[Review: 2 tables; 1[MD1] figure[SC2]]

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

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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[CP4][MD5]

[Abstract]

Executive functions are a ~~set~~ collection of ~~skills~~ 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 ~~the development of~~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 ~~positive deflection~~[CP6][MD7] [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.

[Boxed text to appear on page 2]

What this review adds[SC8][MD9]

- 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.

[Main text]

Executive functions are a ~~set~~ collection of cognitive processes that help us to ~~control and~~ 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] ~~as well as~~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 ~~skills~~.[4] Basic executive functions, ~~such as the ability to~~including the ~~-~~inhibit~~ion of~~ an inappropriate motor response, emerge early in life and subsequently lay down the foundations for ~~the~~later development of higher-order ~~more complex~~ executive functions, ~~such as reasoning,~~ 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 ~~emergence~~gradual integration of executive ~~skills~~functions and the prolonged ~~maturation~~development of the prefrontal cortex,[16–18] but it is also clear that executive functions ~~rely~~depend up~~on~~ a wider ~~brain network~~neural system.[19] In addition to magnetic resonance imaging (MRI) studies, event-related potential (ERP) measures ~~of~~

~~brain electrical activity~~ have contributed to our increasing comprehension of ~~understanding of~~ the developing ~~ment of the~~ neural substrates ~~of~~ underlying ~~executive functions~~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 ~~the development of~~ executive function~~s~~ 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 ~~research in~~ this ~~area~~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 I). As previously described, executive functions are often compromised by acquired brain injury and ~~across a range of~~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][CP10]

THE N200: INHIBITORY CONTROL AND INFERENCE SUPPRESSION[CP11]

The N200 component of ERP is ~~thought~~believed to reflect the cognitive control ~~required~~ necessary for successful inhibitory control and interference suppression.[29] Inhibitory control ~~refers to~~is the ability to ~~suppress~~ control a dominant, ~~automatic, or~~ 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 ~~resist~~ control interference from irrelevant~~distracting~~ stimuli and to inhibit a pre-potent 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 ~~as well as~~ 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/~~NoGo~~No-go[CP12][MD13] paradigm where the participant ~~has to~~ responds to a 'Go' stimulus but ~~not to~~ignores the ~~a~~ '~~NoGo~~No-go' stimulus, a larger peak is seen when '~~NoGo~~No-go' stimuli ~~share~~ have some similar dimensions ~~with~~ as the 'Go' stimuli, or when there is increased pressure to respond faster.[33,34][CP14][MD15] The N200 can also be observed in other paradigms, the most common of which are the Go/~~NoGo~~No-go task, the Stop-signal task, the Stroop task, and the Flanker task (see Table II for a detailed description of these tasks[CP16]).

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.[~~40~~35] In a combined Go/~~NoGo~~No-go–Flanker task with 14 young adults, the incongruous flanker ~~condition~~, ~~requiring~~ which requires ~~interference the~~ suppression of distracting information, elicited a more central~~ly~~ ~~distributed~~ topography ~~with~~and a ~~later~~ more delayed N200 peak than the ~~NoGo~~No-go condition ~~that required~~which requires ~~response~~ inhibition of a prepotent response.[~~36~~41] 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.[~~42~~37,38] However, 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.[29,44][CP17][MD18][39] The N200 has also been compared with the error-related negativity (ERN), [SC19] which is elicited in trials where commissions are made. Some research suggests that the ERN may reflect error detection or inhibition. However, 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.[45][40] One study showed that distinct cortical areas were associated with response inhibition, commission errors, and behavioural correction using electroencephalography (EEG [SC20] [MD21]) and functional MRI during a Go/No-go task.[41][6] Error detection was correlated with activation of the anterior cingulate and pre-supplementary motor area, whereas behavioural correction was related to with the anterior cingulate as well as the left prefrontal cortex.

Development of the N200[CP22]

Developmental research on the N200 typically reports a decrease in amplitude and latency with increasing age.[35][42–38][CP23][MD24][45] However, some studies have observed no age effect on N200 responses.[39][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.[38][45] Lamm et al.[38][CP25][MD26][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 ~~decreased~~diminished with age but were not ~~predicted by~~related to task performance.

THE P300: INFORMATION PROCESSING[CP27]

The P300 is a ~~late~~ positive waveform that appears at ~~a latency of~~ 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 ~~that it is a~~on its description as a neurophysiological index of information processing and updating in working memory ~~updating~~.[48] ~~Both t~~The latency and amplitude of the P300 have both been ~~associated with~~connected to behavioural ~~performance~~ 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' ~~is~~ activate~~s~~d in passive oddball tasks ~~in response~~as a reaction to novel ~~targets~~stimuli that do not ~~require~~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 ~~is required to~~ respond~~s~~ to the novel stimulus, often by button press~~ing a button~~.[49] The P3a is observed when a task requires orienting or novelty detection and has a frontocentral topography. The P3a is likely to originate ~~from sources~~ in the frontal ~~lobe~~ 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 ~~NoGo~~No-go P3 is thought to reflect inhibitory control as it is observed in response to distractor items[~~40~~5,52] and shows a different topography to P3a and P3b with maximum peaks in centro-parietal channels.

Development of the P300[CP28]

It has been proposed that the latency and amplitude of the P300 ~~index~~ reflect different ~~aspects of brain maturation~~developmental processes in the brain. ~~, with L~~latency is thought to index~~ing~~ neural speed ~~or brain~~ and efficiency and amplitude ~~indexing reflects neural power or~~growing cognitive resources, ~~which~~ 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] However, a recent systematic review by van Dinteren et al.[60][CP29][MD30][MD31] 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 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[CP32]

The contralateral delay activity (CDA) is a lateralized ERP over the parietal cortex that ~~is indicative of~~reflects the ~~number~~ amount of target~~s or~~ and distractor stimuli~~s~~ that are ~~selectively~~ encoded or maintained from one hemi-field ~~of~~ 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 ~~refers to~~is the ability to temporarily mentally ~~keep~~store and manipulate information ~~in mind and mentally manipulate it~~. 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 ~~amount~~ number of units of information that can be kept in working memory, is ~~an~~ important ~~factor for~~in the development of academic ~~skills~~ skills and for general learning ~~such as reading and mathematics and fluid intelligence~~.[62]

Development of the CDA[CP33]

~~Developmental studies~~It is ~~have consistently shown~~typically reported that mature working memory capacity is ~~only reached during~~achieved by ~~late childhood or~~ adolescence. ~~Whereas~~ Although some ~~studies report~~research has provided evidence for mature visuospatial working memory capacity ~~around~~ by 10 to 12 years,[63,64] other ~~studies~~ report~~s~~ suggest that ~~mature~~ adult-like ~~working memory~~ capacity is not reached before ~~the age of~~ 16 years.[65] ~~Such developmental~~These differences ~~seem~~ in findings

are though to reflect ~~to depend on~~ the level of executive control ~~processes~~ that ~~are needed~~is required to perform ~~a specific working memory~~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 ~~12- to 16-year-olds~~teenagers (*n*=21) than adults, and that CDA amplitudes were positively ~~correlated~~associated with successful ~~performance measures of~~ 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 front~~oal~~o-parietal 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[CP34]

ERPs have also been used to investigate other preparatory and inhibitory processes during cued attention and working memory tasks. In tasks where children are ~~instructed to make an eye movement~~required to look towards a cued ~~target~~ location and to ignore ~~elements at~~ a distractor location, a series of ERP responses are observed. Early directing attention negativity, possibly reflecting early parietal

activ~~ity~~ation within the frontoparietal[SC35][MD36] network, precedes frontal ~~activation~~ activity reflected by the anterior directing-attention negativity. After these ~~These~~ responses, ~~are then followed by~~ a late widespread contralateral positivity is observed, which is thought to represent ~~both~~ the oculomotor programming of the ~~upcoming~~ planned eye movement as well as ~~attentional~~ the orienting of attention.[68] Studies show that these early attentional responses are related to working memory abilities. Shimi et al.[69] ~~demonstrated~~ report that age-related differences in ~~the temporal dynamics of~~ attention~~al~~ orienting ~~mechanisms~~ processes before and after encoding ~~items~~ stimuli in visual working memory (VWM)[CP37][MD38] can ~~contributed to~~ explain differences in VWM performance between ~~children and adults~~ the developing brain and the adult brain. ~~Importantly, individual d~~ Differences on an individual level in the ~~temporal dynamics of the preparatory~~ attention~~al~~ orienting ~~mechanisms~~ processes before encoding can be ~~that~~ bias~~ed~~ so that ~~the encoding of~~ relevant items are more efficiently encoded into VWM ~~discriminated between~~ in children with high ~~and low~~ VWM capacity.[69] A further study showed that children with large cue~~ing~~ benefits in VWM capacity elicite~~dd~~ ~~an~~ adult-like ~~contralateral negativity~~ responses ~~following attentional~~ after selection of the ~~to-be-encoded item~~ stimuli, whereas children with low capacity did not elicit a contralateral negativity,~~ whereas children with low VWM capacity did not elicit this component~~.[4383]

ERROR-RELATED NEGATIVITY: RESPONSE MONITORING[CP39]

~~Another important aspect of executive function is t~~ 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 (~~such as the~~ Go/~~NoGo~~ No-go paradigm, Eriksen Flanker task, and the Simon task) are marked by

specific ERP components following error. ~~Error-related negativity (ERN or Ne[SC40][MD41])~~The ERN is a negative deflection between 80 and 150 milliseconds with maximal amplitudes over fronto-central channels[70] ~~that is likely~~thought to be ~~generated~~produced ~~by~~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 ~~is followed by~~appears before a positive deflection (error-related positivity, Pe) with a maximum response over centro-parietal channels with a peak between 200 and 500 milliseconds.[73] In contrast to ERN, the Pe ~~does~~ depend[SC42][MD43]s on conscious error awareness and is not present in all error-trials. ~~For this reason, the Pe is thought to reflect the conscious evaluation of errors.~~[74] ~~At the~~On a behavioural level, increases in response accuracy, reaction time, and a reduction in response variability have been found ~~in studies~~ using the Go/~~NoGo~~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[CP44]

In parallel with improvements in task performance, increases in ERN amplitude have been documented. ERN can be ~~reliably~~ detected ~~in children~~ 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 girls and about 15 years for boys.[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 ~~neural~~ generators in the anterior cingulate ~~cortex~~ in children~~, adolescents,~~ 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/~~NoGo~~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/~~NoGo~~No-go task.[83] ~~Interestingly, this~~ 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 ~~influence~~ 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] However, studies comparing age groups from mid-childhood to adolescence do not find ~~significant~~ 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[SC45][MD46].

FEEDBACK-RELATED NEGATIVITY: FEEDBACK MONITORING[CP47]

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 medio-frontal electrodes with a peak around 270 milliseconds after feedback onset.[87] This component is called described as the feedback-related negativity (FRN). The amplitude of this component is consistently found to be larger in response to negative feedback when compared withthan positive feedback.[87] The dorsal anterior cingulate cortex is the proposed generator 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[CP48]

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 girls have been found to showed higher FRN amplitude in response to wins[91] and smaller amplitudes for losses,[92] whereas boys 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[CP49]

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[CP50]

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/~~NoGo~~No-go tasks in children with ADHD ~~have been interpreted as~~are thought to reflect early signs of atypical attention development.[98,99] Further, reduced P200 and N200 effects during Go/~~NoGo~~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.[10~~1~~5][CP51][MD52] However, differences are not consistently found across studies with either increased, reduced, or absent amplitude and latency effects in different studies.[10~~1~~2] 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.[10~~2~~3] 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[CP53]

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 between 12 and 16 years old 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.[10~~1~~3] The CDA showed that ~~, initially, all groups encoded and maintained distracting information, but~~ adults were able to more efficiently remove distracting ~~information~~ details from memory later in the retention interval, resulting in better working memory ~~performance~~. ~~The only effect of~~ ADHD diagnosis ~~was~~ was related to smaller CDA amplitude in adolescents and adults with ADHD than

in the comparison group when maintaining a low ~~one item~~ load, which ~~was possibly related to~~could reflect an inability to ~~keep~~maintain focused attention ~~focused at~~to cued stimuli ~~with~~when there are low task demands. Thus, overall, the ERP results discussed here suggest ~~that~~no differences in ~~the development of~~filtering efficiency and visuospatial working memory storage capacity in adolescents and adults with ADHD ~~is not different to typically developing peers~~.

ERP MARKERS OF REWARD PROCESSING AND RESPONSE MONITORING IN ADHD[CP54]

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 [SC55] [MD56] with ADHD as well as intermediate amplitudes in unaffected siblings than in a matched comparison group.[10~~5~~4] 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[CP57]

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/~~Nogo~~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 ~~affected~~ impacted by the ~~presence of~~ 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 ~~individual's~~ 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][CP58][MD59] recently showed that the N200 difference waveform and the P3b mean 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. However, 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 ~~taken into~~ consider~~ed~~ation in the development of future ~~research~~ 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 ~~considerable~~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 ~~to assist in~~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[CP60].

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Table I: Event-related potential components of executive functioning

Component	Peak latency time window (ms)	Maximum amplitude topography	Task paradigms	Proposed neural generators	Associated cognitive function	Illustrative examples
N200	180–325	Frontocentral	Auditory Oddball, Go/~~NoGo~~No-go, Eriksen Flanker	Auditory cortex, inferior frontal gyrus	Updating of stimulus presentation in memory trace, response inhibition, conflict monitoring	Figure 1 in Lamm et al. ~~38~~45
Error-related negativity	80–150	Frontocentral	Go/~~NoGo~~No-go, Eriksen Flanker, Simon task following an erroneous motor response	Anterior cingulate cortex, dorsolateral prefrontal cortex	Unconscious error monitoring, conflict monitoring	Figure 1 in Richardson et al.[78]
Error-related positivity	200–500	Centro-parietal	Go/~~NoGo~~No-go, Eriksen Flanker, Simon task following an erroneous motor response	Posterior cingulate cortex	Conscious error monitoring	Figure 1 in Arbel and Donchin[86]
Feedback-related negativity	230–270	Frontocentral	Go/~~NoGo~~No-go, Eriksen Flanker, Simon task following outcome feedback	Anterior cingulate cortex	Outcome monitoring, reinforcement learning	Figure 2 in Gao et al.[93]

Contralateral delay activity	300–1000	Parietal contralateral to stimuli	Visual working memory tasks	Intraparietal sulcus	Visuospatial working memory	Figure 2 in Sander et al.[67]
P3a	250–280	Frontocentral	Oddball	Frontal lobe, hippocampus	Attention, orienting, novelty detection	Figure 1 in Fuchigami et al.[57]
P3b	250–500	Centro-parietal	Task-relevant Oddball, dual task paradigms	Temporal lobe, parietal lobe, ACC	Attention, cognitive workload	Figure 1 in Fuchigami et al.[57]
Early directing attention negativity	250–325	Centro-parietal	Spatial cueing paradigm, visual search paradigm	Parietal lobe	Visuospatial orienting, cue processing	Figure 4 in Shimi et al.[69]
Anterior directing attention negativity	325–425	Frontocentral	Spatial cueing paradigm, visual search paradigm	Frontal lobe	Attention control deployment	Figure 4 in Shimi et al.[69]
Late directing attention positivity	550–700	Parietal	Spatial cueing paradigm, visual search paradigm	Parietal lobe, occipital lobe	Oculomotor programming, attentional orienting	Figure 4 in Shimi et al.[69]

Table II: Commonly used classical experimental paradigms[a]

Go/~~NoGo~~No-go task	In the Go/~~NoGo~~No-go task, participants are trained to respond quickly to one type of stimulus and withhold the response when a deviant stimulus is presented. Visual or auditory stimuli may be used in this paradigm. For example, in a visual version of this task the participant may be required to press a button in response to every letter except the letter 'x'.
Stop-signal task	The Stop-signal task is a variation of the Go/~~NoGo~~No-go task. The participant is instructed to refrain from responding to a repetitive task whenever a stop sign appears during the task.
Stroop task	The Stroop task is designed to investigate the 'Stroop effect'. This is the effect that interference from distracting or conflicting information has on the reaction time in a task. For example, in the classical Stroop paradigm, the colour of ink is different to the name denoted by the text, which slows down the participant's reaction or causes them to make errors.
Oddball paradigm	In oddball tasks, an infrequent stimulus (often referred to as the oddball or deviant stimulus) is presented among a series of frequent stimuli (or standards). The ratio of infrequent to frequent stimuli can be altered to measure the impact of probability on the response. Oddball paradigms have been used to measure executive function in different modalities, including vision, audition, and somatosensation. For example, in a visual task the participant may have to press a button in response to an infrequent stimulus appearing on screen, whereas in an audio version of the task the participant will be instructed to press a button in response to hearing an infrequent stimulus presented through loudspeakers or headphones.
Eriksen Flanker task	The Eriksen Flanker task requires participants to quickly press a button following the direction of a central arrow presented on a screen. In the congruent condition, the central arrow is surrounded by arrows pointing in the same direction. In the incongruent condition the central arrow is surrounded by arrows that point in the opposite direction. When the surrounding arrows are pointing in a different direction to the target stimulus, it should be more difficult for the participant to inhibit this distracting information and may slow down their response or cause them to respond incorrectly.
Simon task	In the Simon task, participants learn to press a button with either their left or right hand following a stimulus on the screen. The location of the stimulus can either be congruent or incongruent with the required response.

[a]These paradigms are commonly used in conjunction with event-related potentials to investigate neural responses associated with executive function. Typically for the visual version of these tasks, the stimuli are presented on computer screens and the participant presses buttons in response to stimuli, as instructed.

Figure 1: Fronto-parietal or 'executive' network (highlighted) and associated event-related potential components depicting the proposed underlying regions.[SC81][MD82]