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The cognitive and neural basis of developmental prosopagnosia

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ABSTRACT

Developmental prosopagnosia (DP) is a severe impairment of visual face recognition in the absence of any apparent brain damage. The factors responsible for DP have not yet been fully identified. This article provides a selective review of recent studies investigating cognitive and neural processes that may contribute to the face recognition deficits in DP, focusing primarily on event-related brain potential (ERP) measures of face perception and recognition. Studies that measured the face-sensitive N170 component as a marker of perceptual face processing have shown that the perceptual discrimination between faces and non-face objects is intact in DP. Other N170 studies suggest that faces are not represented in the typical fashion in DP. Individuals with DP appear to have specific difficulties in processing spatial and contrast deviations from canonical upright visual-perceptual face templates. The rapid detection of emotional facial expressions appears to be unaffected in DP. ERP studies of the activation of visual memory for individual faces and of the explicit identification of particular individuals have revealed differences between DPs and controls in the timing of these processes and in the links between visual face memory and explicit face recognition. These observations suggest that the speed and efficiency of information propagation through the cortical face network is altered in DP. The nature of the perceptual impairments in DP suggests that atypical visual experience with the eye region of faces over development may be an important contributing factor to DP.

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Prosopagnosia is a severe deficit of visual face recognition in the absence of low-level visual problems and intellectual disability. Individuals who have this condition may also experience difficulties in interpreting other aspects of faces, such as expressions of emotion, or may even show more general deficits in object recognition, but the core impairment in prosopagnosia concerns the recognition of individual faces. Traditionally, prosopagnosia was regarded as a relatively rare consequence of acquired head injury or stroke (Bodamer, 1947). However, another type of prosopagnosia of a developmental origin that emerges over the lifespan, and in the absence of any apparent brain damage, has recently been found to be much more common than prosopagnosia resulting from brain damage (acquired prosopagnosia; AP). These individuals with developmental prosopagnosia (DP)

typically report first experiencing face recognition problems in childhood or adolescence (for recent reviews, see: Susilo & Duchaine, 2013; Towler & Eimer, 2012). Individuals with DP often become most acutely aware of their face recognition difficulties because of the increased social demands during adulthood. Due to the increasing public awareness of DP, they are now able to get in contact with psychologists and neuroscientists who are investigating the condition.

There are currently no officially established diagnostic criteria for DP. However, particular standards are generally present in the research literature. DPs must experience face recognition difficulties on a regular basis in typical situations where recognition is unproblematic for individuals without face processing impairments. Face memory in DP is typically measured using a combination of tests involving

unfamiliar face learning and long-term memory for familiar faces. Typical diagnostic criteria are that DPs must score below two standard deviations from the mean on a number of different face recognition tests. The majority of published research on DP has employed the Cambridge Face Memory Test (CFMT; Duchaine & Nakayama, 2006) as an important diagnostic tool. This test involves learning six unfamiliar male faces from different images that show the same individual face from different viewpoints, under different lighting conditions, and with varying degrees of visual noise. DPs can have trouble with remembering identical images of unfamiliar faces over short durations and with interleaved items; however, impairments are generally more marked when identification involves changes in the image between sample and test. DP, as assessed by the CFMT, can therefore be described as an impairment in learning new face identities across transformations in the retinal image from which they were originally encountered. Because face learning and face recognition both depend on the ability to perceptually process face images, performance deficits measured with the CFMT could also reflect perceptual face processing impairments. Some DPs do indeed perform poorly in tests of face perception such as simultaneous face matching that are less dependent on memory, demonstrating that problems in perceptual aspects of face processing can contribute to DP. However, other DPs show no such impairments in face perception tests (e.g., Garrido, Duchaine, & Nakayama, 2008).

Estimates of the prevalence of developmental prosopagnosia (DP) range from 1.9% to 2.5% in different populations (Kennerknecht et al., 2006; Kennerknecht, Pluempke, & Welling, 2008), and both family and twin studies suggest strong genetic heritability for the capacity to recognize faces across the entire distribution of this ability (Duchaine, Germine, & Nakayama, 2007; Lee, Duchaine, Wilson, & Nakayama, 2010; Wilmer et al., 2010; Zhu et al., 2010). DP has only recently become the focus of systematic research, and the neurodevelopmental origins of DP are currently unknown. Investigations into the cognitive and neural mechanisms responsible for face recognition impairments in DP have benefited from the extensive psychological and neuroscientific work that has been conducted on face processing in neurotypical individuals without face processing impairments. A general picture is beginning to emerge about the cognitive and neural mechanisms that are

responsible for the persistent face recognition difficulties in DP.

In the current article, we present a selective review of cognitive and neuroscientific research on face processing in individuals with unimpaired face recognition abilities and in individuals with DP. This review is selective because it focuses on recent studies from our own lab that have employed event-related brain potential (ERP) markers of different stages of face processing to investigate specific impairments of face perception and recognition in DP. The results from these studies are discussed in the context of other research on the cognitive and neural basis of DP, and are also linked to general assumptions about the architecture of the face processing system in the human brain. We start with a brief overview of the cognitive and neural architecture of the face processing system in individuals without face processing impairments, which is important to understand the nature of the face processing deficits in DP. In the subsequent sections, we review ERP studies of perceptual stages of face processing in DPs and unimpaired control participants. These studies have used the face-sensitive N170 component to study the neural processes involved in the detection of faces versus non-face objects, as well as different aspects of configural face processing, and the importance of the eye region in early perceptual face representations. Results show that although the basic ability to detect face images and discriminate faces from other types of objects appears intact in DP, there are systematic differences between DPs and control participants in the perceptual configural analysis of faces and the weight given to perceptual representation of the eye region. Next, we provide evidence that the rapid detection of emotional facial expression appears to be unimpaired in DP, which illustrates that there are important dissociations between aspects of face perception that are intact and others that are selectively impaired in DP. In the subsequent section, we review recent ERP evidence that compared processes that are directly involved in face recognition (such as the activation of visual face memories and the explicit recognition of particular faces) between DPs and control participants. In the final section, the main findings from these ERP studies and their implications are summarized and considered within a more general conceptual framework about the nature and causes of neurodevelopmental disorders.

The neural architecture of the face processing system

Functional neuroimaging and direct neuronal recordings in humans and non-human primates have uncovered a network of spatially clustered face-selective patches of cortex in the occipital and temporal lobes (e.g., Haxby, Hoffman, & Gobbini, 2000, 2002; Kanwisher, McDermott, & Chun, 1997; Moeller, Freiwald, & Tsao, 2008; Tsao & Livingstone, 2008; Tsao, Schweers, Moeller, & Freiwald, 2008). In particular, neuroimaging studies in humans have identified a number of brain regions that are more strongly responsive to faces than to other object categories. The inferior occipital gyrus ("occipital face area"—OFA), the middle lateral fusiform gyrus ("fusiform face area"—FFA), and the posterior part of the superior temporal sulcus (STS) are key regions involved in the visual processing of faces (e.g., Haxby et al., 2000, 2002; Kanwisher et al., 1997). These brain areas can be reliably located in most neurotypical individuals, and the coordinated operation of these cortical regions is thought to underlie our ability to perceive and recognize individual faces. For example, the classical lesion sites for patients who present with AP are in the occipital and temporal lobes including core posterior face processing regions such as the OFA and FFA (e.g., Barton, 2008; Kanwisher & Barton, 2011; Meadows, 1974). Additional evidence for the causal role of these core face-selective regions in face processing comes from studies employing brain stimulation. Transcranial magnetic stimulation (TMS) is one such technique that has been successfully applied to alter performance in face processing tasks when face-selective regions are stimulated. Stimulation of the OFA by TMS has been shown to cause impairments in face identity and expression matching performance (Pitcher, Garrido, Walsh, & Duchaine, 2008; Pitcher, Walsh, Yovel, & Duchaine, 2007). Perhaps the most compelling evidence for the causal role of the core face network in face processing comes from direct neuronal stimulation of these brain regions in pre-surgical epileptic patients. When electrical current is directly applied to neurons in the OFA of these patients, they experience temporary prosopagnosia while trying to recognize famous faces, while no such effect is present at other electrode sites (Jonas et al., 2012). When the FFA is electrically stimulated, patients experience striking perceptual distortions of facial structure, with facial features appearing to move within the face and face identity changing to that of other different

individuals (Parvizi et al., 2012; Rangarajan et al., 2014). No such distortions are reported for non-face objects. Together, these lines of evidence firmly establish causal links between the core posterior face processing regions and face perception and recognition.

Outside of the core posterior face-selective areas described by Haxby et al. (2000), additional face-selective brain regions have been identified. One of these is located anterior to the FFA in inferior temporal cortex (Kriegeskorte, Formisano, Sorger, & Goebel, 2007; Rajimehr, Young, & Tootell, 2009). The involvement of the temporal lobes in semantic memory is well documented (e.g., Martin & Chao, 2001), and there is some debate whether this region represents visual properties of faces, or more conceptual or abstract semantic information about known individuals. Although patients with brain lesions or atrophy to the anterior temporal lobe may have difficulties recognizing known individuals, this impairment may reflect a loss of semantic knowledge about people, rather than a primarily visual deficit (Ellis, Young, & Critchley, 1989; Gainotti, 2013; Gainotti, Barbier, & Marra, 2003). However, more recent evidence favours the view that the anterior temporal lobe is also linked to visual-perceptual stages of face processing (for a review, see Collins & Olson, 2014). Neurophysiological recordings in the macaque visual cortex have identified an anterior temporal area as the most face-selective region in the face network, which contains the most image-invariant representations of facial identity that are tolerant to rotations of the head (Meyers, Borzello, Freiwald, & Tsao, 2015). Neuroimaging studies in humans have shown that a homologous face-selective anterior temporal region represents individual face identities in an image-invariant fashion that is also largely tolerant to changes in head rotation (Anzellotti, Fairhall, & Caramazza, 2013). Evidence for a causal role of this region in perceptual face processing comes from the observation that intracranial electrical stimulation of a face-selective region in the right anterior temporal lobe caused transient prosopagnosia, similar to the effects of OFA stimulation previously discussed (Jonas et al., 2015). Along similar lines, two patients with focal lesions to the anterior temporal lobe showed visual-perceptual deficits both in face identity matching and in matching the configuration of random dot patterns (Olson, Ezzyat, Plotzker, & Chatterjee, 2015). A patient with a focal right anterior temporal lobe lesion showed remarkably specific impairments in holistic face perception across a number of perceptual tasks (Busigny et al., 2014).

A posterior-to-anterior gradient in the cortical basis of face processing impairments in developmental prosopagnosia

Because individuals with DP have problems in recognizing known individuals and often also in perceptually discriminating between different face identities, most neuroimaging studies of DP have focused on the presence or absence of face-selective activations in the core posterior face processing regions. Surprisingly, these functional magnetic resonance imaging (fMRI) experiments have generally observed relatively normal fMRI activation patterns within these regions for individuals with DP (Avidan & Behrmann, 2009; Avidan, Hasson, Malach, & Behrmann, 2005; Avidan et al., 2014; Furl, Garrido, Dolan, Driver, & Duchaine, 2011; Hasson, Avidan, Deouell, Bentin, & Malach, 2003). A study with a larger sample size found that the FFA was reduced in size and showed less face-selectivity in DPs than in a control group (Furl et al., 2011), although these regions were generally present in most DPs and showed normal sensitivity to face identity repetitions. Because a core deficit in DP is the inability to recognize known or learned faces across transformations in the retinal image, a likely candidate for a neural basis of DP is the face-selective anterior temporal region that represents individual faces in an image-invariant fashion (Anzellotti et al., 2013). A recent neuroimaging study (Avidan et al., 2014) has tested this hypothesis. As expected, face-selective responses were present in the entire face processing network in control participants without face processing difficulties, including the anterior temporal region. In a group of DPs, face-selective activation was also present in the core posterior face-selective regions, consistent with previous reports. However, and critically, such activations were entirely absent in the most anterior temporal face-selective region in DPs (Avidan et al., 2014). These findings cast doubt on the hypothesis that perceptual face processing impairments in DP are primarily associated with the posterior face processing network. They suggest that expert face processing requires the integrated functioning of the entire occipito-temporal face network rather than a single cortical site such as the FFA, and point to the anterior temporal face-selective region as a possibly critical locus of the perceptual face processing impairments in DP. However, because the absence of face-selective activation of this region in DP may also be linked to impairments at post-perceptual face processing

stages, more evidence that this region is directly associated with face perception deficits in DP is needed. More generally, there may be a gradient of increasing cortical dysfunction from posterior to more anterior regions of occipito-temporal cortex in DP.

Alongside reductions of functional brain activity in face-selective regions in DP, there may also be subtle alterations in structural neuroanatomy. In an early investigation, structural MRI suggested a smaller than average right temporal lobe in a single case of DP (Bentin, Deouell, & Soroker, 1999). More recently, Garrido et al. (2009) reported reduced grey matter volumes in middle and anterior temporal lobe regions such as the anterior inferior temporal lobe, superior temporal sulcus, and middle temporal gyrus, as well as the right middle fusiform gyrus and inferior temporal gyrus. Behrmann, Avidan, Gao, and Black (2007) found differences in the volume of anterior temporal lobe structures between individuals with DP and neurotypical controls, and a correlation between decreased grey matter volume in the anterior fusiform gyrus and face recognition deficits in DPs. These findings are consistent with a gradient of increasing cortical dysfunction from the posterior to anterior regions of the occipito-temporal pathway in DP. Whether and how specific reductions in the size of temporal lobe structures are related to the reductions of face-selective functional specialization in these regions remains to be determined. In addition to the size of particular cortical regions linked to face processing, white-matter fibres that connect different face processing regions may also be altered in DP. Thomas et al. (2009) reported selective reductions in the number of white-matter fibres in the inferior longitudinal fasciculus (which runs from the occipital to the anterior temporal lobe and connects ventral face-selective regions) in a group of DPs as compared to control participants without face processing impairments. There were additional reductions in the number of fibres in the fronto-occipital fasciculus (which connects the occipital and frontal lobes) in DPs as compared to controls, but no differences between groups in additional control white-matter tracts. A recent study with a larger sample size has shown that white-matter abnormalities in DP may be selectively restricted to areas that are close to face-selective regions such as the FFA (Song et al., 2015). Taken together, these studies suggest that the size of face processing regions in the temporal lobe and the connections between occipital and temporal,

and occipital and frontal brain regions may play a key role for normal face recognition and in the development of face recognition impairments.

Outside of the occipito-temporal face processing network, abnormalities in the neural activity in response to familiar as compared to unfamiliar faces in DPs have been reported in the left precuneus, posterior cingulate cortex, and anterior paracingulate cortex (Avidan & Behrmann, 2009). These regions form part of the extended face processing network for the recognition of familiar faces and are probably linked to post-perceptual face processing stages such as the retrieval of semantic or episodic memories about specific individuals (Gobbini & Haxby, 2007). Because these brain regions are associated with post-perceptual face recognition stages, we discuss them in the section “ERP evidence for delay and disconnection in the face recognition system in DP”. In summary, the emerging view from neuroimaging studies of DP is that the neural locus of face recognition difficulties is more pronounced at higher level cognitive stages of cortical face processing than at lower level perceptual stages. Deficits are most apparent in brain regions that process image-invariant representations of facial identity and in regions that are involved in post-perceptual face recognition processes, while earlier face-sensitive perceptual areas appear to operate largely normally in DP. While this may suggest that the visual-perceptual processing of faces is not selectively impaired in DP, behavioural tests often reveal severe face perception deficits in many individuals with DP (e.g., Duchaine, Yovel, & Nakayama 2007). How can the neuroimaging data be reconciled with the known behavioural impairments in face perception in DP? Studies employing neuroimaging techniques can inform about the presence or absence of face-selective brain regions in DP, but cannot provide crucial information about the timing of neural activity in the posterior face network. In contrast, event-related brain potential (ERP) measures allow more precise insights into the time course of face processing and into how specific stages of early face perception differ between DPs and individuals with unimpaired face recognition. In the subsequent sections of this article, recent results from ERP studies of DP are discussed.

The N170 component shows normal face sensitivity of visual processing in DP

Most ERP investigations of face processing have focused on the face-sensitive N170 component, and

this component has also featured prominently in DP research. In the following sections, we focus on studies investigating perceptual face processing impairments in DP by measuring N170 components. Research on other aspects of face processing in DP (the detection of emotional facial expression and the recognition of facial identity) has employed different ERP components, and is discussed later.

The N170 is an enhanced negativity to faces versus non-face objects that typically emerges between 140 and 200 ms after stimulus onset over lateral occipito-temporal areas (e.g., Bentin, Allison, Puce, Perez, & McCarthy, 1996; Eimer, 2000a; Eimer, Gosling, Nicholas, & Kiss, 2011; Eimer, Kiss, & Nicholas, 2010; Rossion & Jacques, 2011). Source localization studies (Bötzel, Schulze, & Stodieck, 1995; Itier & Taylor, 2004b; Rossion, Joyce, Cottrell, & Tarr, 2003; Watanabe, Kakigi, & Puce, 2003) have suggested that the N170 component is generated in structures such as the middle fusiform gyrus, inferior occipital gyrus, and superior temporal sulcus, which are part of the posterior core face-selective processing network (Haxby et al., 2000). Evidence from intracranial studies with pre-surgical patients indicates that face-sensitive N170-like potentials can be observed in lateral and ventral occipito-temporal cortex, including the OFA and FFA (Jonas et al., 2012; Parvizi et al., 2012). Studies of patients with AP have suggested that the integrity of posterior face processing regions, and in particular the fusiform gyrus, is essential to elicit a face-sensitive N170 response on the scalp (Dalrymple et al., 2011; Prieto, Caharel, Henson, & Rossion, 2011). At the cognitive level, the N170 component is believed to reflect early perceptual encoding stages of face processing (“structural encoding”; Bruce & Young, 1986). At the neural level, N170 components can be used to study the core posterior face processing system during face perception.

Several studies have addressed the question of whether the generic face-sensitivity of the N170 component (i.e., the enhancement of N170 amplitudes to faces versus non-face images) is preserved or abolished in DP. In experiments with small sample sizes, face-sensitive N170 components were present in some individuals with DP and absent in others (Bentin, DeGutis, D’Esposito, & Robertson, 2007; Bentin et al., 1999; Harris, Duchaine, & Nakayama, 2005; Kress & Daum, 2003; Minnebusch, Suchan, Ramon, & Daum, 2007; Németh, Zimmer, Schweinberger, Vakli, & Kovács, 2014; Righart & de Gelder, 2007; Rivolta, Palermo, Schmalzl, & Williams, 2012). These

findings underline the apparent heterogeneity of DP and suggest that face recognition deficits in DP may have different causes in different individuals. For DPs with normal N170 components, impairments may primarily be present at later memory-related processing stages. The reduction or absence of face-sensitive N170 components in other individuals suggests a reduced functional specialization for faces at early perceptual processing stages, which will have obvious knock-on effects on subsequent face recognition processes. A study from our lab (Towler, Gosling, Duchaine, & Eimer, 2012) tested a larger sample (16 DPs and 16 age-matched controls) and found enhanced N170 components to faces versus non-face objects (houses) in both DPs and controls, with no difference in the face-sensitivity of the N170 between these two groups (as shown in Figure 1, top panel). These observations suggest that the perceptual processes involved in the visual discrimination between faces and non-face objects generally operate normally in most individuals with DP. This is consistent with normal face-selective activations within the core face processing regions observed in fMRI studies of DP (as discussed earlier) and extends these observations by showing that such activations are elicited with their normal time course within 200 ms after encountering a face. This is also consistent with the fact that individuals with DP do not have problems distinguishing faces from other non-face objects, but find it difficult to discriminate between different individual faces.

The presence of similar face-sensitive N170 components in DPs and control participants does not necessarily imply that the posterior visual face processing network operates in an entirely typical fashion in DP. For example, the apparently normal N170 components to faces versus non-face objects found for participants with DP (Towler et al., 2012) may be driven by salient local features such as the eyes, which are known to trigger large N170 components in neurotypical individuals even when presented in isolation (e.g., Bentin et al., 1996). There is some evidence that perceptual deficits in DP may be primarily associated with the processing of global rather than local features (Avidan, Tanzer, & Behrmann, 2011), although another study (Duchaine, Yovel et al., 2007) did not find such a bias towards the visual processing of local features in DP. To investigate how DPs process global face configurations, we recently measured N170 components to two-tone Mooney faces versus Mooney houses in a group of control participants with normal face recognition abilities (Eimer et al.,

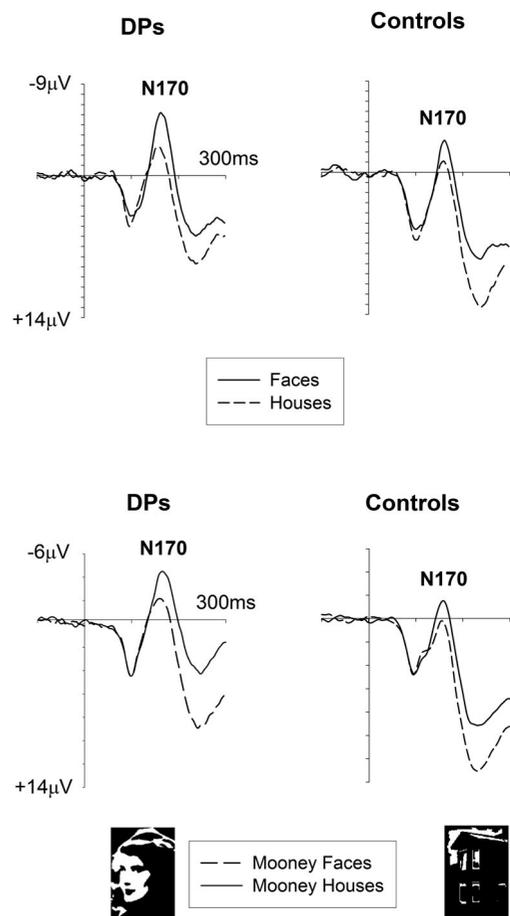


Figure 1. Top panel: Grand-averaged event-related brain potentials (ERPs) elicited by upright faces and houses at right occipito-temporal electrode P8 in the 300-ms interval after stimulus onset, in a group of 16 participants with developmental prosopagnosia (DPs; left) and 16 age-matched control participants. Enhanced N170 amplitudes to faces versus houses are triggered in a similar fashion in both groups. Data from Towler et al. (2012), reproduced in a different format. Bottom panel: Grand-averaged ERPs elicited by upright Mooney faces and Mooney houses at right occipito-temporal electrode P8 in the 300-ms interval after stimulus onset, in a group of 16 participants with DP (left) and a group of 12 participants with unimpaired face recognition abilities (right). Mooney faces triggered larger N170 components relative to Mooney houses in both groups. Data taken with permission from Towler, Gosling et al. (2016) and Eimer et al. (2011), reproduced in a different format.

2011) and in a group of participants with DP (Towler, Gosling, Duchaine, & Eimer, 2016). Both groups showed essentially the same pattern of face-sensitive N170 responses to Mooney faces, with larger N170 components to Mooney faces than to Mooney houses (as shown in Figure 1, bottom panel), in spite of the fact that the individual parts of these Mooney faces are recognizable only within the global context

of the whole face. This result demonstrates that individuals with DP are able to extract coarse spatially global information for categorical discriminations between faces and non-face objects, even in the absence of salient local facial features (for corresponding behavioural evidence for normal processing of Mooney faces in DP, see Le Grand et al., 2006). These N170 findings with Mooney faces suggest that DPs can form coarse low spatial frequency face representations to detect and categorize facial cues that are present in Mooney faces. They show that early stages of face perception in DP are not entirely based on local part-based processes, and strongly suggest that DPs have an internal global template of face structure. However, the question remains whether other aspects of this face template and its use during early stages of face processing in the posterior visual face network are also typical in DP.

The N170 component reveals atypical configural face processing in DP

The fMRI and ERP results discussed so far suggest that perceptual stages of face processing operate normally in DP. While this may be the case for particular aspects of face perception such as the discrimination of faces and non-face objects, there is behavioural evidence that other aspects that are more directly linked to the encoding of face identity might be selectively impaired in DP. Stimulus inversion makes face recognition more difficult (e.g., Yin, 1969), and this is usually interpreted as demonstrating the important role of configural face processing, as inverting faces disrupts their prototypical first-order configuration (e.g., eyes above nose, nose above mouth; Maurer, Le Grand, & Mondloch, 2002). Relative to unimpaired control participants, individuals with DP tend to have absent or reduced face inversion effects in tasks involving identity perception (e.g., Duchaine, 2011; Duchaine, Yovel et al., 2007). Performance differences between DPs and controls have also been observed in tasks of holistic face processing. Matching the identity of the top half of face pairs while ignoring their bottom halves is more difficult when the two face halves are spatially aligned than when they are misaligned, suggesting that aligned face halves are integrated into a single holistic face representation (Hole, 1994; Young, Hellawell, & Hay, 1987). For some individuals with DP, this composite face effect tends to be reduced (Avidan et al., 2011; Liu & Behrmann, 2014; Palermo et al., 2011; for an individual DP with

normal holistic face processing, see Susilo et al., 2010). Performance in part-whole face matching tasks (Tanaka & Farah, 1993) is typically better when task-relevant face parts are presented in the context of an intact upright face than when they are shown in isolation or among other scrambled facial features. Individuals with DP show such a whole-face benefit when asked to match mouths, but not when they are required to match the eye region (DeGutis, Cohan, Mercado, Wilmer, & Nakayama, 2012). Such behavioural findings suggest that perceptual mechanisms that are specifically tuned to analyse upright faces and their prototypical spatial configuration might be selectively impaired in DP.

Initial electrophysiological evidence for atypical configural face processing in DP was provided by other results of our ERP study that showed no difference in the face selectivity of the N170 component between DPs and controls (Towler et al., 2012). In this study, face images were presented either in their normal upright orientation or inverted, which allowed us to compare N170 components to upright and inverted faces. For participants with unimpaired face recognition, N170 components are highly sensitive to the orientation of faces, with larger N170 amplitudes and delayed N170 peak latencies for inverted than for upright faces (e.g., Eimer, 2000a; Rossion et al., 1999; Towler et al., 2012). These typical N170 face inversion effects were also observed for the control participants in our study (Towler et al., 2012; as illustrated in Figure 2, right panel). In contrast, the typical N170 amplitude enhancement to inverted faces was absent for participants with DP. There was even a non-significant trend towards larger N170 amplitudes for upright faces in the DP group (as shown in Figure 2, left panel). These observations suggest that posterior face processing areas are not selectively tuned to the canonical upright orientation of faces in DP. As a result, DPs may process upright and inverted faces in a similar fashion, perhaps because DPs are less efficient than unimpaired individuals in utilizing the prototypical spatial configuration specifically present in upright faces (for a more detailed discussion, see: Towler & Eimer, 2012; Towler et al., 2012).

The presence of atypical N170 face inversion effects suggests that DPs may be unable to encode recognizable facial features relative to a spatial template of the canonical locations of facial features within an upright face. However, face inversion alters not only the prototypical spatial relationships

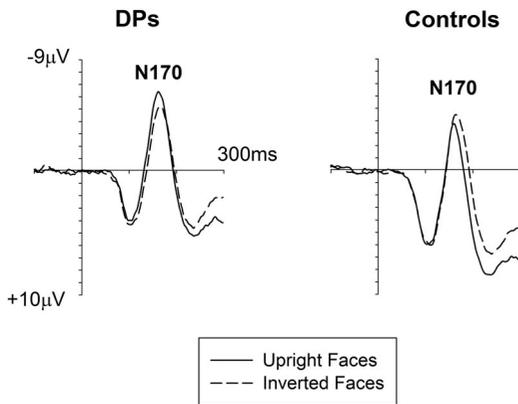


Figure 2. Grand-averaged event-related brain potentials (ERPs) elicited by upright faces and inverted faces at right occipito-temporal electrode P8 in the 300-ms interval after stimulus onset, for a group of 16 participants with developmental prosopagnosia (DPs; left panel) and a group of 16 age-matched controls (right panel). Control participants showed enhanced N170 amplitudes to inverted faces, whereas DPs did not show this typical N170 face inversion effect. Data taken with permission from Towler et al. (2012), reproduced in a different format.

between facial features, but also the orientation of these features themselves. Inversion-induced N170 enhancements could in principle reflect orientation-specific neural mechanisms that are tuned to individual face parts. For this reason, the atypical N170 face inversion effects in DPs (Towler et al., 2012) may not primarily be due to a reduced sensitivity to the prototypical spatial configuration of an upright face template in DP, but could instead be linked to differences processing of upright and inverted facial features between DPs and controls. If atypical N170 face inversion effects in DP are linked to a lack of sensitivity to the canonical positions of facial features within upright faces, other disruptions of this prototypical spatial configuration should also trigger an atypical pattern of N170 components in individuals with DP. We tested this prediction in a recent study (Towler, Parketny, & Eimer, 2016) where we measured N170 components in response to intact upright faces and to face images where the eyes, the nose, and the mouth were spatially scrambled but retained their individual upright orientations (as illustrated in Figure 3, top panel). Previous studies have shown that N170 components triggered by scrambled faces tend to be enhanced and delayed relative to the N170 in response to intact faces (e.g., Bentin et al., 1996; Zion-Golumbic & Bentin, 2007; for similar N170 modulations caused by other disruptions of the canonical facial configuration, see Jacques & Rossion, 2010; Letourneau & Mitchell, 2008). The fact that face

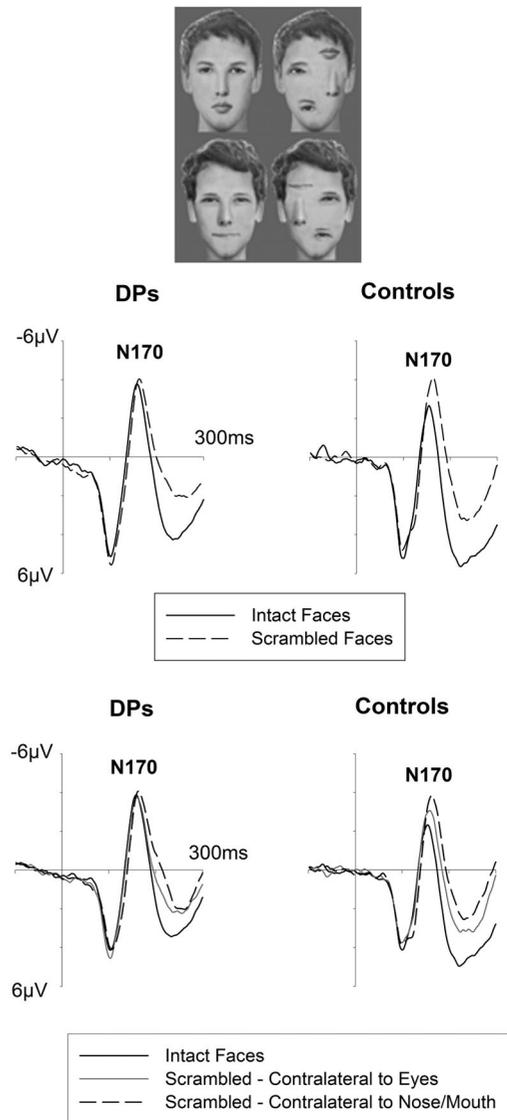


Figure 3. Top panel: Examples of intact and scrambled faces images used by Towler, Parketny et al. (2016). In scrambled faces, the two eyes always appeared on one side, and the nose and mouth were presented together on the other side. Middle panel: Grand-averaged event-related brain potentials (ERPs) elicited by intact and scrambled faces at right occipito-temporal electrode P8 in the 300-ms interval after stimulus onset, in a group of 10 participants with developmental prosopagnosia (DPs; left) and 10 age-matched control participants. For controls, N170 amplitude was enhanced for scrambled as compared to intact faces. No such N170 amplitude effect was present for the DP group. Bottom panel: ERPs elicited at lateral temporo-occipital electrodes P7/P8 in response to intact faces (averaged across P7/8) and to scrambled face images at electrodes contralateral to the side of the two eyes and for electrodes contralateral to the side of the mouth and nose. For control participants (right panel), the N170 enhancement and delay to scrambled faces was larger contralateral to the side of the nose/mouth. For DPs (left panel), no such position-specific N170 modulations were present. Data taken with permission from Towler, Parketny et al. (2016), reproduced in a different format.

inversion and the spatial scrambling of facial features have similar effects on the N170 emphasize the sensitivity of this N170 component to deviations from a canonical upright face template and suggests that these two manipulations may affect the same stages of configural face processing. In our study (Towler, Parketny et al., 2016) scrambled faces triggered the typical enhanced and delayed N170 components relative to intact faces for control participants, but there was no such enhancement of N170 amplitudes for scrambled faces as compared to intact faces in the group of DPs (see Figure 3, middle panel). There was a tendency for the N170 to be delayed for scrambled versus intact faces in the DP group, but this was not statistically reliable. The absence of an N170 amplitude enhancement for scrambled as compared to intact faces for DPs is similar to the absence of the typical face inversion effect on N170 amplitudes observed in our earlier study (Towler et al., 2012) for the DP group. This suggests that atypical N170 modulations to face inversion and feature scrambling may both reflect a reduced sensitivity to deviations from an upright visual face template in DP.

Further evidence for the hypothesis that canonical face templates are impaired in DP was obtained in the same study (Towler, Parketny et al., 2016). Visual face representations, as reflected by the N170, are strongly position dependent (e.g., Rousselet, Ince, van Rijsbergen, & Schyns, 2014; Smith, Gosselin, & Schyns, 2004). When a face and a non-face object are simultaneously presented in opposite visual fields, the face-sensitive N170 component is confined to the contralateral hemisphere (Towler & Eimer, 2015). If the N170 reflects the activation of position-dependent visual representations of faces and facial features, N170 components to scrambled face images might also be sensitive to the location of specific face parts in the visual field, and in particular to the deviation of these parts from the canonical upright face template. To test this prediction, the scrambled faces used in our experiment were always asymmetric (as shown in Figure 3, top panel). The two eyes were always located on one side of these faces, and one eye was located in its canonical position. The mouth and nose occupied non-canonical positions on the opposite side. Faces were presented at fixation, so that these two sides were each projected to the opposite (contralateral) hemisphere. In the control group, N170 components to scrambled faces were larger at posterior electrodes contralateral to the side where the nose and mouth both appeared in non-canonical

positions than at electrodes over the hemisphere that was contralateral to the two eyes (Figure 3, bottom right panel). Both N170 components to scrambled faces were larger than the N170 to intact faces for control participants. This suggests that deviation of face parts from a canonical upright face template are registered and represented in a position-dependent fashion, with more extreme deviations triggering larger contralateral N170 components. In contrast to the control group, the DP group showed no reliable amplitude differences between N170 components triggered in response to scrambled faces contralateral to the nose/mouth and contralateral to the eyes and N170 components to intact faces (Figure 3, bottom left panel). These findings suggest that deviations from the canonical face template are registered in retinotopic coordinates in the normal face perception system, and that sensitivity to deviations from this template are absent or strongly reduced in developmental prosopagnosics.

Visual-spatial configural templates of facial structure may be necessary for holistic stages of face perception. Facial features are encoded and perceived in a context-dependent fashion relative to the surrounding facial features and head outline. The apparent lack of sensitivity to the typical retinotopic locations of facial features in participants with DP, as reflected by atypical N170 amplitude modulations in response to inverted or scrambled faces, may be linked to deficits in holistic face processing in DP. If individuals with DP do not have neural mechanisms that are tuned to the typical locations of features within a face, holistic aspects of face processing may be impaired as a result. In line with this hypothesis, markers of holistic face processing such as the composite face illusion are also often reduced in DP (Avidan et al., 2011; Palermo et al., 2011), which is notable because this visual illusion may be dependent on a canonical visual template of the whole face. The fact that DPs show normal face-sensitive N170 components to Mooney faces versus houses (as discussed in the previous section) suggests that face processing and face detection in DP can be based on a coarse global face template. This fact is consistent with evidence suggesting that holistic face processing in DP is generally not entirely absent. In fact, DPs are also capable of representing the identity of particular facial features such as the mouth within the context of the rest of the face as shown by normal part-whole effects for the mouth region (DeGutis, Cohan et al., 2012). The primary difficulty for DPs appears to

be in the use of these global contextual cues about face shape for face recognition specifically when processing the eye region (DeGutis, Cohan et al., 2012). In this context, it is notable that for participants without face processing impairments, N170 amplitude enhancements caused by face inversion appear to be largely driven by the presence of the eyes in faces (Kloth, Itier, & Schweinberger, 2013). This suggests that perceptual face processing impairments in DP might be specifically associated with the eye region. In the next section, we discuss neural evidence from the N170 component that processing of the eyes within the face template is disrupted in DP.

The N170 component reveals a specific impairment in processing the contrast polarity of the eye region in DP

The contrast polarity of faces is crucial for face perception and recognition. Reversing the contrast polarity of familiar faces dramatically impairs their recognizability (e.g., Galper, 1970; Johnston, Hill, & Carman, 1992). This effect appears to be specific to face perception, as the recognition of non-face objects is much less sensitive to contrast reversal (Nederhouser, Yue, Mangini, & Biederman, 2007; Vuong, Peissig, Harrison, & Tarr, 2005). Contrast inversion of faces has also been shown to eliminate face-selective activity in posterior face processing regions such as the FFA (e.g., George et al., 1999). Neurophysiological studies of face perception in the macaque have found not only that facial features are encoded relative to an upright face template (e.g., Freiwald, Tsao, & Livingstone, 2009), but also that the polarity of the face parts is crucial in modulating the responses of these same face-selective neural populations in inferior temporal cortex (Ohayon, Freiwald, & Tsao, 2012). Face-selective neurons exhibit the strongest and earliest responses to face images only when the face parts occupy their canonical spatial positions and when they were also in the correct contrast polarity. The fact that disruptions to the contrast-sensitive spatial layout of the face template delay and attenuate face-selective neural responses suggests that the face template is strongly sensitive not only to spatial locations of features but also to the typical contrast values of these features.

The importance of contrast signals from the eye region for face recognition was demonstrated by Gilad, Meng, and Sinha (2009) with “contrast chimera” images that include face parts in normal

contrast and other parts that are contrast inverted. When the eye region of famous faces are restored to normal contrast, while the rest of the face is contrast inverted, recognition performance for these positive-eyes chimeras improves to approximately 90% of the level observed with faces in normal contrast (see also Sormaz, Andrews, & Young, 2013). Gilad et al. (2009) also showed that fMRI activity in the FFA elicited by positive-eyes chimeras was indistinguishable from the response to normal contrast faces. Such observations suggest that the effects of contrast inversion on face processing might be primarily or even exclusively driven by the contrast of the eye region within whole upright faces.

The N170 component is sensitive not only to manipulations of face orientation but also to manipulations of face contrast. Similar to the effects of face inversion and the spatial scrambling of face parts, contrast-inverted faces elicit delayed and enhanced N170 components relative to faces with normal positive contrast (e.g., Itier, Latinus, & Taylor, 2006; Itier & Taylor, 2002). Such N170 modulations have been attributed to disruptive effects on perceptual face processing caused by changing the typical contrast polarity of faces (Itier et al., 2006). To test whether these effects of contrast inversion on the N170 are primarily driven by contrast signals from the eye region, and whether this may depend on which part of a face is currently fixated, we recently ran a study where the contrast of the eye region and the contrast of the rest of a face was varied orthogonally (Fisher, Towler, & Eimer, 2015; as illustrated in Figure 4, top panel). Face images appeared unpredictably either in the upper or lower visual field, so that eye gaze was centred either between both eyes (upper fixation condition) or between the nose and mouth (lower fixation condition). Contrast-reversal of the eye region resulted in delayed and enhanced N170 components, independently of the contrast of other face parts, and regardless of gaze location. Contrast-reversal of the rest of the face produced similar N170 modulations, but only when participants fixated between the nose and mouth, and not when their gaze was close to the eye region. This demonstrates that the contrast polarity of the eye region has a privileged role during early stages of face processing.

To investigate whether the perceptual processing of face contrast and the role of contrast signals from the eye region in DP, we used procedures identical to those of Fisher et al. (2015) in a study where a group of 11 individuals with DP and 11 age-matched

control participants were compared (Fisher, Towler, & Eimer, *in press*). The N170 results for the upper fixation condition are shown in Figure 4 for DPs and controls. ERPs to faces with normal contrast in the eye region (positive eyes) and with inverted contrast in this region (negative eyes) are shown separately for images where the rest of the face was contrast-positive or contrast-negative. For the control group, inverting the contrast polarity of the eye region delayed and enhanced the N170, independently of the contrast polarity of the rest of the face. The contrast polarity of the rest of the face had no impact on the N170 component (as in Fisher et al., 2015). For DPs, there was no reliable N170 amplitude enhancement for faces with contrast-inverted versus contrast-normal eye regions, regardless of whether the rest of the face appeared in normal or inverted contrast. Similar to the control group, the contrast polarity of the rest of the face did not affect N170 amplitudes in the DP group. In the lower fixation condition (not shown in Figure 4), inverting the contrast of the rest of the face produced similar N170 amplitude enhancements both for control participants and for DPs, demonstrating that perceptual face processing in DP is not generally insensitive to variations of face contrast across the whole face. The contrast polarity of face parts outside the eye region affects early stages of perceptual face processing in DPs and controls, but only when gaze is directed away from the eyes.

The absence of N170 amplitude modulations caused by contrast inversion of the eye region in the DP group strongly suggests that DPs have a specific impairment in processing contrast signals from the eye region, even when fixation is close to this region. This atypical effect appears to be specific to the eye region. Given that contrast information in the eye region is important for face recognition (Gilad et al., 2009; Sormaz et al., 2013), a failure to perceptually process and utilize this information may contribute to the face recognition deficits in DP. It should be noted, however, that while N170 amplitudes were insensitive to the contrast of the eye region in the DP group, N170 latencies showed a similar sensitivity to contrast inversion in both groups. As can be seen in Figure 4, N170 peak amplitudes were delayed to faces with contrast-inverted as compared to contrast-normal faces both for DPs and for control participants, with no statistical difference in N170 latency delays between the two groups. The implications of this apparent dissociation between N170 amplitude and latency measures are discussed below.

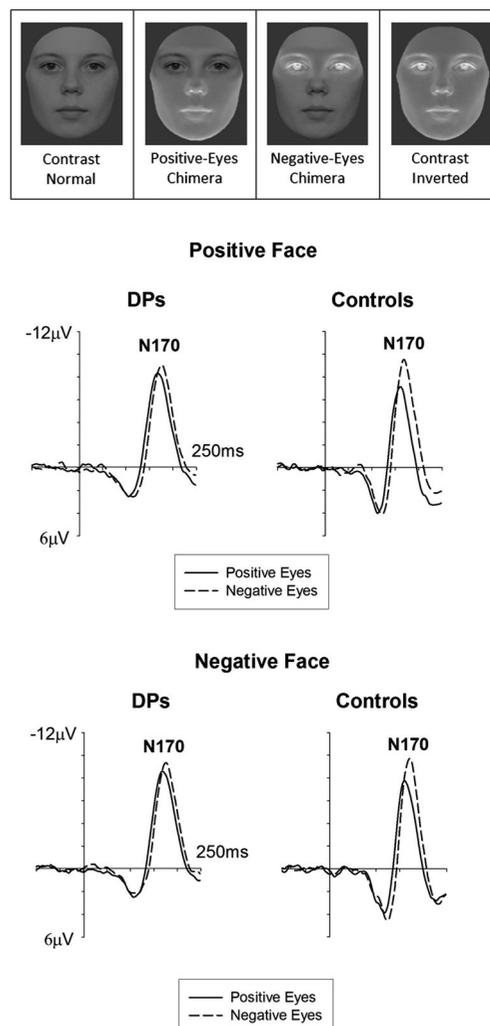


Figure 4. Top panel: Examples of the different types of face images used in the study by Fisher et al. (*in press*). In addition to contrast-normal and fully contrast-inverted faces, there were also face images with contrast-normal eye regions and contrast inversion of the rest of the face (positive-eyes chimeras) and faces with a contrast inverted eye region within an otherwise normal contrast face (negative-eyes chimeras). Middle and bottom panels: Grand-averaged event-related brain potentials (ERPs) elicited by faces with positive-contrast eye regions (positive eyes) and faces with contrast-inverted eye regions (negative eyes) at right occipito-temporal electrode P10 in the 250-ms interval after stimulus onset, for trials where the participant's gaze was focused on the nasion near the eye region. ERPs are shown for a group of 11 participants with developmental prosopagnosia (DPs; left) and 11 age-matched control participants (right). For both groups, ERP waveforms are presented separately for images where the rest of the face was contrast-positive or contrast-negative. For controls, contrast inversion of the eye region triggered an N170 amplitude enhancement. No such effect was present for participants with DP. Data taken with permission from Fisher et al. (*in press*).

The implications of atypical N170 amplitude effects for the nature of perceptual face processing impairments in DP

The N170 studies of perceptual face processing in DP discussed in the previous sections suggest that there are systematic differences between DPs and control participants during early stages of face-selective visual processing within the first 200 ms after encountering a face. Even though DPs have face-selective brain regions that are activated within the same time range as in individuals without face recognition impairments, the pattern of atypical N170 responses to face inversion, contrast inversion, and the spatial scrambling of face parts suggests that some aspects of early face processing operate in a qualitatively different fashion in DPs and control participants. It is notable that while DPs consistently showed atypical effects for N170 amplitudes, N170 latencies were not systematically different between DPs and control groups across the four ERP studies (Fisher et al., *in press*; Towler et al., 2012; Towler, Gosling et al., 2016; Towler, Parketny et al., 2016). To explain this dissociation, it is important to consider how N170 amplitude and latency modulations are usually interpreted. N170 latency delays to inverted faces or faces with scrambled internal features are believed to reflect delayed responses of face-selective neural populations that are linked to the increased difficulty of detecting faces or face parts that lack some of the canonical visual properties of prototypical upright faces (e.g., Rossion et al., 1999). The fact that individuals with DP do not tend to differ from control participants in terms of N170 latencies suggests that the activation of face-selective neural populations in response to faces that deviate from the canonical face template is delayed in both groups.

N170 amplitude modulations associated with face inversion, the scrambling of face parts, and the inversion of face contrast have been interpreted in two different ways (e.g., Sadeh & Yovel, 2010). According to the quantitative account, face-selective neurons increase their firing rates in response to faces that do not correspond to the canonical upright face template, because these face images are more difficult to encode than standard upright faces (e.g., Rossion et al., 1999). According to an alternative qualitative account, atypical face images recruit separate neural populations in addition to those that are activated during the perceptual processing of canonical

upright faces, thereby producing enhanced N170 amplitudes (e.g., Sadeh & Yovel, 2010). For example, N170 amplitude enhancements may reflect increased firing rates of visual neurons that are not exclusively face-selective but more broadly tuned, or neurons that are selective for non-face object categories. When an orientation-inverted or contrast-reversed face is encountered, face-selective neurons take longer to classify the stimulus, and other non-face-selective neurons become simultaneously active and contribute to the neural activation that gives rise to the N170 component. In line with this idea, Rosburg et al. (2010) found that inverted faces produced enhanced neural firing in both face-selective and house-selective neurons in lateral occipital cortex, but not in ventral temporal cortex.

In summary, N170 amplitude enhancements and latency delays reflect perceptual violations to the average statistical visual properties (such as spatial configuration and contrast polarity) that are present in canonical face images, provided that deviant face images can be still be perceptually interpreted as faces and are therefore at least partially processed by specialized face-selective neural populations. This account has direct consequences for how the pattern of atypical N170 modulations in DP can be interpreted. We suggest that the absence of characteristic N170 amplitude enhancements in response to faces that deviate from the canonical spatial configuration or contrast values of prototypical upright faces is due to the fact that the perceptual analysis of upright faces is atypical in DP. Individuals with DP process upright faces in a similar way to faces with non-canonical features (e.g., inverted faces, scrambled faces, faces with contrast-inverted eye regions), resulting in similar N170 amplitudes to canonical and non-canonical face images. If the typical N170 amplitude increase to non-standard faces reflects an increased activation of face-selective neural populations in response to faces that are more difficult to encode (as suggested by the quantitative account), the absence of such N170 amplitude effects in DP would suggest that the difficulty of encoding standard upright and non-standard faces does not differ systematically in individuals with DP, perhaps because both are treated as deviating equally from the atypical face template in DP. Alternatively, and in line with the qualitative account of N170 amplitude modulations, the lack of such modulations in DP may reflect the recruitment of additional object-selective neuronal populations not just during the processing of non-canonical

faces, but also when standard upright faces are perceptually analysed. We return to this point in the General Discussion where the implications of these N170 findings in DP are discussed in the context of neurodevelopmental models.

More generally, the overall pattern of N170 results for individuals with DP suggests that the processing of upright faces in the posterior core face processing system operates in an atypical fashion, despite the presence of face-selective regions. Face representations in the occipito-temporal network are less finely tuned to the visual statistical properties of canonical upright faces in DP. Additional evidence for this hypothesis comes from a recent fMRI study that used pattern classification to decode the difference between faces with typical or disrupted spatial configurations (Zhang, Liu, & Xu, 2015). For control participants, face configuration was selectively decodable from the right FFA, but this was not possible for a group of participants with DP, suggesting that configural information is less precisely represented in DP. In this context, the observation that DPs show face-selective activation within posterior face processing areas, but not in the most anterior temporal face-selective region (Avidan et al., 2014), may also be important. As damage to this anterior temporal region has been shown to produce specific perceptual deficits in holistic face processing (e.g., Busigny et al., 2014), it may be involved in the activation of canonical upright face templates, which will facilitate the perceptual analysis of template-matching face images in areas such as the FFA. If the face-selective anterior temporal region does not operate normally in DP, it may not be able to serve this specific function. The direction of causality between impairments in anterior and posterior regions of the visual face processing system remains to be determined. On the one hand, atypical face representations in the FFA may result in an impaired ability to extract and maintain configural face templates in anterior temporal region. On the other hand, anterior temporal dysfunctions may directly affect perceptual face processing in more posterior regions, as reflected by the N170 component. In this case, impaired feedback connections from the anterior temporal region to the FFA may result in canonical and non-canonical faces being processed in the same inefficient fashion. Such atypical interactions between the FFA and the anterior temporal region could be a major contributing factor to the face recognition impairments in DP. Further implications of this hypothesis are discussed in the final section in the

context of a general neurodevelopmental framework of DP.

The rapid detection of emotional facial expression is preserved in DP

The previous sections have discussed how processes involved in the structural perceptual encoding of faces might be impaired in DP. According to Bruce and Young (1986), the function of structural encoding is to construct visual representations of seen faces that are suitable for subsequent face recognition and identification. However, faces provide information not only about identity, but also about other socially important attributes, such as a person's gender, age, eye gaze, and emotional state. Although there is some degree of heterogeneity among DPs as a group, DPs are not generally impaired in their recognition of these other facial attributes (e.g., Duchaine, Murray, Turner, White, & Garrido, 2009). This is a key dissociation from other disorders that often co-occur with face recognition deficits, such as autism spectrum disorder. Most DPs also are relatively normal in their ability to recognize basic emotional facial expressions (Duchaine, Parker, & Nakayama, 2003; Humphreys, Avidan, & Behrmann, 2007), demonstrating that they have intact categorical long-term visual and semantic memories of these expressions. Match-to-sample tasks for facial expressions in DPs also often yield normal scores (Bentin et al., 2007; Garrido et al., 2009; Lee et al., 2010), suggesting that representations of facial expressions can be successfully maintained in working memory. DPs are also able to make comparative judgements about the intensity of basic expressions of two simultaneously presented photographs (Duchaine et al., 2003). This suggests that DPs are able not only to categorize different basic emotional expressions but also to perceive more subtle differences in the strength of these emotions. Some DPs can identify a large variety of subtle and complex emotions from faces in naturalistic photographs (Duchaine et al., 2003; Duchaine, Yovel et al., 2007), and this ability is inversely correlated with autistic traits (Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001). DPs have also been shown to rate faces for attractiveness and trustworthiness similarly to control participants, although they show more inconsistent and random patterns for facial distinctiveness ratings (Carbon, Grüter, Grüter, Weber, & Lueschow, 2010; Todorov & Duchaine, 2008). In addition, they are able to make correct gender

judgments of whole faces and face parts (DeGutis, Chatterjee, Mercado, & Nakayama, 2012) and to successfully sort morphed faces according to age and gender (Chatterjee & Nakayama, 2012). Gender judgments are impaired both for controls and for DPs when faces are inverted or facial features are scrambled (Chatterjee & Nakayama, 2012; DeGutis, Cohan et al., 2012). Such observations suggest that face perception and recognition in DP can be comparable to those in controls in tasks where facial identity is not relevant. However, this does not imply that the processing of emotional expression and other facial attributes apart from identity is normal in every individual DP. Some studies have identified DPs with gender perception deficits (Behrmann, Avidan, Marotta, & Kimchi, 2005; De Haan & Campbell, 1991; Duchaine, Yovel, Butterworth, & Nakayama, 2006) and below-average expression perception and recognition (e.g., Dobel, Bölte, Aicher, & Schweinberger, 2007; Duchaine et al., 2003; Duchaine et al., 2006; Duchaine et al., 2009; Minnebusch et al., 2007).

The presence of dissociations between the recognition of identity and expression in DP supports the hypothesis proposed by Bruce and Young (1986) that facial identity and expression are processed in functionally distinct parallel pathways. Initial evidence for this claim was provided by studies of patients with AP, who were reported to have no expression recognition deficits (e.g., Bruyer et al., 1983; Fox, Hanif, Iaria, Duchaine, & Barton, 2011; Tranel, Damasio, & Damasio, 1988; Young, Newcombe, de Haan, Small, & Hay, 1993). According to Haxby et al. (2000), identity information is processed in the fusiform face area (FFA), whereas changeable face features such as expression are processed in the superior temporal sulcus (STS) as well as in regions of the extended face processing system such as the amygdala. Others have challenged this idea that the processing of expression and identity can be fully dissociated in the face network at the cognitive level and at the neural implementation level (e.g., Calder & Young, 2005). For example, FFA activity has been shown to be modulated by facial expression (Fox, Moon, Iaria, & Barton, 2009; Furl et al., 2011; Ganel, Valyear, Goshen-Gottstein, & Goodale, 2005; Van den Stock, van de Riet, Righart, & de Gelder, 2008; Vuilleumier, Armony, Driver, & Dolan, 2001), and the STS was found to be responsive to facial identity (Baseler, Harris, Young, & Andrews, 2013; Winston, Henson, Fine-Goulden, & Dolan, 2004). As the debate about dissociations between the processing of facial identity

and expression remains unresolved, investigating the processing of emotional expression in DP is obviously important. Neural response to emotional faces in brain regions that preferentially respond to emotional expressions, such as the fusiform gyrus, the STS, and the amygdala, appears to be normal in DP (Avidan et al., 2014; Dinkelacker et al., 2011; Furl et al., 2011; Van den et al., 2008), suggesting that the processing of facial identity and expression may indeed be dissociable.

If DPs have atypical visual face templates at early perceptual stages of face processing, they may use atypical or compensatory strategies to recognize facial emotions, despite their generally competent performance at such tasks. ERP measures may reveal differences in the time course of facial expression processing between DPs and control participants. In a study by Eimer and Holmes (2002), a neural marker of emotional face processing was found to emerge as early as 120 ms after the onset of a face image. Relative to neutral faces, fearful faces elicited an enhanced positivity at anterior frontal electrode sites that was present between around 120–200 ms post stimulus and was followed by a more broadly distributed sustained positivity across frontocentral electrodes that emerged at about 250 ms after stimulus onset (see also Eimer, Holmes, & McGlone, 2003, for similar effects in response to facial expressions other than fear). The early differential ERP response was interpreted in terms of a rapid detection of emotional facial expression that is based on signals from posterior visual areas that are transmitted to medial prefrontal regions involved in the fast detection of emotionally salient stimuli. Lesion and brain imaging studies have also suggested an integral role for the prefrontal cortex in facial emotion recognition and the evaluation of social signals (e.g., Adolphs 2002, 2003). Additional causal evidence for the role of the medial prefrontal cortex in emotion recognition comes from TMS stimulation, which has been shown to disrupt recognition of positive and negative emotional expressions (Harmer, Thilo, Rothwell, & Goodwin, 2001; Mattavelli, Cattaneo, & Papagno, 2011).

To test whether rapid anterior ERP responses to fearful versus neutral faces would also be observed in participants with DP, we tested a group of 16 DPs and 16 age-matched controls (Towler, Fisher, Grubert, Gosling, & Eimer, unpublished), using the same stimulus procedures as those in Eimer and Holmes (2002). Figure 5 shows ERPs triggered at frontopolar midline electrode Fpz in response to upright

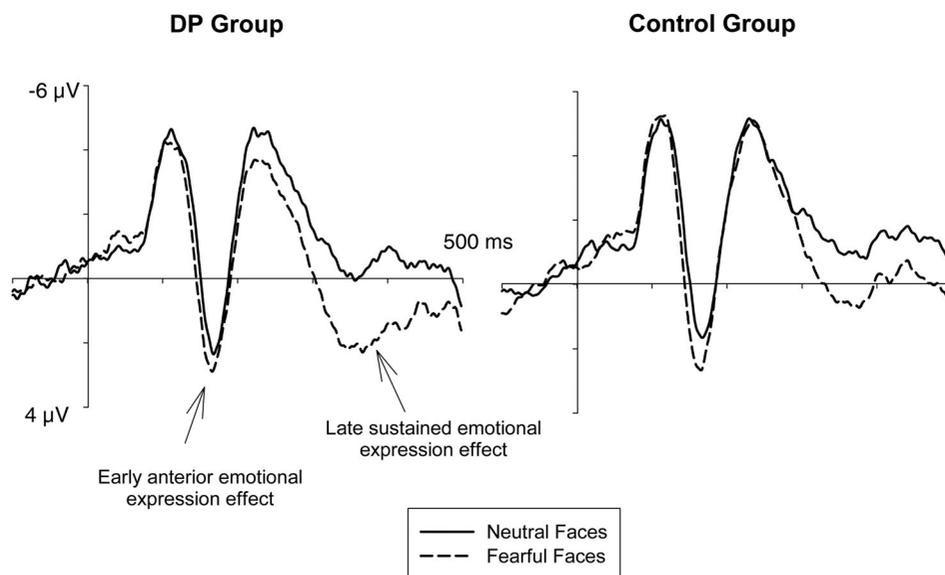


Figure 5. Grand-averaged event-related brain potentials (ERPs) elicited by neutral and fearful faces at frontopolar electrode Fpz in the 500-ms interval after stimulus onset, for a group of 16 participants with developmental prosopagnosia (DPs; left panel) and a group of 16 age-matched controls (right panel). In both groups, an early anterior emotional expression effect (an enhanced positivity to fearful versus neutral faces) was present and was followed by a later sustained positivity to fearful faces both for DPs and for controls. Data taken with permission from Towler et al. (unpublished).

fearful and neutral faces, separately for DPs and control participants. Rapid differential ERP responses to emotional faces were clearly present in both groups. As in the original study (Eimer & Holmes, 2002), the frontocentral positivity for fearful as compared to neutral faces started around 120 ms after stimulus onset, and this effect was reliably present not just in the control group but also for the DPs. The onset and size of this rapid anterior emotional expression effects did not differ between the two groups, demonstrating that the cortical detection of facial expression operates just as rapidly in DP as in unimpaired control participants. In addition, the subsequent sustained positivity to fearful versus neutral faces that started around 250 ms post stimulus was also found to be equivalent across both groups. This later sustained effect, which has also been observed in response to emotionally salient non-face stimuli (Schacht & Sommer, 2009), may be associated with the in-depth cognitive processing and explicit recognition of emotional facial expression. In line with behavioural studies showing normal recognition of emotional expressions in DP and fMRI studies showing that DPs have normal activity in brain regions sensitive to facial expression, these ERP results suggest that the processing of emotional facial expression does not differ between DPs and controls.

The apparent dissociation between the processing of facial identity (which is severely impaired in DP) and the rapid detection of facial emotion signals (which appears to operate normally) is in line with the ideas originally proposed by Bruce and Young (1986). In spite of the fact that DPs have impaired structural templates for face recognition, the processing of fearful versus neutral faces in prefrontal regions appears to follow a normal time course in DP, suggesting that DPs do not use atypical compensatory strategies to detect emotional facial expressions. It is important to note that the detection of emotional expression does not necessarily require a full configural analysis of face images. Salient facial features such as the eyes or the mouth that are diagnostic for specific expressions can be registered during the rapid perceptual analysis of visual input and can then activate anterior areas involved in the detection of facial emotion even before the configural processing of face images in posterior visual areas has been completed.

ERP evidence for delay and disconnection in the face recognition system in DP

The ERP studies discussed so far have focused on early stages of face processing that are activated within the first 200 ms post stimulus. The N170 component is

linked to perceptual stages of face processing and does not directly reflect the recognition of facial identity. ERP components associated with face recognition emerge at post-stimulus latencies of about 220 ms. At this point in time, an enhanced occipito-temporal negativity is elicited in response to the faces of familiar individuals or previously learned target faces (N250 component; Gosling & Eimer, 2011; Tanaka, Curran, Porterfield, & Collins, 2006; see also Schweinberger, Pfütze, & Sommer, 1995). Such N250 components are assumed to reflect the activation of a stored representation of a specific face in visual memory that is triggered when there is a match with a currently presented face. The N250 component is typically followed by a broadly distributed sustained positivity (P600f) to familiar faces (Bentin & Deouell, 2000; Eimer, 2000a, 2000b), which has been linked to later stages of face recognition, such as name retrieval or access to episodic or semantic information about a specific individual (e.g., Gosling & Eimer, 2011).

Because these components are assumed to be elicited during the activation of an individual face representation in visual memory (N250) and the subsequent explicit recognition of a particular face (P600f), it is important to find out whether they are preserved or eliminated in individuals with DP in tasks that require the explicit recognition of individual faces. A study by Eimer, Gosling, and Duchaine (2012) investigated these ERP markers of face recognition in DP. Twelve participants with DP were presented with photographs of famous and non-famous faces and had to classify each face as known/familiar or unfamiliar/unknown. Famous faces were those of actors, politicians, musicians, sports personalities, and other celebrities well known in the UK. Control participants correctly classified more than 80% of these famous faces (Gosling & Eimer, 2011), whereas DPs recognized less than 30% of the same famous faces. However, on those relatively few trials where DPs correctly classified a particular face as known or familiar, these faces elicited N250 and P600f components that were virtually identical to the components observed for control participants (as shown in Figure 6, top panel, for the N250 component). These similarities suggest that there are no fundamental qualitative differences in the brain mechanisms responsible for the recognition of famous faces between these two groups.

An interesting dissociation between some DPs and the control group emerged in the analysis of ERPs elicited by those famous faces that were not recognized. None of the control participants showed any

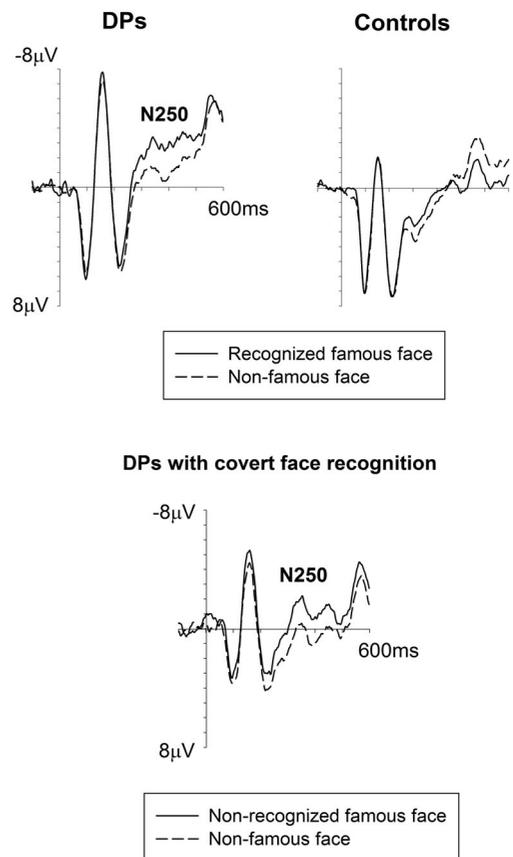


Figure 6. Top panel: Grand-averaged event-related brain potentials (ERPs) to correctly recognized famous faces and to non-famous faces at right occipito-temporal electrode P8 for 12 participants with developmental prosopagnosia (DPs; left) and 16 participants without face recognition impairment (right). N250 components to successfully recognized famous faces are very similar in both groups. Bottom panel: ERP correlates of covert face recognition for six participants with DP who showed a reliable N250 to famous faces on trials where they failed to recognize these faces. Data taken with permission from Gosling and Eimer (2011) and Eimer et al. (2012), reproduced in a different format.

differential ERP response between non-recognized famous and unfamiliar faces, confirming that they indeed failed to identify these particular famous faces. In contrast, a clear and statistically reliable N250 component was elicited by non-recognized famous faces for six of the 12 DPs tested (as shown in Figure 6, bottom panel). Even though these six DPs had reported these famous faces to be unknown, N250 components distinguished between these faces and other objectively unfamiliar faces. In other words, non-recognized famous faces activated their associated stored representations in visual face memory in these individuals with DP. The presence of a differential physiological response to a non-

recognized familiar face is described as covert face recognition (Diamond, Valentine, Mayes, & Sandel, 1994). The presence of an N250 component to non-recognized famous faces in DP can thus be interpreted as evidence for covert recognition at the level of visual face memory. These observations also indicate that the activation of visual memory representations of individual faces is not sufficient for overt face recognition, in line with current functional models of normal face processing (Bruce & Young, 1986; Burton, Bruce, & Johnston, 1990). Conscious recognition may require that faces are also processed at subsequent post-perceptual semantic stages, which are associated with the later P600f component. In line with this prediction, non-recognized famous faces did not trigger a P600f component for those six DPs who showed covert recognition effects for the N250 component. While six of the 12 DPs tested in our study (Eimer et al., 2012) showed N250 components to non-recognized famous faces, this was not the case for the other six participants with DPs. This subgroup of DPs apparently did not activate any visual memory representations of famous faces when they failed to recognize these faces explicitly. The absence of N250 components could reflect either the absence of long-term visual memories of famous faces, or an inability to access such long-term memory representations on the basis of the current perceptual input.

The presence of an N250 component to non-recognized famous faces in some DPs indicates that these faces were able to activate corresponding representations in visual memory. The absence of a P600f component in response to the same faces shows that this activation of visual face memory was insufficient to trigger processes that lead to the explicit recognition of these faces. The inability of these DPs to consciously identify and report a particular famous face even when this face activated a matching memory representation may thus be due to a disruption of links between stored visual representations of familiar individuals and semantic or episodic representations in long-term memory. This interpretation is consistent with previous views of prosopagnosia as a disconnection phenomenon, where face recognition deficits are described as the result of damaged links between stored visual representations of familiar individuals and associated semantic memory traces (Burton, Young, Bruce, Johnston, & Ellis, 1991). In the face processing architecture proposed by Bruce and Young (1986), this disconnection would be located between

visual face recognition units (FRUs) and subsequent semantic person identity nodes (PINs). A similar disconnection hypothesis was suggested by Breen, Caine, and Coltheart (2000) to account for the patterns of dissociation between overt and covert face recognition observed in AP and in the Capgras delusion (the belief that a close relative or friend has been replaced by an impostor).

In terms of the standard neuroanatomical model of face recognition (Gobbini & Haxby, 2007; Haxby et al., 2000), these ERP findings suggest that the activation of visual face memories in the occipito-temporal regions of the face processing network in response to a perceptual match is insufficient for conscious face recognition. The dissociation between the presence of N250 and absence of P600f components to non-recognized famous faces in some DPs suggests a disconnection between the core occipito-temporal face processing system and areas within the extended face processing network that are involved in the recognition of familiar people, such as the anterior paracingulate, posterior cingulate, precuneus, and anterior temporal cortex. A previous fMRI study has shown that activation of anterior temporal regions during face recognition is reduced in DPs as compared to controls (Avidan et al., 2014). The P600f component may reflect the activation of these extended regions that are involved in access to episodic and semantic memory, and the interpretation of personality traits and mental states associated with particular individuals. Because DPs do not appear to have general semantic or episodic memory deficits, it is likely that the absence of activations in these regions in face recognition tasks reflects the inability of face memory signals to propagate from an atypical occipito-temporal face network to more anterior regions in the extended face processing system that are necessary for conscious face recognition.

While the ERP study by Eimer et al. (2012) investigated the recognition of famous faces, which are likely to be represented in long-term memory, a recent experiment (Parketny, Towler, & Eimer, 2015) studied the processes that are responsible for the recognition of previously unfamiliar but now task-relevant faces in DP. In an experimental paradigm developed by Tanaka et al. (2006), single face images were presented sequentially, and participants had to respond to a previously studied but otherwise unknown target face ("Joe"), while ignoring other task-irrelevant distractor faces. One of these distractors was the participant's own face. Ten participants with DP

and a group of 10 age-matched control participants were tested. The pattern of N250 and P600f components in response to target faces revealed systematic differences in the identity-related processing of these faces between the two groups. Target faces triggered reliable N250 components both for DPs and for controls, demonstrating that a visual face memory trace of a previously unfamiliar learned task-relevant face was activated by a match with the currently seen face in both groups. Importantly, the onset of the N250 to target faces in the right hemisphere was delayed by approximately 40 ms in the DP group. This onset delay is illustrated in Figure 7, which shows difference waveforms obtained by subtracting ERPs to nontarget faces from ERPs to target faces at posterior right-hemisphere electrode P8, separately for the DP and control groups. The delay of the target N250 component in the DP group provides the first direct evidence that the time-course of face identity processing (i.e., the activation of short-term memory representations of a recently learned individual face) is altered in DP. The later onset of the N250 component in DPs could reflect a deficit in perceptual face processing, such as an impairment in analysing and representing identity-specific properties of a currently seen face. Alternatively, it could also be associated with deficits in visual face memory (e.g., lower precision of, or impaired access to, stored visual representations of the learned target face).

In addition to these differences between DPs and control participants in the timing of the N250 component, the subsequent P600f component to target faces was attenuated and emerged almost 70 ms later in the DP group relative to the control group. At right-hemisphere electrode P8, the P600f was clearly visible for control participants, but was virtually eliminated for DPs (as shown in Figure 7). As the P600f is linked to explicit face recognition (see above), this observation indicates that DPs identified target faces considerably slower than did control participants. In line with this hypothesis, reaction times to correctly detected target faces were about 150 ms slower in the DP group than in the control group. The inclusion of participants' own faces in this study (Parketny et al., 2015) allowed us to investigate whether face-based self-recognition processes might also be impaired in DP. Overall, own faces elicited very similar N250 and P600f components in DPs and controls, suggesting that the activation of long-term representations of one's own face in visual memory and the subsequent explicit recognition of this face are not generally

Difference Waves: Target Face - Nontarget Face

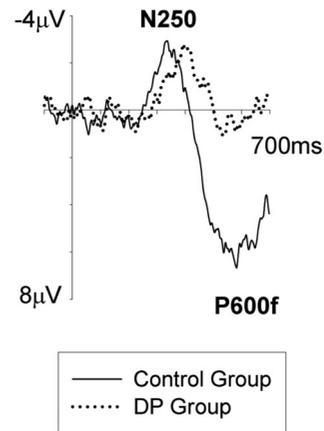


Figure 7. Difference waveforms computed for right occipito-temporal electrode P8 by subtracting event-related brain potentials (ERPs) to task-irrelevant nontarget faces from ERPs in response to a learned previously unfamiliar target face, shown separately for a group of 10 participants with developmental prosopagnosia (DPs) and a group of 10 age-matched controls. The N250 component to target faces was delayed in the DP group, and the subsequent P600f component was strongly attenuated for DPs as compared to controls. Data taken with permission from Parketny et al. (2015), reproduced in a different format.

selectively impaired in DP. This shows that prolonged experience with particular faces can result in normal face recognition processes in individuals with DP. However, two of the 10 DPs tested reported after the experiment that they had been unaware of the presence of their own face during testing. For these two DPs, P600f components to their own faces were entirely absent, demonstrating that the explicit recognition of the own face can be strongly impaired in some individuals with DP.

Overall, the results from these two ERP investigations of the recognition of famous faces and of previously unfamiliar learned target faces in DP show that there are both similarities and differences in the identity-related processing of faces between DPs and controls. The fact that N250 and P600f components were elicited during the successful recognition of individual faces in DPs shows that the activation of visual face memory representations and the subsequent explicit recognition of faces do not operate in a qualitatively different fashion in DP. However, the delay of the N250 to previously unfamiliar target faces in the DP group (Parketny et al., 2015) and the observation that visual face memory representations of a famous face can be activated in the absence of explicit face

recognition in some DPs (Eimer et al., 2012) point to systematic differences in the identity-related processing of faces between DPs and controls. It will be important to investigate whether and to what degree these differences are the consequence of processes that take place during earlier sensory-perceptual face processing stages. If face perception in DP cannot be effectively guided by canonical face templates, as suggested earlier, the quality of visual-perceptual representations of individual faces that are used during subsequent face recognition processes may often not be sufficient to provide the basis for a fast and effortless identification of a particular individual.

The observation that the delay and attenuation to the P600f to target faces in DPs versus controls was greater than the corresponding effects observed for the N250 (Parketny et al., 2015) suggests that disruptions to explicit face recognition mechanisms in more anterior regions are not simply linear knock-on effects from the poor visual perceptual processing of face images in upstream posterior areas. Lesion studies indicate that posterior and anterior sites may comprise functionally distinct components of the face recognition system. Damage to posterior occipito-temporal cortex can cause specific problems with the visual perception of faces (“apperceptive prosopagnosia”, e.g., Barton, 2008). Damage to the anterior temporal lobes can have specific effects on face and person memory and can lead to a deficit in activating and integrating new semantic associations related to an individual person (e.g., Collins & Olson, 2014). This region has been shown to be reduced in size in some individuals with DP (Behrmann et al., 2007; Bentin et al., 1999; Garrido et al., 2009), and some DPs have reduced density in the white matter tracts that connect posterior face areas with the anterior temporal lobe and occipital with frontal lobes (Thomas et al., 2009). If impaired perceptual representations of faces are transmitted through this defective pathway, this could further delay or even interrupt the propagation of signals between activated visual face memory representations and stored semantic and episodic information about individual faces (see also Avidan et al., 2014). The anterior temporal face-selective region may serve a dual purpose in the typical face processing system. It may be a key brain area that links high-level perceptual representations of facial identity with post-perceptual semantic and episodic associations about particular individuals. Additionally, this region may also form

part of the core face processing system for the perceptual representation of faces. Dysfunction in the anterior temporal lobe may contribute to both perceptual and post-perceptual face processing deficits in DP.

General discussion

This review has described the present state of research on the cognitive and neural deficits that characterize developmental prosopagnosia. Behaviourally, individuals with DP share common deficits in familiar face recognition and unfamiliar face learning. Many DPs also have difficulties discriminating individual faces during perceptual tasks such as simultaneous face matching. Based on evidence from the face-sensitive N170 component, we have argued that perceptual face processing deficits in DP that emerge during the first 200 ms after encountering a face arise from atypical visual-perceptual face templates. Although these templates are sensitive to global face shape, they are less sensitive to deviations from the canonical face configuration or changes to the contrast polarity of the eye region in DPs relative to control participants. We have also described ERP correlates of impairments in the recognition of famous and newly learned faces in DP. In some DPs, famous faces will activate visual face memory (as reflected by N250 components) without being explicitly recognized (as shown by the absence of P600f components), suggesting a disconnection scenario, where visual face memory traces are not propagated to higher order mechanisms responsible for conscious face recognition. During the recognition of newly learned faces, DPs show delayed N250 and P600f components, demonstrating that the time course of identity-related stages of face processing is altered in DP. Some of these face recognition impairments may be a direct result of earlier perceptual face processing deficits, which are likely to affect the speed with which stored visual representations of individual faces are activated or the precision of these activations. Other deficits, such as the apparent disconnection between visual face memory activations and explicit face recognition, may be linked to reduced anatomical and functional connections between cortical areas that are involved in different aspects of face perception and recognition.

The neuroimaging literature suggests that there may be a posterior-to-anterior gradient of dysfunction in the occipito-temporal face processing network in

DP. More posterior face processing regions often show more typical face-selective activation patterns, while such activations appear to be reduced or entirely absent in the most anterior temporal region. Of course, the mere presence of posterior face-selective regions in DP does not necessarily imply that they operate in the same manner as in individuals with normal face processing. For example, although individuals with DP have readily localizable FFAs, this region may not encode the spatial configuration of facial features in the same way as in control participants. The atypical N170 amplitude modulations discussed earlier suggest that DPs have atypical visual face templates in posterior face processing regions. Our suggestion that face perception deficits in DP arise because visual face templates are less well tuned to canonical upright faces is consistent with a leading neurodevelopmental account of the typical development of cognitive systems. According to this interactive specialization account (Johnson, 2011; Johnson, Grossmann, & Kadosh, 2009), the cognitive development of the face processing system occurs through increases in the number of face-selective neurons and, importantly, decreases in the face responsiveness of other populations of visual neurons across developmental time. As a result, upright faces are processed by specialized face processing mechanisms in adulthood. Inverted faces will also activate the same mechanisms, but because they only loosely fit the canonical face template, they will be processed more like other types of objects and therefore also activate additional neuronal populations that are specialized for processing non-face objects (see Haxby et al., 1999; Pitcher, Walsh, & Duchaine, 2011; Yovel & Kanwisher, 2005, for fMRI and TMS evidence in support of this hypothesis in neurotypical adults). These considerations are in line with the ideas that N170 amplitude enhancements to inverted faces are linked to increases in the response of non-face object-selective neurons to non-canonical face images. If the selectivity of visual processing areas for faces and other non-face object categories was generally less well developed in DP, this could also explain the fact that many (but not all) DPs also show impairments in the recognition of individual exemplars of non-face objects (e.g., Duchaine & Nakayama, 2005). These object recognition problems, which are usually less extreme than the face recognition difficulties in DP, could be due to a reduction in the degree to which object recognition areas in ventral visual cortex are selectively tuned to

particular objects or object categories. A reduction in the functional specialization of these areas would make them less sensitive to visual properties that are diagnostic for particular object categories, resulting in a generic deficit of object recognition.

If DPs show no N170 amplitude modulations in response to inverted versus upright faces or faces with contrast-negative versus positive eye regions because canonical and non-canonical faces both activate object-selective neurons, this would reflect a lower degree of neural specialization for standard upright faces in DP. Importantly, typical face inversion and contrast reversal effects on the N170 component are not present during early childhood, but only emerge over developmental time (Itier & Taylor, 2004a; Taylor, Batty, & Itier, 2004). In fact, the atypical N170 modulations caused by face inversion in adults with DP appear very similar to the normal N170 inversion effects found in children under the age of 11 (see Towler & Eimer, 2012, for further discussion), suggesting that functional specialization in the occipito-temporal pathway may only be partial in DP. Even though the number of face-selective neural populations in the posterior occipito-temporal face selective regions may increase over developmental time in DP, this increase may not be accompanied by a corresponding decrease in the responsiveness of other object-selective populations to upright faces. As a result, genuinely face-selective and more generic object-selective neural populations will compete for the processing of upright faces, resulting in the formation of less efficient representations of those visual properties of individual faces that are relevant for their subsequent recognition. This neurodevelopmental interpretation suggests that DP may be the result of a general developmental delay in the occipito-temporal pathway, but the question remains as to which factors may be responsible for this delay.

According to the interactive specialization account (Johnson, 2011; see also Karmiloff-Smith, 1998), the developmental trajectory of cognitive systems is strongly affected by perceptual experience and the history of interactions of these systems with the sensory environment. Face recognition abilities are largely dependent on visual experience with faces. Individuals who are deprived of patterned visual input during the first six months of life due to congenital cataracts do not develop holistic face processing, despite years of experience with faces after surgery to remove the cataracts (Le Grand, Mondloch, Maurer, & Brent, 2001, 2003, 2004). Importantly, such individuals

also appear to lack face-sensitive N170 components, strongly suggesting that early visual experience with faces or face-like stimuli is critical for the formation of a typical occipito-temporal face network (Röder, Ley, Shenoy, Kekunnaya, & Bottari, 2013). Along similar lines, the other-race effect (a recognition impairment for individual other-race faces) emerges gradually during the first year of life (Kelly et al., 2009; Sangrigoli & de Schonen, 2004). Such observations strongly suggest that visual experience in childhood, especially during a critical period in the first year of life, shapes the processing of facial properties that are relevant for the recognition and discrimination of individual faces. The impairments of face perception and recognition processes in individuals with DP may therefore be linked to atypical early patterns of interaction with their visual environment. In order to develop normal canonical face templates, faces must be fixated frequently, in a stereotyped manner, and with more fixations to the eye region than to other face regions. Unimpaired individuals usually move their gaze towards human faces in naturalistic visual scenes (e.g., Yarbus, 1967) and often fixate on the eye region or just below the eyes (Hsiao & Cottrell, 2008). The visual properties within this eye region are preserved under different naturalistic image transformations such as the direction of illumination on the face (Sinha, 2002), and this statistical visual regularity may be especially important for the development of canonical perceptual face templates that are tuned for the detection and recognition of faces across naturalistic changes in the retinal image. There is some evidence that adults with DP do indeed scan faces in an atypical fashion during face recognition (Schwarzer et al., 2007). Patterns of gaze fixations during visual exploration of faces in this group of DPs showed more diffuse scan paths, with fewer and shorter fixations on internal facial features and more reliance on external facial features such as the hair. A case study of a four-year-old child with suspected DP revealed similar atypical gaze patterns during face recognition (Schmalzl, Palermo, Green, Brunsdon, & Coltheart, 2008; for a similar case, see: Pizzamiglio et al., 2015). Face identity training reduced the face recognition impairments, and these improvements were accompanied by an increase in normal gaze fixation patterns and an increased focus on the eye region during face recognition.

A different link between eye contact, face recognition performance, and DP was suggested by studies investigating the neuropeptide oxytocin.

Intranasal inhalation of oxytocin has been shown to increase the number of fixations and total gaze time spent on the eye region of faces in neurotypical participants (Guastella, Mitchell, & Dadds, 2008). Importantly, intranasal inhalation of oxytocin improved face learning and recognition performance in DPs, but not in control participants (Bate et al., 2014). Links between oxytocin and parent–infant bonding during the first year of life are well established (e.g., Donaldson & Young, 2008; Lim & Young, 2006). Because early visual experience is crucial for the development of normal face perception and recognition, such links suggest that factors such as eye contact and the pattern of gaze fixations on a face may play a crucial role in shaping the early visual experience responsible for atypical perceptual face templates in DP.

Such links between the face processing deficits in DP and an atypical processing of the eye region during early development may explain why individuals with DP appear to have a disproportionate impairment in the ability to use the canonical spatial configuration and contrast polarity of the eye region to process the eyes within the context of the whole face (DeGutis, Cohan et al., 2012; Fisher et al., *in press*; Towler et al., 2012). Holistic processing of the eye region is present in typically developing children from as young as four years of age (Pellicano & Rhodes, 2003) and may emerge during the first year of life (Schwarzer & Zauner, 2003). Specific sensitivity of cortical responses to the contrast polarity of the eye region also emerges during the first year of life (Otsuka et al., 2013). Given the importance of signals from the eye region for face recognition (e.g., Peterson & Eckstein, 2012) and the development of canonical upright face templates, an atypical pattern of interacting with visual information from the eyes during early development may result in long-term changes in the architecture of the face processing system, which can produce some of the differences in face perception and recognition between DPs and controls that were discussed in this review.

From a neurodevelopmental perspective, dissociations between impaired and apparently normal functions within the same cognitive domain are particularly important, because they can provide insights into the factors that determine particular trajectories within different sub-systems during development. As discussed previously, there appears to be a dissociation between the processing of facial identity and emotional facial expression in DP. Similar to

neurotypical participants, individuals with DP show enhanced neural responses to fearful as compared to neutral faces over medial frontal electrodes with an onset of 120 ms after encountering a face. This contrast between the apparently intact rapid detection of emotional expression and the impairment of face perception and identity recognition in DP suggests that the normal development of detecting and evaluating salient emotional signals is not dependent upon the entirely typical operation of other parts of the face processing network. Although perceptual face representations in DPs lack sensitivity to the eye region and the spatial configuration of face parts, this does not result in impairments in the processing and recognition of facial expression. Such a dissociation suggests that in contrast to the processing of facial identity, the analysis of emotional expression is less sensitive to the relative locations of face parts or to the contrast of the eye region (see Bombari et al., 2013; Savage & Lipp, 2015, for behavioural evidence that emotion recognition can be independent of configural cues). In line with this suggestion, neuroimaging studies have shown that the face-selective STS represents facial emotions in a part-based rather than holistic fashion (Flack et al., 2015), and that contrast inversion reduces neural responses in identity-selective but not in emotion-selective regions of the posterior face processing network (Harris, Young, & Andrews, 2014). Such differences between identity and emotion processing systems in their sensitivity to particular facial properties may be a principal reason why emotional expression recognition can develop normally in a face processing system that is poorly tuned for identity recognition.

The focus on the neurodevelopmental trajectory of the face processing system in DP, as proposed by the interactive specialization account, does not rule out additional contributions of genetic factors, which may influence domain-specific face processing mechanisms directly, or domain-relevant neural systems indirectly. It is sometimes assumed that genetic and developmental explanations are mutually exclusive. This would imply that atypical visual face templates in DP have to be interpreted as the result either of innate factors or of atypical developmental trajectories. In the former case, a genetically determined inability to form canonical representations of faces and facial configurations would result in the perceptual face processing deficits described above. In the latter case, a failure to interact with faces in the typical way during cognitive development would

lead to poor visual face templates, which is then reflected by atypical perceptual face processing in adulthood. We believe that such unidirectional causal explanations of the origin of DP that focus exclusively on genetics or on development are not particularly helpful. As in other neurodevelopmental disorders, genetic and experience-based developmental factors will inevitably interact and thus contribute jointly to the pattern of face recognition problems in adults with DP. For example, oxytocin expression in the brain is regulated by an oxytocin receptor gene, and variations in this gene have been shown to produce systematic differences in face recognition ability (Skuse et al., 2014). This observation illustrates the point that heritable factors can influence a specific cognitive ability through indirect routes, such as the expression of a neuropeptide that regulates social motivation and social behaviours such as eye contact. Other genetic factors may contribute more directly to the impairments of specialized face processing mechanisms in the occipital and temporal lobes (see Susilo & Duchaine, 2013, for the suggestion of genetic influences on neuronal migration during early prenatal development in DP). In addition, individual differences in the preferential allocation of attention to particular objects in visual scenes may also have a genetic basis. For example, the fact that newborn infants preferentially orient towards face-like visual patterns (Farroni et al., 2005; Johnson, Dziurawiec, Ellis, & Morton, 1991) may be sufficient to prioritize faces and more specifically the eye region during early development. Atypical visual orienting towards faces during infancy may be a genetically determined domain-specific factor that could cause atypical visual perceptual face templates in adults with DP. Importantly, genetic and developmental factors should not be seen as mutually exclusive contributors to DP. It is likely that these two types of factors will often interact. For example, an atypical pattern of eye contact during early development that is associated with a particular genetically influenced level of oxytocin expression may contribute to the development of impaired canonical upright face templates and can eventually result in a reduced perceptual ability to discriminate between individual upright faces in an adult with DP. Along similar lines, small individual differences in the pattern of visual-perceptual interactions with faces during early development may or may not produce an atypical development of the face processing system and subsequent face recognition impairments, depending on the existence of other

specific and presumably genetically determined vulnerabilities within this system.

It is sometimes assumed that the aetiology of DP reflects a linear progression from genetics to brain structure within a domain-specific face processing system, to the functional specialization within this domain-specific network and the resulting pattern of cognitive ability and impairment. However, this unidirectional view is unlikely to provide a satisfactory account of the factors that contribute to DP. According to the alternative interactive view proposed here, DP is a neurodevelopmental disorder that reflects the joint contribution of heritable and experience-dependent factors and needs to be understood in the context of its particular developmental trajectory. In this review, we have presented evidence for specific impairments of cortical function at multiple temporal and anatomical processing stages in the face recognition system in adults with DP and have suggested that the specific nature of these impairments are most likely the long-term consequence of early atypical visual experience with faces.

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References

- Adolphs, R. (2002). Neural systems for recognizing emotion. *Current Opinion in Neurobiology*, 12(2), 169–177. doi:10.1016/S0959-4388(02)00301-X
- Adolphs, R. (2003). Cognitive neuroscience: Cognitive neuroscience of human social behaviour. *Nature Reviews Neuroscience*, 4(3), 165–178. doi:10.1038/nrn1056
- Anzellotti, S., Fairhall, S. L., & Caramazza, A. (2013). Decoding representations of face identity that are tolerant to rotation. *Cerebral Cortex*, 24(8), 1988–1995. doi:10.1093/cercor/bht046
- Avidan, G., & Behrmann, M. (2009). Functional MRI reveals compromised neural integrity of the face processing network in congenital prosopagnosia. *Current Biology*, 19(13), 1146–1150. doi:10.1016/j.cub.2009.04.060
- Avidan, G., Hasson, U., Malach, R., & Behrmann, M. (2005). Detailed exploration of face-related processing in congenital prosopagnosia: 2. *Functional neuroimaging findings*. *Journal of Cognitive Neuroscience*, 17(7), 1150–1167. doi:10.1162/0898929054475145
- Avidan, G., Tanzer, M., & Behrmann, M. (2011). Impaired holistic processing in congenital prosopagnosia. *Neuropsychologia*, 49(9), 2541–2552. doi:10.1016/j.neuropsychologia.2011.05.002
- Avidan, G., Tanzer, M., Hadj-Bouziane, F., Liu, N., Ungerleider, L. G., & Behrmann, M. (2014). Selective dissociation between core and extended regions of the face processing network in congenital prosopagnosia. *Cerebral Cortex*, 24(6), 1565–1578. doi:10.1093/cercor/bht007
- Baron-Cohen, S., Wheelwright, S., Hill, J., Raste, Y., & Plumb, I. (2001). The “Reading the Mind in the Eyes” test revised version: A study with normal adults, and adults with Asperger syndrome or high-functioning Autism. *Journal of Child Psychology and Psychiatry*, 42(2), 241–251. doi:10.1017/S0021963001006643
- Barton, J. J. S. (2008). Structure and function in acquired prosopagnosia: Lessons from a series of 10 patients with brain damage. *Journal of Neuropsychology*, 2(1), 197–225. doi:10.1348/174866407X214172
- Baseler, H. A., Harris, R. J., Young, A. W., & Andrews, T. J. (2013). Neural responses to expression and gaze in the posterior superior temporal sulcus interact with facial identity. *Cerebral Cortex*, 24(3), 737–744. doi:10.1093/cercor/bhs360
- Bate, S., Cook, S. J., Duchaine, B., Tree, J. J., Burns, E. J., & Hodgson, T. L. (2014). Intranasal inhalation of oxytocin improves face processing in developmental prosopagnosia. *Cortex*, 50, 55–63. doi:10.1016/j.cortex.2013.08.006
- Behrmann, M., Avidan, G., Gao, F., & Black, S. (2007). Structural imaging reveals anatomical alterations in inferotemporal cortex in congenital prosopagnosia. *Cerebral Cortex*, 17(10), 2354–2363. doi:10.1093/cercor/bhl144
- Behrmann, M., Avidan, G., Marotta, J. J., & Kimchi, R. (2005). Detailed exploration of face-related processing in congenital prosopagnosia: 1. *Behavioral Findings*. *Journal of Cognitive Neuroscience*, 17(7), 1130–1149. doi:10.1162/0898929054475154
- Bentin, S., Allison, T., Puce, A., Perez, E., & McCarthy, G. (1996). Electrophysiological studies of face perception in humans. *Journal of Cognitive Neuroscience*, 8(6), 551–565. doi:10.1162/jocn.1996.8.6.551
- Bentin, S., DeGutis, J. M., D’Esposito, M., & Robertson, L. C. (2007). Too many trees to see the forest: Performance, event-related potential, and functional magnetic resonance imaging manifestations of integrative congenital prosopagnosia. *Journal of Cognitive Neuroscience*, 19(1), 132–146. doi:10.1162/jocn.2007.19.1.132
- Bentin, S., & Deouell, L. Y. (2000). Structural encoding and identification in face processing: ERP evidence for separate mechanisms. *Cognitive Neuropsychology*, 17(1–3), 35–55. doi:10.1080/026432900380472
- Bentin, S., Deouell, L. Y., & Soroker, N. (1999). Selective visual streaming in face recognition: Evidence from developmental prosopagnosia. *Neuroreport*, 10(4), 823–827. doi:10.1097/00001756-199903170-00029
- Bodamer, J. (1947). Die Prosop-Agnosie: Die Agnosie des Physiognomieerkenntens. *Archiv für Psychiatrie und Nervenkrankheiten Vereinigt mit Zeitschrift für die Gesamte Neurologie und Psychiatrie*, 179, 6–53.
- Bombardi, D., Schmid, P. C., Schmid Mast, M., Birri, S., Mast, F. W., & Lobmaier, J. S. (2013). Emotion recognition: The role of

- featural and configural face information. *The Quarterly Journal of Experimental Psychology*, 66(12), 2426–2442. doi:10.1080/17470218.2013.789065
- Bötzel, K., Schulze, S., & Stodieck, S. G. (1995). Scalp topography and analysis of intracranial sources of face-evoked potentials. *Experimental Brain Research*, 104(1), 135–143. doi:10.1007/BF00229863
- Breen, N., Caine, D., & Coltheart, M. (2000). Models of face recognition and delusional misidentification: A critical review. *Cognitive Neuropsychology*, 17(1–3), 55–71. doi:10.1080/026432900380481
- Bruce, V., & Young, A. (1986). Understanding face recognition. *British Journal of Psychology*, 77, 305–327. doi:10.1111/j.2044-8295.1986.tb02199.x
- Bruyer, R., Laterre, C., Seron, X., Feyereisen, P., Strypstein, E., Pierrard, E., & Rectem, D. (1983). A case of prosopagnosia with some preserved covert remembrance of familiar faces. *Brain and Cognition*, 2(3), 257–284. doi:10.1016/0278-2626(83)90014-3
- Burton, A. M., Bruce, V., & Johnston, R. A. (1990). Understanding face recognition with an interactive activation model. *British Journal of Psychology*, 81(3), 361–380. doi:10.1111/j.2044-8295.1990.tb02367.x
- Burton, A. M., Young, A. W., Bruce, V., Johnston, R. A., & Ellis, A. W. (1991). Understanding covert recognition. *Cognition*, 39(2), 129–166. doi:10.1016/0010-0277(91)90041-2
- Busigny, T., Van Belle, G., Jemel, B., Hosein, A., Joubert, S., & Rossion, B. (2014). Face-specific impairment in holistic perception following focal lesion of the right anterior temporal lobe. *Neuropsychologia*, 56, 312–333. doi:10.1016/j.neuropsychologia.2014.01.018
- Calder, A. J., & Young, A. W. (2005). Understanding the recognition of facial identity and facial expression. *Nature Reviews Neuroscience*, 6(8), 641–651. doi:10.1038/nrn1724
- Carbon, C.-C., Grüter, T., Grüter, M., Weber, J. E., & Lueschow, A. (2010). Dissociation of facial attractiveness and distinctiveness processing in congenital prosopagnosia. *Visual Cognition*, 18(5), 641–654. doi:10.1080/13506280903462471
- Chatterjee, G., & Nakayama, K. (2012). Normal facial age and gender perception in developmental prosopagnosia. *Cognitive Neuropsychology*, 29(5–6), 482–502. doi:10.1080/02643294.2012.756809
- Collins, J. A., & Olson, I. R. (2014). Beyond the FFA: The role of the ventral anterior temporal lobes in face processing. *Neuropsychologia*, 61, 65–79. doi:10.1016/j.neuropsychologia.2014.06.005
- Dalrymple, K. A., Oruç, I., Duchaine, B., Pancaroglu, R., Fox, C. J., Iaria, G., ... Barton, J. J. S. (2011). The anatomic basis of the right face-selective N170 in acquired prosopagnosia: A combined ERP/fMRI study. *Neuropsychologia*, 49(9), 2553–2563. doi:10.1016/j.neuropsychologia.2011.05.003
- DeGutis, J., Chatterjee, G., Mercado, R. J., & Nakayama, K. (2012). Face gender recognition in developmental prosopagnosia: Evidence for holistic processing and use of configural information. *Visual Cognition*, 20(10), 1242–1253. doi:10.1080/13506285.2012.744788
- DeGutis, J., Cohan, S., Mercado, R. J., Wilmer, J., & Nakayama, K. (2012). Holistic processing of the mouth but not the eyes in developmental prosopagnosia. *Cognitive Neuropsychology*, 29(5–6), 419–446. doi:10.1080/02643294.2012.754745
- De Haan, E. H. F., & Campbell, R. (1991). A fifteen year follow-up of a case of developmental prosopagnosia. *Cortex*, 27(4), 489–509. doi:10.1016/S0010-9452(13)80001-9
- Diamond, B. J., Valentine, T., Mayes, A. R., & Sandel, M. E. (1994). Evidence of covert recognition in a prosopagnosic patient. *Cortex*, 30(3), 377–393. doi:10.1016/S0010-9452(13)80336-X
- Dinkelacker, V., Grüter, M., Klaver, P., Grüter, T., Specht, K., Weis, S., ... Fernandez, G. (2011). Congenital prosopagnosia: Multistage anatomical and functional deficits in face processing circuitry. *Journal of Neurology*, 258(5), 770–782. doi:10.1007/s00415-010-5828-5
- Dobel, C., Bölte, J., Aicher, M., & Schweinberger, S. R. (2007). Prosopagnosia without apparent cause: Overview and diagnosis of six cases. *Cortex*, 43(6), 718–733. doi:10.1016/S0010-9452(08)70501-X
- Donaldson, Z. R., & Young, L. J. (2008). Oxytocin, vasopressin, and the neurogenetics of sociality. *Science*, 322(5903), 900–904. doi:10.1126/science.1158668
- Duchaine, B. (2011). Developmental prosopagnosia: Cognitive, neural, and developmental investigations. In A. J. Calder et al. (Eds.), *The Oxford handbook of face perception* (pp. 821–838). Oxford, UK: Oxford University Press.
- Duchaine, B. C., Parker, H., & Nakayama, K. (2003). Normal recognition of emotion in a prosopagnosic. *Perception*, 32(7), 827–838. doi:10.1068/p5067
- Duchaine, B. C., Yovel, G., Butterworth, E. J., & Nakayama, K. (2006). Prosopagnosia as an impairment to face-specific mechanisms: Elimination of the alternative hypotheses in a developmental case. *Cognitive Neuropsychology*, 23(5), 714–747. doi:10.1080/02643290500441296
- Duchaine, B., Germine, L., & Nakayama, K. (2007). Family resemblance: Ten family members with prosopagnosia and within-class object agnosia. *Cognitive Neuropsychology*, 24(4), 419–430. doi:10.1080/02643290701380491
- Duchaine, B., Murray, H., Turner, M., White, S., & Garrido, L. (2009). Normal social cognition in developmental prosopagnosia. *Cognitive Neuropsychology*, 26(7), 620–634. doi:10.1080/02643291003616145
- Duchaine, B., & Nakayama, K. (2005). Dissociations of face and object recognition in developmental prosopagnosia. *Journal of Cognitive Neuroscience*, 17(2), 249–261. doi:10.1162/0898929053124857
- Duchaine, B., & Nakayama, K. (2006). The Cambridge face memory test: Results for neurologically intact individuals and an investigation of its validity using inverted face stimuli and prosopagnosic participants. *Neuropsychologia*, 44(4), 576–585. doi:10.1016/j.neuropsychologia.2005.07.001
- Duchaine, B., Yovel, G., & Nakayama, K. (2007). No global processing deficit in the Navon task in 14 developmental prosopagnosics. *Social, Cognitive, and Affective Neuroscience*, 2(2), 104–113. doi:10.1093/scan/nsm003
- Eimer, M. (2000a). Effects of face inversion on the structural encoding and recognition of faces: Evidence from event-related brain potentials. *Cognitive Brain Research*, 10(1–2), 145–158. doi:10.1016/S0926-6410(00)00038-0
- Eimer, M. (2000b). Event-related brain potentials distinguish processing stages involved in face perception and recognition. *Clinical Neurophysiology*, 111(4), 694–705. doi:10.1016/S1388-2457(99)00285-0

- Eimer, M., Gosling, A., & Duchaine, B. (2012). Electrophysiological markers of covert face recognition in developmental prosopagnosia. *Brain*, 135(Pt 2), 542–554. doi:10.1093/brain/awr347
- Eimer, M., Gosling, A., Nicholas, S., & Kiss, M. (2011). The N170 component and its links to configural face processing: A rapid neural adaptation study. *Brain Research*, 1376, 76–87. doi:10.1016/j.brainres.2010.12.046
- Eimer, M., & Holmes, A. (2002). An ERP study on the time course of emotional face processing. *Neuroreport*, 13(4), 427–431. doi:10.1097/00001756-200203250-00013
- Eimer, M., Holmes, A., & McGlone, F. P. (2003). The role of spatial attention in the processing of facial expression: An ERP study of rapid brain responses to six basic emotions. *Cognitive, Affective, & Behavioral Neuroscience*, 3(2), 97–110. doi:10.3758/CABN.3.2.97
- Eimer, M., Kiss, M., & Nicholas, S. (2010). Response profile of the face-sensitive N170 component: A rapid adaptation study. *Cerebral Cortex*, 20(10), 2442–2452. doi:10.1093/cercor/bhp312
- Ellis, A. W., Young, A. W., & Critchley, E. M. (1989). Loss of memory for people following temporal lobe damage. *Brain: A Journal of Neurology*, 112(6), 1469–1483. doi:10.1093/brain/112.6.1469
- Farroni, T., Johnson, M. H., Menon, E., Züljan, L., Faraguna, D., & Csibra, G. (2005). Newborns' preference for face-relevant stimuli: Effects of contrast polarity. *Proceedings of the National Academy of Sciences of the United States of America*, 102(47), 17245–17250.
- Fisher, K., Towler, J., & Eimer, M. (2015). Effects of contrast inversion on face perception depend on gaze location: Evidence from the N170 component. *Cognitive Neuroscience*, 1–10. doi:10.1080/17588928.2015.1053441
- Fisher, K., Towler, J., & Eimer, M. (in press). The face-sensitive N170 component reveals that developmental prosopagnosics are not sensitive to the contrast polarity of the eyes.
- Flack, T. R., Andrews, T. J., Hymers, M., Al-Mosaiwi, M., Marsden, S. P., Strachan, J. W., ... & Young, A. W. (2015). Responses in the right posterior superior temporal sulcus show a feature-based response to facial expression. *Cortex*, 69, 14–23. doi:10.1016/j.cortex.2015.03.002
- Fox, C. J., Hanif, H. M., Iaria, G., Duchaine, B. C., & Barton, J. J. S. (2011). Perceptual and anatomic patterns of selective deficits in facial identity and expression processing. *Neuropsychologia*, 49(12), 3188–3200. doi:10.1016/j.neuropsychologia.2011.07.018
- Fox, C., Moon, S., Iaria, G., & Barton, J. (2009). The correlates of subjective perception of identity and expression in the face network: An fMRI adaptation study. *NeuroImage*, 44(2), 569–580. doi:10.1016/j.neuroimage.2008.09.011
- Freiwald, W. A., Tsao, D. Y., & Livingstone, M. S. (2009). A face feature space in the macaque temporal lobe. *Nature Neuroscience*, 12(9), 1187–1196. doi:10.1038/nn.2363
- Furl, N., Garrido, L., Dolan, R. J., Driver, J., & Duchaine, B. (2011). Fusiform gyrus face selectivity relates to individual differences in facial recognition ability. *Journal of Cognitive Neuroscience*, 23(7), 1723–1740. doi:10.1162/jocn.2010.21545
- Gainotti, G. (2013). Is the right anterior temporal variant of prosopagnosia a form of "Associative Prosopagnosia" or a form of "Multimodal Person Recognition Disorder"? *Neuropsychology Review*, 23(2), 99–110. doi:10.1007/s11065-013-9232-7
- Gainotti, G., Barbier, A., & Marra, C. (2003). Slowly progressive defect in recognition of familiar people in a patient with right anterior temporal atrophy. *Brain*, 126(4), 792–803. doi:10.1093/brain/awg092
- Galper, R. E. (1970). Recognition of faces in photographic negative. *Psychonomic Science*, 19(4), 207–208. doi:10.3758/BF03328777
- Ganel, T., Valyear, K., Goshen-Gottstein, Y., & Goodale, M. A. (2005). Greater fMRI activation in the "fusiform face area" for the processing of expression than the processing of identity: Implications for face-recognition models. *Journal of Vision*, 4(8), 135–135. doi:10.1167/4.8.135
- Garrido, L., Duchaine, B., & Nakayama, K. (2008). Face detection in normal and prosopagnosic individuals. *Journal of Neuropsychology*, 2(1), 119–140. doi:10.1348/174866407X246843
- Garrido, L., Furl, N., Draganski, B., Weiskopf, N., Stevens, J., Tan, G. C.-Y., ... Duchaine, B. (2009). Voxel-based morphometry reveals reduced grey matter volume in the temporal cortex of developmental prosopagnosics. *Brain*, 132(Pt 12), 3443–3455. doi:10.1093/brain/awp271
- George, N., Dolan, R. J., Fink, G. R., Baylis, G. C., Russell, C., & Driver, J. (1999). Contrast polarity and face recognition in the human fusiform gyrus. *Nature Neuroscience*, 2(6), 574–580. doi:10.1038/9230
- Gilad, S., Meng, M., & Sinha, P. (2009). Role of ordinal contrast relationships in face encoding. *Proceedings of the National Academy of Sciences of the United States of America*, 106(13), 5353–5358. doi:10.1073/pnas.0812396106
- Gobbini, M. I., & Haxby, J. V. (2007). Neural systems for recognition of familiar faces. *Neuropsychologia*, 45(1), 32–41. doi:10.1016/j.neuropsychologia.2006.04.015
- Gosling, A., & Eimer, M. (2011). An event-related brain potential study of explicit face recognition. *Neuropsychologia*, 49(9), 2736–2745. doi:10.1016/j.neuropsychologia.2011.05.025
- Guastella, A. J., Mitchell, P. B., & Dadds, M. R. (2008). Oxytocin increases gaze to the eye region of human faces. *Biological Psychiatry*, 63(1), 3–5. doi:10.1016/j.biopsych.2007.06.026
- Harmer, C. J., Thilo, K. V., Rothwell, J. C., & Goodwin, G. M. (2001). Transcranial magnetic stimulation of medial-frontal cortex impairs the processing of angry facial expressions. *Nature Neuroscience*, 4, 17–18.
- Harris, A. M., Duchaine, B. C., & Nakayama, K. (2005). Normal and abnormal face selectivity of the M170 response in developmental prosopagnosics. *Neuropsychologia*, 43(14), 2125–2136. doi:10.1016/j.neuropsychologia.2005.03.017
- Harris, R. J., Young, A. W., & Andrews, T. J. (2014). Brain regions involved in processing facial identity and expression are differentially selective for surface and edge information. *NeuroImage*, 97, 217–223. doi:10.1016/j.neuroimage.2014.04.032
- Hasson, U., Avidan, G., Deouell, L. Y., Bentin, S., & Malach, R. (2003). Face-selective activation in a congenital prosopagnosic subject. *Journal of Cognitive Neuroscience*, 15(3), 419–431. doi:10.1162/089892903321593135
- Haxby, J. V., Hoffman, E. A., & Gobbini, M. I. (2000). The distributed human neural system for face perception. *Trends in Cognitive Sciences*, 4(6), 223–233. doi:10.1016/S1364-6613(00)01482-0
- Haxby, J. V., Hoffman, E. A., & Gobbini, M. I. (2002). Human neural systems for face recognition and social communication. *Biological Psychiatry*, 51(1), 59–67. doi:10.1016/S0006-3223(01)01330-0
- Haxby, J. V., Ungerleider, L. G., Clark, V. P., Schouten, J. L., Hoffman, E. A., & Martin, A. (1999). The effect of face inversion on activity in human neural systems for face and object perception. *Neuron*, 22(1), 189–199. doi:10.1016/S0896-6273(00)80690-X

- Hole, G. J. (1994). Configurational factors in the perception of unfamiliar faces. *Perception*, 23(1), 65–74. doi:10.1068/p230065
- Hsiao, J. H., & Cottrell, G. (2008). Two fixations suffice in face recognition. *Psychological Science*, 19(10), 998–1006. doi:10.1111/j.1467-9280.2008.02191.x
- Humphreys, K., Avidan, G., & Behrmann, M. (2007). A detailed investigation of facial expression processing in congenital prosopagnosia as compared to acquired prosopagnosia. *Experimental Brain Research*, 176(2), 356–373. doi:10.1007/s00221-006-0621-5
- Itier, R. J., Latinus, M., & Taylor, M. J. (2006). Face, eye and object early processing: What is the face specificity? *NeuroImage*, 29(2), 667–676. doi:10.1016/j.neuroimage.2005.07.041
- Itier, R. J., & Taylor, M. J. (2002). Inversion and contrast polarity reversal affect both encoding and recognition processes of unfamiliar faces: A repetition study using ERPs. *NeuroImage*, 15(2), 353–372. doi:10.1006/nimg.2001.0982
- Itier, R. J., & Taylor, M. J. (2004a). Face recognition memory and configural processing: A developmental ERP study using upright, inverted, and contrast-reversed faces. *Journal of Cognitive Neuroscience*, 16(3), 487–502. doi:10.1162/089892904322926818
- Itier, R. J., & Taylor, M. J. (2004b). Source analysis of the N170 to faces and objects. *Neuroreport*, 15(8), 1261–1265. doi:10.1097/01.wnr.0000127827.73576.d8
- Jacques, C., & Rossion, B. (2010). Misaligning face halves increases and delays the N170 specifically for upright faces: Implications for the nature of early face representations. *Brain Research*, 1318, 96–109. doi:10.1016/j.brainres.2009.12.070
- Johnson, M. H. (2011). Interactive specialization: A domain-general framework for human functional brain development? *Developmental Cognitive Neuroscience*, 1(1), 7–21.
- Johnson, M. H., Dziurawiec, S., Ellis, H., & Morton, J. (1991). Newborns' preferential tracking of face-like stimuli and its subsequent decline. *Cognition*, 40(1), 1–19. doi:10.1016/0010-0277(91)90045-6
- Johnson, M. H., Grossmann, T., & Kadosh, K. C. (2009). Mapping functional brain development: Building a social brain through interactive specialization. *Developmental Psychology*, 45(1), 151–159. doi:10.1037/a0014548
- Johnston, A., Hill, H., & Carman, N. (1992). Recognising faces: Effects of lighting direction, inversion, and brightness reversal. *Perception*, 21(3), 365–375. doi:10.1068/p210365
- Jonas, J., Desclois, M., Koessler, L., Colnat-Coulbois, S., Sauvée, M., Guye, M., ... Maillard, L. (2012). Focal electrical intracerebral stimulation of a face-sensitive area causes transient prosopagnosia. *Neuroscience*, 222, 281–288. doi:10.1016/j.neuroscience.2012.07.021
- Jonas, J., Rossion, B., Brissart, H., Frismand, S., Jacques, C., Hossu, G., ... Maillard, L. (2015). Beyond the core face-processing network: Intracerebral stimulation of a face-selective area in the right anterior fusiform gyrus elicits transient prosopagnosia. *Cortex*, 72, 140–155. doi:10.1016/j.cortex.2015.05.026
- Kanwisher, N., & Barton, J. J. S. (2011). The functional architecture of the face system: Integrating evidence from fMRI and patient studies. In A. J. Calder (Ed.), *The Oxford handbook of face perception* (pp. 111–129). Oxford, UK: Oxford University Press.
- Kanwisher, N., McDermott, J., & Chun, M. M. (1997). The fusiform face area: A module in human extrastriate cortex specialized for face perception. *The Journal of Neuroscience*, 17(11), 4302–4311.
- Karmiloff-Smith, A. (1998). Development itself is the key to understanding developmental disorders. *Trends in Cognitive Sciences*, 2(10), 389–398. doi:10.1016/S1364-6613(98)01230-3
- Kelly, D. J., Liu, S., Lee, K., Quinn, P. C., Pascalis, O., Slater, A. M., & Ge, L. (2009). Development of the other-race effect during infancy: Evidence toward universality? *Journal of Experimental Child Psychology*, 104(1), 105–114. doi:10.1016/j.jecp.2009.01.006
- Kennerknecht, I., Grueter, T., Welling, B., Wentzek, S., Horst, J., Edwards, S., & Grueter, M. (2006). First report of prevalence of non-syndromic hereditary prosopagnosia (HPA). *American Journal of Medical Genetics Part A*, 140A(15), 1617–1622. doi:10.1002/ajmg.a.31343
- Kennerknecht, I., Pluemp, N., & Welling, B. (2008). Congenital prosopagnosia—a common hereditary cognitive dysfunction in humans. *Frontiers in Bioscience*, 13, 3150–3158.
- Kloth, N., Itier, R. J., & Schweinberger, S. R. (2013). Combined effects of inversion and feature removal on N170 responses elicited by faces and car fronts. *Brain and Cognition*, 81(3), 321–328. doi:10.1016/j.bandc.2013.01.002
- Kress, T., & Daum, I. (2003). Event-related potentials reflect impaired face recognition in patients with congenital prosopagnosia. *Neuroscience Letters*, 352(2), 133–136. doi:10.1016/j.neulet.2003.08.047
- Kriegeskorte, N., Formisano, E., Sorger, B., & Goebel, R. (2007). Individual faces elicit distinct response patterns in human anterior temporal cortex. *Proceedings of the National Academy of Sciences of the United States of America*, 104(51), 20600–20605. doi:10.1073/pnas.0705654104
- Lee, Y., Duchaine, B., Wilson, H. R., & Nakayama, K. (2010). Three cases of developmental prosopagnosia from one family: Detailed neuropsychological and psychophysical investigation of face processing. *Cortex*, 46(8), 949–964. doi:10.1016/j.cortex.2009.07.012
- Le Grand, R., Cooper, P. A., Mondloch, C. J., Lewis, T. L., Sagiv, N., de Gelder, B., & Maurer, D. (2006). What aspects of face processing are impaired in developmental prosopagnosia? *Brain and Cognition*, 61(2), 139–158. doi:10.1016/j.bandc.2005.11.005
- Le Grand, R., Mondloch, C. J., Maurer, D., & Brent, H. P. (2001). Neuroperception: Early visual experience and face processing. *Nature*, 410(6831), 890–890. doi:10.1038/35073749
- Le Grand, R., Mondloch, C. J., Maurer, D., & Brent, H. P. (2003). Expert face processing requires visual input to the right hemisphere during infancy. *Nature Neuroscience*, 6, 1108–1112. doi:10.1038/nn1121
- Le Grand, R., Mondloch, C. J., Maurer, D., & Brent, H. P. (2004). Impairment in holistic face processing following early visual deprivation. *Psychological Science*, 15(11), 762–768. doi:10.1111/j.0956-7976.2004.00753.x
- Letourneau, S. M., & Mitchell, T. V. (2008). Behavioral and ERP measures of holistic face processing in a composite task. *Brain and Cognition*, 67(2), 234–245. doi:10.1016/j.bandc.2008.01.007
- Lim, M. M., & Young, L. J. (2006). Neuropeptidergic regulation of affiliative behavior and social bonding in animals. *Hormones and Behavior*, 50(4), 506–517. doi:10.1016/j.yhbeh.2006.06.028
- Liu, T. T., & Behrmann, M. (2014). Impaired holistic processing of left-right composite faces in congenital prosopagnosia. *Frontiers in Human Neuroscience*, 8. doi:10.3389/fnhum.2014.00750

- Martin, A., & Chao, L. L. (2001). Semantic memory and the brain: Structure and processes. *Current Opinion in Neurobiology*, 11(2), 194–201. doi:10.1016/S0959-4388(00)00196-3
- Mattavelli, G., Cattaneo, Z., & Papagno, C. (2011). Transcranial magnetic stimulation of medial prefrontal cortex modulates face expressions processing in a priming task. *Neuropsychologia*, 49(5), 992–998. doi:10.1016/j.neuropsychologia.2011.01.038
- Maurer, D., Le Grand, R., & Mondloch, C. J. (2002). The many faces of configural processing. *Trends in Cognitive Sciences*, 6(6), 255–260. doi:10.1016/S1364-6613(02)01903-4
- Meadows, J. C. (1974). The anatomical basis of prosopagnosia. *Journal of Neurology, Neurosurgery, and Psychiatry*, 37(5), 489–501. doi:10.1136/jnnp.37.5.489
- Meyers, E. M., Borzello, M., Freiwald, W. A., & Tsao, D. (2015). Intelligent information loss: The coding of facial identity, head pose, and non-face information in the macaque face patch system. *The Journal of Neuroscience*, 35(18), 7069–7081. doi:10.1523/JNEUROSCI.3086-14.2015
- Minnebusch, D. A., Suchan, B., Ramon, M., & Daum, I. (2007). Event-related potentials reflect heterogeneity of developmental prosopagnosia. *European Journal of Neuroscience*, 25(7), 2234–2247. doi:10.1111/j.1460-9568.2007.05451.x
- Moeller, S., Freiwald, W. A., & Tsao, D. Y. (2008). Patches with links: A unified system for processing faces in the macaque temporal lobe. *Science*, 320(5881), 1355–1359. doi:10.1126/science.1157436
- Nederhouser, M., Yue, X., Mangini, M. C., & Biederman, I. (2007). The deleterious effect of contrast reversal on recognition is unique to faces, not objects. *Vision Research*, 47(16), 2134–2142. doi:10.1016/j.visres.2007.04.007
- Németh, K., Zimmer, M., Schweinberger, S. R., Vakli, P., & Kovács, G. (2014). The background of reduced face specificity of N170 in congenital prosopagnosia. *PLoS ONE*, 9(7), e101393. doi:10.1371/journal.pone.0101393
- Ohayon, S., Freiwald, W. A., & Tsao, D. Y. (2012). What makes a cell face selective? The importance of contrast. *Neuron*, 74(3), 567–581. doi:10.1016/j.neuron.2012.03.024
- Olson, I. R., Ezzyat, Y., Plotzker, A., & Chatterjee, A. (2015). The end point of the ventral visual stream: face and non-face perceptual deficits following unilateral anterior temporal lobe damage. *Neurocase*, 21(5), 554–562. doi:10.1080/13554794.2014.959025
- Otsuka, Y., Motoyoshi, I., Hill, H. C., Kobayashi, M., Kanazawa, S., & Yamaguchi, M. K. (2013). Eye contrast polarity is critical for face recognition by infants. *Journal of Experimental Child Psychology*, 115(3), 598–606. doi:10.1016/j.jecp.2013.01.006
- Palermo, R., Willis, M. L., Rivalta, D., McKone, E., Wilson, C. E., & Calder, A. J. (2011). Impaired holistic coding of facial expression and facial identity in congenital prosopagnosia. *Neuropsychologia*, 49(5), 1226–1235. doi:10.1016/j.neuropsychologia.2011.02.021
- Parketry, J., Towler, J., & Eimer, M. (2015). The activation of visual face memory and explicit face recognition are delayed in developmental prosopagnosia. *Neuropsychologia*, 75, 538–547. doi:10.1016/j.neuropsychologia.2015.07.009
- Parvizi, J., Jacques, C., Foster, B. L., Witthoft, N., Witthoft, N., Rangarajan, V., ... Grill-Spector, K. (2012). Electrical stimulation of human fusiform face-selective regions distorts face perception. *The Journal of Neuroscience*, 32(43), 14915–14920. doi:10.1523/JNEUROSCI.2609-12.2012
- Pellicano, E., & Rhodes, G. (2003). Holistic processing of faces in preschool children and adults. *Psychological Science*, 14(6), 618–622. doi:10.1046/j.0956-7976.2003.psci.1474.x
- Peterson, M. F., & Eckstein, M. P. (2012). Looking just below the eyes is optimal across face recognition tasks. *Proceedings of the National Academy of Sciences of the United States of America*, 109(48), E3314–3323. doi:10.1073/pnas.1214269109
- Pitcher, D., Garrido, L., Walsh, V., & Duchaine, B. C. (2008). Transcranial magnetic stimulation disrupts the perception and embodiment of facial expressions. *The Journal of Neuroscience*, 28(36), 8929–8933. doi:10.1523/JNEUROSCI.1450-08.2008
- Pitcher, D., Walsh, V., & Duchaine, B. (2011). The role of the occipital face area in the cortical face perception network. *Experimental Brain Research*, 209(4), 481–493. doi:10.1007/s00221-011-2579-1
- Pitcher, D., Walsh, V., Yovel, G., & Duchaine, B. (2007). TMS evidence for the involvement of the right occipital face area in early face processing. *Current Biology*, 17(18), 1568–1573. doi:10.1016/j.cub.2007.07.063
- Pizzamiglio, M. R., De Luca, M., Di Vita, A., Palermo, L., Tanzilli, A., Dacquino, C., & Piccardi, L. (2015). Congenital prosopagnosia in a child: Neuropsychological assessment, eye movement recordings and training. *Neuropsychological rehabilitation*, 1–40.
- Prieto, E. A., Caharel, S., Henson, R., & Rossion, B. (2011). Early (N170/M170) face-sensitivity despite right lateral occipital brain damage in acquired prosopagnosia. *Frontiers in Human Neuroscience*, 5, 138. doi:10.3389/fnhum.2011.00138
- Rajimehr, R., Young, J. C., & Tootell, R. B. H. (2009). An anterior temporal face patch in human cortex, predicted by macaque maps. *Proceedings of the National Academy of Sciences of the United States of America*, 106(6), 1995–2000. doi:10.1073/pnas.0807304106
- Rangarajan, V., Hermes, D., Foster, B. L., Weiner, K. S., Jacques, C., Grill-Spector, K., & Parvizi, J. (2014). Electrical stimulation of the left and right human fusiform gyrus causes different effects in conscious face perception. *The Journal of Neuroscience*, 34(38), 12828–12836. doi:10.1523/JNEUROSCI.0527-14.2014
- Righart, R., & de Gelder, B. (2007). Impaired face and body perception in developmental prosopagnosia. *Proceedings of the National Academy of Sciences*, 104(43), 17234–17238. doi:10.1073/pnas.0707753104
- Rivalta, D., Palermo, R., Schmalzl, L., & Williams, M. A. (2012). Investigating the features of the M170 in congenital prosopagnosia. *Frontiers in Human Neuroscience*, 6. doi:10.3389/fnhum.2012.00045
- Röder, B., Ley, P., Shenoy, B. H., Kekunnaya, R., & Bottari, D. (2013). Sensitive periods for the functional specialization of the neural system for human face processing. *Proceedings of the National Academy of Sciences of the United States of America*, 110(42), 16760–16765. doi:10.1073/pnas.1309963110
- Rosburg, T., Ludwig, E., Dümpelmann, M., Alba-Ferrara, L., Urbach, H., & Elger, C. E. (2010). The effect of face inversion on intracranial and scalp recordings of event-related potentials. *Psychophysiology*, 47(1), 147–157. doi:10.1111/j.1469-8986.2009.00881.x
- Rossion, B., Delvenne, J. F., Debatisse, D., Goffaux, V., Bruyer, R., Crommelinck, M., & Guérit, J. M. (1999). Spatio-temporal localization of the face inversion effect: An event-related potentials

- study. *Biological Psychology*, 50(3), 173–189. doi:10.1016/S0301-0511(99)00013-7
- Rossion, B., & Jacques, C. (2011). The N170: Understanding the time-course of face perception in the human brain. In S. Luck & E. Kappenman (Eds.), *The Oxford handbook of ERP components* (pp. 115–142). Oxford, UK: Oxford University Press.
- Rossion, B., Joyce, C. A., Cottrell, G. W., & Tarr, M. J. (2003). Early lateralization and orientation tuning for face, word, and object processing in the visual cortex. *NeuroImage*, 20(3), 1609–1624. doi:10.1016/j.neuroimage.2003.07.010
- Rousselet, G. A., Ince, R. A. A., van Rijsbergen, N. J., & Schyns, P. G. (2014). Eye coding mechanisms in early human face event-related potentials. *Journal of Vision*, 14(13), 7–7. doi:10.1167/14.13.7
- Sadeh, B., & Yovel, G. (2010). Why is the N170 enhanced for inverted faces? An ERP competition experiment. *NeuroImage*, 53(2), 782–789. doi:10.1016/j.neuroimage.2010.06.029
- Sangrigoli, S., & de Schonen, S. (2004). Recognition of own-race and other-race faces by three-month-old infants. *Journal of Child Psychology and Psychiatry*, 45(7), 1219–1227. doi:10.1111/j.1469-7610.2004.00319.x
- Savage, R. A., & Lipp, O. V. (2015). The effect of face inversion on the detection of emotional faces in visual search. *Cognition and Emotion*, 29(6), 972–991.
- Schacht, A., & Sommer, W. (2009). Emotions in word and face processing: Early and late cortical responses. *Brain and Cognition*, 69(3), 538–550. doi:10.1016/j.bandc.2008.11.005
- Schmalzl, L., Palermo, R., Green, M., Brunsdon, R., & Coltheart, M. (2008). Training of familiar face recognition and visual scan paths for faces in a child with congenital prosopagnosia. *Cognitive Neuropsychology*, 25(5), 704–729. doi:10.1080/02643290802299350
- Schwarzer, G., Huber, S., Grüter, M., Grüter, T., Groß, C., Hipfel, M., & Kennerknecht, I. (2007). Gaze behaviour in hereditary prosopagnosia. *Psychological Research*, 71(5), 583–590. doi:10.1007/s00426-006-0068-0
- Schwarzer, G., & Zauner, N. (2003). Face processing in 8-month-old infants: Evidence for configural and analytical processing. *Vision Research*, 43(26), 2783–2793. doi:10.1016/S0042-6989(03)00478-4
- Schweinberger, S. R., Pfützte, E.-M., & Sommer, W. (1995). Repetition priming and associative priming of face recognition: Evidence from event-related potentials. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 21(3), 722–736. doi:10.1037/0278-7393.21.3.722
- Sinha, P. (2002). Qualitative representations for recognition. In H. H. Bülthoff, C. Wallraven, S.-W. Lee, & T. A. Poggio (Eds.), *Biologically motivated computer vision* (Vol. 2525, pp. 249–262). Berlin, Heidelberg: Springer Berlin Heidelberg. Retrieved from http://link.springer.com/10.1007/3-540-36181-2_25
- Skuse, D. H., Lori, A., Cubells, J. F., Lee, I., Conneely, K. N., Puura, K., ... & Young, L. J. (2014). Common polymorphism in the oxytocin receptor gene (OXTR) is associated with human social recognition skills. *Proceedings of the National Academy of Sciences*, 111(5), 1987–1992.
- Smith, M. L., Gosselin, F., & Schyns, P. G. (2004). Receptive fields for flexible face categorizations. *Psychological Science*, 15(11), 753–761. doi:10.1111/j.0956-7976.2004.00752.x
- Song, S., Garrido, L., Nagy, Z., Mohammadi, S., Steel, A., Driver, J., ... & Furl, N. (2015). Local but not long-range microstructural differences of the ventral temporal cortex in developmental prosopagnosia. *Neuropsychologia*, 78, 195–206. doi:10.1016/j.neuropsychologia.2015.10.010
- Sormaz, M., Andrews, T. J., & Young, A. W. (2013). Contrast negation and the importance of the eye region for holistic representations of facial identity. *Journal of Experimental Psychology: Human Perception and Performance*, 39(6), 1667–1677. doi:10.1037/a0032449
- Susilo, T., & Duchaine, B. (2013). Advances in developmental prosopagnosia research. *Current Opinion in Neurobiology*, 23(3), 423–429. doi:10.1016/j.conb.2012.12.011
- Susilo, T., McKone, E., Dennett, H., Darke, H., Palermo, R., Hall, A., ... Rhodes, G. (2010). Face recognition impairments despite normal holistic processing and face space coding: Evidence from a case of developmental prosopagnosia. *Cognitive Neuropsychology*, 27(8), 636–664. doi:10.1080/02643294.2011.613372
- Tanaka, J. W., Curran, T., Porterfield, A. L., & Collins, D. (2006). Activation of preexisting and acquired face representations: The N250 event-related potential as an index of face familiarity. *Journal of Cognitive Neuroscience*, 18(9), 1488–1497. doi:10.1162/jocn.2006.18.9.1488
- Tanaka, J. W., & Farah, M. J. (1993). Parts and wholes in face recognition. *The Quarterly Journal of Experimental Psychology*, 46(2), 225–245. doi:10.1080/14640749308401045
- Taylor, M. J., Batty, M., & Itier, R. J. (2004). The faces of development: A review of early face processing over childhood. *Journal of Cognitive Neuroscience*, 16(8), 1426–1442. doi:10.1162/0898929042304732
- Thomas, C., Avidan, G., Humphreys, K., Jung, K., Gao, F., & Behrmann, M. (2009). Reduced structural connectivity in ventral visual cortex in congenital prosopagnosia. *Nature Neuroscience*, 12(1), 29–31. doi:10.1038/nn.2224
- Todorov, A., & Duchaine, B. (2008). Reading trustworthiness in faces without recognizing faces. *Cognitive Neuropsychology*, 25(3), 395–410. doi:10.1080/02643290802044996
- Towler, J., & Eimer, M. (2012). Electrophysiological studies of face processing in developmental prosopagnosia: Neuropsychological and neurodevelopmental perspectives. *Cognitive Neuropsychology*, 29(5–6), 503–529. doi:10.1080/02643294.2012.716757
- Towler, J., & Eimer, M. (2015). Early stages of perceptual face processing are confined to the contralateral hemisphere: Evidence from the N170 component. *Cortex*, 64, 89–101. doi:10.1016/j.cortex.2014.09.013
- Towler, J., Fisher, K., Grubert, A., Gosling, A., & Eimer, M. (unpublished) Normal rapid detection of fear in developmental prosopagnosia.
- Towler, J., Gosling, A., Duchaine, B., & Eimer, M. (2012). The face-sensitive N170 component in developmental prosopagnosia. *Neuropsychologia*, 50(14), 3588–3599. doi:10.1016/j.neuropsychologia.2012.10.017
- Towler, J., Gosling, A., Duchaine, B., & Eimer, M. (2016). Normal perception of mooney faces in developmental prosopagnosia: Evidence from the N170 component and rapid neural adaptation. *Journal of Neuropsychology*, 10(1), 15–32. doi:10.1111/jnp.12054
- Towler, J., Parketny, J., & Eimer, M. (2016). Perceptual face processing in developmental prosopagnosia is not sensitive to the canonical location of face parts. *Cortex*, 74, 53–66. doi:10.1016/j.cortex.2015.10.018
- Tranel, D., Damasio, A. R., & Damasio, H. (1988). Intact recognition of facial expression, gender, and age in patients with impaired

- recognition of face identity. *Neurology*, 38(5), 690–690. doi:10.1212/WNL.38.5.690
- Tsao, D. Y., & Livingstone, M. S. (2008). Mechanisms of face perception. *Annual Review of Neuroscience*, 31(1), 411–437. doi:10.1146/annurev.neuro.30.051606.094238
- Tsao, D. Y., Schweers, N., Moeller, S., & Freiwald, W. A. (2008). Patches of face-selective cortex in the macaque frontal lobe. *Nature Neuroscience*, 11(8), 877–879. doi:10.1038/nn.2158
- Van den Stock, J., van de Riet, W. A. C., Righart, R., & de Gelder, B. (2008). Neural correlates of perceiving emotional faces and bodies in developmental prosopagnosia: An event-related fMRI-Study. *PLoS ONE*, 3(9). doi:10.1371/journal.pone.0003195
- Vuilleumier, P., Armony, J. L., Driver, J., & Dolan, R. J. (2001). Effects of attention and emotion on face processing in the human brain: An event-related fMRI study. *Neuron*, 30(3), 829–841. doi:10.1016/S0896-6273(01)00328-2
- Vuong, Q. C., Peissig, J. J., Harrison, M. C., & Tarr, M. J. (2005). The role of surface pigmentation for recognition revealed by contrast reversal in faces and Greebles. *Vision Research*, 45(10), 1213–1223. doi:10.1016/j.visres.2004.11.015
- Watanabe, S., Kakigi, R., & Puce, A. (2003). The spatiotemporal dynamics of the face inversion effect: A magneto- and electro-encephalographic study. *Neuroscience*, 116(3), 879–895. doi:10.1016/S0306-4522(02)00752-2
- Wilmer, J. B., Germine, L., Chabris, C. F., Chatterjee, G., Williams, M., Loken, E., ... Duchaine, B. (2010). Human face recognition ability is specific and highly heritable. *Proceedings of the National Academy of Sciences*, 107(11), 5238–5241. doi:10.1073/pnas.0913053107
- Winston, J. S., Henson, R. N. A., Fine-Goulden, M. R., & Dolan, R. J. (2004). fMRI-adaptation reveals dissociable neural representations of identity and expression in face perception. *Journal of Neurophysiology*, 92(3), 1830–1839. doi:10.1152/jn.00155.2004
- Yarbus, A. L. (1967). Eye movements during perception of complex objects. In A. L. Yarbus, *Eye movements and vision* (pp. 171–211). Boston, MA: Springer US. Retrieved from http://link.springer.com/10.1007/978-1-4899-5379-7_8
- Yin, R. K. (1969). Looking at upside-down faces. *Journal of Experimental Psychology*, 81(1), 141–145. doi:10.1037/h0027474
- Young, A. W., Hellawell, D., & Hay, D. C. (1987). Configurational information in face perception. *Perception*, 16, 747–759. doi:10.1068/p160747
- Young, A. W., Newcombe, F., de Haan, E. H., Small, M., & Hay, D. C. (1993). Face perception after brain injury. *Selective impairments affecting identity and expression*. *Brain: A Journal of Neurology*, 116, 941–959.
- Yovel, G., & Kanwisher, N. (2005). The neural basis of the behavioral face-inversion effect. *Current Biology*, 15(24), 2256–2262. doi:10.1016/j.cub.2005.10.072
- Zhang, J., Liu, J., & Xu, Y. (2015). Neural decoding reveals impaired face configural processing in the right fusiform face area of individuals with developmental prosopagnosia. *The Journal of Neuroscience*, 35(4), 1539–1548. doi:10.1523/JNEUROSCI.2646-14.2015
- Zhu, Q., Song, Y., Hu, S., Li, X., Tian, M., Zhen, Z., ... Liu, J. (2010). Heritability of the specific cognitive ability of face perception. *Current Biology*, 20(2), 137–142. doi:10.1016/j.cub.2009.11.067
- Zion-Golombic, E., & Bentin, S. (2007). Dissociated neural mechanisms for face detection and configural encoding: Evidence from N170 and induced gamma-band oscillation effects. *Cerebral Cortex*, 17(8), 1741–1749. doi:10.1093/cercor/bhl100