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Research report

The neural development of the biological motion processing system does not rely on early visual input

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ABSTRACT

Naturally occurring sensory deprivation in humans provides a unique opportunity to identify sensitive phases for the development of neuro-cognitive functions. Patients who had experienced a transient period of congenital visual deprivation due to bilateral dense cataracts (congenital cataract, cc) have shown, after visual re-afferentation, deficits in a number of higher visual functions including global motion and face processing. By contrast, biological motion (BM) perception seemed to be spared. The present study investigated the neural correlates of BM processing in a sample of 12 congenital cataract-reversal individuals who had underwent visual restoration surgery at the age of a few months up to several years. The individual threshold for extracting BM from noise was assessed in a behavioral task while event-related potentials (ERPs) were recorded in response to point-light displays of a walking man and of a scrambled version of the same stimuli. The threshold of the cc group at detecting BM did not differ from that of a group of matched controls (mc). In both groups, the N1 was modulated by BM. These largely unimpaired neural responses to BM stimuli together with a lack of behavioral group differences suggest that, in contrast to the neural systems for faces the neural systems for BM processing specialize independent of early visual input.

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1. Main text

Brain development comprises phases of enhanced neural plasticity during which the effects of experience are particularly strong (Hensch, 2004; Knudsen, 2004). Such phases are termed sensitive periods (Knudsen, 2004; Lewis & Maurer, 2005). The time course and the degree of experience dependence of neuro-functional development differ across brain regions and even within functional domains, as, for example, for different aspects of vision (Maurer, Lewis, & Mondloch, 2005) or language (Schachter, 1996).

Sensitive periods are properties of emerging neural circuitries (Knudsen, 2004) and have mostly been investigated in animal research using a visual deprivation approach (see pioneer work of Wiesel & Hubel, 1965). In humans only few models exist that allow for a systematic investigation of the time course and neural mechanisms of sensitive periods: such opportunities arise for example, when the re-afferentation of a deprived modality is possible. Individuals born with bilateral dense cataracts (opaque lenses that prevent patterned light to reach the retina) and whose cataracts were surgically removed at different ages provide such a rare opportunity (see Maurer et al., 2005 for a review). Studies in humans have shown that visual deprivation from birth can results in permanent visual impairments at different levels of visual processing. For example, individuals with a history of congenital cataracts often show lower visual acuities, impaired peripheral vision and higher thresholds for the judgment of local motion direction (see Maurer, Mondloch, & Lewis, 2007 for a review). Furthermore, these individuals perform at a lower level compared to controls in tasks relying on an automatic binding of visual features including global motion perception (Ellemberg, Lewis, Maurer, Liu, & Brent, 2002), global form perception (Lewis et al., 2002), configural face processing (Robbins, Nishimura, Mondloch, Lewis, & Maurer, 2010), the ability to recognize faces from different perspectives (Geldart, Mondloch, Maurer, de Schonen, & Brent, 2002; Putzar, Hötting, & Röder, 2010), and the perception of illusory contours (Putzar, Hötting, Rösler, & Röder, 2007). Despite significant impairments at perceiving the direction of local motion (Ellemberg et al., 2005) as well as at extracting global motion (Ellemberg et al., 2002; Hadad, Maurer, & Lewis, 2012), recent evidence has suggested that the behavioral sensitivity to human biological motion (BM) is largely unaffected by periods of congenital visual deprivation (Hadad et al., 2012). However, to finally reject the idea of a sensitive period for BM processing, it must be demonstrated that congenital cataract-reversal individuals engage the same neural system for BM processing as controls rather than using alternative routes, as for instance a more controlled processing mediated by later visual processing stages (Fieger, Röder, Teder-Sälejärvi, Hillyard, & Neville, 2007). As an example, it has been shown that even though congenital and late permanently blind individuals show similarly enhanced auditory localization, they use different neural systems (Fieger et al., 2007).

In the present study we made use of the high temporal resolution of non-invasive event-related potential (ERP) recordings to investigate the effects of a transient visual deprivation on the development of the neural systems of BM: typically sighted controls detect BM within the first 250 msec post stimulus onset, resulting in an enhanced N1 response of the ERP (Hirai, Senju, Hirokata, & Hiraki, 2005; Hirai, Watanabe, Honda, & Kakigi, 2009; Jokisch, Daum, Suchan, & Troje, 2005; Krakowski et al., 2011). In a previous ERP study in a group of cataract-reversal individuals (Röder, Ley, Shenoy, Kekunnaya, & Bottari, 2013) we demonstrated a lack of a functional specialization of the neural systems for face processing. In the present study, we recorded ERPs to BM and scrambled BM stimuli in a sample of 12 individuals who had been totally deprived of patterned visual input from birth for a few months up to 16 years. Differences in the ERPs between cc individuals and matched controls (mc) despite a lack of behavioral group differences would suggest a sensitive period for the functional specialization of the neural systems for BM processing and an alternative route serving functional recovery. By contrast, if cc individuals and their controls would not differ, either at the behavioral or at the neural level, we would reject the hypothesis of a sensitive phase for the functional specialization of the BM processing system.

2. Methods

2.1. Participants

The cc group comprised 12 individuals with a history of congenital, bilateral, dense cataracts (cc: mean age = 17.8 years, ranging between 10 and 35 years of age, for details see Table 1). All cc individuals were recruited at the LV Prasad Eye Institute in Hyderabad, India. Cataract history was confirmed from the medical records. Cataracts presence was diagnosed at different ages, therefore, the following additional criteria were applied to guarantee that only individuals with total ccs were included in this group: the presence of a nystagmus, a density of the lenticular opacity, an invisibility of the fundus prior to surgery, a family history and family reports. Prior to surgery, most of the participants had only light perception (see Table 1). cc individuals underwent surgery on average at the mean age of 94 months (range: 4–192 months). The duration of visual deprivation (time since surgery) was on average 119 months (range: 12–396 months). Mean visual acuity, measured post surgery at the best eye, was on average .14 (range: .05–.50). All cc individuals took part in the BM EEG experiment. A subset of them, comprising 7 individuals (3 females, mean age: 18.5 years, range: 11–35 years, mean visual acuity: .2, range: .05–.50; mean age at surgery: 86 months, range: 4–168 months, see Table 1) was tested in an additional behavioral task which assessed the behavioral threshold for detecting BM (BM behavioral task; see Table 1). All cc participants were right-handed and neurologically healthy according to self report and medical examination by a physician.

A group of aged matched healthy participants was recruited as control (mc) in Hamburg, Germany, for the BM EEG experiment and the BM behavioral task. All mc participants had normal or corrected to normal vision and were neurologically healthy according to self report. A group of 12 individuals participated in the BM EEG experiment (6 females, mean age: 18 years, range: 8–37 years). In addition, a sample

Table 1 – Description of participants and performance in the four different tasks.

Participant	Age (years)	Gender	Cataract onset	Age at surgery (months)	Fundus visibility pre-surgery best eye	Nystagmus	Presurgical visual acuity in best eye*	Last postsurgical visual acuity in best eye		Mean accuracy in BM EEG task, %	BM detection task, noise dots n**
								Decimal	logMar		
cc-a	23	M	Congenital	48	Unknown	Yes	Unknown	.16	.80	100.0	46.3
cc-b	35	M	Congenital	24	Unknown	Yes	Unknown	.50	.30	93.8	13.7
cc-c	17	F	Congenital	168	No view	Yes	FC:0.5	.13	.90	100.0	–
cc-d	17	F	Congenital	192	No view	Yes	PL+, PR+	.02	1.78	97.8	–
cc-e	10	M	Congenital	108	No view	Yes	PL+, PR+	.02	1.78	100.0	–
cc-f	11	F	Congenital	120	No details	Yes	FC:0.5	.05	1.30	96.4	0.2
cc-g	31	M	Congenital	72	Unknown	Yes	No vision	.05	1.30	100.0	27.4
cc-h	11	F	Congenital	120	No view	Yes	PL+, PR+	.16	.80	100.0	10.2
cc-i	11	M	Congenital	96	No view	Yes	PL+, PR+	.32	.50	100.0	22.7
cc-l	13	M	Congenital	120	No view	Yes	PL+, PR+	.02	1.78	100.0	28.7
cc-m	21	M	Congenital	4	Unknown	Yes	Unknown	.13	.90	100.0	–
cc-n	13	M	Congenital	60	No view	Yes	Unknown	.13	.90	97.4	–
Mean	17.8			94.3				.14	1.09	99.8	21.3

* PL+: able to perceive light; PR+: able to report the location of light; FC: able to count fingers at *n* meters.
BM: Biological motion** Degree if noise at which a hit rate of 82% was reached (high values indicate high sensitivity) in the behavioral BM task.

of 7 individuals participated in the BM behavioral task (3 females, mean age: 19.0 years, range: 10–27 years).

Participants and, for minors, legal guardians provided informed consent after experiments had been explained. The study was approved by the ethical committee of the German Society of Psychology and by the ethical committee of the Hyderabad Eye Research Foundation.

2.2. *Stimuli and apparatus*

2.2.1. *BM behavioral task*

BM detection thresholds were assessed with an adapted version of the subtest "Detection Test" of the Biological Motion Perception Test Battery (BML test battery, for details see Saunders & Troje, 2011). One stimulus consisted of a walker made out of point-lights (Troje & Westhoff, 2006) embedded in scrambled walker noise. The second display showed the same number of dots but only scrambled walker motion. The participants' task was to report whether the walker was presented in the first or in the second display. The inter-trial-interval (stimulus offset to stimulus onset, ITI) between displays was set to 1 sec, starting from the participants' response. The number of masking noise dots was set by a QUEST procedure. The detection threshold was defined as the number of dots comprising the mask at which the participant achieved a hit rate of 82%. The test terminated after 40 trials. The behavioral task lasted on average 10 min.

2.2.2. *BM EEG experiment*

Stimuli consisted of three categories of dynamic motion point-light displays: (1) walkers, (2) scrambled walkers and (3) a walking cat (for details on the stimuli see Troje & Westhoff, 2006). The walker subtended a height of 8.6°, and a width of 4.3°. In the scrambled walker stimuli (scrambled BM, SBM), the location of each point-light trajectory was randomly swapped with the location of another trajectory. This scrambling procedure preserves the overall shape of the walker while scrambling local motion. At the same time this procedure insures that the size of the stimuli within each stimulus category is matched. During locomotion, the walker remained at the same central location. Most importantly, the starting phase of each walker was randomized, so that incidental shape differences between intact and scrambled walkers were averaged out. Eight different walkers were created, four facing left, four facing right; during the locomotion the figures remained at the same central location. In addition, point-light displays depicting the profile view of a walking cat (facing either to the left or to the right) were used as behavioral targets. Each stimulus was presented for 2000 msec. The inter-trial interval ranged pseudo-randomly from 2000 to 2700 in steps of 100 msec. A total of 48 target trials ($p = .17$) were randomly intermixed with 240 trials ($p = .83$), half with intact and half with scrambled walkers. A total of 6 blocks were presented including 48 trials each. Participants were asked to detect the walking cat and to respond via mouse button press or verbally.

Stimuli were presented with a Dell laptop on a 22 inches Dell LCD monitor with a refresh rate of 60 Hz. Stimuli where created with MatLab© and the psychotoolbox 3 software

(Brainard, 1997; Pelli, 1997) and presented using the Presentation® software (http://www.neurobs.com/). Using a custom made photo diode circuit we tested the latency of the stimuli presented in the EEG tasks directly on the screen. We adjusted the EEG markers accordingly to the measured delay in the EEG analysis.

During the experimental session participants sat at a distance of 60 cm from the monitor and were instructed to maintain their head and gaze towards fixation. Compliance with fixation was controlled by the experimenter, sitting beside the participant. Written and verbal task instructions were provided; when necessary a translator instructed the participants in their native language. Prior to the start of the experiment, one block of trials was run as practice. The total experiment, including EEG task, EEG application and removal, took on average 2 h. The EEG experiment was run after the behavioral task in each participant.

2.3. EEG recording

The EEG was continuously recorded (analog bandwidth: .01–200 Hz, sampling rate: 500 kHz, BrainAmp, http://www.brainproducts.com/) with 30 passive Ag/AgCl electrodes attached to an elastic cap (Easy cap) at standard 10–20 sites including Fp1, Fp2, F7, F3, Fz, F4, F8, T7, C3, Cz, C4, T8, P7, P3, Pz, P4, P8, O1, and O2. Additional electrodes at intermediate sites were mounted at FC5, FC1, FC2, FC6, TP9, CP5, CP1, CP2, TP10, F9 and F10. All scalp recordings were performed against the right ear lobe. Horizontal eye movements were monitored with a bipolar montage comprising two electrodes close to the left and right outer canthi of the eyes (F9, F10). Vertical eye-movements were monitored with frontal electrodes (Fp1, Fp2). Offline, data were down sampled to 250 Hz and re-referenced to Fz. To eliminate artifacts related to eye movements and heart beat we performed an Independent Component Analysis (ICA; Comon, 1994) runica version, implemented on EEGLAB (Delorme & Makeig, 2004) running in MatLab©. The ICA was conducted for each participant on the whole data recording decomposing the data into 31 components. For every component, we visually inspected the dynamic, the distribution on the scalp, the distribution across the trials, and the power spectrum. Only components clearly showing eye related activity and heart beat (when detectable) were removed. In addition, trials with signals exceeding 100 μV (130 μV for participant cc-c and cc-f) were eliminated prior to averaging. For all participants, at least 40% artifact free trials remained after the artifact elimination. ERP analyses were conducted for the frequent standard stimuli (the walkers), which did not require any response. Electrophysiological recordings were analyzed with the EEGLAB (Delorme & Makeig, 2004) and the fieldtrip software (Oostenveld, Fries, Maris, & Schoffelen 2011).

2.4. ERP analysis

ERPs were averaged over a period of 1500 msec including a 500 msec pre-stimulus epoch. All ERPs were baseline corrected using the −100–0 msec pre-stimulus epoch. Finally, the ERPs were digitally filtered (low-pass filter with a 40-Hz upper cut-off). The N1 (negative deflection peaking between 160 and 260 msec after stimulus onset) of the ERP was the main target of the analysis as the N1 has repeatedly been demonstrated to be enhanced to visual BM compared to the corresponding scrambled stimuli (Hirai et al., 2005; Jokisch et al., 2005; Krakowski et al., 2011). Nevertheless, following the observation of clear between group differences at an earlier latency, the P1 (positive deflection peaking between 90 and 140 msec after stimulus onset) of the ERP was analyzed as well.

Mean amplitude measure of P1 and N1 waves: in order to compensate for latency differences across individuals (partially due to different ages), the P1 and N1 peaks were assessed individually and semi-automatically with a custom made Matlab program. The P1 peak was searched within a time window between 72 and 172 msec, the N1 within a time window between 140 and 300 msec after stimulus onset. The peaks were identified across the most posterior electrodes (TP9-10, P3-4, P7-8, O1-2) for each individual and condition. For each participant the peak latency of the P1 and N1 was defined as the average of the peak latencies measured across the conditions BM and scrambled BM for the N1, and across the conditions BM, scrambled BM and targets for the P1. The latter was done to compare the P1 mean amplitude between groups across all stimulus types, since the P1, in contrast to the N1 amplitude did not vary as a function of BM. Centered at the so defined peak latency, a 64 msec (±8 data samples at 250 Hz) long window was created and used to extract the mean amplitude at all electrodes; these mean amplitude served as dependent variable for the statistical analysis. Mean amplitudes were extracted for each participant, condition and electrode.

The mean amplitudes of the ERPs were statistically analyzed at the electrodes for which visual ERPs are known to reach their highest amplitudes: T7, T8 (temporal), TP9, TP10 (temporal-occipital), P7, P8 (parietal-occipital), and O1, O2 (occipital). The P1 was analyzed at P7, P8 and O1, O2, as the P1 has a more posterior scalp distribution. For the statistical analysis, mean amplitudes of the N1 and the P1 were separately submitted to repeated-measures ANOVAs with Hemisphere (left and right), Electrode location (depending on the ERP wave, four or two electrodes per hemisphere for the N1 and P1, respectively) and Condition (BM *vs* Scrambled BM for the N1 analysis and BM, Scrambled BM *vs* Target for the P1 analysis) as within-subject factors, and Group (cc and mc) as between-subjects factor.

In the cc group the following correlations (Pearson's *r*) were calculated between the BM effect (N1 difference amplitude: BM-SBM) and the duration of visual deprivation (the time from birth to surgery of the first eye), the amount of visual experience (the time from the first cataract surgery to the day of testing) and the post surgery visual acuity.

The latency of the N1 and the P1 peak was compared between groups with an ANOVA comprising the repeated measurement factor condition (BM and SBM; BM, Scrambled BM *vs* Target for the P1 analysis) and the between-subject factor Group (cc and mc).

In case of a sphericity violation, the Greenhouse–Geisser or the Huynh–Feldt correction (the latter when the Epsilon value was greater than .75) was applied.

3. Results

3.1. Behavioral results

Thresholds as assessed with the "Detection test" of the Biological Motion Perception Test Battery (BML test battery, for details see Saunders & Troje, 2011) were compared between groups with a one-way ANOVA with group (cc and mc) as between-subject factor. The performance of the cc group ($n = 7$, see Table 1) and the mc group ($n = 7$; see Fig. 1a) did not significantly differ [$F(1,12) = 1.7$, $p > .2$].

In the EEG session, all groups displayed a high detection accuracy with no differences between groups [target detection: cc = 98.8% SE = .6%, mc = 98.6% SE = 1.0%; $F(1,22) = .2$, $p > .8$; see Fig. 1b]. False alarms rates were below 1% in all groups.

3.2. ERP results

3.2.1. N1

The ERPs to BM and SBM are displayed in Fig. 1c, d. The N1 was higher in amplitude for BM than SBM stimuli in both groups. This amplitude effect was indistinguishable between groups (see Figs. 1 and 2). The N1 was characterized by a posterior topography in both groups. An ANOVA was run with Group as between participant factor (cc and mc) and the repeated measurement factors Electrode location (temporal, temporo-occipital, parieto-occipital, occipital), Hemisphere (left and right) and Condition (BM and SBM). A main effect of Condition [$F(1,22) = 9.9$, $p < .01$] indicated larger N1 amplitudes to BM than to SBM. The interaction of Group and Condition was not significant [$F(1,22) = .1$, $p > .7$]. Follow up ANOVAs were run separately for each group and confirmed that BM stimuli elicited larger N1 amplitudes than SBM stimuli in the mc group [$F(1,11) = 4.8$, $p = .05$] and in the cc group [$F(1,11) = 5.1$, $p < .05$ see Figs. 1c, d and 2]. No other main effect or interaction involving the factor group was significant (all Fs < 2.1). A Lavenen's test verified the equality of variances (homogeneity of variance) of the N1 effect (BM-SBM) between the two groups ($p > .3$).

In the cc group, neither the duration of visual deprivation nor the amount of visual experience, nor the post surgery visual acuity correlated with the size of the N1 effect (BM-SBM; $r = .1$, $p > .7$; $r = -.1$, $p > .9$ and $r = -.1$, $p > .7$, respectively; see Supplementary Fig. 1).

The latency of the N1 peak was compared between the two groups in an ANOVA with Group as between participant factor (cc and mc) and Condition (BM and SBM) as within participant factor. The main effect of Group was not significant

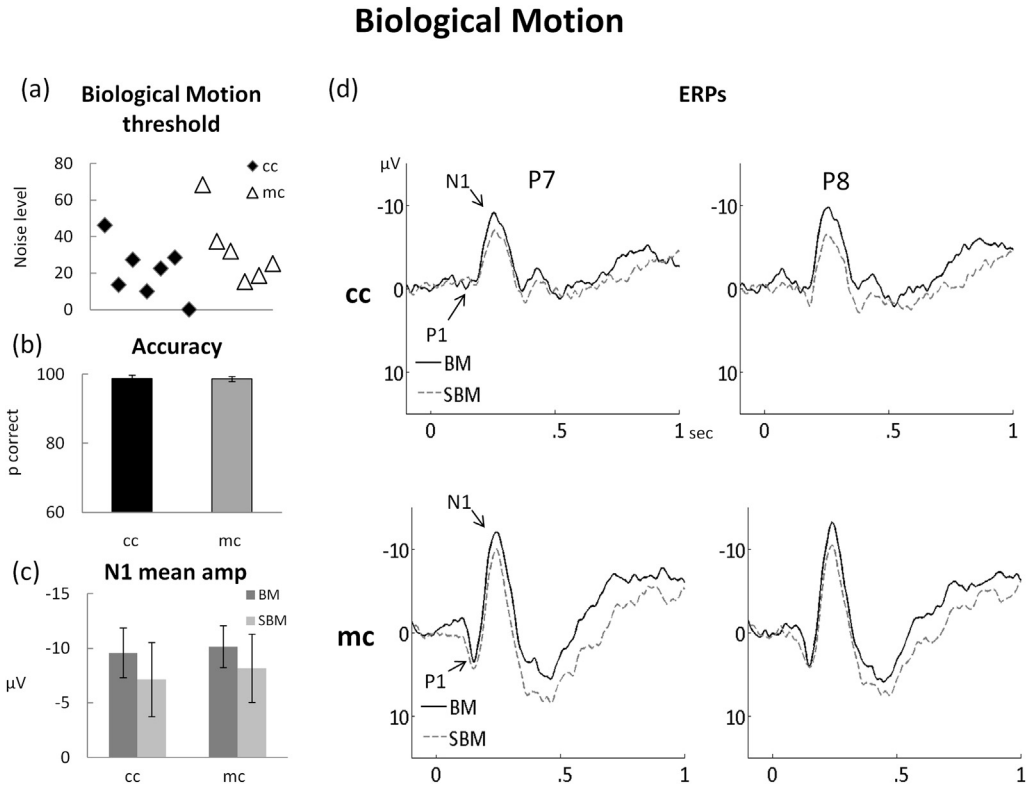

Fig. 1 – Biological motion (BM) detection thresholds as well as behavioral results and event-related brain potentials. (a) Thresholds of single participants [black diamonds = cc (congenital cataract individuals), white triangle = mc (matched controls)] for detecting BM: high values indicate high sensitivity. (b) Hit rates in the biological EEG task separately shown for the cc and the mc groups. (c) N1 amplitudes (average between the electrodes: T7-8, TP9-10, P7-8, O1-2) to biological motion (BM) and scrambled biological motion (SBM) stimuli separately averaged for the cc and mc group. (d) Event-related brain potentials at the parietal electrodes P7 and P8 (left and right hemisphere respectively) averaged across all participants of the cc group and the mc group. Traces for BM and SBM are superimposed.

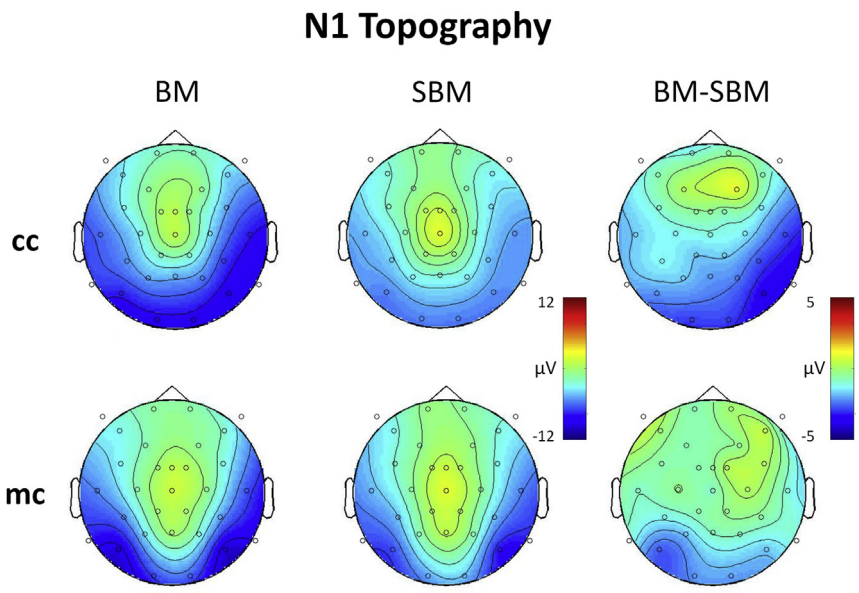

Fig. 2 – Topographic maps of the N1 (averaged on a 10 msec time window centered on each group N1 peak, cc = 234 msec, mc = 212 msec) to BM, SMB as well as for the difference BM-SBM separately shown for the cc group (upper panel) and the mc group (lower panel).

[$F(1,22) = 2.3$, $p > .1$] suggesting indistinguishable latencies between the two groups (cc group mean = 234 msec, SE = 7.9; and mc group mean = 212 msec, SE = 5.1), irrespective of the condition (interaction of group and condition: $p > .2$).

These results suggest that the N1 specific response to BM was preserved in individuals with a history of a transient congenital blindness after vision had been restored.

3.2.2. P1

The P1 of the cc group was reduced in amplitude compared to the mc group (see Fig. 1d and Supplementary Fig. 2). An ANOVA with Group (cc and mc) as between participant factor and Condition (BM, SBM *vs* Targets), Electrode location (parieto-occipital, occipital) and Hemisphere (left *vs* right) as within participant factors revealed a significant main effect of Group [$F(1,22) = 4.8$,$p < .04$] due to a reduction of the P1 amplitude in the cc group (mean = .1 μV, SE = .8) compared to the mc group (mean = 5.3 μV, SE = 2.2). No interactions involving the factors condition and group were significant (all *ps* > .2) suggesting that the P1 was overall reduced in amplitude in the cc group that is independent of the stimulus category (see Fig. 1d and Supplementary Fig. 2). Indeed, a reanalysis of another data set of 12 cc and 12 mc from the study of Röder et al. (2013) confirmed the overall reduced P1 amplitude of cc individuals compared to controls for a second stimulus set (static faces and houses as well as their scrambled versions) and by and large independent sample (8 new cc individuals; see Supplementary material). The P1 peak latency was compared between the two groups with an ANOVA with Group as between participant factor (cc and mc) and Condition (BM, SBM and Targets) as within participant factor. The main effect of Group was not significant [$F(1,22) = 1.8$, $p > .19$] suggesting no latency differences between the two groups (cc group mean = 125 msec, SE = 5.2; and mc group mean = 134 msec, SE = 4.2), irrespectively of the condition (interaction of group and condition: $p > .17$).

4. Discussion

The present study combined a behavioral assessment of biological motion processing (BM) and the recording of ERPs in individuals who had experienced a transient period of visual deprivation lasting up to several years prior to visual restoration by means of cataract surgery (cc individuals). In order to reject the hypothesis of a sensitive period for the development neural systems for BM, as has been suggested by previous behavioral results, it had to be demonstrated that cc individuals do not only show unimpaired behavioral performance but, in addition, that they employ the same neural systems to perform the task. In line with a previous study (Hadad et al., 2012), we did not find evidence for an impairment in BM processing in cc individuals in either of our two behavioral BM tasks. Moreover, the N1 of the ERPs was modulated by BM in both the cc group and a matched control group, confirming the results of previous studies in healthy individuals (Hirai et al., 2005, 2009; Jokisch et al., 2005; Krakowski et al., 2011). Finally, the N1 latency and the topography of this effect were indistinguishable between cc individuals and controls, suggesting the involvement of similar neural circuits, independent of group. These results argue against a sensitive phase for the setting up of a neural system for visual BM processing.

There are three possible explanations that might account for the present findings:

(1) The neural circuitries associated with the processing of BM can specialize in late childhood or adulthood. That is, as soon as visual input becomes available, initiates the

functional maturation of the BM system. Alternatively the neural systems for BM might mature independently of vision. (2) Either they are shaped crossmodally or (3) they mature independent of experience.

In favor of the first hypothesis are findings of a prolonged sensitive phase if adequate input is not available to functionally tune a neural system, such as for ocular dominance (Mower, 1991). Thus, it could be assumed that the neural systems for BM remain in a pre-sensitive period state and mature following visual restoration. Although we are not able to finally exclude this hypothesis, we consider this account of the present data as unlikely, since the maintenance of a pre-sensitive period status has yet only been shown for a few months (Cynader & Mitchell, 1980; Fagiolini, Pizzorusso, Berardi, Domenici, & Maffei, 1994; Mower, 1991; Mower, Caplan, Christen, & Duffy, 1985) rather than for up to 16 years.

Alternatively, it might be speculated that the neural systems for visual BM are initially driven by multiple modalities, such as auditory motion signals. In typical development such crossmodal activation disappears leaving predominantly visual processing in these areas (see Johannsen & Röder, 2014). Indeed, in congenitally permanently blind individuals a crossmodal activation of visual areas for faces (Hölig, Föcker, Best, Röder, & Büchel, 2014) or motion (Bedny, Konkle, Pelphrey, Saxe, & Pascual-Leone, 2010; Collignon et al., 2011; Collignon, Voss, Lassonde, & Lepore, 2009; Poirier et al., 2006; Ricciardi et al., 2007) by functionally analog stimulation (voices and auditory motion stimuli, respectively), has been reported (see Lomber, Meredith, & Kral, 2010 for an animal model). Thus, this account would assume that if visual input becomes available late, it might make use of the crossmodally tuned neural circuits. However, other authors have argued that such a crossmodal recruitment during the phase of sensory deprivation might be detrimental rather than advantageous for functional recovery (Kral & Eggermont, 2007; Lee et al., 2001).

The third hypothesis would propose that neural systems for BM develop independent of visual input, e.g., genetically determined or driven by spontaneous activity of the retina (Katz & Crowley, 2002) and/or within the neural circuits (Balmer & Pallas, 2013). Indeed, studies in chicks observed neural systems which were sensitive to foot motion without any prior visual experience (Vallortigara & Regolin, 2006). In particular, newly hatched chicks, which were reared in total darkness, displayed a preference for BM stimuli (a point-light display of a hen) moving in an upright rather than an upside-down orientation (Vallortigara & Regolin, 2006). Similarly, newborns prefer BM compared to SBM stimuli without any prior visual experience (Bardi, Regolin, & Simion, 2011; Simion, Regolin, & Bulf, 2008). Finally, the ERP topography in response to BM stimuli in 8 months infants has been reported to be adult like (Hirai & Hiraki, 2005; Reid, Hoehl, & Striano, 2006, respectively) suggesting that no substantial reorganization of the BM system takes place from infancy to adulthood. In the present study cc individuals were not only indistinguishable from controls in behavioral BM processing, they were indistinguishable from their controls at a neural level as well. Thus, our ERP data suggest that cc individuals use the same neural system as controls for processing BM. These data make a sensitive phase for BM processing highly unlikely. Together with the arguments above, we conclude that the neural systems for BM specialize independently of visual experience. Nevertheless, this conclusion should be confirmed in a longitudinal study which assesses behavioral indices and ERPs to BM processing as soon as possible after surgery.

It could be argued that the present study lacked sufficient power to demonstrate differences in BM related systems and behavior between groups. We consider this possibility as unlikely since we were able to demonstrate group differences in the P1 amplitude (lower amplitudes in the cc group). Additionally, one might doubt that BM is processed in a specialized neural system. However, this idea would be incompatible with neuropsychological data demonstrating a selective loss of BM in patients with lesions to the superior temporal sulcus (STS; Vaina & Gross, 2004). In addition, this account would be incompatible with previous reports in cc individuals. We have shown, partially in the same individuals, that the neural systems for face processing do not specialize for the processing of faces in cc individuals (Röder et al., 2013). The N170, an ERP known to indicate the structural encoding of faces, was of the same size in cc individuals for all tested object categories (faces, houses) as well as for their scrambled version. In Supplementary Fig. 3, ERPs for BM and SBM and for faces and scrambled faces as assessed by Röder et al. (2013) are shown in three cc individuals who participated in both studies. While these three cc individuals demonstrate a BM effect in the N1 they do not demonstrate a face-specific effect in the N170. Therefore, the present study argues for a dissociation of the neural systems for the processing of faces and for the processing of BM. Interestingly, these systems have often been considered as tightly linked (Engell & McCarthy, 2013). Our data, thus, provide strong evidence for independently developing neural systems resulting in functional distinct neural circuits.

We can only speculate why these different developmental trajectories for faces and BM emerge: BM is characteristic for any type of living being and the major properties are shared across species. Combining physiological and neuroimaging data on humans and animals, Giese and Poggio (2003) proposed a feed-forward model comprising in parallel organized ventral (form related) and dorsal (optic flow related) visual pathways associated with the processing of BM. By contrast, faces are highly specific for a species and biases for the processing of faces from our own ethnicity and age have been shown (Wiese, Kaufmann, & Schweinberger, 2012). Thus, a system that is born with a by and large matured processing system for BM is well adapted for the environment as for instance when faced with catching prey or escaping from predators (see Puce & Perrett, 2003). By contrast, the face system must be tuned not only to one's own species and one's own social group (Sangrigoli, Pallier, Argenti, Ventureyra, & de Schonen, 2005).

5. Conclusions

In sum, our data demonstrates a lack of a sensitive phase for the development of the neural systems mediating BM processing. This finding is in stark contrast to numerous

impairments previously observed in this population. Thus, our data provides evidence for a dissociation of visual systems for BM and face processing and thus independent developmental trajectories within the visual system.

Competing financial interest

The authors declare no competing financial interests.

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Supplementary material

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