khadem F, Gadian DG, de Haan M, Baldeweg T. Heterogeneity in the patterns of neural abnormality in autistic spectrum disorders: evidence from ERP and MRI. *Cortex* 2007; **43**: 686–99.

24. de Sonneville LM, Huijbregts SC, Licht R, Sergeant JA, van Spronsen FJ. Pre-attentive processing in children with early and continuously-treated PKU. Effects of concurrent Phe level and lifetime dietary control. *J Inherit Metab Dis* 2011; **34**: 953–62.

25. Stevens C, Sanders L, Neville H. Neurophysiological evidence for selective auditory attention deficits in children with specific language impairment. *Brain Res* 2006; **1111**: 143–52.

26. Johnson MH, Halit H, Grice SJ, Karmiloff-Smith A. Neuroimaging of typical and atypical development: a perspective from multiple levels of analysis. *Dev Psychopathol* 2002; **14**: 521–36.

27. Scerif G, Kotsoni E, Casey BJ. The functional neuroimaging of development. In: Cabeza R, Kingstone A, editors. Functional Neuroimaging of Cognition. Cambridge, MA: MIT Press, 2006: 351–78.

28. Tarver J, Daley D, Sayal K. Attention-deficit hyperactivity disorder (ADHD): an updated review of the essential facts. *Child Care Health Dev* 2014; **40**: 762–74.

29. Jodo E, Kayama Y. Relation of a negative ERP component to response inhibition in a Go/No-go task. *Electroencephalogr Clin Neurophysiol* 1992; **82**: 477–82.

30. Best JR, Miller PH, Jones LL. Executive functions after age 5: changes and correlates. *Dev Rev* 2009; **29**: 180–200.

31. Blair C, Razza RP. Relating effortful control, executive function, and false belief understanding to emerging math and literacy ability in kindergarten. *Child Dev* 2007; **78**: 647–63.

32. Huster R, Westerhausen R, Pantev C, Konrad C. The role of the cingulate cortex as neural generator of the N200 and P300 in a tactile response inhibition task. *Hum Brain Mapp* 2010; **31**: 1260–71.

33. Azizian A, Freitas AL, Parvaz MA, Squires NK. Beware misleading cues: perceptual similarity modulates the N2/P3 complex. *Psychophysiology* 2006; **43**: 253–60.

34. Bruin K, Wijers A. Inhibition, response mode, and stimulus probability: a comparative event-related potential study. *Clin Neurophysiol* 2002; **113**: 1172–82.

35. Brydges CR, Anderson M, Reid CL, Fox AM. Maturation of cognitive control: delineating response inhibition and interference suppression. *PLoS ONE* 2013; **8**: e69826.

36. Brydges CR, Clunies-Ross K, Clohessy M, et al. Dissociable components of cognitive control: an event-related potential (ERP) study of response inhibition and interference suppression. *PLoS ONE* 2012; **7**: e34482.

37. Mazza V, Turatto M, Caramazza A. Attention selection, distractor suppression and N2pc. *Cortex* 2009; **45**: 879–90.

38. Shimi A, Nobre AC, Scerif G. ERP markers of target selection discriminate children with high vs. low working memory capacity. *Front Syst Neurosci* 2015; **9**: 153.

39. Donkers FC, Van Boxtel GJ. The N2 in go/no-go tasks reflects conflict monitoring not response inhibition. *Brain Cogn* 2004; **56**: 165–76.

40. Falkenstein M, Hoormann J, Hohnsbein J. ERP components in Go/No-go tasks and their relation to inhibition. *Acta Psychol* 1999; **101**: 267–91.

41. Garavan H, Ross T, Murphy K, Roche R, Stein E. Dissociable executive functions in the dynamic control of behavior: inhibition, error detection, and correction. *NeuroImage* 2002; **17**: 1820–29.

42. Davis EP, Bruce J, Snyder K, Nelson CA. The X-trials: neural correlates of an inhibitory control task in children and adults. *J Cogn Neurosci* 2003; **15**: 432–43.

43. Johnstone SJ, Pleffer CB, Barry RJ, Clarke AR, Smith JL. Development of inhibitory processing during the go/No-go task. *J Psychophysiol* 2005; **19**: 11–23.

44. Lewis MD, Lamm C, Segalowitz SJ, Stieben J, Zelazo PD. Neurophysiological correlates of emotion regulation in children and adolescents. *J Cogn Neurosci* 2006; **18**: 430–43.

45. Lamm C, Zelazo PD, Lewis MD. Neural correlates of cognitive control in childhood and adolescence: disentangling the contributions of age and executive function. *Neuropsychologia* 2006; **44**: 2139–48.

46. Johnstone SJ, Dimoska A, Smith JL, et al. The development of stop-signal and Go/No-go response inhibition in children aged 7–12 years: performance and event-related potential indices. *Int J Psychophysiol* 2007; **63**: 25–38.

47. Donchin E, Coles MG. Is the P300 component a manifestation of context updating. *Behav Brain Sci* 1988; **11**: 357–27.

48. Picton TW. The P300 wave of the human event-related potential. *J Clin Neurophysiol* 1992; **9**: 456–79.

49. Escera C, Alho K, Winkler I, Näätänen R. Neural mechanisms of involuntary attention to acoustic novelty and change. *J Cogn Neurosci* 1998; **10**: 590–04.

50. Knight RT. Distributed cortical network for visual attention. *J Cogn Neurosci* 1997; **9**: 75–91.

51. Polich J. Neuropsychology of P300. In: Kappenman ES, Luck SJ, editors. The Oxford Handbook of Event-Related Potential Components. New York, NY: Oxford University Press, 2012: 159–88.

52. Azizian A, Freitas A, Watson T, Squires N. Electrophysiological correlates of categorization: P300 amplitude as index of target similarity. *Biol Psychol* 2006; **71**: 278–88.

53. Boucher O, Bastien CH, Muckle G, Saint-Amour D, Jacobson SW, Jacobson JL. Behavioural correlates of the P3b event-related potential in school-age children. *Int J Psychophysiol* 2010; **76**: 148–57.

54. Ruhnau P, Wetzel N, Widmann A, Schröger E. The modulation of auditory novelty processing by working memory load in school age children and adults: a combined behavioral and event-related potential study. *BMC Neurosci* 2010; **11**: 1.

55. Rozhkov VP, Sergeeva EG, Soroko SI. Age dynamics of evoked brain potentials in involuntary and voluntary attention to a deviant stimulus in schoolchildren from the northern region. *Neurosci Behav Physiol* 2009; **39**: 851–63.

56. Tsai M-L, Hung K-L, Tung WT-H, Chiang T-R. Age-changed normative auditory event-related potential value in children in Taiwan. *J Formos Med Assoc* 2012; **111**: 245–52.

57. Fuchigami T, Okubo O, Ejiri K, et al. Developmental changes in P300 wave elicited during two different experimental conditions. *Pediatr Neurol* 1995; **13**: 25–28.

58. Brumback TY, Arbel Y, Donchin E, Goldman MS. Efficiency of responding to unexpected information varies with sex, age, and pubertal development in early adolescence. *Psychophysiology* 2012; **49**: 1330–39.

59. Pfueller U, Oelkers-Ax R, Gmehlin D, et al. Maturation of P300 amplitude and short-term learning as reflected by P300 habituation between trial blocks in children. *Int J Psychophysiol* 2011; **79**: 184–94.

60. van Dinteren R, Arns M, Jongsma MLA, Kessels RPC. P300 development across the lifespan: a systematic review and meta-analysis. *PLoS ONE* 2014; **9**: e87347.

61. Vogel EK, Machizawa MG. Neural activity predicts individual differences in visual working memory capacity. *Nature* 2004; **428**: 748–51.

62. Gathercole SE, Pickering SJ, Ambridge B, Wearing H. The structure of working memory from 4 to 15 years of age. *Dev Psychol* 2004; **40**: 177.

63. Van Leijenhorst L, Crone EA, Van der Molen MW. Developmental trends for object and spatial working memory: a psychophysiological analysis. *Child Dev* 2007; **78**: 987–1000.

64. Riggs KJ, McTaggart J, Simpson A, Freeman RP. Changes in the capacity of visual working memory in 5- to 10-year-olds. *J Exp Child Psychol* 2006; **95**: 18–26.

65. Luciana M, Conklin HM, Hooper CJ, Yarger RS. The development of nonverbal working memory and executive control processes in adolescents. *Child Dev* 2005; **76**: 697–12.

66. Spronk M, Vogel EK, Jonkman LM. Electrophysiological evidence for immature processing capacity and filtering in visuospatial working memory in adolescents. *PLoS ONE* 2012; **7**: e42262.

67. Sander MC, Werkle-Bergner M, Lindenberger U. Contralateral delay activity reveals life-span age differences in top-down modulation of working memory contents. *Cereb Cortex* 2011; **21**: 2809–19.

68. Praamstra P, Kourtis D. An early parietal ERP component of the frontoparietal system: EDAN ≠ N2pc. *Brain Res* 2010; **1317**: 203–10.

69. Shimi A, Kuo B-C, Astle DE, Nobre AC, Scerif G. Age group and individual differences in attentional orienting dissociate neural mechanisms of encoding and maintenance in visual STM. *J Cogn Neurosci* 2014; **26**: 864–77.

70. Falkenstein M, Hohnsbein J, Hoormann J, Blanke L. Effects of crossmodal divided attention on late ERP components. II. Error processing in choice reaction tasks. *Electroencephalogr Clin Neurophysiol* 1991; **78**: 447–55.

71. Segalowitz SJ, Davies PL, Santesso D, Gavin WJ, Schmidt LA. The development of the error negativity in children and adolescents. In: Ullsperger M, Falkenstein M, editors. Errors, Conflicts, and the Brain: Current Opinions on Performance Monitoring. Leipzig: Max Planck Institute for Cognitive Neuroscience, 2004: 177–84.

72. Nieuwenhuis S, Ridderinkhof KR, Blom J, Band GP, Kok A. Error-related brain potentials are differentially related to awareness of response errors: evidence from an antisaccade task. *Psychophysiology* 2001; **38**: 752–60.

73. Falkenstein M, Hoormann J, Christ S, Hohnsbein J. ERP components on reaction errors and their functional significance: a tutorial. *Biol Psychol* 2000; **51**: 87–107.

74. Wiersema JR, van der Meere JJ, Roeyers H. Developmental changes in error monitoring: an event-related potential study. *Neuropsychologia* 2007; **45**: 1649–57.

75. Santesso DL, Segalowitz SJ. Developmental differences in error-related ERPs in middle- to late-adolescent males. *Dev Psychol* 2008; **44**: 205.

76. van Meel CS, Heslenfeld DJ, Rommelse NN, Oosterlaan J, Sergeant JA. Developmental trajectories of neural mechanisms supporting conflict and error processing in middle childhood. *Dev Neuropsychol* 2012; **37**: 358–78.

77. Torpey DC, Hajcak G, Kim J, Kujawa A, Klein DN. Electrocortical and behavioral measures of response monitoring in young children during a go/no-go task. *Dev Psychobiol* 2012; **54**: 139–50.

78. Richardson C, Anderson M, Reid CL, Fox AM. Neural indicators of error processing and intraindividual variability in reaction time in 7 and 9 year-olds. *Dev Psychobiol* 2011; **53**: 256–65.

79. Hogan AM, Vargha-Khadem F, Kirkham FJ, Baldeweg T. Maturation of action monitoring from adolescence to adulthood: an ERP study. *Dev Sci* 2005; **8**: 525–34.

80. Davies PL, Segalowitz SJ, Gavin WJ. Development of response-monitoring ERPs in 7- to 25-year-olds. *Dev Neuropsychol* 2004; **25**: 355–76.

81. Fjell AM, Westlye LT, Amlien IK, Walhovd KB. A multi-modal investigation of behavioral adjustment: post-error slowing is associated with white matter characteristics. *NeuroImage* 2012; **61**: 195–05.

82. Kim EY, Iwaki N, Uno H, Fujita T. Error-related negativity in children: effect of an observer. *Dev Neuropsychol* 2005; **28**: 871–83.

83. Torpey DC, Hajcak G, Kim J, et al. Error-related brain activity in young children: associations with parental anxiety and child temperamental negative emotionality. *J Child Psychol Psychiatry* 2013; **54**: 854–62.

84. Brooker RJ, Buss KA, Dennis TA. Error-monitoring brain activity is associated with affective behaviors in young children. *Dev Cogn Neurosci* 2011; **1**: 141–52.

85. Grammer JK, Carrasco M, Gehring WJ, Morrison FJ. Age-related changes in error processing in young children: a school-based investigation. *Dev Cogn Neurosci* 2014; **9**: 93–105.

86. Arbel Y, Donchin E. When a child errs: the ERN and the Pe complex in children. *Psychophysiology* 2011; **48**: 55–63.

87. Holroyd CB, Coles MG. The neural basis of human error processing: reinforcement learning, dopamine, and the error-related negativity. *Psychol Rev* 2002; **109**: 679.

88. Crowley MJ, Wu J, Hommer RE, et al. A developmental study of the feedback-related negativity from 10–17 years: age and sex effects for reward versus nonreward. *Dev Neuropsychol* 2013; **38**: 595–12.

89. Mueller EM, Burgdorf C, Chavanon ML, et al. The COMT Val158Met polymorphism regulates the effect of a dopamine antagonist on the feedback-related negativity. *Psychophysiology* 2014; **51**: 805–09.

90. Meyer M, Bekkering H, Janssen DJ, de Bruijn ER, Hunnius S. Neural correlates of feedback processing in toddlers. *J Cogn Neurosci* 2014; **26**: 1519–27.

91. Santesso DL, Dzyundzyak A, Segalowitz SJ. Age, sex and individual differences in punishment sensitivity: factors influencing the feedback-related negativity. *Psychophysiology* 2011; **48**: 1481–89.

92. Grose-Fifer J, Migliaccio R, Zottoli TM. Feedback processing in adolescence: an event-related potential study of age and gender differences. *Dev Neurosci* 2014; **36**: 228–38.

93. Gao Y, Chen H, Jia H, Ming Q, Yi J, Yao S. Dysfunctional feedback processing in adolescent males with conduct disorder. *Int J Psychophysiol* 2016; **99**: 1–9.

94. Sheffield JG, Crowley MJ, Bel-Bahar T, et al. Reward-related neural activity and adolescent antisocial behavior in a community sample. *Dev Neuropsychol* 2015; **40**: 363–78.

95. Senderecka M, Grabowska A, Szewczyk J, Gerc K, Chmylak R. Response inhibition of children with ADHD in the stop-signal task: an event-related potential study. *Int J Psychophysiol* 2012; **85**: 93–105.

96. Bell AS. A critical review of ADHD diagnostic criteria: what to address in the DSM-V. *J Atten Disord* 2011; **15**: 3–10.

97. Trujillo-Orrego N. N200: an electrophysiological signal associated with inhibitory control, as an endophenotype candidate in attention deficit hyperactivity disorder. (In Spanish). *Rev Neurol* 2011; **53**: 35–43.

98. Spronk M, Jonkman L, Kemner C. Response inhibition and attention processing in 5- to 7-year-old children with and without symptoms of ADHD: an ERP study. *Clin Neurophysiol* 2008; **119**: 2738–52.

99. Yamamuro K, Ota T, Iida J, et al. Event-related potentials reflect the efficacy of pharmaceutical treatments in children and adolescents with attention deficit/hyperactivity disorder. *Psychiatry Res* 2016; **242**: 288–94.

100. Janssen TWP, Bink M, Geladé K, van Mourik R, Maras A, Oosterlaan J. A randomized controlled trial investigating the effects of neurofeedback, methylphenidate, and physical activity on event-related potentials in children with attention-deficit/hyperactivity disorder. *J Child Adolesc Psychopharmacol* 2016; **26**: 344–53.

101. Albrecht B, Banaschewski T, Brandeis D, Heinrich H, Rothenberger A. Response inhibition deficits in externalizing child psychiatric disorders: an ERP-study with the Stop-task. *Behav Brain Func* 2005; **1**: 1.

102. Cao J, Wang S, Ren Y, et al. Interference control in 6-11 year-old children with and without ADHD: behavioral and ERP study. *Int J Dev Neurosci* 2013; **31**: 342–49.

103. Karayanidis F, Robaey P, Bourassa M, Koning D, Geoffroy G, Pelletier G. ERP differences in visual attention processing between attention-deficit hyperactivity disorder and control boys in the absence of performance differences. *Psychophysiology* 2000; **37**: 319–33.

104. Spronk M, Vogel EK, Jonkman LM. No behavioral or ERP evidence for a developmental lag in visual working memory capacity or filtering in adolescents and adults with ADHD. *PLoS ONE* 2013; **8**: e62673.

105. Albrecht B, Brandeis D, Uebel H, et al. Action monitoring in boys with ADHD, their nonaffected siblings and normal controls: evidence for an endophenotype. *Biol Psychiatry* 2008; **64**: 615.

106. Zhang J-S, Wang Y, Cai R-G, Yan C-H. The brain regulation mechanism of error monitoring in impulsive children with ADHD—an analysis of error related potentials. *Neurosci Lett* 2009; **460**: 11–15.

107. Burgio-Murphy A, Klorman R, Shaywitz SE, et al. Error-related event-related potentials in children with attention-deficit hyperactivity disorder, oppositional defiant disorder, reading disorder, and math disorder. *Biol Psychol* 2007; **75**: 75–86.

108. van Meel CS, Oosterlaan J, Heslenfeld DJ, Sergeant JA. Telling good from bad news: ADHD differentially

affects processing of positive and negative feedback during guessing. *Neuropsychologia* 2005; **43**: 1946–54.

109. Holroyd CB, Baker TE, Kerns KA, Müller U. Electrophysiological evidence of atypical motivation and reward processing in children with attention-deficit hyperactivity disorder. *Neuropsychologia* 2008; **46**: 2234–42.

110. Moore DR, Pindus DM, Raine LB, et al. The persistent influence of concussion on attention, executive control and neuroelectric function in preadolescent children. *Int J Psychophysiol* 2016; **99**: 85–95.

111. Hogan AM, Vargha-Khadem F, Saunders DE, Kirkham FJ, Baldeweg T. Impact of frontal white matter lesions on performance monitoring: ERP evidence for cortical disconnection. *Brain* 2006; **129**: 2177–88.

112. Ozonoff S, Jensen J. Specific executive function profiles in three neurodevelopmental disorders. *J Autism Dev Disord* 1999; **29**: 171–77.

113. Levin HS, Hanten G. Executive functions after traumatic brain injury in children. *Pediatr Neurol* 2005; **33**: 79–93.

114. Brydges CR, Fox AM, Reid CL, Anderson M. Predictive validity of the N2 and P3 ERP components to executive functioning in children: a latent-variable analysis. *Front Hum Neurosci* 2014; **8**: 80.

115. Stevens C, Lauinger B, Neville H. Differences in the neural mechanisms of selective attention in children from different socioeconomic backgrounds: an event-related brain potential study. *Dev Sci* 2009; **12**: 634–46.

116. Drollette ES, Scudder MR, Raine LB, et al. Acute exercise facilitates brain function and cognition in children who need it most: an ERP study of individual differences in inhibitory control capacity. *Dev Cogn Neurosci* 2014; **7**: 53–64.

117. Lewis MD, Granic I, Lamm C, et al. Changes in the neural bases of emotion regulation associated with clinical improvement in children with behavior problems. *Dev Psychopathol* 2008; **20**: 913–39.

118. Duncan CC, Barry RJ, Connolly JF, et al. Event-related potentials in clinical research: guidelines for eliciting, recording, and quantifying mismatch negativity, P300, and N400. *Clin Neurophysiol* 2009; **120**: 1883–908.

119. Espinet SD, Anderson JE, Zelazo PD. Reflection training improves executive function in preschool-age children: behavioral and neural effects. *Dev Cogn Neurosci* 2013; **4**: 3–15.