COMMENTARY

Neural tuning of human face processing

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Within the first 6 mo of life, our vision improves many times over and continues to improve during childhood (1). During these early years, we encounter multiple and varied sensory experiences, whose overall patterns can differ considerably between individuals. For example, some of us grow up among a large social group with many races and languages, whereas others grow within a smaller, more uniform cohort. Nevertheless, despite the multitude of environments in which we are raised, there is a surprising convergence in our behavioral capacities. One such capacity is recognizing individual faces. Like other visual skills, face recognition requires experience. Although we have a reasonable idea of when our perception of faces gets refined behaviorally and even some of its neural correlates (2, 3), we don't really know the mechanisms by which experience tunes our face-processing circuits. Part of the problem, of course, is that one cannot do perturbation experiments in humans as we would with an animal model system (4). A new electrophysiology study by Röder et al. (5) capitalizes on a naturally occurring experiment: a unique clinical population of individuals born blind because of cataracts, who then later have their sight restored following cataract removal (5). The study revealed that the neural signature for face processing is present in these individuals but not sufficiently tuned by experience.

Two clinical populations were tested through the Prasad Eye Institute in India. In one group were individuals born with congenital cataracts, a condition where the lenses of the eyes are opaque at birth. It is one of the most common forms of childhood blindness (estimates range from 5 to 20% of births, worldwide), but also one of the most treatable. Before treatment, these individuals undergo a period of visual deprivation and upon restoration of sight, the perceptual consequences of this deprivation become apparent. Remarkably, despite the months or years of living with blindness, these individuals go on to recognize faces and facial expressions (6–10). However, they are unable to discriminate between individual human faces that differ based on the configuration of facial features, such as the distance between eyes (11). Typically developing humans are able to do this almost reflexively. This deficit is likely a result of missing critical visual input during a sensitive period when typical experience with faces in one's environment tunes (or "narrows") perceptual sensitivity to match one's everyday social world (e.g., one's own species, one's own race) (12). As an elegant part of the study's design, Röder et al. (5) included a second clinical population comprised of individuals born with normal vision but whom, over time, developed cataracts in one or both eyes during childhood and later received the same corrective surgery as the congenital cataract group. Thus, these individuals served as control participants who had early visual experience but who were then severely visually impaired for a time (though not necessarily blind).

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A Neurophysiological Marker for Face Processing

To investigate neural processing, Röder et al. (5) used electroencephalography to detect traces of neural electrical activity along the scalp and to measure their responses to sensory events (known as event-related potentials, or ERPs). These are neural responses whose specific pattern depends on the type of sensory input. For faces, ERPs reliably exhibit a robust negative peak in the signal at ∼170 ms after stimulus presentation. Known as the "N170," this peak is absent or reduced in amplitude when other categories of visual stimuli are presented (13). The key question is this: Is this face-sensitive N170 response present in those individuals deprived of early visual experience versus those who had experience? The two clinical groups, as well as age-matched control groups, viewed black-and-white images of faces, houses, and image-scrambled versions of each. Interspersed throughout the sequence of images were target images of butterflies to which the participants were instructed to respond by lifting their right hand. This simple task served to keep overall attention levels high. Surprisingly, all groups showed a robust N170 response to faces but the N170 was nonspecific for those individuals who previously had congenital cataracts (and thus lacked early visual experience). In these participants, robust N170 responses were elicited by houses, scrambled houses, and scrambled faces, and the responses were indistinguishable from those elicited by faces. Importantly, there was no correlation between the lack of N170 specificity and the age at which participants underwent corrective surgery or with their subsequent visual acuity. Patients who previously had congenital cataracts could, for example, discriminate faces, houses, and butterflies just as well as all of the other participants in the study. Thus, although the N170 response is present in all participants, only those lacking early visual experience have N170 neural responses that are "untuned" (5).

Mechanisms: Tuning or Pruning?

The developmental mechanisms that underlie the tuning of the N170 ERP response are unknown. To explain both the acquisition and decline of certain perceptual and motor skills, developmental psychologists are quick to invoke "neural pruning," whereby exuberant, somewhat nonspecific synaptic connections are lost over the course of neural development in an adaptive manner. For example, a recent functional MRI study reported that visual representations of faces are "pruned back" during early childhood (14). Such purported explanations fail to account for the fact that the brain does not shrink during postnatal development but grows in size, and that the loss of synaptic connections is only one phenomenon in the context of several other mechanisms of

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selective synaptic growth and elaboration (15, 16). In the present context, the lack of N170 tuning in congenital cataract participants would typically evoke a "pruning" explanation: a failure to eliminate incorrect synapses. Eschewing such simplistic explanations, Röder et al. (5) suggest that the lack of tuning is because of the failure of inhibitory processes to be shaped by experience. This idea is consistent with the well-established mechanisms that underlie critical period visual plasticity and tuning in animal models (17). For example, in mice early visual experience modulates the expression of growth factors, which in turn influence the growth of inhibitory connections (18); these connections shape the specificity of neural responses. This is a reasonable framework within which to interpret what we now know is the experience-dependent tuning of the face-specific N170 response in humans. It is also worth noting that neural pruning implies the permanent shedding of neural connections, whereas tuning through modulation of inhibitory circuits could theoretically allow for recurrence and reversibility that is consistent with the overall restoration of sight and visual skills in cataract patients.

By the clever juxtaposition of two clinical populations with restored vision—those who had congenital cataracts versus those whose cataracts developed postnatally—Röder et al. (5) demonstrate the existence of a sensitive period for the tuning of a face-sensitive neural response in humans. The exact time at which visual experience is necessary for face-specificity remains unknown and could not be addressed in this study because of the varied timing of sight restoration (and thus the amount of visual experience) of each clinical participant. The constructive mechanism by which face-sensitive neural responses are tuned may be similar to that seen in animal models, but it is likely to occur on a different time scale relative to the development of other visual processes (19). Regardless of these unknowns, the ability of these formerly blind individuals to use visual information to navigate their social world to any extent at all speaks volumes about the resiliency of the human brain.

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