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Early childhood development of visual texture segregation in full-term and preterm children

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ABSTRACT

To date, very little is known about the normal development trajectory of visual texture segregation, or how it is affected by preterm birth. The goal of this study was to characterize the development of visual texture segregation using texture segregation visual evoked potentials (tsVEPs) in children born full-term and children born preterm without major neurological impairment. Forty-five full-term and 43 preterm children were tested at either 12, 24 or 36 months of age (corrected age for prematurity at 12 and 24 months old). VEPs were obtained using two lower-level stimuli defined by orientation (oriVEP) and two higher-level stimuli defined by texture (texVEP). TsVEP was obtained by dividing by two the subtraction of oriVEP from texVEP. Results show a clear maturation of the processes underlying visual texture segregation in the full-term group, with a significant N2 latency reduction between 12 and 36 months of age for all conditions. Significant N2 amplitude reduction was observed for oriVEP between 12 and 24 months, as well as for texVEP between 12 and 24 months, and 12 and 36 months. Comparison between full-term and preterm children indicated significantly lower N2 amplitude for the preterm group at 12 months for oriVEP and texVEP. These differences were no longer apparent at 24 months of age, suggesting that children born preterm catch up with their full-term counterparts somewhere between 12 and 24 months of age. Our results appear to reflect a maturational delay in preterm children in both lower-level and higher-level visual processing during, at least, early childhood.

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1. Introduction

During the last decade, numerous articles have been published about vision development and its different components, such as acuity, visual fields, visual attention, visual pathways and other aspects of visual perception (e.g., stereopsis, contrast and orientation sensitivity). Those publications have contributed to a better comprehension of normal visual development in children by providing age-dependent normative data that can be further applied to the study of atypical brain development. In fact, measures of visual system integrity constitute reliable indicators of

neurological status and brain function integrity, especially in preterm children (Cioni et al., 2000; Guzzetta et al., 2001). Now that it is recognized that early visual function has an impact on cognitive development (Cioni et al., 2000; Mercuri et al., 1999; O'Reilly et al., 2010), the identification of early visual impairments or dysfunctions is essential; the earlier they are discovered, the sooner intervention programs can be implemented, which can positively influence the cognitive outcome of visually impaired children (Fazzi et al., 2005).

Throughout the years, it has been demonstrated that visual functions not only mature during the gestational period, but continue to develop and specialize after birth. Electrophysiological studies, for instance, have shown that the visual system matures rapidly during infancy, gradually during childhood, continuing into adulthood (Brecelj, 2003; Brecelj et al., 2002). Visual development has been studied using psychophysiological methods such as visual evoked potentials (VEPs), a technique that does not necessitate active participation of the child. In infants and children,

maturational changes are indicated by modifications in VEP waveform, amplitude and latency (Brecelj, 2003; Lenassi et al., 2008; Lippe et al., 2007). Although knowledge about VEP maturation is continually expanding and being refined, the exact developmental course of vision functioning remains unknown.

The majority of the studies of visual system development in children using VEPs have used "lower-level" stimuli defined by contrasts and spatial frequencies, which solicit principally the fields of V1 (Braddick & Atkinson, 2011). However, there are many analytical processes involved in the perception of a visual scene, such as the encoding of stimulus features, the processing of top-down information, which bring to segmentation of forms and recognition of stimuli (Bach & Meigen, 1990, 1992). Hence, in the last two decades researchers have been looking further into more integrative "higher-level" visual processes. Visual texture segregation, for instance, relies on many features (e.g. motion, stereo, color, luminance, spatial frequency) and constitutes a necessary mechanism for segregation of a figure from its background (Bach & Meigen, 1998; Kastner, De Weerd, & Ungerleider, 2000). This process, closely related to pop-out (Julesz & Bergen, 1983), occurs spontaneously and is required for the perception and recognition of objects, such as faces, numbers, and letters. Therefore, it occupies a central position in social and cognitive development (van den Boomen, Lamme, & Kemner, 2014).

Many animal studies, such as those who specifically stimulated the receptive field of neurons in the monkey visual cortex, have contributed to the development of our knowledge regarding visual texture segregation. In fact, by using single-cell recording, these studies demonstrated that the perception of an object's features is dependent of the context in which it is observed, a phenomenon called contextual modulation (Kapadia et al., 1995; Sillito & Jones, 1996). For example, it has been shown that cells in V1 give larger response to textured than homogeneous stimuli (Lamme, 1995; Zipser, Lamme, & Schiller, 1996), and that this modulation activity is suppressed when the animal is anesthetized (Lamme, Zipser, & Spekreijse, 1998). Regarding the segregation processes per se, other studies highlighted the importance of the difference between boundary detection and scene segmentation (Nakayarna, Shirnojo, & Silverman, 1989). These differences have been specifically studied by Scholte et al. (2008) using EEG and fMRI techniques in humans. Their results indicate that there is a feedforward detection of texture boundaries; they are first detected in lower-level then in higher-level visual areas (i.e. from occipital region to peri-occipital, temporal, and parietal regions), whereas scene segregation is represented in a "reverse hierarchical" manner (i.e. from temporal areas to peri-occipital, parietal and occipital regions) through feedback connections. Furthermore, some other authors suggest that both feedforward and horizontal connections are implicated in boundary detections (Roelfsema et al., 2002). This model, on the role of different types of neural connectivity in the processing of specific visual stimuli such as visual texture segregation, has been put forward by many other researchers in order to understand its underlying mechanisms (ex. boundary detection, grouping, etc.) (Roelfsema et al., 2002; van den Boomen, Lamme, & Kemner, 2014).

During the last decade, animal research has allowed translation to human studies of visual texture segregation, including relationships between event-related potentials and localization of sources (Lamme, Van Dijk, & Spekreijse, 1992; Roelfsema et al., 2002). It has been demonstrated, for instance, that this process can be detected in adults with particular VEPs, namely texture segregation visual evoked potentials (tsVEPs) and that it can be obtained in response to stimuli defined by luminance, orientation, motion and stereo (Bach & Meigen, 1997). Electrophysiological studies that have investigated this process in normal adults have shown a negative component peaking around 200 ms after stimulus onset (texture-related negativity) obtained from the difference wave between VEP obtained by textured stimuli versus homogeneous one (Caputo & Casco, 1999; Lamme, Van Dijk, & Spekreijse, 1992). This component originates from the V1 area and is thought to reflect combination of information from V2 and V3 associative visual areas through feedback connection circuits (Scholte et al., 2008). Therefore, tsVEPs give an intermediary measure of visual processing between lower-level VEPs, which peak at around 100 ms, and cognitive event-related responses, which usually peak after 300 ms after stimulus appearance (Lachapelle et al., 2004).

Few studies of visual texture segregation have been conducted in children to date, so very little is known about its normal developmental pattern. Early behavioral studies found it to appear around 9–12 months of age (Rieth & Sireteanu, 1994; Sireteanu & Rieth, 1992), while VEP studies suggested that the ability to discriminate texture-defined stimuli emerges around 14–18 weeks of age (Atkinson & Braddick, 1992). Using VEPs, Arcand et al. (2007) demonstrated a clear developmental pattern characterized by changes in amplitude, latency and scalp distribution of texture segregation processes during the first year of life; tsVEPs appear approximately at 3 months of age, continue to develop until 12 months but are still immature at this age. Furthermore, van den Boomen, Lamme, and Kemner (2014) have studied the developmental trajectory of visual texture segregation in typically developing children aged from 7 to 18 years old. They found significant differences in event-related potentials between age groups 7–8 and 9–10 years, as well as between age groups 11–12 and 13–14 years, which they considered as the strongest developmental periods for this process. According to these authors, visual texture segmentation continues to develop until early puberty, where it reaches adult-like EEG responses. However, its developmental pattern during early childhood (i.e. between 12 months and school-age) remains unknown. Consequently, a better understanding of this developmental trajectory is needed so it may be subsequently used to further study developmental disorders associated with altered visual texture segregation processing or "higher-order" visual analysis, such as autistic spectrum disorder (Rivest et al., 2013), Williams Syndrome (Palomares & Shannon, 2013), and prematurity (Thibault et al., 2007).

Although no study has investigated the effect of prematurity on texture segregation VEPs, it is proposed that preterm birth can disrupt the development of feedforward connections such as it alters the development of the primary visual pathways (e.g. magnocellular-dorsal), as shown by VEPs and source analyses (Hammarrenger et al., 2007; Lassonde et al., 2010; O'Reilly et al., 2010; Tremblay et al., 2014). As higher-level visual processing relies on both magnocellular and parvocellular pathways, the developmental course of texture segregation could also be compromised by prematurity. Moreover, recent imaging studies have shown white matter microstructural alterations in the visual cortex of children born preterm during childhood and adolescence, which have been related to higher risks of visual impairment in this population (Kelly et al., 2014; Oros et al., 2014; Thompson et al., 2014). These studies also support the idea that connections allowing visual processing might be affected in preterm children. Therefore, we hypothesize that altered development of the visual pathways also has a deleterious effect on higher-level visual function development, such as texture segregation processes. Consequently, the goal of the present study was to characterize the developmental pattern of visual texture segregation processing during early childhood using tsVEPs in (1) children born full-term, and (2) children born preterm without major neurological impairment.

2. Method

2.1. Participants

Using a cross sectional study design, 88 children, 46 full-term and 43 preterm distributed in 3 age groups, were tested at either 12, 24 or 36 months old, corrected age for prematurity at 12 and 24 months old (see Table 1 for participants' characteristics). One full-term child was excluded from further analyses because of excessive cries. Preterm children were recruited at the gynecology-obstetrics and neonatology Departments of the Sainte-Justine University Hospital in Montreal and also in collaboration with Préma-Québec, a non-profit organization that supports preterm children and their families. Full-term children were recruited in different daycares around the Sainte-Justine University Hospital area and through publicity in the hospital and its internet site section about ongoing research projects. Children with a gestational age of 36 weeks or less were accepted in the preterm groups, and those with a gestational age of 37 weeks or more, with no health problems, and no prenatal and/or neonatal complications were included in the full-term groups. For both groups, exclusion criteria were: intra-uterine growth restriction (<10^e percentile), infectious disease during pregnancy (e.g., tuberculosis, genital herpes, AIDS), and known genetic, metabolic, neurologic or chromosomal anomalies. For the preterm groups, additional exclusion criteria were: abnormal ultrasonography (e.g., presence of periventricular leukomalacia, ventriculomegaly, hemorrhagic lesions), retinopathy of prematurity (ROP) > stage 3, or treatment by Avastin, cryotherapy or laser therapy in at least one eye.

2.2. Procedure

Only one visit to our laboratory was required for each participant to complete the experiment. The duration never exceeded 45 min, including electrode placement and completion of the questionnaire. Short breaks were given as necessary.

Table 1
Full-term and preterm group characteristics, in terms of their mean age, gender, and gestational age in weeks (GA). Preterm category according to GA is added for the preterm groups.

Full-term groups	N	Mean age (MO) – (SD)	Gender	Mean GA (SD)
12 months	15	12.2 (1.4)	M: 8 F: 7	39.07 (1.39)
24 months	15	24.2 (2.8)	M: 3 F: 12	39.53 (0.92)
36 months	15	35.5 (2.4)	M: 6 F: 9	39.67 (0.98)

Preterm groups	N	Mean age (MO) – (SD)	Gender	Mean GA (SD)	Preterm category, according to GA[a]
12 months	15	12.7 (1.5)	M: 11 F: 4	28.33 (4.03)	Late preterm: 2 Moderate: 5 Severe: 2 Extreme: 6
24 months	15	24 (1.6)	M: 6 F: 9	29.93 (3.61)	Late preterm: 4 Moderate: 6 Severe: 4 Extreme: 1
36 months	13	38.4 (2.3)	M: 10 F: 3	30.31 (3.54)	Late preterm: 4 Moderate: 6 Severe: 1 Extreme: 2

MO = months; M = male; F = female; W = weeks, SD = standard deviation.
[a] Late preterm = 33–36 weeks; moderate = 28–32 weeks; severe: 26–27 weeks; extreme: less than 26 weeks.

During EEG recording, visual stimulations were presented in a blocked design to the child (see next section for more details). Developmental and socio-demographic information were collected using a semi-structured questionnaire completed by a parent or legal guardian. The research protocol was developed in accordance with the The Code of Ethics of the World Medical Association (Declaration of Helsinki), and was approved by the ethics committee of the Sainte-Justine's University Hospital Research Centre. An informed and written consent of a parent or legal guardian was obtained before children participated in the study.

2.3. Visual stimuli

The same stimuli as in the study from Arcand et al. (2007) were used. They were composed of four different stimuli, two lower-level (orientation) and two higher-level (textured), and were presented binocularly on a 40.5×30.5 cm ViewSonic monitor (ViewSonic, Canada) at a distance of 114 cm from the child's eyes. The lower-level orientation stimuli were composed of parallel lines consistently oriented to the left or right (see Fig. 1a and b). The higher-level textured stimuli were composed of an orientation-defined checkerboard with 90° line gradients oriented concentrically or outward (see Fig. 1c and d). Their physical characteristics consisted of a spatial frequency of 1 cycle/degree at 95% contrast level. Luminance stayed stable at 30 candelas/m². Each stimulus was presented for 150 ms, alternating with a 850 ms gray mask (rate of 1 Hz). One block composed of 120 presentations of each stimulus was usually sufficient to obtain a strong response; additional blocks were presented when needed. Stimuli were produced by the E-Prime software (Psychology Software Tools, Inc.) on a Dell PC (model GX150).

2.4. Electrophysiological recordings

The visual evoked potentials (VEPs) were acquired using a high density electrophysiological system, the Geodesic Sensor Net system consisting of 128 electrodes (Electrical Geodesic Inc. Eugene, OR). EEG signals were recorded at a sampling rate of 250 Hz with an analog band pass filter from 0.1 to 100 Hz and were then amplified by Net Amps 200 (Electrical Geodesics Inc., Eugene, OH, USA). They were acquired using the Net Station program operating on a
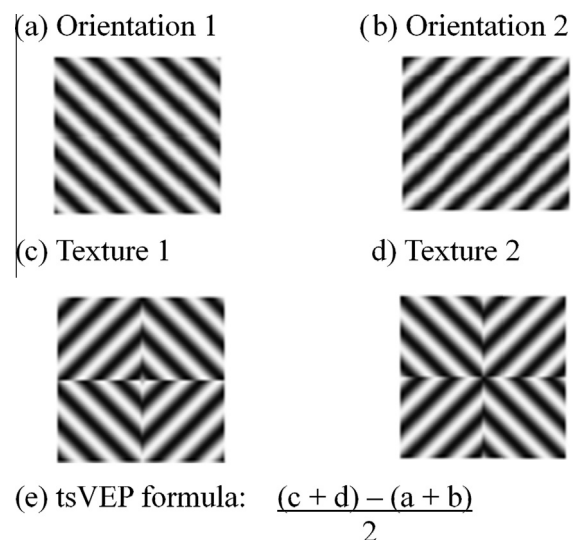

Fig. 1. Examples of stimuli used for orientation (a and b) and texture (c and d), and method for extracting the tsVEP (e). Responses to orientation stimuli are subtracted from the responses to the textured stimuli and then divided by two.

G4 Macintosh computer. The vertex was used as reference. As suggested by Tucker (1993), electrode impedance was kept under 40 kΩ.

During the recording session, the child was seated on his/her parent/guardian's lap in a faraday room. The experimenter present in the recording room used a small and noisy toy to catch and maintain the child's attention to the center of the screen, a common technique employed in research with children (Roy et al., 1995). The experimenter also used a green/red LED, in order to signal to the experimenters in the adjoining control room to reject the trials when the child was not looking at the screen. Moreover, the child's behavior was observable via an infrared camera placed in the recording room.

2.5. Data analysis

Electrolophysiological data were analyzed using Brain Vision Analyzer software, version 2 (Brain Vision Products, Germany). For pre-processing analyses, EEG data were first filtered offline with a band-pass filter of 1–50 Hz. Second, they were corrected for eye movements using the independent component analysis (ICA) method (Vigário, 1997) and epoched into 700 ms segments (−100 ms before stimulus appearance and 600 ms after). Then, artifacts were automatically rejected on voltage criteria ±100 μV and each trial was visually examined thereafter to confirm artefact rejection. Finally, the EEG data were re-referenced to an averaged reference, and a 100 ms pre-stimulus interval was used to baseline correct the recorded VEPs.

In order to compare lower-level and higher-level VEPs, the same technique as stated in Arcand et al. (2007) was used, which consists of adding together the two orientation VEPs responses (oriVEP; number of trials ranged between 65 and 133 trials, mean number of trials: 95.72) with the two textured VEPs (texVEP; number of trials ranged between 58 and 131, mean number of trials: 95.25). As proposed by Bach and Meigen (1990, 1992), the underlying principle for data analysis is based on the hypothesis that texture segregation is formed of both orientation processing and texture processing. Therefore, to obtain the negative wave corresponding to texture segregation (tsVEP), the oriVEP was removed from the texVEP, and then divided by two (see Fig. 1e). As a result, the activation associated with the lower-level processing was suppressed and the resulting negative wave, a difference potential, reflects only the tsVEP response (Arcand et al., 2007; Bach & Meigen, 1992; Bach et al., 2000; Lachapelle et al., 2004). For each oriVEP, texVEP and tsVEP waveform obtained for each participant, the N2 peak (or the texture-segregation N2 in the case of the subtraction) was identified for the electrode sites O1, O2 and Oz using semi-automatic detection. These electrodes were chosen because they are standard for VEP measurement (Odom et al., 2010) and reflect the strongest VEP response. N2 peak was defined as the maximum negative peak within a ±200 ms time-window (160 ms to 320 ms). Although a positive drift of the VEPs was sometimes noticeable, an event that can occur when using multichannel recordings with children (Luck, 2005), we decided to keep the baseline-to-peak technique (instead of calculating the area under the curve, for instance), since the negative deflection of the N2 peak was easily identifiable. Latencies and baseline-to-peak of this component were determined according to two independent judges.

2.6. Statistical analyses

All statistical analyses were performed using SPSS software (version 17.0). First, Pearson correlations were applied to determine the impact of age at testing, gestational age and birth weight on N2 amplitude and latency in each condition. Then, for the EEG data, analyses of variance with repeated measurements were used to compare groups, with two between-subjects factors (group: full-term and preterm; age group: 12, 24 and 36 months) and one within-subject factor (conditions: oriVEP, texVEP and tsVEP). All ANOVAs were executed separately for latency and amplitude measures of the N2 peak and for each selected occipital electrode (i.e. O1, O2 and Oz). Significant interactions and main effects were examined with post hoc pairwise comparisons.

3. Results

3.1. Participants

According to the responses collected in the developmental questionnaire, no medical or psychiatric conditions (children or parents) were revealed in any of the participants. Parents of eight preterm children (18.6%) reported visual problems in their child (e.g., astigmatism, myopia, etc.), but none of them were corrected to normal vision by wearing glasses because of the low severity of the problem and the young age of the children (i.e. 36 months or less). Ten preterm children (23.6%) were receiving or had received at least one specialized evaluation or intervention (e.g., speech-language therapy, psychology, physiotherapy, occupational therapy, etc.). See Table 2 for details on the type of visual problems and interventions reported in the questionnaire. None of the parents of full-term children reported that their child had developmental problems, visual problems or had received specialized interventions.

Chi-square tests for independence indicated a higher number of male than female preterm children (χ^2 (1, $n = 88$) = 5.503, $p = 0.019$, phi = −0.250), which reflects the gender prevalence of male preterm birth as stated in previous studies (Brettell, Yeh, & Impey, 2008; Melamed, Yogev, & Glezerman, 2010). Results also showed significant differences between full-term and preterm groups, where preterm children reported more visual problems (χ^2 (1, $n = 87$) = 9.439, $p = 0.002$, phi = 0.329) and specialized interventions (χ^2 (1, $n = 87$) = 9.166, $p = 0.02$, phi = 0.325) than full-term children. However, no significant differences were found between groups for other socio-demographic variables (all p's > 0.1), which indicate that the preterm and full-term samples did not differ in terms of family income or mother's education level. Results from independent t test performed on each age group showed no significant differences between full-term and preterm groups in regard of head circumference (all p's > 0.05).

3.2. Development of visual texture segregation in full-term children

Because texture segregation VEPs have never been studied between 12 months and school-age, results are presented for

Table 2
Detailed information about the type of visual problems and interventions received as reported in the developmental questionnaire for the preterm participants.

	12 months	24 months	36 months
Vision problems			
Astigmatism	1	0	2
Retinopathy of prematurity (⩽grade 3)	1	1	0
Myopia	1	0	1
Glaucoma	0	0	1
Intervention or consultation			
Physiotherapy	2	0	1
Occupational therapy	1	0	1
Speech-language therapy	1	1	1
Psychology/neuropsychology			
Evaluation of development	0	5	1
Intervention	0	0	1
Neurology	0	0	1

full-term children in order to describe development of the N2 peak in response to oriVEP, texVEP and tsVEP (or texture-segregation N2). Description of N2 development in preterms as well as comparisons between full-term and preterm groups, are subsequently presented.

3.2.1. General development of VEP responses

The grand averaged VEPs for each full-term age group in each condition are illustrated in Fig. 2 (black lines), and mean latency and amplitude values are presented in Table 3. Analyses performed to explore the relationship between N2 (or texture-segregation N2 in the case of subtraction) latency and age at testing, for all three conditions, show a strong negative Pearson correlation between the two variables, with shortening of N2 latency with increasing age for oriVEP ($r = -0.328$, $n = 45$, $p < 0.05$), texVEP ($r = -0.445$, $n = 45$, $p < 0.01$), and tsVEP ($r = -0.536$, $n = 45$, $p < 0.001$). Pearson correlations were also performed to explore the relationship between N2 amplitude and age at testing; however, none of the comparisons were significant (all p's > 0.05), whether for oriVEP, texVEP or tsVEP.

3.2.2. N2 latency and amplitude

In order to further investigate these findings, N2 latency and amplitude were also compared between age groups for ori, tex, and tsVEP for three occipital electrodes: O1, O2 and Oz using analyses of variance with repeated measures. Since the analyses showed similar results for each of these electrodes, only results from the Oz electrode are reported.

Firstly, concerning the latency measures, results revealed no significant interaction between conditions, groups and age ($F(4, 164) = 2.034$, $p = 0.103$, partial $\eta^2 = 0.047$). A significant interaction was found between conditions and age groups ($F(4, 164) = 5.377$, $p < 0.001$, partial $\eta^2 = 0.116$). The main effect of condition was also significant ($F(2, 164) = 4.955$, $p < 0.01$, partial $\eta^2 = 0.057$),

indicating longer N2 latency in more complex conditions when all age groups are taken together. The main effect of age was significant as well ($F(2, 164) = 18.614$, $p < 0.001$, partial $\eta^2 = 0.057$), showing N2 latency reduction with increasing age. More specifically, for oriVEP, post hoc pairwise comparisons show a significant latency reduction between 12 and 36 months ($p < 0.01$) and a tendency for shorter latency between 24 and 36 months ($p = 0.07$). For texVEP, significant latency reduction is found between 12 and 24 months ($p < 0.05$) and between 12 and 36 months ($p < 0.001$). Finally, for tsVEP, pairwise comparisons indicate similar results, with significant texture-segregation N2 latency reduction between 12 and 24 months ($p < 0.001$) and between 12 and 36 months ($p < 0.05$) (see Fig. 3a).

Secondly, regarding the N2 amplitude, results indicated a significant interaction between conditions, groups and age ($F(4, 164) = 3.002$, $p < 0.05$, partial $\eta^2 = 0.065$). Thus, significant differences between variables were identified using post hoc pairwise comparisons. All age groups taken together, a smaller N2 amplitude is found in more complex conditions ($F(4, 164) = 30.486$, $p < 0.001$, partial $\eta^2 = 0.271$). Furthermore, N2 amplitude also tends to diminish with age ($F(2, 82) = 2.929$, $p = 0.059$, partial $\eta^2 = 0.067$). More specifically, analyses revealed significant amplitude reduction between 12 and 24 months for oriVEP ($p < 0.01$). N2 amplitude also decreases between 12 and 24 months ($p < 0.05$), and between 12 and 36 months for texVEP ($p < 0.05$). No significant amplitude differences were found between the three age groups for the texture-segregation N2 amplitude for tsVEP (all p's > 0.1) (see Fig. 3b).

3.3. Development of visual texture segregation in preterm vs. full-term children

To ensure that the results could not be explained by visual problems, all analyses were performed with and without the
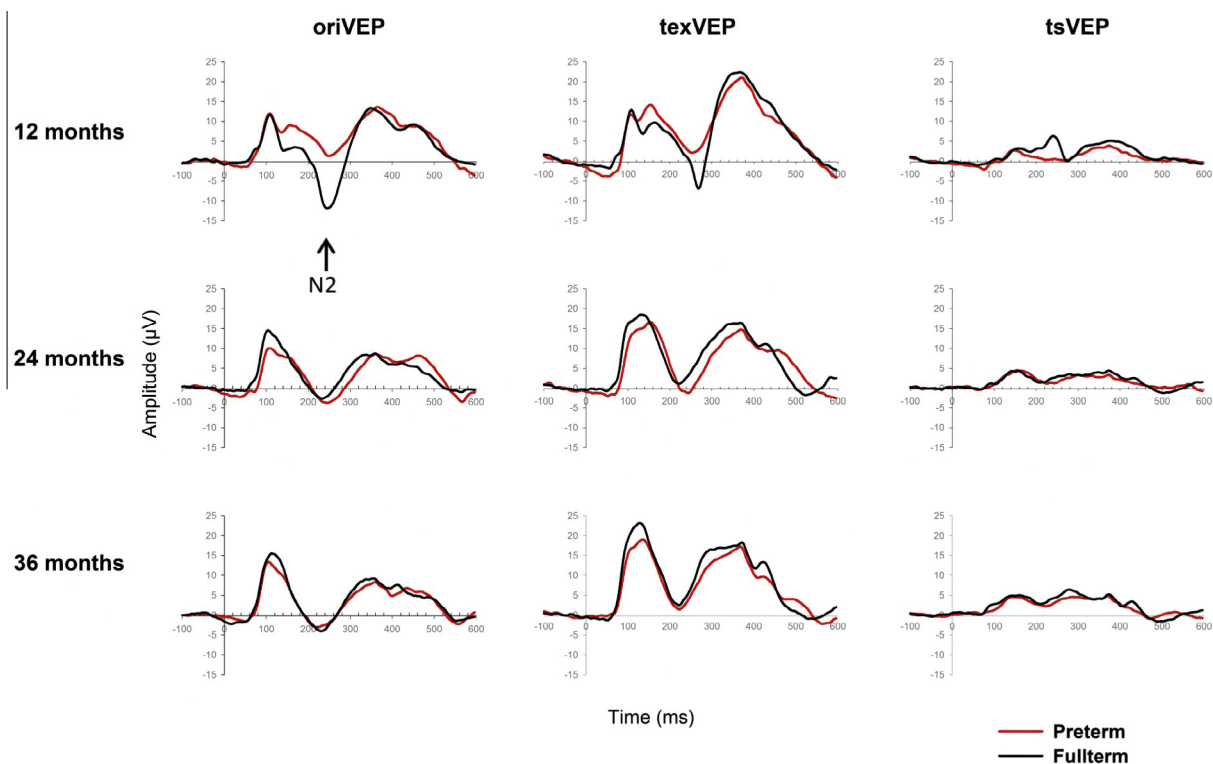

Fig. 2. Grand average VEPs from Oz (central occipital electrode) for full-term (black) and preterm (red) for each age group (12, 24 and 36 months) and each condition (oriVEP, texVEP and tsVEP). There are all presented on the same scale. Amplitude is in μV on the ordinate, latency is in ms on the abscissa. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Table 3
Mean values (standard deviation in parentheses) for N2 latency (msec) and amplitude (μV) at Oz for each group (full-term and preterm) at 12, 24 and 36 months in each condition (oriVEP, texVEP, tsVEP).

		oriVEP		texVEP		tsVEP	
		Latency	Amplitude	Latency	Amplitude	Latency	Amplitude
12 months	Full-term	233.87 (28.08)	−6.54 (5.38)	245.87 (31.78)	−4.45 (5.72)	265.6 (30.34)	−0.62 (4.47)
	Preterm	241.6 (26.7)	−0.53 (2.02)	248.0 (25.12)	−0.005 (3.21)	251.47 (20.89)	−1.26 (3.84)
24 months	Full-term	226.67 (24.87)	−2.47 (2.72)	224.0 (19.53)	−0.54 (3.67)	214.4 (20.22)	0.351 (2.79)
	Preterm	240.0 (17.37)	−2.6 (1.71)	240.27 (15.3)	−0.998 (2.52)	238.4 (27.12)	0.25 (1.78)
36 months	Full-term	203.73 (36.83)	−4.3 (4.86)	215.2 (22.13)	−0.35 (4.86)	218.67 (19.22)	1.48 (4.33)
	Preterm	215.08 (25.31)	−2.63 (1.43)	217.85 (17.02)	0.14 (1.73)	219.0 (20.0)	1.79 (2.6)

participants with reported visual problems ($n = 9$). The results obtained are similar in both cases and consequently, we decided to report all the participants in the following results sections.

3.3.1. Effect of prematurity on general VEP development

The grand averaged VEPs for each preterm age group in each condition are illustrated in Fig. 2 (red lines). Initial analyses explored the relationship between N2 latency/amplitude and the two criteria for prematurity (Beck et al., 2010): preterm level (i.e. number of gestational weeks) and birth weight, for the three conditions. First, Pearson correlations showed significant relationships between variables for oriVEP, with a prolonged N2 latency

($r = −0.220$, $n = 88$, $p < 0.05$) and smaller N2 amplitude ($r = −0.336$, $n = 88$, $p < 0.05$) as gestational age decreases. No significant correlations were found between preterm level and N2 latency/amplitude in texVEP and tsVEP experimental conditions (all p's > 0.1).

Second, regarding relationship between N2 latency and birth weight, Pearson correlations also showed negative correlations between variables for oriVEP, with a decrease in N2 amplitude as birth weight decreases ($r = −0.341$, $n = 85$, $p < 0.001$) and a tendency for longer latency as birth weight decreases ($r = −0.193$, $n = 85$, $p = 0.077$). A tendency for smaller amplitude as birth weight decreases was also obtained for texVEP ($r = −0.194$, $n = 85$,
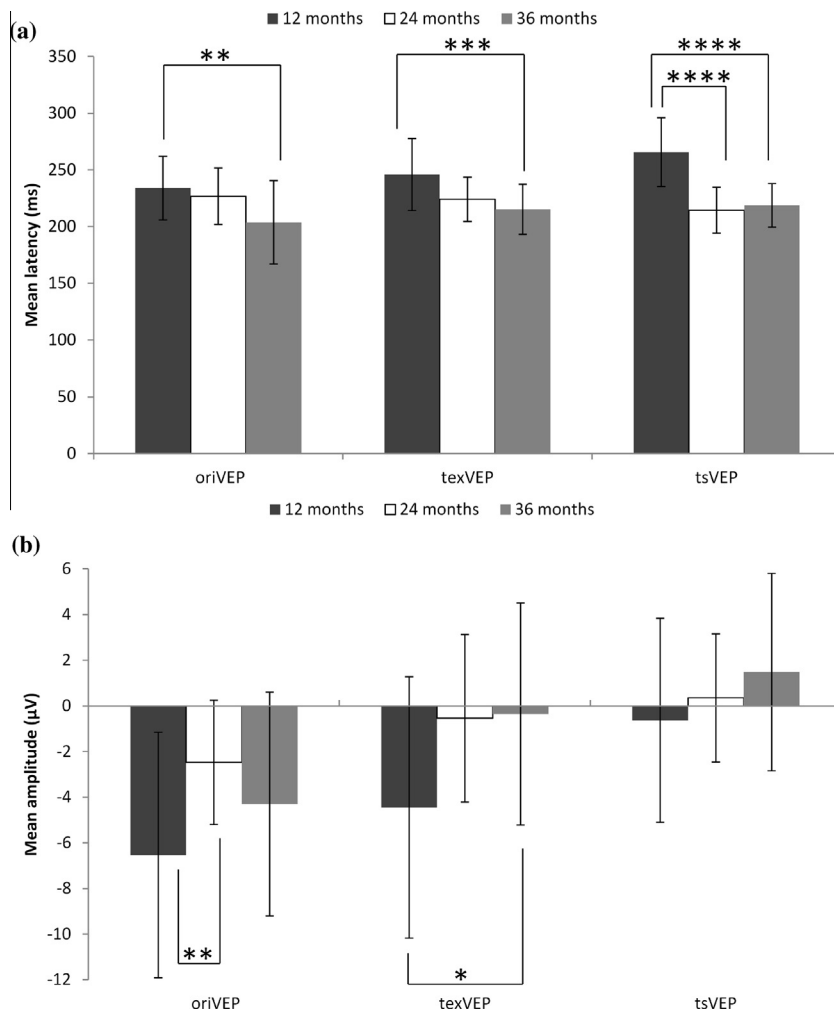

Fig. 3. (a) Mean latency values and (b) mean amplitude values for N2 component for oriVEP, texVEP and tsVEP at Oz electrode for each full-term age group. Errors bars represent standard deviations. Asterisks indicate that differences were statistically significant or tendencies. $^*p < 0.1$ (tendency), $^{**}p < 0.05$; $^{***}p < 0.01$; $^{****}p < 0.001$.

$p = 0.075$). No significant correlations were found between birth weight and texture-segregation N2 latency/amplitude in tsVEP experimental condition (all p's > 0.1).

3.3.2. N2. latency and amplitude

Results presented in this section were obtained with the same (between-subjects for group and age, within subject for condition) analyses of variance with repeated measures that included the full-term children. Therefore, only results related to preterm children at 12, 24, and 36 months for each condition, as well as comparisons with full-term children, are described in the following paragraphs.

First, concerning the latency measures, results revealed no significant interactions between conditions and groups ($F(4, 164) = 0.346$, $p = 0.355$, partial $\eta^2 = 0.013$) and between groups and ages $F(2, 82) = 1.736$, $p = 0.183$, partial $\eta^2 = 0.041$). There was no main effect for group either ($F(1, 82) = 2.666$, $p = 0.107$, partial $\eta^2 = 0.031$). Pairwise comparisons do indicate that preterm childrens' VEPs follow a similar developmental course as the one found in full-term children, with a significant N2 latency reduction between 12 and 36 months for oriVEP ($p < 0.01$), and a tendency for shorter latency between 24 and 36 months ($p = 0.053$). Comparable results were found for texVEP, with a significant N2 latency reduction between 12 and 36 months ($p < 0.01$) and between 24 and 36 months ($p < 0.05$). Results also indicated a significant texture-segregation N2 latency reduction between 12 and 36 months for tsVEP ($p < 0.01$).

Second, regarding the N2 amplitude, results revealed that preterm children do not follow the same VEP developmental pattern than full-term children. In fact, for the preterm groups, pairwise comparisons showed no significant difference in N2 amplitude either for oriVEP or texVEP between the three age groups (all p's > 0.1). However, for tsVEP, results suggest a tendency for smaller texture-segregation N2 amplitude between 12 and 36 months ($p = 0.065$). Moreover, comparisons between preterm and full-term children indicate significantly greater amplitude in full-term compared to preterm at 12 months for oriVEP ($p < 0.001$) and texVEP ($p < 0.01$). No significant differences were found between preterm and full-term children for tsVEP, either for 12, 24 or 36 months of age (all p's > 0.1).

4. Discussion

The purpose of this study was to characterize the developmental pattern of higher-level visual processing, namely texture segregation, during early childhood in typically developing full-term children and preterm children without major neurological impairment. Using tsVEPs and a cross-sectional design, we assessed 15 full-term children in each age group (12, 24 and 36 months) and compared their EEG responses to those of 43 preterm children of the same age (corrected age for prematurity at 12 and 24 months).

4.1. Electrophysiological results for children born full-term

As expected, our results suggest that developmental modifications take place in the visual cortex between 12 and 36 months of age in full-term children. In fact, our VEP results indicate a significant N2 (or texture-segregation N2 in the case of subtraction) latency reduction between 12 months and 36 months group for orientation, texture and texture segregation processes. We also found a significant N2 amplitude reduction between the groups aged 12- and 24-month groups for oriVEP, and a tendency for lower amplitude between 12 and 36 months for texVEP. Consequently, these findings appear consistent with previous studies which showed that maturation of the visual system is demonstrable through VEP latency, amplitude and waveform changes (Brecelj, 2003; Lippe et al., 2007), whereby VEP latency and amplitude measures decrease as children grow older.

In fact, maturation of electrophysiological pattern response follows different time courses in regard of the processing level required. Authors that have specifically studied maturation of brain responses to simple and complex stimulus are in agreement with this idea; they found that EEG responses evoked by simple orientation stimuli appear between 2 and 5 months of age and begin to resemble adult-like patterns within the first year of age (Norcia et al., 2005). However, our findings suggest that VEP responses to orientation continue to develop beyond this age, as we found significant amplitude reduction for oriVEP between 12 and 24 months, accompanied with a significant latency reduction between 12 and 36 months. This is nonetheless consistent with the results of Lewis et al. (2007) who found evidences that the mechanism underlying orientation discrimination is still immature at 5 years old for both first-order (luminance-modulated) and higher-order (contrast-modulated) stimuli. Similar conclusions can be drawn from our results concerning the EEG response pattern obtained using more complex stimuli (texVEP), and with the texture segregation difference wave (tsVEP). We obtained a significantly shorter latency at 36 months in comparison to 12 months for texVEP. A tendency for lower amplitude was also observed between 12 and 36 months for texVEP. For the tsVEP, results indicated a significant latency reduction between 12 and 24 months, and between 12 and 36 months. Thus, these findings complement those of previous studies who showed that VEP in response to a complex stimulus such as texture segregation emerges early in life (around 3 months of age) (Arcand et al., 2007; Atkinson & Braddick, 1992) and continues to develop in terms of latency and amplitude reduction during childhood, probably until early puberty (13–14 years of age) (van den Boomen, Lamme, & Kemner, 2014).

Neuroanatomical studies also suggest that, in the cortical visual areas, the developmental course of the activity related to feedforward connections varies from the activity of recurrent connections (Burkhalter, 1993; Burkhalter, Bernardo, & Charles, 1993; Lamme, Supèr, & Spekreijse, 1998; Roelfsema et al., 2002). This hypothesis proposes that processing of a simple stimulus (i.e. orientation) that needs little integration of details is principally managed by feedforward connections in the visual cortex, while processing of more complex stimuli (i.e. texture segregation) that requires more integration, is supposedly supported by recurrent (feedback) connections. Accordingly, such a theory implies that feedforward connections are completely functional at an earlier age than recurrent connections, which are thought to develop more slowly. This is supported by the study of Burkhalter (1993), who found that recurrent connections are still immature at 5 years old in V1 and that recurrent connections between V1 and V2 develop more slowly than feedforward connections. Again, these findings suggest that texture segregation mechanisms will continue to undergo maturational changes until at least late childhood. In this regard, it has been specifically shown that texture boundaries are processed by both feedforward and horizontal connections, while scene segregation is processed by recurrent connections (Roelfsema et al., 2002; Scholte et al., 2008). Although the stimuli used in our study were not optimal to allow distinction between boundary detection and scene segmentation, when looking at our tsVEPs we could speculate: (1) that boundary detection is present in all our groups, since it is considered as a previous step before texture segregation and that this later is already observable through the texture-segregation N2; (2) that scene segmentation appears to develop later and more slowly, as suggested by the latency and amplitude reductions found between 12 and 36 months of age for both full-term and preterm groups. However, more studies are needed to confirm these hypotheses.

In addition to visual pathway development, anatomical changes that arise within the maturing visual cortex could also account, at least partially, for the differences in the developmental rates of simple and more complex visual processing described above. These include (1) synaptogenesis, which peaks around 9–15 months, followed by a continuing synaptic density decrement until adulthood (Huttenlocher & Dabholkar, 1997), and (2) myelinogenesis, which develops from birth to adulthood in the visual cortex (Paus et al., 2001). How these two anatomical changes affect VEP development is not clearly understood yet. However, it has been proposed by Vaughan and Kurtzberg (1992) that synaptogenesis might be related to the inverted-U shape theory about VEP amplitude (i.e. increase in voltage until 3–6 months then reduction until adulthood); accordingly, the synaptic density reduction could account, at least partially, for VEP amplitude reduction with increasing age. Myelogenesis, on the other hand, is thought to be responsible for the speed of transmission along neurons fibers resulting in shorter latencies with increasing age (Paus et al., 2001). Therefore, our findings in regards of amplitude and latency reduction between 12 and 36 months might reflect, in addition to maturation of visual texture segregation processes per se, anatomical brain changes such as synaptic density and myelinogenesis that occur simultaneously.

4.2. Electrophysiological results for children born preterm

Although no significant differences were found between preterm and full-term children regarding N2 latency, results indicated that preterm children follow the same developmental pattern to the one observed in the full-term children (i.e. significant N2 (or texture-segregation N2 in the case of subtraction) latency reduction between 12 and 36 months for orientation, texture and texture segregation processes). However, this was not the case for N2 amplitude; in fact, no significant differences were found between the three age groups except for a tendency for smaller amplitude between 12 and 36 months for tsVEP. Comparisons between preterm and full-term children revealed that at 12 months, preterm children show smaller N2 amplitude in comparison to full-term for both oriVEP and texVEP. Therefore, these findings suggest that preterm birth could have an impact on cortical vision development, at least during early childhood. Results of the Pearson correlations support this idea as well, by showing relationship between N2 latency/amplitude and gestational age, where the N2 component appears later and with smaller amplitude as gestational age decreases for oriVEP. Although not as strong as the relationship between N2 latency/amplitude and gestational age, similar results were obtained between N2 latency (tendency) and amplitude and birth weight for oriVEP, and N2 amplitude for texVEP (tendency).

Several lines of evidence have shown that preterm birth is a risk factor for sensory impairment, such as vision functioning. Although some authors have hypothesized that, in comparison with full-term born infants, preterm infants without evident brain injury might benefit from additional visual experiences, (Hunnius et al., 2008; Norcia et al., 1987; Ricci et al., 2008), the majority of studies published to date stated otherwise (Birtles et al., 2007; Hammarrenger et al., 2007; Jakobson, Frisk, & Downie, 2006; MacKay et al., 2005; Taylor et al., 2009). In fact, because that maturation of the visual system not only takes place during the prenatal period, but also continues during the first years of life (Chau, Taylor, & Miller, 2013), it is considered immature at birth in full-term children, even more so in preterm children. Throughout the years, many VEP studies have supported this view, by showing lower amplitudes in preterm samples (Feng et al., 2011; Hammarrenger et al., 2007; Kuba et al., 2008) in response to pattern-reversal stimuli, which were interpreted as a disruption

of normal visual development. Consequently, our results regarding lower N2 amplitudes in preterms at 12 months for oriVEP and texVEP suggest that processing of orientation and textured visual stimuli, which is related to feedforward connections, is poorer for these children when compared to their full-term counterparts of the same age. Regarding the tsVEP, the waveforms are immature in both full-term and preterm children and therefore, the texture-segregation N2 is of lower amplitude compared to the N2 the oriVEP and texVEP conditions. Because of this, we can hardly draw direct conclusions on development of higher-level visual processing such as visual texture segregation. Therefore, we cannot completely exclude the idea that recurrent connections are abnormal in preterm children.

Another explanation regarding the lower N2 amplitude found in preterm at 12 months would be that at this age, preterm children have not yet reached their highest N2 amplitude, according to the inverted-U shape theory. These findings could also be interpreted as an effect of neuronal synchronization, where higher N2 amplitude in full-term children reflects a better neuronal synchrony in response to orientated and textured visual stimulations in comparison to preterm children. In fact, some authors suggested that neuronal synchrony correlates with EEG amplitudes (Uhlhaas & Singer, 2006), where a high neural synchrony might be reflected in higher VEP amplitudes. In addition, problems regarding neuronal synchronization have been suggested in other populations with brain disorders such as autism (Just et al., 2004; Uhlhaas & Singer, 2007) and schizophrenia (Spencer et al., 2003). Consequently, we cannot exclude the idea that this factor could have had an influence on our VEP results.

In this context, our results concerning lower amplitudes in preterm might reflect a maturational delay in both orientation and textured visual processing during, at least, early childhood. Nonetheless, preterm children seem to catch up to their full-term counterparts somewhere between 12 and 24 months, since no VEP differences are found between groups beyond this age. Wave morphologies also support this idea. However, because EEG response patterns (1) can vary between subjects, especially during development, and (2) they are still immature in the tested age groups, we cannot exclude the possibility that some differences might have been present in higher-level visual processing (tsVEP) at 12 months, although our study was unsuccessful in demonstrating significant ones.

In summary, results obtained in the present study add to the available knowledge on the development of lower- and higher-level visual processing in developing children. Because of the lack of information concerning visual texture segregation development in both full-term and preterm children however, our findings cannot easily be interpreted in relation with previous studies. Although the impact of preterm birth on more complex visual processes needs more investigation, it is likely that being born preterm somehow affects the development of processes underlying visual texture segregation, as indicated by our VEP results.

4.3. Limitations

The first limitation that can be highlighted regarding our study is that conclusions about age-related differences are based on a cross-sectional design, compared to a longitudinal study that allows comparisons between the same participants at different time points. However, a longitudinal design was not possible due to time restrictions to conduct this project. The second limitation is that amplitude is an electrophysiological parameter than can vary from one subject to another (inter-subject variability), particularly during development. Although we cannot totally exclude the potential influence of this variable on our VEP results, correlations

between gestational age, birth weight and N2 latency and amplitude data were significant, which supports the idea that the more preterm is the birth, the more delayed and reduced in amplitude is the N2 component. The third limitation is that our results did not differentiate preterm children according to their preterm categories (late, moderate, severe and extreme preterm birth) because of the small number of participants in each category. In the current study, we decided to include all preterm children who met our restricted inclusion/exclusion criteria, regardless of their level of prematurity, in order to recruit a sufficient number of participants for reaching a good statistical power.

Finally, another consideration that one might raises in regard of our VEP results is the developmental variation in skull thickness, which has been shown to increase as the child grows older (Epstein, 1974) which may, in turn, impact the VEP amplitude. Because this effect is described as minimal (Hagemann et al., 2008; Tierney et al., 2013) and because the age of our preterm groups were corrected to normal, it is unlikely that skull thickness explain differences found in the VEP amplitude between preterm and full-term children of the same age. Moreover, although not a direct measure of the skull thickness, the head circumference is often used to exclude the potential influence of this anatomical factor (ex. brain volume) on the EEG data (Bartholomeusz, Courchesne, & Karns, 2002). In our case, results indicated no significant differences between groups (preterm and full-term) at any age, which give more support to our VEP results.

Despite these limitations, our study presents new findings about the development of visual texture segregation process between 12 months and 36 months of age, for full-term and preterm children. It is clear, however, that further investigations are needed to continue documenting its typical development, since to our knowledge, no study has investigated its maturation from 3 years old to school-age. The relationship between preterm birth and visual texture segregation process needs to be studied further as well. In this context, longitudinal studies for both populations, full-term and preterm children, might be very useful to investigate in more depth the maturation of this process.

5. Conclusions

Our study is likely the first one to investigate the development of visual texture segregation processes during childhood in both typically developing full-term and preterm born children using VEPs. In fact, very few studies have explored these mechanisms in normal children, even less in preterm children. Our study suggests that in the full-term age groups, developmental modifications take place in the visual cortex between 12 and 36 months of age, resulting in a significant N2 (or texture-segregation N2 in the case of subtraction) latency reduction for ori, tex, and tsVEPs, a significant N2 amplitude reduction between 12 and 24 months for oriVEPs, and a tendency for lower amplitude between 12 and 36 months for texVEPs. As for the preterm groups, our findings indicate similar developmental pattern regarding N2 latency, but not amplitude. In fact, preterm children exhibit smaller N2 amplitude at 12 months for oriVEP and texVEP, when compared to the full-terms. These findings may be related to a maturational delay of cortical visual areas in preterm born children at least during early childhood, and therefore, emphasize the importance of early visual assessment.

As mentioned earlier, the development of visual texture segregation processes in children needs to be further explored, in full-term and in preterm children. We identified only three research papers that have studied visual texture segregation using a developmental perspective: one during the first weeks of life (Atkinson, 1992), one during infancy (Arcand et al., 2007) and the other during late childhood, adolescence and early adulthood (van den Boomen, Lamme, & Kemner, 2014). Therefore, a wide gap exists between infancy and late childhood, and our study proposes new knowledge in regard of visual texture segregation development in both populations, and constitutes a basis for future research as well.

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