Sparing of sensitivity to biological motion but not of global motion after early visual deprivation

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Abstract

Patients deprived of visual experience during infancy by dense bilateral congenital cataracts later show marked deficits in the perception of global motion (dorsal visual stream) and global form (ventral visual stream). We expected that they would also show marked deficits in sensitivity to biological motion, which is normally processed in the superior temporal sulcus via input from both the dorsal and ventral streams. When tested on the same day for sensitivity to biological motion and to global motion at two speeds (4 and 18° s⁻¹), patients, as expected, displayed a large deficit in processing global motion at both speeds. Surprisingly, they performed normally in discriminating biological motion from scrambled displays, tolerating as much noise as their age-matched controls. Networks bypassing damaged portions of the dorsal and the ventral streams must mediate the spared sensitivity to biological motion after early visual deprivation.

Introduction

Studies in humans, like those in monkeys, indicate that early visual input is necessary for normal visual development, even for aspects of vision that are very poor at birth and that have a protracted period of development. In humans, the long-term effects of early visual deprivation are evident from permanent visual deficits in individuals born with dense central cataracts in both eyes, despite treatment to remove the cataracts during infancy and many years of subsequent visual input. In such patients, a few visual capabilities are spared: sensitivity to low spatial frequencies and high temporal frequencies (Ellemberg, Lewis, Maurer, Liu & Brent, 1999) and discrimination of large shapes of high contrast (Maurer, Lewis & Brent, 1989). However, most visual capabilities are impaired. In addition to low-level visual deficits in acuity, peripheral vision, and perceiving the direction of local motion (see Maurer, Mondloch & Lewis, 2007 for a review), there are deficits in capabilities mediated at higher levels of the visual pathway where information from local signals is integrated: processing of local motion (Ellemberg, Lewis, Maurer, Brar & Brent, 2002), processing of global form (e.g. Lewis, Ellemberg, Maurer, Wilkinson, Wilson, Dirks & Brent, 2002), and many aspects of face processing (e.g. Le Grand, Mondloch, Maurer & Brent, 2004; Robbins, Nishimura, Mondloch, Lewis & Maurer, 2010). The lack of correlation between these higher level deficits and acuity suggests that they likely reflect damage to higher levels of the visual pathway, rather than being merely the result of poor visual input from neurons in the damaged primary visual cortex.

Here, we evaluated the effects of early visual deprivation on another aspect of vision that is dependent on higher levels of the visual pathway and that requires the integration of local signals, namely the processing of point light animations depicting biological motion (Johansson, 1973). Studies using fMRI and transcranial magnetic stimulation show that the posterior region of the superior temporal sulcus (pSTS), an area that receives substantial input from both the dorsal and ventral visual streams, plays an important role in the perception of biological motion (Puce & Perrett, 2003). Because early visual deprivation compromises sensitivity to global motion, which depends on the dorsal visual stream (e.g. Maunsell & Newsome, 1987), and sensitivity to global form, which depends on the ventral visual stream (Gallant, Connor, Rakshit, Lewis & van Essen, 1996), we predicted that such deprivation would also compromise sensitivity to biological motion.

In a pilot study, we found that six patients treated for bilateral congenital cataract were normal in discriminating biological motion from scrambled motion, tolerating as much noise as their age-matched controls. The sparing of biological motion is surprising, given documented impairments in this cohort in sensitivity to global motion (Ellemberg et al., 2002), global form (Lewis et al., 2002), and holistic face processing (Le Grand et al., 2004;
Robbins et al., 2010), all of which require a similar type of spatial and temporal integration. However, the patients were on average older when tested on biological motion than the other tasks, and it is possible that deficits in these integration processes decrease with age. The present experiment examined this possibility by testing eight patients on three tasks on the same day: a biological motion task requiring the discrimination of biological from scrambled motion, sensitivity to global motion with the dots moving at 4° s⁻¹ (the average speed of movement in the biological motion task), and sensitivity to global motion with the dots moving at 18° s⁻¹ (the speed in our previous study of patients’ sensitivity to global motion – Ellenberg et al., 2002). Results confirm the sparing of biological motion but not of global motion after early visual deprivation.

**Method**

**Participants**

Visually deprived patients

The patient group consisted of eight patients treated for bilateral congenital cataracts, aged 8 to 29 years at the time of testing. Patient details are summarized in Table 1. Patients were included in the sample only if they had been diagnosed on the first eye exam before 6 months of age with bilateral dense central cataracts that blocked all patterned visual input. Treatment involved surgical removal of the cataractous lenses and fitting of compensatory contact lenses. We assumed that these patients had been deprived from birth because it would be unusual to have dense cataracts develop rapidly between birth and 6 months. Consequently, we defined duration of deprivation for each eye as the period extending from birth until the age of first optical correction after surgery to remove the cataract (i.e. the first time the infant received focused visual input). After normal correction, visual input was only nearly normal because contact lenses focused input perfectly for only one distance, and the eyes could not accommodate for other distances. Detailed inclusion and exclusion criteria for bilaterally deprived patients are described elsewhere (Lewis, Maurer & Brent, 1995).

**Visually normal controls**

Results of patients were compared to those of visually normal control subjects in four age groups (n = 20 per group) tested under the same conditions (Hadad, Maurer & Lewis, 2011): 6- to 8-year-olds (mean age = 7.10; range = 6.0–8.9 years; 9 females), 9- to 11-year-olds

### Table 1 Clinical details of patients (in order of age)

<table>
<thead>
<tr>
<th>Patient/Age (years)</th>
<th>Refraction*</th>
<th>Diagnosis (days)</th>
<th>Snellen acuity*</th>
<th>Eye Tested</th>
<th>Nystagmus</th>
<th>Additional details</th>
<th>Biological Motion d’/staircase (% signal)</th>
<th>Global Motion 4/18° s⁻¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>JO (8.5) OD +29.00</td>
<td>OS +29.00</td>
<td>0 17 20/63 OS</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>2.39 88.51</td>
<td>31.75 100</td>
</tr>
<tr>
<td>DO (10.11) OD +13.00</td>
<td>OS +17.00</td>
<td>49 61 20/200 OS</td>
<td>Fine horizontal</td>
<td>Strabismus surgery</td>
<td>OU at 7 years</td>
<td>106.25 36.05</td>
<td>3.3 41.87</td>
<td>87.25 25.46</td>
</tr>
<tr>
<td>BL (10.11) OS +38.00</td>
<td>OS +11.00</td>
<td>2 11 20/32 OD</td>
<td>–</td>
<td>–</td>
<td>Minimal</td>
<td>microphthalmia OU</td>
<td>4.9 81.28</td>
<td>56.75 38.99</td>
</tr>
<tr>
<td>JB (13.30) OD</td>
<td>OS +38.00</td>
<td>2 11 20/64</td>
<td>Pendular OU</td>
<td>Strabismus surgery</td>
<td>OU at 5 years</td>
<td>73.25 42.55</td>
<td>4.9 55.08</td>
<td>79 79.98</td>
</tr>
<tr>
<td>RA (15.10) OD</td>
<td>OS +25.0</td>
<td>45 102 20/40 OD</td>
<td>Fine Vertical</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>4.64 100</td>
<td>73.25 42.55</td>
</tr>
<tr>
<td>CB (24.30) OD +7.50</td>
<td>OS +5.50</td>
<td>30 91 20/25 OD</td>
<td>Pendular OU</td>
<td>LET &amp; RET surgery at 1 year, 6 months</td>
<td>4.9 55.08</td>
<td>79 79.98</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IW (27.50) OD +14.5</td>
<td>OS +16.5</td>
<td>92 181 20/160 OS</td>
<td>Horizontal constant</td>
<td>OS Strabismus surgery at 3 years, 3 months; Strabismus surgery at 5 years, 10 months LET OU; Strabismus surgery OD at 10 years, 1 month</td>
<td>4.32 44.15</td>
<td>75.25 18.45</td>
<td></td>
<td></td>
</tr>
<tr>
<td>AC (28.70) OD +11.00</td>
<td>OS +16.50</td>
<td>123 196 20/50 OD</td>
<td>Pendular horizontal manifest nystagmus OU with latent component</td>
<td>Secondary membrane removal OS at 1 year; Strabismus surgery bimedial recession at 3 years, 7 months; conjunctival cyst OS removed at 4 years, 4 months</td>
<td>4.64 59.17</td>
<td>44 39.9</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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(mean age = 10.00; range = 9.0–11.9 years; 10 females), 12- to 14-year-olds (mean age = 12.74; range = 12.0–14.9 years; 9 females), and adults (mean age = 20.02; range = 18.0–26.2 years, 11 females). All controls reported that they had no history of eye problems and all met our criteria on a visual-screening exam. Detailed criteria for visual screening for this group of controls are described elsewhere (Hadad et al., 2011).

**General procedure**

The experimental protocol was approved by the Research Ethics Board of McMaster University and of The Hospital for Sick Children, Toronto. Before testing, the procedures were explained, and written consent was obtained from the parents of the children and from the adults who participated. Each participant was tested with three tasks on the same day: biological motion, global motion with the dots moving at 4° s⁻¹, and global motion with the dots moving at 18° s⁻¹. All testing was monocular, from a viewing distance of 50 cm. Patients were tested with the better eye, based on acuity at the time of the test or, when acuity was equal, with the better eye based on medical history (see Table 1). When necessary, patients wore an additional optical correction over the tested eye to focus it at the testing distance. Half in each of the control groups were tested with the left eye, whilst the remaining half were tested with the right eye. The eye not being tested was patched with 3 M Micropore™ tape.

Half the participants in each group were tested first with the biological motion task, and half were tested first with the global motion tasks. For the biological motion tasks, half of the participants were tested first with dots moving at 4° s⁻¹, and half were tested first with dots moving at 18° s⁻¹. Before each task (biological motion, global motion at 4°, and global motion at 18° s⁻¹), participants were given demonstration and criterion trials, with experimenter feedback, until the participant was correct on four consecutive trials, a criterion usually met within the first four practice trials. Participants were then given two test runs for each task without feedback except for periodic praise by the experimenter, who sat to the side of the monitor and controlled presentation of displays, but could not see the displays themselves. The experimenter watched the participant’s viewing eye to ensure that he/she was looking at the centre of the screen and reminded him/her to do so as necessary. Participants were given a short break between the two test runs and the results are based on the mean. Completion of the biological motion task and the two global motion tasks took about half an hour.

**Apparatus and stimuli**

**Biological motion**

A detailed description of the stimuli and procedure has been reported elsewhere (Freire, Lewis, Maurer & Blake, 2006; Grossman & Blake, 1999). Briefly, video recordings were made of an adult engaged in a variety of familiar activities, including running, kicking, climbing, throwing, and jumping. The phase-scrambled animations consisted of the same number of individual dots undergoing the same local motions as in the normal animations from which they were derived, but with their temporal phases scrambled. This form of scrambling perturbs the hierarchical, pendular motions characteristic of biological motion while preserving local motion trajectories. When produced in this way, the scrambled and biological patterns cannot be distinguished by local motion cues. Noise dots in the biological motion task moved at the same speed as the signal dots. The moving trajectories of these noise points were generated in the same way as the trajectories for the scrambled animations.

Stimuli were presented on a Macintosh G3 computer and a monochrome monitor, 29° high by 37° wide, with a refresh rate of 75 Hz with MatLab® and the Psycho-physics Toolbox software (Brainard, 1997). A total of 48 animations, 24 depicting normal biological motion and 24 depicting phase-scrambled sequences, were used in the yes/no and staircase tasks described below. In all animations, dots appeared black against a light gray background (60 cd/m²). Individual dots subtended approximately 10 arc min at the viewing distance of 50 cm, and the biological motion figures subtended approximately 6° × 3° of visual angle. The duration of each animation was 1 s and average speed within a sequence was about 4° s⁻¹. On each trial, the spatial location of the biological motion or scrambled stimulus was displaced in a random direction and by a variable distance within the 19.2° × 14.4° display window; for the displays masked with noise dots, this spatial jittering prevented participants from making judgments based on just a few tell-tale dots located at a given location in the display window.

Participants completed two tasks: a yes/no task and a two-interval staircase task. For the yes/no task, participants were shown 25 biological motion and 25 scrambled stimuli. Following each 1-sec-long animation, participants judged whether or not they saw a person. For the two-interval staircase task, a trial consisted of two successive point-light animations, one a biological motion stimulus, and the other a scrambled version of the same stimulus. Participants judged whether the person appeared in the first or second 1-sec interval.

The first two trials in the staircase included no noise dots. Six noise dots were added to each display after two consecutive correct responses, and six were subtracted after one incorrect response. The staircase terminated after 20 reversals. Subsequent to the first 12 reversals, the number of noise dots added or removed was lowered to three, in order to obtain a more precise estimate of the participant’s threshold. Threshold was defined as the mean number of noise dots in the final six reversals and represents an estimate of the noise level producing a
percent correct value of 71% (Grossman, Blake & Kim, 2004).

Global motion

Stimuli were generated by an Apple Macintosh G3 computer by means of VPixx software™ and were displayed on a monochrome monitor, 29° high by 37° wide, with a refresh rate of 75 Hz. Stimuli consisted of limited lifetime random-dot kinematogram displays (RDKs) similar to those described by Newsome and Paré (1988). Each frame contained 300 dots, giving a density of 0.75 dots/deg². The black dots were presented against a 17.5° × 17.5° gray background square and were 0.5° × 0.5° when viewed from the testing distance of 50 cm. Each dot had a mean luminance of 14 cd/m² against a 17.5° square, with a Michelson contrast between the dots and their background was 78%. A subset of dots (signal), randomly chosen for a number of frames, was constrained to move in the same direction at a specified speed. The other (noise) dots in the display moved at the same speed but in random directions, covering the entire 360° range. Signal strength was manipulated by varying the proportion of signal dots. Coherence thresholds, defined as the minimum percentage of signal dots required to accurately determine the overall direction of motion, were measured for dots that moved at speeds of 4° and 18° s⁻¹. The duration of each trial was 2 seconds.

To ensure that the overall direction of motion could not be determined by local motion detectors, the dots had a limited lifetime of 200 msec (15 frames) or 400 msec (30 frames) for the slower and faster speeds, respectively. When expired, each dot reappeared at a random position within the display area for a subsequent lifetime. The phase of each dot’s lifetime cycles was shifted randomly to prevent all dots from being reborn at the same time. Signal dots moved coherently in a common direction across lifetimes, giving the impression of a global motion, while the noise dots had a random initial direction of displacements every subsequent lifetime. The direction of the global pattern could thus be determined only by integrating the local signals over a larger summation field and not by following a single dot (e.g. Bex & Dakin, 2003).

Participants were instructed to fixate a cross at the centre of the screen followed by the RDK, and were asked to judge whether the global motion of the dots was upward or downward. The percentage of signal dots was varied over trials using the VPixx VPEST adaptive staircase that is similar to Harvey’s (1986) ML-TEST. Thresholds were defined as the minimum percentage of dots that had to be moving in the same direction for the subject to detect the overall direction of motion with 82% accuracy. The staircase terminated when the 95% confidence interval of the estimated threshold was within ±0.1 log units.

Results

Patients’ thresholds for each of the tasks were converted into z-scores based on the mean and standard deviation of the age-matched controls (mean and standard deviation for controls are summarized in Table 2; for the full set of data for the control groups see Hadad et al., 2011). Negative z-scores indicate a deficit compared to normal and positive z-scores reflect above-average performance. Raw thresholds for each patient for each of the tasks are shown in Table 1. The number of trials completed in each of the tasks was not significantly different between the patients and the control group (t(20) = 1.07, p > .29; t(20) = 0.83, p > .42, for 4 and 18 deg/sec, respectively; t(20) = 1.99, p > .06; t(20) = 1.03, p > .32, for yes/no and threshold biological motion tasks, respectively).

Biology motion

Yes/no task

We calculated d’ for each participant as the differences between the z-scores of hits (responding ‘biological’ when a sequence was biological) and those of false alarms (responding ‘biological’ when a sequence was phase-scrambled). d’ scores expressed in z-scores are shown in Figure 1a. The patients performed normally at discriminating biological from scrambled motion when no noise was presented. T-tests confirmed that the overall mean of z-scores (mean z = –0.07; range = 1.62 to –2.5) of the patients was not significantly different from zero, t(7) = 0.16, p > .80.

Staircase task

Z-scores of the individual thresholds for the eight patients are shown in Figure 1b. The patients performed normally at detecting the biological motion, tolerating just as many noise dots as their age-matched controls. T-tests confirmed that the overall mean z-score (mean z = –0.15; range = 2.14 to –2.3) of the patients was not significantly different from zero, t(7) = 0.27, p > .70.

Global motion

Z-scores of the motion coherence thresholds for each of the speed conditions are shown in Figure 2a. For clarity, the z-scores of the individual thresholds for the eight patients are shown in Figure 1b. The patients performed normally at detecting the biological motion, tolerating just as many noise dots as their age-matched controls. T-tests confirmed that the overall mean z-score (mean z = –0.15; range = 2.14 to –2.3) of the patients was not significantly different from zero, t(7) = 0.27, p > .70.

Table 2 Mean thresholds for controls in each of the tasks

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Global motion (% signal)</th>
<th>Biological motion</th>
</tr>
</thead>
<tbody>
<tr>
<td>6–8.9 yrs</td>
<td>62.62 44.05 3.41 48.53</td>
<td>47.53 37.51 3.95 60.03</td>
</tr>
<tr>
<td>9–11.9 yrs</td>
<td>47.53 37.51 3.95 60.03</td>
<td>18.53 10.51 4.3 69.75</td>
</tr>
<tr>
<td>12–14.9 yrs</td>
<td>27.34 24.95 4.3 69.75</td>
<td>19.41 14.69 4.3 80.57</td>
</tr>
<tr>
<td>18–26 yrs</td>
<td>47.53 37.51 3.95 60.03</td>
<td>18.53 10.51 4.3 69.75</td>
</tr>
</tbody>
</table>
z-scores were multiplied by minus 1 so that negative z-scores indicate a deficit as in the graphs for biological motion. In clear contrast to the biological motion task, most patients performed abnormally both with dots moving at 4° s⁻¹ and with dots moving at 18° s⁻¹. T-tests revealed a large deficit in processing the slower (mean z-score = −3.85), t(7) = 4.75, p < .002, and the faster speed (mean z-score = −4.21), t(7) = 2.99, p < .02. The deficits did not differ across the two speeds, t(7) = 1.14, p > .20, although, as for their age-matched controls, most patients had better coherence thresholds for the faster than the slower speed. Comparing Figure 2a to Figure 1b also indicates that, for each patient, z-scores for global motion were always worse than those for biological motion tested with a staircase procedure.

As shown in Figure 2b, there was no significant correlation between the deficits in global motion and visual acuity in the same eye (r (8) = −0.33, p > .40, and r (8) = −0.01, p > .90, for slow and fast speeds, respectively), suggesting that these deficits likely reflect damage to higher levels of the visual pathway, rather than being merely the result of poor visual input from neurons in the damaged primary visual cortex. There was also no correlation between duration of deprivation and the extent of the deficits in global motion processing (r (8) = −0.04, p > .90, and r (8) = 0.10, p > .80 for slow and fast speeds, respectively; see Figure 2a).

Discussion

The results show that early visual deprivation leads to deficits in integrating local information into global percepts of motion direction for both relatively slow (4° s⁻¹) and fast (18° s⁻¹) speeds but, nevertheless, spares the perception of biological motion. Although both global...
and biological motion have a protracted period of development, lasting until 12 to 14 years of age (Hadad et al., 2011), the normal development of global motion appears to depend on normal early visual input while the normal development of biological motion does not. Thus, damage from abnormal visual input cannot be predicted from the rate of normal development.

The deficit in global motion is unlikely to arise from a general difficulty in segregating signal from noise (e.g. Thompson, Troje, Hansen & Hess, 2008) because the patients tolerated a normal amount of noise in the biological motion task. Their elevated coherence thresholds must be caused at least in part by a compromised global motion system. This deficit is an example of a sleeper effect (Maurer et al., 2007): visual deprivation in the first few months of life prevents the development of sensitivity to global motion, a sensitivity that develops postnataally (e.g. Wattam-Bell, 1992) and that normally is not adult-like until much later in childhood (Hadad et al., 2011). This sleeper effect indicates that patterned visual input immediately after birth plays a critical role in the construction and/or preservation of the neural architecture that will later mediate sensitivity to global motion. Our results suggest that deprivation for as little as the first 2–3 months of life is sufficient to compromise the necessary architecture, with no additional detriment for deprivation lasting longer during the first year of life. It is possible, of course, that periods of deprivation shorter or longer than those we studied might lead to smaller or larger deficits in global motion processing than those we observed.

The sparing of biological motion is surprising given the impairments in this cohort in sensitivity to global motion, which implicates the dorsal visual pathway, and the documented impairments in similar cohorts in sensitivity to global form (Lewis et al., 2002) and holistic face processing (Le Grand et al., 2004; Robbins et al., 2010), both of which implicate the ventral pathway. Biological motion requires a similar type of spatial and temporal integration and implicates a network involving pSTS, an area that receives input from both the dorsal and ventral pathways (e.g. Puce & Perrett, 2003). A similar sparing of biological motion demonstrated in a patient with bilateral lesions in the posterior visual pathways (Vaina et al., 1990), suggests that global motion analyses might not be a necessary prerequisite for intact biological motion perception with the latter being mediated by an additional spared mechanism. It is not yet clear, however, whether this additional mechanism involves an interaction between temporal and spatial integration, or may be attributed specifically to the biological aspects of motion. This could be examined further by testing performance of these patients on other tasks involving interaction between temporal and spatial integration when no biological aspects are involved (e.g. structure-from-motion). Intact performance in a structure-from-motion task along with intact biological motion has been demonstrated in Vaina et al.’s (1990) patient with impaired motion mechanisms, suggesting that, at least in his case, sparing of biological motion is not specifically attributed to the biological aspects of motion. However, unlike in our patients, bilateral lesions have been documented in this patient involving his posterior visual pathways, affecting the lateral parietal-temporal-occipital cortex and the underlying white matter.

How, then, does one account for the finding that the processing of biological motion appears robust in the face of visual deprivation in early infancy and consequent deficits in other aspects of global integration? One possibility is that infants’ actions are sufficient to stimulate neurons involved in the perception of biological motion and hence to offset any adverse effect of visual deprivation. Single-cell recordings have identified a population of ‘mirror neurons’ in the macaque frontal cortex, in area F5, that fire during both the production and observation of a given action and its observation. In humans, there is overlapping cortical activation when a motor action is observed with the goal of imitation, and when the same action is executed (Iacoboni, Woods, Brass, Bekkering, Mazziotta & Rizzolatti, 1999). As in monkeys (Ferrari, Gallesse, Rizzolatti & Fogassi, 2003), no such activation occurs for comparable non-biological motions (Tai, Scherler, Brooks, Sawamoto & Castiello, 2004). Furthermore, the ventral premotor cortex that is thought to be part of this mirror neuron system (e.g. Binkofski & Buccino, 2006) has been shown to respond selectively to point light biological motion (Saygin, 2007). If a homologue of the macaque mirror system exists in human infants, it may play an especially important role in the development of sensitivity to biological motion when early visual input is missing, as in our patients, all of whom are able-bodied. In the absence of patterned visual input at birth, establishment of the common coding system may be driven initially by the intact motor system. Consistent with this possibility, the sound of an action engages human mirror system brain areas even in the congenitally blind (Ricciardi, Bonino, Sani, Vecchi, Guazzelli, Haxby, Fadiga & Pietrini, 2009).

Another possibility is that alternative networks are recruited that may be functioning in early infancy,
resilient to early visual deprivation and capable of mediating normal sensitivity to biological motion but not global form, global motion, or holistic face processing. In addition to pSTS, areas implicated in the processing of biological motion include the medial and lateral cerebellum, intraparietal cortex, middle temporal gyrus, posterior inferior frontal gyrus, premotor cortex, kinetic-occipital area (KO), fusiform face area (FFA), amygdala, and the ventral portion of V3 (e.g. Bonda, Petrides, Ostry & Evans, 1996; Grèzes, Fonlupt, Bertenthal, Delon-Martin, Segebarth & Decety, 2001; Saygin, 2007; Saygin, Bentin, Harel, Rees & Gilaie-Dotan, 2010). One contributing factor to the sparing of processing of biological motion may simply be that the vast number of structures implicated compensate for any deficits arising from compromised development within the geniculostriate pathway and the higher areas it feeds, including pSTS. Some of these alternative pathways may also mediate the sensitivity to biological motion that has been demonstrated in newborn babies (Simion, Regolin & Bulf, 2008) at a time when the primary visual cortex appears to be too immature to mediate most other forms of visual integration (e.g. Burkhalter, 1993). The possibility of such reorganization after early visual deprivation is strengthened by recent findings showing that even during normal development, there is considerable reorganization between 5 months and adulthood in the pathways mediating global form and global motion (Wattam-Bell, Birtles, Nyström, von Hofsten, Rosander, Anker, Atkinson & Braddick, 2010).

In summary, we showed different resilience to early visual deprivation of two visual abilities involving extrastriate processing and the integration of local motions across space and time. This resilience of biological motion to visual deprivation might be explained by the unusually wide distribution of the network for detecting biological motion and/or by a neural network that is stimulated by body movements. In any case, the present results suggest that biological motion is a special case of motion integration, perhaps because of its contribution to the identification of living creatures, an ability crucial for survival and social interaction.

References


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