

The Development of Visual P3a and P3b

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The relationship of visual P3a and P3b to age and neuropsychological performance was investigated in 26 healthy children (6.8–15.8 years) and 129 adult volunteers (20.0–88.8 years). Within the sample of children, an effect of age on midline topography was observed, with higher frontal amplitudes in the youngest compared to the oldest children. Increasing age was associated with lower P3a and P3b amplitude and shorter P3b latency at Fz. Performance on neuropsychological tests (matrix reasoning from WASI, digit span from WAIS, word order and hand movement from Kaufman) was only weakly associated with measures of P3a and P3b. The analyses were then repeated with the full life-span sample ($n = 155$). It was found that for P3a, amplitude decreased and latency increased with age. For P3b, the pattern was more complex, with a nonlinear amplitude reduction and no latency change with age. It appears that the development of P3a in children represents the start of processes that later continue in the adult life-span, but that the automatic processes indexed by P3a seems to mature earlier than the controlled processes reflected by P3b. Finally, it was demonstrated that the relationships between neuropsychological test scores (matrix reasoning, digit span) and P3 parameters were complex, following a mix of linear and nonlinear patterns. It is suggested that the neuropsychological significance of the different P3a and P3b parameters may change from childhood to the adult life-span.

This study focuses on how visual P3a and P3b develop and change in childhood, relative to adulthood, and how this is related to cognitive performance. P3a and P3b are distinct positive-going components of event-related potentials (ERP). P3a is assumed to be part of the orienting reflex (Friedman, Cycowicz, & Gaeta, 2001; Escera et al., 1998; Courchesne, 1978) and is thought to reflect relatively automatic processing of unexpected stimuli (Fuchigami et al., 1995), but it has also been suggested that the component may reflect inhibition (Jonkman, Lansbergen, & Stauder, 2003). The component may be viewed as an electrophysiological manifestation of distractibility or involuntary attention shifts (Alho et al., 1998; Gumenyuk et al., 2004; Squires, Squires, & Hillyard, 1975), since P3a is typically elicited in discrimination paradigms by stimuli extraneous to the task, and studies have shown prolonged reaction times to target stimuli following irrelevant P3a-eliciting sounds (Escera et al., 1998; Grillon et al., 1990; Woods, 1992). The P3a-eliciting stimuli can either be novel or repeated, but typically deviate much from standard and target stimuli on central characteristics. In adults, P3a has fronto-central maximum amplitude (Friedman, Cycowicz, & Gaeta, 2001; Katayama & Polich, 1998; Schröger & Wolff, 1998) and a peak latency of 300–450 msec in visual paradigms (Walhovd & Fjell, 2004). P3b, in contrast, is thought to reflect controlled information processing, in the sense that it is typically elicited by task-relevant stimuli to which the participant is actively paying attention (Jonkman, Lansbergen, & Stauder, 2003; Friedman, Cycowicz, & Gaeta, 2001; Fuchigami et al., 1995). P3b has parietal maximum and peak latency around 350–450 msec when studied in adults (Courchesne, Hillyard & Galambos, 1975; Fjell & Walhovd, 2004).

Research has mapped how P3a and P3b change during the adult life span (e.g., Fjell & Walhovd, 2004; 2002; Friedman, Kazmerski, & Fabiani, 1997; Iragui et al., 1993; Barrett, Neshige, & Shibasaki, 1987). Most studies have focused on P3b (Key, Dove, & Maguire, 2005), while less research has focused on P3a adult life span changes (e.g., Fjell & Walhovd, 2003; Friedman, Simpson, & Hamberger, 1993). Fjell and Walhovd (2004) have demonstrated electrophysiological age changes in visual P3a in a large-scale study ($n = 103$, aged 20–92 years), indicating longer latency and lower amplitude of P3a with increasing age. In this study, visual P3b was not associated with age to the same degree. Paradigm characteristics may have been responsible for diminishing P3b in the above-mentioned study. In a smaller auditory study ($n = 31$, aged 22–95), Walhovd and Fjell (2001) found comparable age changes in P3a and P3b. Since P3a has a more frontal scalp distribution than P3b, and aging lead to greater volumetric neuroanatomical reductions in anterior relative to other brain areas (e.g., Allen et al., 2005; Cowell et al., 1994; Jernigan et al., 2001), one may speculate P3a could be more vulnerable to the aging process. This may be accompanied by a decrease in frontally based cognitive functions and reflected by electrophysiological changes in P3a.

As a parallel to the frontal age-changes, Gogtay et al. (2004), in a study of brain maturation found that higher-order brain areas, the frontal lobes, matured after lower-order areas, e.g. somatosensory areas. This fits with knowledge about neuropsychological development. Other studies of frontal lobe development have found grey matter increases in pre-adolescence, peaking around the age of 12 years (Giedd et al., 1999), followed by maturational reductions in post-adolescent years (Sowell et al., 1999). One might expect such behavioral and structural development to be accompanied by electrophysiological changes. However, while electrophysiological aging has received much attention, less is known about electrophysiological development in childhood and adolescence, and relatively few studies have investigated how and when P3a and P3b develop and mature in children.

When P3b has been studied in nonpathological groups of children (e.g., Segalowitz & Davies, 2004; Harada et al., 2002; Van der Stelt et al., 1998; Oades, Dittmann Balcar, & Zerbin, 1997; Thomas & Nelson, 1996; Yordanova & Kolev, 1997), one has generally found broader peaks compared to adults, and decreased latency with increasing age, with more inconsistent results concerning amplitude change during childhood. However, Harada et al. (2002) reported decreased P3b amplitude with increasing age across 6–81 years, and Berman et al. (1990) have similarly reported decreasing amplitude with age across 7–30 years ($n = 42$, aged).

Research on P3a in children is relatively scarce. Most studies have focused on children with different pathological conditions, including ADHD (Banaschewski et al., 2003; Brandeis et al., 2002; Taylor et al., 1993), autism (Gomot et al., 2002), and extreme prematurity (Lavoie et al., 1997). There is surprisingly little research on the normal development of P3a. Further, while pathology studies include both auditory (Gomot et al., 2002; Lavoie et al., 1997) and visual (Banaschewski et al., 2003; Brandeis et al., 2002; Taylor et al., 1993) P3a, there have only been studies of the normal development of auditory P3a. One such auditory and exceptionally large study is that of Fuchigami et al. (1995; $n = 108$, ages 1–21 years for P3a, $n = 175$, ages 4–21 years for P3b). The results from this study indicated that the latency of both P3a and P3b decreased with age, and that P3a matures earlier than P3b, as latency for P3a stabilized around 12 years, while latency for P3b continued to decrease until 17 years. However, Segalowitz and Davies (2004; $n = 77$, ages 7–25) found results pointing in a different direction, where the auditory P3b was similar in children and adults, with a posterior maximum, while the auditory P3a showed inconsistent patterns in the children. The children were 13 years old before the P3a started to resemble that of the adults. Further, a recent study by Èeponienë et al. (2004, $n = 11$, ages 9–13 yrs) reported that overall amplitude of auditory P3a appear relatively mature in children aged 9–13 years. However, the topography seemed to differ from what is reported in adult samples when dividing the component into early and late P3a, where late P3a (250–350 msec poststimuli) lacked

frontal predominance among the children. Gumenyuk et al. (2004; $n = 26$, ages 8–13 years) reported results that indicated that amplitude for the late auditory P3a differed between the youngest and oldest children in their sample, where the 8–9-year-olds showed greater amplitude on late P3a than was the case for the 12–13-year-olds. These researchers also reported a centrally dominant scalp distribution.

The above research has only applied auditory stimuli to elicit P3a in children. Further, the child studies generally lack an adult life span sample with which the results can be compared. In order to accurately map the development of P3a and P3b during the life-span, one must apply the same paradigm and the same stimuli in a large life-span sample that include children as well as adults and elderly. The present study attempts to shed light on possible age changes in visual P3a and P3b during the normal life-span. Based on previous studies of childhood and adult life-span development, the following hypotheses were formulated:

(1) P3a and P3b will show differential maturational patterns. This is based on the view that P3a indexes more automatic and fundamental cognitive processing than the P3b, which is assumed more related to controlled processing. This hypothesis will be tested by comparing latency, amplitude and topography within the child sample, and across the full life-span.

(2) Neuropsychological performance will be related to P3a and P3b latency and amplitude. Previous studies on P3 and neuropsychological performance in adult life-span samples have found positive correlations between test scores and amplitude, and negative correlations between scores and latency. It is not given that the same pattern will be seen in children, however. For instance, increased amplitude may be a function of immaturity of the cognitive system, linked to disinhibition of attention. In such cases, P3-neuropsychology correlations may be in the opposite direction of what is found in adults. This may yield complex relationships between test and P3 parameters, possibly including quadratic relationships.

METHODS

ERP Stimuli

We used a three-stimuli visual oddball task, with 210 trials and .10 target and .10 distractor probability for the adult sample, and the same oddball task with 250 trials, but with identical probabilities for the children. The greater number of trials in the child sample reflects an assumption of greater task demands for the children as compared to the adults, as well as an assumption of possible lower signal-noise ratio in the child recordings due to movement artefacts. That is, we assumed that a

lesser number of trials would be included in the analysis relative to the total number or trials for children compared to adults. Counting of accepted trials in each of four age groups, showed similar numbers across groups. The mean number of accepted trials for distractor and target were 21.88/22.50 (age 6–19), 19.84/19.70 (age 20–44), 20.12/20.22 (age 45–70), and 19.86/19.08 (age 70–90). When dividing the youngest group by the median age (12.4 years), the accepted trial numbers for distractor/target were 22.58/22.50 and 21.29/22.00 for the youngest and the oldest child group, respectively. However, number of accepted trials still correlated significantly with age ($r = -.22$ for distractor, $-.19$ for target, $p < .05$, whole sample). Still, in light of the small actual difference between the groups, we decided not to exclude trials in the younger participants.

The paradigm used is a variation of one used by Comerchero and Polich (1999) which has been shown to elicit P3a (Comerchero & Polich, 1999; Polich & Comerchero, 2003). Demiralp et al. (2001) have demonstrated that such a paradigm produced virtually identical P3a potentials as those obtained by using novel stimuli, which has been employed traditionally. The component elicited by such a three-stimuli oddball task seems to be the same component as novelty P3a (Simons et al., 2001). In the present task, the standard stimulus, which the participant was asked not to respond to, was a blue elliptic shape with a height of 12 cm and a width of 8 cm. The target stimulus, to which the participant was asked to press a button, was a blue elliptic shape with height and width of 17.5 and 14.5 cm, respectively. The distractor stimulus, which the participant was told to ignore, was a large blue rectangle of 21×17 cm. The stimuli were presented on a 21-in. computer screen with a black background, and the distance from the participants' eyes to the screen was about 100 cm, with a visual field of about $9^\circ \times 7^\circ$, $10^\circ \times 8^\circ$, and $12^\circ \times 10^\circ$ for the standard, target, and distractor stimuli, respectively. The stimuli were presented for 0.5 sec, and inter-stimuli-interval was set to 1.5 sec offset to onset. Before recording, an example task including nine standard and two target stimuli was presented to ascertain that all participants could discriminate targets from standards, and to prime them for the task. The small difference between the target and the standard, and the large difference between the target and the distractor in the present task, were chosen to maximize the P3a curve (Comerchero and Polich, 1999). In a series of experiments, Comerchero and Polich (e.g., 1999) have demonstrated that stimulus context as defined by the target/standard discrimination difficulty determines P3a generation: When discrimination was easy, P3 amplitude was larger for the target (P3b) than the distractor (P3a), and both had parietal maximums. When discrimination was difficult, distractor amplitude was larger and earlier than the target P3 over the frontal/central electrode sites, whereas target amplitude was larger parietally and occurred later. The present task involved a rather difficult target/standard discrimination. However, as it was studied in a life span sample, and since we needed to ensure that different age groups could all perform the

task satisfactorily, we have employed a somewhat easier discrimination task than the one originally used by Comerchero and Polich (1999).

ERP Procedures

Subjects were seated in a reclining chair within a sound attenuating recording chamber. The electrodes were placed in accordance with the international 10–20-system. For the adult sample, a total of 20 electrodes (Ag/AgCl) were used for recording; Fp1, Fp2, F7, F3, Fz, F8, F4, T3, C3, Cz, C4, T4, T5, P3, Pz, P4, T6, O1, Oz, and O2, referred to the left mastoid. For the child sample, a total of 10 electrodes (Ag/AgCl) were used; F3, Fz, F4, C3, Cz, C4, P3, Pz, P4 and Oz, referred to the left mastoid. In this paper, results from the midline electrodes only will be reported, because the P3 is generally most pronounced at these electrode sites (Fjell & Walhovd, 2004). Results from other electrodes would also be of interest, since scalp topography may change during development, and such changes may also be seen at other than midline sites (e.g., Karhu et al., 1997). Still, in consideration of the length and complexity of the present manuscript, analyses of additional electrode sites are not reported here. For both samples, a VEOG channel was obtained by placing one electrode above and one below the left eye, and ground was placed anteriorly on the right side, with equal distances to Fz, F4, Cz, and C4. Inter-electrode impedance was generally less than 10 kOhm. For the recording of EEG activity, A/D rate was 500 Hz, and filter-setting was 0.10 Hz (high pass) and 70 Hz (low pass). In addition, a 50 Hz notch filter was applied. The signals were amplified by a SynAmp DC amplifier (Neuroscan Inc., El Paso, TX). Epochs were analyzed offline and were rejected from averaging if amplitude exceeded an absolute value of ± 110 microVolts relative to baseline. Eye blinks were corrected statistically in accordance with Semlitsch et al.'s (1986) recommendations. Ideally, an additional horizontal EOG channel should have been used to remove artifacts resulting from horizontal eye movement. However, other data ($n = 10$) from our lab have shown correlations between HEOG-corrected and noncorrected amplitudes in the range of $r = .99$ to $r = .98$ at the central electrode (see also Fjell et al., 2005). Thus, it is very unlikely that the lack of HEOG correction will have any substantial impact on the data presented in this article. For the adult sample, data were discarded for Fz for one participant, Cz for another, and Pz for a third due to bad electrodes. Averaging was performed for targets and distractors separately. EEG was segmented in epochs of 900 msec duration (-100 msec to 800 msec relative to stimulus onset). All data average files were digitally filtered (15 Hz low pass) and baseline corrected before statistical measures of component latency or amplitude were made. Neuroscan software was used to present stimuli as well as for recording and analyses of EEG-activity. P3s were determined by algorithmically (rather than by investigator's evaluation), in accordance with Pfefferbaum et al.'s (1990) recommendations, and were defined as the most positive peak within 250 and 650 msec post

stimulus. P3a was defined in the each participant's average wave for the distractor stimuli, and P3b was defined in each participant's average wave for the target stimuli.

Cognitive Tests

In the adult sample, all participants were examined with the Norwegian version of the Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999). Only participants with an IQ score of 85 or above were included. The matrix reasoning subtest of the WASI was administered to the child sample, and children had to score no more than 1.5 standard deviation (*SD*) below the mean ($T = 35$) to be included. None of the children scored below this value. The "matrix reasoning" and "digit span" from the Norwegian version of WASI, as well as the "word order" and "hand movement" from the Kaufman battery (Kaufman & Kaufman, 1983) were administered to the child sample. The Kaufman battery has norms up to 12.5 years only. For the oldest children, in order to avoid roof-effects, we prolonged the distraction interval on "word order" from 5 to 10 sec, while "hand movement" was kept identical for both age groups. For all tests, raw scores were used for further analyses.

Sample

Table 1 summarizes the full sample and sub-samples satisfying inclusion criteria (see above). The included participants were 155 healthy volunteers (88 females), age range 6 years 9 months through 88 years 9 months. The participants in the adult sample (age range 20.0–88.8 years) were recruited among employees from a

TABLE 1
Summary of the Demographic Variables in the Different Samples used in
the Statistical Analyses

	<i>n</i>	<i>% females</i>	<i>Age</i>		<i>Matrix Reasoning</i>	
			<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
<i>Sub groups in the child sample</i>						
Group 1: 6.8–12.4 years	13	69.2	11.0	1.8	52.1	8.7
Group 2: 12.5–15.8 years	13	69.2	14.1	1.2	48.5	5.2
<i>Sub groups in the life span sample</i>						
1. Child sample (6.8–15.8 years)	26	69.2	12.6	2.2	50.3	7.3
2. 20–44 years (20–44.5 years)	50	62.0	27.7	5.9	56.8	5.6
3. 45–69 years (46–69 years)	43	48.8	56.8	7.1	59.4	6.0
4. 70–90 years (70–88.8 years)	36	50.0	77.8	5.1	55.7	10.0
<i>Full sample</i> (6.8–88.8 years)	155	56.8	44.9	44.9	56.3	7.7

local hospital, and through charity organizations, activity centers for elderly, and newspaper ads. They were given a moderate sum of money to refund possible costs related to participation. The children (age range 6.8–15.8 years) were recruited by distributing information letters in schools and at internet sites for youth and parents. Children were given a movie gift card as a surprise at the end of the session.

All participants were community dwellers, screened for diseases and traumas known to affect CNS functioning by a set of health-related questions. Participants were required to have normal or corrected to normal vision and were told to wear their glasses if using such. Criteria for exclusion were neurological conditions or use of medication known to influence CNS functioning. For the child sample, the parents completed a form asking if the child had ever been referred to the school counseling service, and whether they had worries regarding the child's activity level or functioning at school. Children were excluded if parents answered "yes" to any of these questions. One child was excluded from all analyses by this criterion.

For the adult sample, exclusion criteria for the behavioral data from the ERP task were set to > 20% incorrect responses to the standards, > 25% incorrect response to the distractors or < 80% hits. Four adult participants were excluded by the above mentioned criteria, reducing the adult sample to 129 persons. The employed experimental paradigm was originally developed for adults, and children were only included in the study at a later point. Unfortunately, this made it unrealistic to equate discrimination difficulty or performance rates across age groups. The discrimination task was assumed, and turned out to be, more difficult for the children. It was thus assumed that in children, a lower hit rate did not to the same extent as in adults reflect misunderstanding of task instructions or failure to pay attention. The exclusion criterion for the children was thus set to < 60% hits, and > 20% incorrect responses to standards or distractors. Two children were excluded by this criterion. Two additional children were excluded due to movement artefacts in the recordings. Finally, two children were excluded from the P3b analyses only, due to muscle artefacts linked to responses to the targets. Application of the described criteria thus yielded a total number of 26 children for the P3a analyses and 24 children for the P3b analyses. In this sample, mean proportion of target hits minus proportion of false alarms (incorrect responses) were .79 (*SD* .19), .97 (*SD* .04), .96 (*SD* .05), and .91 (*SD* .08) for the child, young adult, middle aged, and old groups, respectively.

Statistical Analyses

Statistical analyses were performed separately for the child and full sample, to see if the same tendencies reported for the adult sample (Fjell & Walhovd, 2004) would be evident in these two samples. The full sample was also divided into four age groups, and statistical analyses were performed to compare these groups and investigate possible differences in P3a and P3b.

Child sample. ANOVAs with two age groups \times three electrodes (Fz, Cz, Pz) were performed to investigate the topography of P3a and P3b, and possible differences in topography for the oldest versus the youngest children. When appropriate, Greenhouse-Geisser corrections were applied. Voltage normalization was then performed as recommended by McCarthy and Wood (1985) for each child in the two age groups, by subtracting the group minimum from each person's peak value, and divide by the group range. Thus, the participants in the group with the highest minimum value and the largest range would get the largest correction of their amplitude values. Additional ANOVAs were performed with these normalized values to study interaction effects of electrode and age group. Linear regressions with age as the independent variable and amplitude and latency of P3a and P3b as dependent variables were performed to see how age was related to these measures. To control for possible curvilinear functions of age on P3a and P3b, additional multiple regression analyses were performed with age and square of age (age^2) as independent variables. A significant contribution from age^2 would indicate that a nonlinear fit best accounts for the variance in the data. Finally, linear regressions were performed with the raw scores from the different tests as independent variables and P3a and P3b amplitude and latency as dependent variables.

Full sample. As for the children, ANOVAs with four age groups \times three electrodes (Fz, Cz, Pz) were performed to study topographical differences of P3a and P3b in the different age groups. When appropriate, Greenhouse-Geisser corrections were applied. Voltage normalization was then performed as for the children and additional ANOVAs were performed with these normalized values, to study interaction effects between electrode and age group. Stepwise regressions with P3a/P3b latency/amplitude as dependent and age, age squared (age^2), and gender as independent variables were performed, where probability of F to enter was less than or equal to 0.05, and probability to remove was greater or equal to 0.1. In cases where square of age was significant in step 1, age was forced into the equation, since square of age gives no meaning without age being a simultaneous predictor. Finally, matrix reasoning was used as dependent variable in a series of stepwise regressions, with P3a/P3b amplitude/latency and the square of P3a/P3b amplitude/latency in turn as predictors.

RESULTS

Grand average ERP-curves for the full sample are presented in Figure 1, and mean amplitudes and latencies are presented in Table 2. A sharp positive deflection, peaking between 400 and 700 msec, was visible for all age groups at all electrodes, both for P3a and P3b. Grand average ERP-curves for the child sample divided into two subgroups are shown in Figure 2. The statistical analyses for the child sample will be reported first, and the analyses for the full sample last.

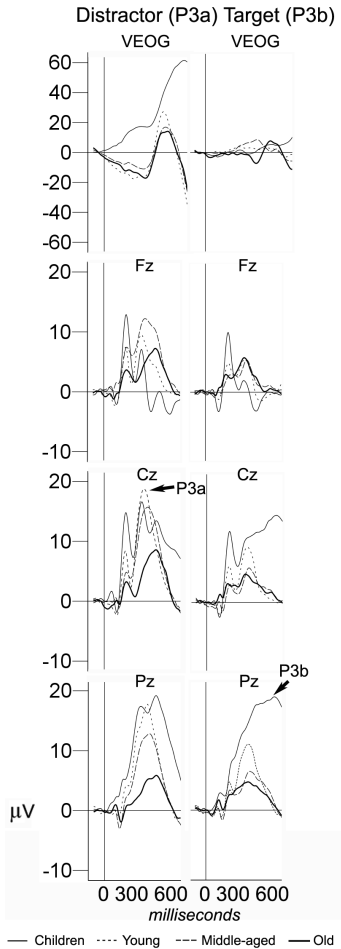


FIGURE 1 Grand average ERP curves for the full sample. The figure shows grand average ERP curves for P3a (left column) and P3b (right column), divided by electrode and age group. The typical P3a and P3b curve is marked with an arrow. The group of children had a mean age of 12.6 years (6.8–15.8 years, $n = 26$), the young group had a mean age of 27.7 (20.0–44.5, $n = 50$), the middle-aged group had a mean age of 56.8 years (46–69 years, $n = 43$), and the old group had a mean age of 77.8 years (70.0–88.8 years, $n = 36$).

Child Sample

ANOVAs with two age groups \times three electrodes (Fz, Cz, Pz) showed a main effect of electrode on P3a amplitude, $F(1.255, 30.11) = 24.45, p = .000$, P3b amplitude, $F(1.256, 27.63) = 37.70, p = .000$, and P3b latency, $F(1.613, 35.48)$

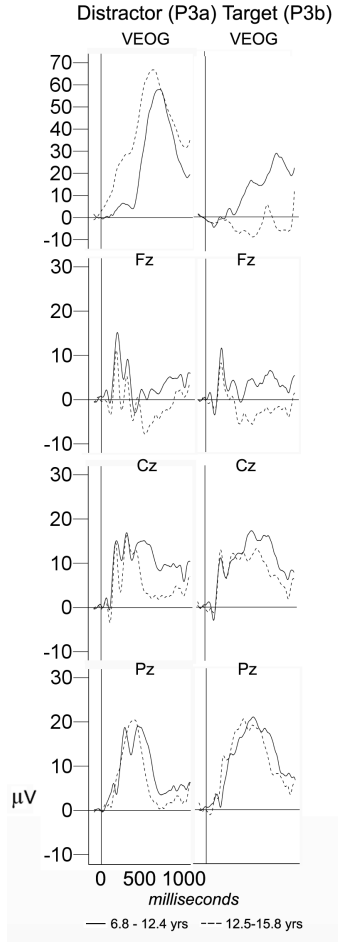


FIGURE 2 Grand average ERP curves for the two groups of children. The figure shows the grand average ERP curves for P3a (left column) and P3b (right column) for the two groups of children (6.8–12.4 years and 12.5–15.8 years) at the midline electrodes. For P3a, each group consisted of 13 participants, while the corresponding number for P3b was 12 (the P3b data from two of the participants were excluded because of muscle artifacts).

= 11.21, $p = .000$, but not P3a latency, $F(1.846, 44.29) = 2.10$, ns . See Table 2 for mean amplitude and latency values. Posthoc tests showed that for P3a, the amplitude at Fz was lower than at Cz ($p < .0001$) and Pz ($p < .0001$), while the difference between Cz and Pz was not significant. For P3b, most contrasts between the midline electrodes were significant for both amplitude and latency.

TABLE 2
Means and Standard Deviations for P3a and P3b Amplitude (mV) and Latency (ms) at Fz, Cz, and Pz in Each Age Group

	<i>Fz Amplitude</i>	<i>Fz Latency</i>	<i>Cz Amplitude</i>	<i>Cz Latency</i>	<i>Pz Amplitude</i>	<i>Pz Latency</i>
	P3a					
Child sample	16.23 (8.15)	381 (119)	26.77 (8.62)	450 (139)	26.78 (9.23)	416 (122)
Life span sample	8.93 (12.57)	384 (95)	21.03 (8.85)	382 (60)	24.30 (10.58)	417 (56)
6.8–12.4 years	12.58 (11.03)	383(106)	23.90 (9.05)	416 (110)	25.54 (9.81)	416 (93)
12.5–15.8 years	12.93 (6.54)	369 (71)	21.71 (8.66)	369 (47)	19.83 (7.07)	370 (45)
Child 6–15 years	14.32 (7.05)	416 (71)	17.20 (6.53)	427 (49)	14.23 (6.35)	417 (55)
Young 20–44 years	9.98 (5.37)	451 (63)	12.00 (6.17)	456 (62)	9.70 (5.18)	449 (88)
Middle 46–69 years						
Old 70–88 years						
	P3b					
Child sample	10.28 (7.72)	409 (115)	19.60 (7.77)	541 (100)	23.57 (8.44)	540 (124)
Life span sample	4.08 (6.02)	379 (90)	17.25 (9.61)	444 (104)	22.75 (10.25)	505 (122)
6.8–12.4 years	7.44 (7.53)	396 (103)	18.52 (8.55)	497 (111)	23.19 (9.12)	524 (122)
12.5–15.8 years	8.41 (5.15)	385 (93)	11.30 (6.55)	392 (68)	12.75 (6.80)	389 (53)
Child 6–15 years	6.69 (5.39)	399 (109)	6.94 (5.15)	439 (95)	7.72 (4.50)	440 (86)
Young 20–44 years	8.43 (5.39)	378 (66)	8.43 (7.58)	410 (79)	8.97 (6.45)	420 (87)
Middle 46–69 years						
Old 70–88 years						

Amplitude at Fz was lower than at Cz ($p < .0001$), and Pz ($p < .0001$), and amplitude at Cz was lower than at Pz ($p < .0001$). P3b latency was shorter at Fz than Cz ($p < .05$) and Pz ($p < .01$), while no differences existed between Cz and Pz. These results correspond to expectations of peak amplitude at Pz for P3b and more centrally distributed peaks for P3a, indicating a valid topographical distribution.

There was no significant main effect of age group, neither for P3a amplitude, $F(1, 24) = 2.81$, *ns*, P3b amplitude, $F(1, 22) = 1.39$, *ns*, P3a latency, $F(1, 24) = .385$, *ns*, nor P3b latency, $F(1, 22) = 2.99$, *ns*. ANOVA with voltage normalized values revealed an age group \times electrode interaction for P3a amplitude, $F(.409, 33.80) = 6.24$, $p = .01$. As can be seen from Table 2, the interaction effect is due to higher fronto-central relative to parietal amplitudes in the youngest child group. No age group \times electrode interactions were observed for P3b amplitude, $F(1.23, 27.07) = .076$, *ns*, P3a latency, $F(1.846, 44.29) = 2.28$, *ns*, or P3b latency, $F(1.613, 35.48) = .885$, *ns*.

Linear regressions with age as the independent variable showed negative effects of age on P3a amplitude at Fz (beta = -2.5 , adjusted $R^2 = .21$, $p < .05$), P3b amplitude at Fz (beta = -1.2 , adjusted $R^2 = .30$, $p < .01$), and P3b latency at Fz (beta = -24.5 , adjusted $R^2 = .24$, $p < .01$). Multiple regressions with age and age² as independent variables and P3a and P3b amplitude/latency as dependent variables revealed no significant ($p > .05$) contributions from the quadratic term, indicating a linear fit to the data. Adding gender as an independent variable in addition to age did not improve the fit to the data in any of the significant regressions, and did not render the age term insignificant.

To investigate possible maturational differences between P3a and P3b, an ANOVA with 3 electrodes (Fz, Cz, Pz) \times 2 components (P3a, P3b) \times 2 age groups (6.8–12.4, 12.5–15.8 years) was performed. The component \times age group term was insignificant, both for amplitude, $F(1,22) = 0.02$, *ns*, and for latency, $F(1, -22) = 0.77$, *ns*.

Correlations between raw scores on the subtests matrix reasoning, digit span (forward + backward), word order and hand movement and P3a and P3b amplitude and latency generally revealed weak and nonsignificant associations between the electrophysiological measures and cognitive performance, with a few exceptions: Raw score on word order correlated with P3a latency at Cz ($r = -.41$, $p < .05$). Raw score on matrix reasoning correlated with P3b latency at Fz ($r = -.43$, $p < .05$), and there was also a nonsignificant trend towards a relationship with P3b amplitude at Fz ($r = -.36$, $p < .10$). Raw score on digit span showed a significant negative relationship with P3a amplitude at Fz ($r = -.54$, $p < .05$), and nonsignificant trend towards a negative relationship with P3b amplitude at Fz ($r = .35$, $p < .10$). Raw scores on hand movement were not significantly related to any of the electrophysiological measures investigated.

Full Sample

ANOVAs were performed with four age groups (children, young, middle-aged, old) \times three electrodes (Fz, Cz, Pz) for the amplitude and latency of P3a and P3b separately. There was a main effect of electrode on P3a amplitude, $F(1.535, 230.28) = 68.04, p = .000$, and P3b amplitude, $F(1.7.09, 252.93) = 63.73, p = .000$, as well as P3b latency, $F(1.658, 245.42) = 25.51, p = .000$, but not for P3a latency, $F(1.843, 276.45) = 1.96, ns$. For P3a amplitude, *t*-tests revealed that Fz amplitude was lower than Cz ($p < .0001$) and Pz amplitude ($p < .0001$), and Cz amplitude was higher than Pz amplitude ($p < .0001$). For P3b, the amplitude was shifted posteriorly, so that Fz amplitude was smaller than Cz amplitude ($p < .0001$), which was smaller than Pz amplitude ($p < .0001$). These results correspond to previous reports on the topography of the P3a and the P3b.

Further, a main effect of age group was found for both P3a and P3b amplitude and latency, $F(3,150) = 15.87, p = .000, F(3,148) = 16.09, p = .000$ for P3a and P3b amplitude, respectively, and $F(3,150) = 17.29, p = .000, F(3,148) = 8.43, p = .000$ for P3a and P3b latency, respectively). Post-hoc tests showed that for P3a amplitude, the child group had higher amplitude than the middle-aged ($p < .05$) and the old group ($p < .0001$), while the young and the middle-aged groups both had higher amplitudes than the old group ($p < .001$ and $p < .01$, respectively). Significant differences were not found between the child group and the young group, or between the young group and the middle-aged group. For P3a latency, the child group had longer latency than the young group ($p < .05$) and shorter latency than the old group ($p < .01$), while there was no significant difference between the child and the middle-aged group. The young group had significantly shorter latency than all other groups, while the middle-aged group had marginally shorter latency than the old group ($p = .068$). For P3b, the child group had higher amplitude than all other groups (all p 's $< .0001$). The young group had higher amplitude than the middle-aged ($p < .01$) but not the old group, while the middle-aged and old group did not differ significantly. For P3b latency, the child group had longer latency than the young and the old group ($p < .01$), and marginally ($p = .057$) longer than the middle-aged group. No other latency-differences were significant. All post-hoc tests were Bonferroni-corrected for number of comparisons.

Finally, ANOVAs with voltage normalized amplitude values showed a significant interaction effect of age group \times electrode for both P3a amplitude, $F(4.725, 236.23) = 3.02, p = .013$, P3b amplitude, $F(4.988, 247.76) = 7.89, p = .000$, and P3b latency, $F(4.975, 245.42) = 4.94, p = .000$. For amplitude, this interaction is explained by the well-known frontal shift in amplitude with increasing age, and the relatively higher parietal amplitude in the group of children. While the young participants have a central or parietal maximum for both P3a and P3b, older participants tend to have a more even distribution of activity, with relatively higher frontal amplitudes compared to the younger participants. The P3b latency effect is

explained by relatively shorter latency at Fz than Cz and Pz in the young age group than the middle-aged and the old, and also the child groups.

Stepwise linear regression analyses were performed with P3a and P3b amplitude and latency as dependent variables, and age, square of age, and gender as independent variables. Age was the single or the strongest predictor in all cases. Square of age added to the amount of explained variance in case of P3a latency at Pz (adjusted $R^2 = .12$, $p < .0001$), and P3b amplitude at Cz (adjusted $R^2 = .23$, $p < .0001$) and Pz (adjusted $R^2 = .38$, $p < .0001$). Gender was significantly related to the amplitude at Cz and Pz both for P3a (Cz: adjusted $R^2 = .28$, $p < .0001$, Pz: adjusted $R^2 = .40$, $p < .0001$) and P3b (Cz: adjusted $R^2 = .29$, $p < .0001$, Pz: adjusted $R^2 = .43$, $p < .0001$). Being female indicated higher amplitude than being male. Scatterplots illustrating individual data points are presented in Figure 3.

Next, we wanted to test developmental differences between P3a and P3b in the life-span perspective. ANOVA with three electrodes (Fz, Cz, Pz) \times two components (P3a, P3b) \times four age groups (children, young, middle-aged, old) was performed. The component \times age group term was significant both for amplitude $F(3, 147) = 8.90$, $p < .0001$, and latency, $F(3, 147) = 10.66$, $p < .0001$. These effects were mainly due to the children having higher P3b amplitudes and longer P3b latencies relative to P3a amplitudes and latencies than the rest of the sample. Further, the component \times age group \times electrode interaction term also reached significance for amplitude, $F(6, 294) = 3.22$, $p < .01$, and marginally for latency, $F(6, 194) = 2.90$, $p = .063$. The amplitude interaction was due to more even topographical distribution in the older than the younger, and more so for P3b than P3a. The latency interaction was mainly caused by short latencies at Fz and markedly longer at Cz and Pz in children, and more so for P3b than P3a.

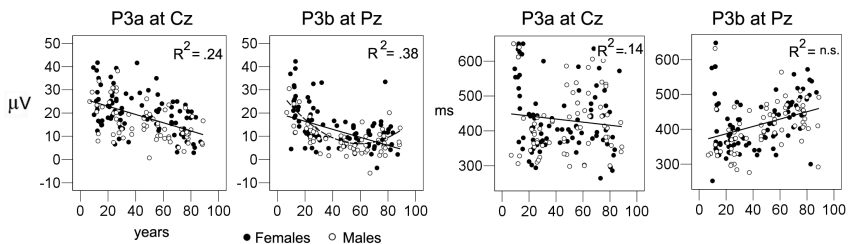


FIGURE 3 Scatterplots of P3 as a function of age. The individual data points are plotted against P3 (P3a or P3b, amplitude or latency) and age. The linear regression line is plotted (solid line), and the adjusted R square is printed in the upper right corner of each scatterplot. In cases where a nonlinear component (age^2) added significantly to the amount of explained variance, the nonlinear regression line (dotted line) is shown in addition to the linear, and the adjusted R^2 is printed. Results from Cz are presented for P3a and Pz from P3b, because the components traditionally have their best expressions at these electrodes.

Finally, the relationship between neuropsychological test scores (matrix reasoning, digit span) and P3 parameters was examined in the full sample. Since correlations in the child sample partly were in directions opposite of what has previously been found in adult samples, the possibility of nonlinear relationships was also tested. Thus, stepwise regressions with matrix reasoning and digit span as dependent variables, and the different P3 parameters and the square of each variable as independent variables, were performed. The relationships between matrix reasoning and the P3 parameters were mainly of a linear nature, and were stronger for P3a. [P3a Cz amplitude: adjusted $R^2 = .09$, $p < .0001$; P3a Fz latency: adjusted $R^2 = .03$, $p < .01$; P3a Cz latency: $R^2 = .11$, $p < .0001$; P3a Pz latency: adjusted $R^2 = .06$, $p < .01$] than P3b (P3b Pz amplitude: adjusted $R^2 = .03$, $p < .05$). Only in one case (P3a Pz amplitude: adjusted $R^2 = .12$, $p < .0001$) did a nonlinear component add significantly to the amount of explained variance. However, when digit span was used as dependent variable, nonlinear fits to the data were more prominent than linear fits, with quadratic fits for P3a Cz latency (adjusted $R^2 = .04$, $p < .01$), P3a Pz latency (adjusted $R^2 = .05$, $p < .01$), P3b Cz latency (adjusted $R^2 = .03$, $p < .05$), and P3b Pz latency (adjusted $R^2 = .04$, $p < .05$), and a linear fit for P3a Pz amplitude (adjusted $R^2 = .03$, $p < .05$).

DISCUSSION

Overall, the results seem to indicate that P3a might mature earlier than P3b, as observed from the grand average-curves. Statistical tests confirmed differential effects of age group on the relationship between P3a and P3b. While both P3a and P3b components show higher amplitude at the central and parietal electrodes in children, there is a more posterior distribution of the P3b in children relative to adults. In adults, P3a and P3b are seen to reflect automated and controlled processing, respectively. Based on the present data, one may speculate that these aspects of information processing, at a neural level, are less differentiated in children relative to adults, with a relatively greater reliance on automated mechanisms. This may be caused by an immature cognitive system. The finding will be elaborated below.

Child Sample

The data indicate changes in both P3a and P3b with increasing age also during childhood. The child sample was unfortunately not of a size sufficient to allow a fine-grained analysis of age changes, and this is a limitation of the present study. However, across the relatively small child (< 12.4 years) and adolescent (> 12.4 years) groups, P3a and P3b amplitude decreased significantly and P3b latency shortened at Fz. P3a latency at Fz, however, was not systematically related to age in the child sample. This may indicate that this aspect of the component might

mature earlier. Further, the grand average figures reveal that the peaks of P3b are broader in the children at Cz and Pz, relative to the adults, while the P3a peaks are more similar in shape to those found in adults. This is in accordance with prior research on P3b in children, where both Segalowitz and Davies (2004) and Thomas and Nelson (1996) have reported broader peaks in children, relative to adults. Of previous studies, Harada et al. (2002) reported a sharp decrease in P3b latency during childhood, while Gumenyuk et al. (2004) reported higher P3a amplitude for the youngest children (8–9 years old).

Fuchigami et al. (1995) also reported earlier maturation of P3a latency relative to P3b, without specifying topography. In the present data, the significant age group \times electrode interaction for P3a amplitude indicated topographical changes across childhood, in that the youngest children had a more fronto-central scalp distribution than the older children. Age-related differences in amplitude and latency were seen at Fz only. This may indicate a link between the ongoing maturational changes of the frontal lobes during childhood and adolescence (Giedd et al., 1999; Sowell et al., 1999) and the electrophysiological expression of P3a and P3b at Fz. The youngest children thus seemed to allocate more attentional resources to both target and distractor stimuli, and the deeper processing associated with controlled attention and P3b took longer time, possibly due to immaturity of frontal circuits. Taken together, the above mentioned results may indicate a difference in maturation processes also within the child age range, where P3b appears to mature later than P3a. Still, the lack of a significant age group \times component interaction when the two child groups only were compared indicate that it is not possible to conclude from the present data that maturational differences between the P3a and the P3b can be found in children of different ages.

There were generally weak relationships between neuropsychological test performance and electrophysiological measures, even though some significant results were found. Raw score on matrix reasoning was negatively related to P3b latency in the current child sample. This has also been reported for adults previously (e.g., Walhovd & Fjell, 2002). The link between longer P3a latency and distractibility was also supported on word order—a task especially designed for children. The amplitude correlations went in the opposite direction of what is usually observed in adults, and were strongest at Fz. The reason for this pattern was that high Fz amplitude is more characteristic of the youngest than the oldest children, while neuropsychological performance is higher in the older than the younger children. Thus, the amplitude results must be interpreted differently when children are studied separately from adult samples. The neuropsychological significance of higher amplitude thus seems different at different ages, and must be interpreted also in light of the topography of the component within the given sample. Still, it may be argued that the negative correlation between digit span score and P3a amplitude at Fz supports a link between P3a amplitude and distractibility in children, as suggested by Gumenyuk et al. (2004).

Full Sample

Analyses of the full life-span sample showed that for amplitude, the childhood development seems to represent a continuation of trends found in the adult sample (Fjell & Walhovd, 2004), where increasing age was associated with lower P3a and P3b amplitude. For latency, this pattern was not so evident, especially for P3b latency, where the group of children had significantly later peaks. Increased P3b latency in children is presumably linked to ongoing maturational changes of the frontal lobes during childhood and adolescence, such as myelination of axons (Giedd et al., 1999), reduction in grey matter volume (Sowell et al., 1999) and synaptogenesis (Huttenlocher & Dabholkar, 1997). In contrast to this, increased latency in older adults may be due to normal aging processes (e.g., Fjell & Walhovd, 2004; Harada et al., 2002), including neuroanatomical volumetric reductions seen in higher age (Walhovd et al., 2005a, 2005b).

Age group \times component interactions were identified both for latency and for amplitude. Thus, there seem to be selective differences in the maturational and age-changes in these components. Differences of P3a and P3b across children and elderly participants lend support to the notion of differential processes leading to high amplitude and long latency in children versus older adults, as outlined above. The P3a seems to mature earlier than the P3b, which corresponds to the established view of P3b as related to controlled cognitive processing, and P3a as more related to an automatic orienting and alerting system.

When the ERP results were related to the neuropsychological tests scores in the full life-span sample, interesting patterns emerged. Adding a quadratic term yielded significantly better fit to the data when digit span was the dependent variable and P3a or P3b latency was used as predictor. This indicates that the neuropsychological significance of the P3 results is nonuniform across different parts of life-span development. Thus, individual differences in latency do not mean the same in child samples as in adult samples, probably related to differential processes causing maturational changes vs. age-related decline. For matrix reasoning, the quadratic term generally did not add significantly to the fit. Thus, at least at central and parietal electrodes, this neuropsychological test may be related to P3 in a more homogenous way from childhood to late adulthood.

CONCLUSION

The present data indicate that P3a and P3b amplitude in children partly represent a continuation of trends demonstrated in adult life-span samples, where increasing age in childhood is associated with lower amplitude. Further the results seem to indicate that P3a mature earlier than P3b, perhaps related to immaturity of the cognitive system supporting the controlled processing involved in the latter. Thus, even

though the development of P3 may be seen as a continuous process from childhood to old age, this may vary with the component and parameter studied, and different neural mechanisms are probably involved in at different stages of the life-span. Finally, quadratic relationships existed between digit span score and P3 parameters across the life-span, while linear relationships generally gave the best fit to the data for matrix reasoning. Thus, it is demonstrated that some relationships between neuropsychological performance and P3a and P3b parameters exist, but that these relationships are complex, and dependent upon the age group in question.

ACKNOWLEDGMENT

The research was supported by The Norwegian Research Council, grant S-03180.

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