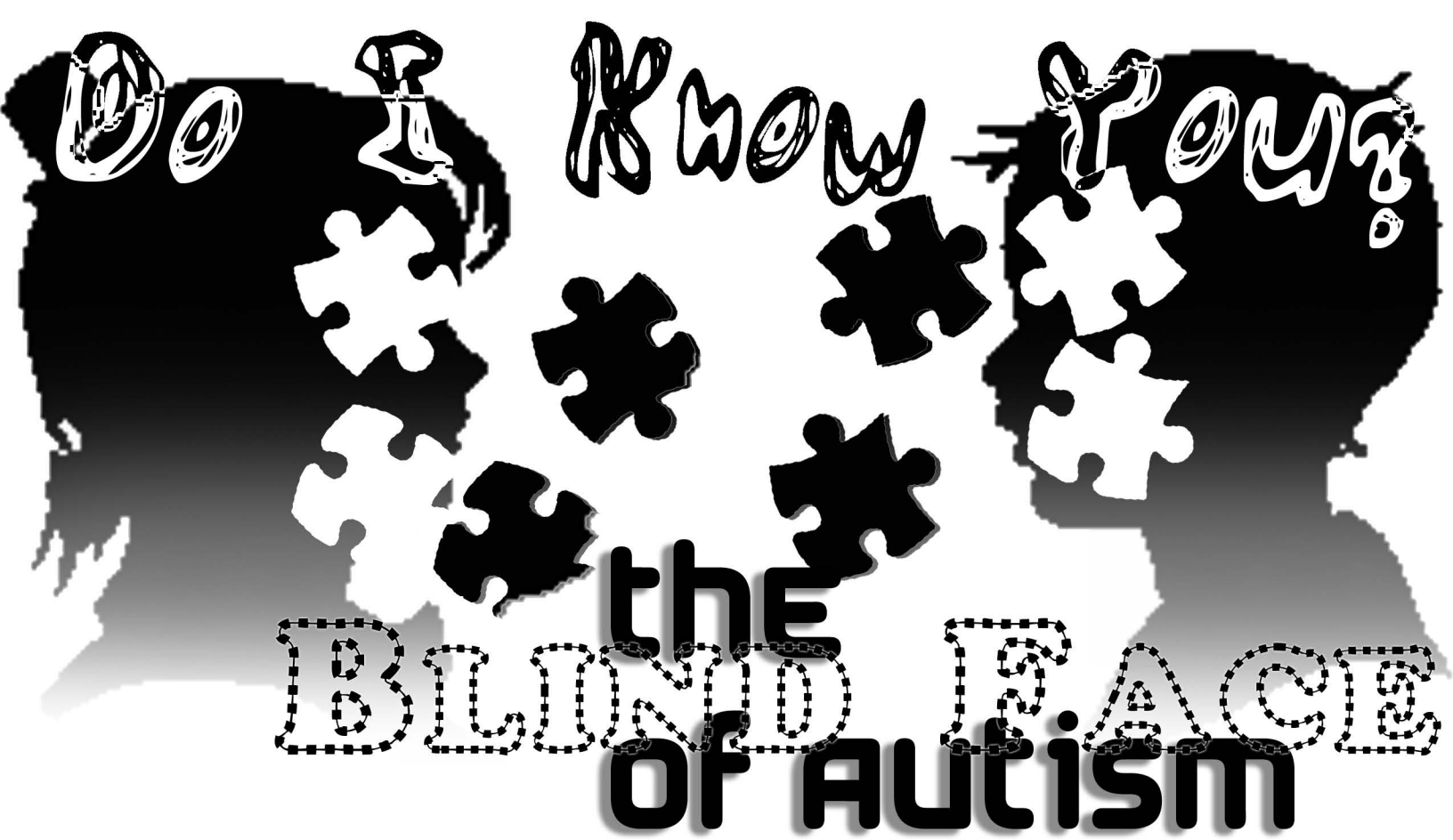

It is one of the greatest acts we can both perform and observe. It transfers to any culture, at any age, anywhere in the world. It is priceless but always free. It requires minimal effort but can turn a bad day around. It is both a silent call and a silent response. It is one of the first gifts we give others—that endearing, precious smile of recognition. But what happens when that recognition doesn't come? When the loving visage of a family member or friend means nothing?

The human brain appears to have an innate interest in faces, as even newborn babies are attracted to face-like stimuli more than others. This is not too surprising, given our lives spent in social situations and the complex set of cues we glean from a momentary glance at a face—how long does it take to see a loved one's happiness after good news? Because of the importance of faces, researchers suggest a system in the human brain that has developed specifically for facial recognition; some mechanisms of this system may be "hardwired" and thus exist at birth (hence the attention to face-like stimuli), whereas others may develop with us, constantly being moulded as we grow by our experiences and the faces we encounter. Face processing and social skills seem, then, to come hand in hand and much of the motivation on face research has been fuelled by social developmental conditions—in particular, the autism spectrum disorders (ASD).

[1 in 10,000...1 in 100...1 in...]

These numbers are harshly disconcerting yet oddly therapeutic at the same time. Year after year, parents of children with an ASD anticipate the next new finding that could shed light on what their child is going through; year after year, new parents open the Science section of the New York Times in stoic apprehension for fear that another factor has been correlated with an increased risk for autism. For some reason, the social suavity and intuition that comes so naturally to many of us—from an act as simple as holding another's gaze—is impaired or lacking in a growing number of our children.

In typically-developing children as young as 6 months, a picture of a mother's face induces different brain activity than the face of a stranger. However, children three to four years old who have been diagnosed with an ASD fail to respond to their mother's faces at all, even though they react normally to an image of their favourite toy. Studies like this, among others, suggest fundamental abnormalities in the face processing mechanisms of the brain. Since our face expertise skills require experience to improve in the same way as our social skills, an initial indifference could lead to poor development of those face areas. The problem then becomes a debate of correlation—a debate with its own history of nuanced complexities: could face processing deficits be an early cause of ASD? Or does ASD itself lead to face processing deficiencies?

Further complicating the story is the existence of individuals who have trouble recognizing faces—known clinically as *prosopagnosics*—who may not necessarily be autistic. As two separate disorders, autism and prosopagnosia require different treatments and therapies, but the latter is not nearly as well known or studied as the former. We tackle this caveat by keeping this in mind; if face recognition is such an important diagnostic criterion for ASD, could certain children be misdiagnosed as autistic when in fact they have prosopagnosia? What about the other way around? Before we delve into this complex question, let us explore what the two disorders are, and what areas in the brain are involved in our face processing abilities in the first place.

[autism]

Categorized as a pervasive developmental disorder, autism is usually regarded as having three hallmark disabilities in social relationships, communication, and repetitive behaviour. It exists on a spectrum that covers a wide range of individuals from mild (*Asperger's*) to severe (*autism*), all of whom share a commonality in social deficits. Currently, ASD can only be diagnosed through standardized clinical assessments as there are yet no known genetic or neurological warning signs. Because these behavioural tests usually are not reliable until a child is two or three years old, researchers have been trying to find earlier markers so the children can obtain beneficial therapy at younger ages. Interestingly, often-cited characteristics which appear within the first year of life and are also used as criteria in later clinical assessments frequently involve abnormal interactions with faces—in particular, lack of interest and gaze aversion. More remarkably, these are the same characteristics often observed in developmental prosopagnosia.

[prosopagnosia]

As a specific type of "loss of knowledge disorder," prosop*agnosia*—or face blindness—is the selective inability to recognize faces while recognition of non-face objects may be typical. Individuals with prosopagnosia usually have no other prior vision or neurological problem, and develop the disorder after acute brain injury. For these individuals, the onset is particularly impactful because something previously as effortless as recognizing a friend is now painfully impossible. However, it is only in the past decade that developmental prosopagnosia (also known as congenital prosopagnosia) was proposed, suggesting that as much as two to three percent of the population, or two to three in 100 (remember the statistic if one in 100 for autism?), may be *born* with impairments to their brain's face-specific areas. In contrast with individuals who acquired prosopagnosia later in life, developmental prosopagnosics (DP) usually do not notice the nature of their impairment until late adolescence or adulthood. Instead, DPs develop their own ways of recognizing people by relying on physical features like hairstyles, voices, and clothing. Accordingly, it is extremely difficult to identify children with DP because they may simply appear to be shy or socially awkward. In fact, only eleven cases of childhood DP have been reported as of last year, and many of these case reports include descriptors consistent with those found in ASD diagnoses because researchers did not think to separate the two disorders.

[typical development of face processing abilities]

As mentioned earlier, infants show preference for faces as early as three days old, and are able to discriminate the facial identities of any primate at six months. By nine months, they become fine-tuned to only discriminate human faces, and this development of face recognition abilities continues to improve until approximately 32 years of age.

A few models of face processing mechanisms attempt to explain how our daily experiences can shape the development of the face-selective areas of our brains. Although the exact neural mechanisms are still unknown, evidence points towards a distributed network of brain areas from

the *temporal lobe* (the region beside our ears that is important for processing speech and visual meanings and also contains the *hippocampus*, our memory center) to the *prefrontal cortex* (the region behind our foreheads where higher order, more conscious processing occurs). Of particular note is the "face fusiform area" (FFA), a region in the temporal lobe strongly supported as playing a key, integral role in face recognition. As face memory improves from childhood to adulthood, not only does face-related activation of these areas selectively increase, but the physical size of the FFA does as well.

Another important area in the temporal lobe is the *amygdala*, a brain area near the hippocampus which is heavily involved with recognizing and processing emotional cues from faces and situations. As early as four months of age, infants can discriminate between their mother's happy and sad facial expressions; by age five, a child's emotional recognition and discrimination abilities have developed to almost adult accuracy. Astoundingly, even children understand that eyes are windows into the soul: eyes alone are often enough to elicit FFA and amygdala responses in normal children because of the amount of identity and emotional information contained in a gaze. We can see, then, how much is lost when those windows no longer capture a child's attention…

[abnormal development]

The direct correlation between maturation of face-selective neural sites and face processing abilities has led to extensive studies on the roles of these areas in social disorders. In children with ASD, the face-specific brain regions develop abnormally and the cortex does not undergo its usual thinning during adolescence. Moreover, there is reduced connectivity between the temporal and frontal lobes and less activation of the FFA. Interestingly, this activation appears to be strongly positively correlated with the amount of time that ASD, and not typically-developing, subjects spent looking at the eye region. If FFA modulation is dependent on fixating on the eyes and children with ASD have habitual gaze avoidance, this chronic under-activation during development could lead to eventual anomalies in the area's function. Similarly, children with ASD have deficits in recognizing facial expression and low amygdala activation, which may be due to—or a cause for—their gaze aversion.

Although less well studied, research has shown that DPs similarly have impaired early face-processing pathways, where the initial 'hardwired' propensity for faces is not operating properly. With a defective mechanism for attending to faces from birth, infant brains may have a hard time developing the specialized processing for faces. Several researchers have found that DPs have smaller FFAs and less brain matter in face-specific areas than controls, with sizes correlated with their face recognition performance. However, study results are inconsistent in their FFA and amygdala activation profiles: while some DP subjects have decreased responses, most respond the same as controls. Genetic factors may also play a role in some cases of DP, but the overall developmental trajectory is relatively unknown. Bottom line—we as a scientific community have much to learn.

[the connection]

The link between ASD and DP is simultaneously robust and fleeting. The main questions are again thesame ones of correlation, causation, and direction: does lack of social interest from autistic deficits prevent the acquisition of face expertise? Does a failure to process faces properly interfere with normal social development? Or are DP and ASD completely separate disorders that simply happen to look similar?

Formal data looking at the two disorders simultaneously is currently lacking, partly because of the relatively new distinction of DP from general prosopagnosia. However, ASD and DP research point equally towards dysfunction in the fusiform gyrus (brain location where the FFA is located) as cause for both social deficit problems and face-processing deficiencies. On the other hand, the little data there is suggests the dissociation of social cognitive functions with face processing abilities, where each can exist in isolation from the other; in fact, the majority of DPs—children and adult—were found to not have problems in their social cognitive abilities after they were subjected to on a battery of tests for both face recognition and ASD. Yet with any study, it is also important to note that much of the above results are from a very small number of DPs—and an even smaller number of whom are children.

[facing the future]

There is still much we do not know and speculate about in the realm of social cognition and faces. If DP and ASD are indeed separate conditions, particular attention should be paid to the method of testing for ASD—especially at younger ages when the criterion for social competency in ASD diagnoses may rely more heavily on face processing skills. We have been so caught up in the increasing prevalence of autism in the world's youth that we must be careful not to turn a blind eye to other details—or in this case, a blind face. It is just as important to realize the substantial percentage of the general population thought to be affected by face recognition difficulties, and to notice the children who may be growing up thinking they are something they are not, receiving treatment for a disorder they do not have, and missing opportunities to live the socially complete life of their friends…at face value.

So next time you see a smile, pause. Pause, and look at the person, the whole person. Look, and imagine what it would be like to not have their visage in your mind's eye. Imagine, and realize where the "*extreme autistic aloneness* that, whenever possible, disregards, ignores, [and] shuts out anything … from the outside"[1] could come from.

[further reading]

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1 As described originally by Leo Kanner, the "founder" of autism, in 1943