Partners Universal International Research Journal (PUIRJ)
Volume: 01 Issue: 02 | April-June 2022 www.puirj.com

Current reflections on Prosopagnosia: Is Prosopagnosia limited to the recognition of faces

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1.INTRODUCTION

Humans have traditionally lived in tiny groups of less than a hundred people throughout our evolutionary history. Our face recognition abilities, on the other hand, appear to enable us to recognize a large number of people, possibly thousands. Even though modern society offers access to a lot of faces, nobody knows how many people know. Prosopagnosia (from Greek próspon, meaning "face," and Agnes, meaning "non-knowledge"), also known as face blindness, is a face perception disorder where the ability to identify familiar faces, including their face (self-recognition), is damaged while other facets of visual processing for example, object discrimination and cognitive functioning (e.g., decision-making) are unaffected.

Face recognition difficulties in the absence of brain injury are referred to as developmental prosopagnosia (DP). DP is seen in 2% of the population and is commonly passed down through generations. DP research has made significant progress in defining the disorder's cognitive and neurological aspects. The development of a true DP taxonomy that will aid many types of study is a major problem. A hereditary form of prosopagnosia, or inability to recognize someone by face alone in absence of sensory or intellectual impairment. It appears to exhibit autosomal dominant inheritance and may affect 0.75-2% of different populations.

Over the course of four years (1976–2017), Geskin and Behrmann (2017) examined over 700 research reports of adults having long-term developmental impairments in individual face-recognition (IFR). The purpose of their review, which is a useful contribution to the field, is to clarify if faces are handled by a separate neural visual recognition process and if face and non-face items are handled by the same system. Here, they focused on the parallel made by the authors, both at the theoretical and methodological levels, between long-term developmental difficulties for IFR - what they call congenital prosopagnosia (commonly named developmental prosopagnosia) and the classic form of prosopagnosia, which follows brain damage in a typically developed individual.

2.CLASSIFICATION OF TYPES

1. Apperceptive prosopagnosia: The inability to even see and process the face is known as apperceptive prosopagnosia. Despite perceiving the face, associative prosopagnosia is characterized as the inability to recognise or attribute any meaning to it.

2. Associative prosopagnosia: Despite perceiving the face, associative prosopagnosia is characterized as the inability to recognise or attribute any meaning to it.

3.Developmental prosopagnosia: In absence of sensory visual abnormalities or intellectual impairment, developmental prosopagnosia is a lifelong condition that inhibits a person's ability to recognize faces. This syndrome affects people who have normal IQ and memory, as well as usual low-level vision and no previous history of brain injury.

Causes

The right fusiform gyrus, a fold in the brain that appears to coordinate the neuronal systems that control facial perception and memory, is hypothesized to be the cause of prosopagnosia. Stroke, traumatic brain injury, and certain neurodegenerative illnesses can all cause prosopagnosia.

Geskin and Behrmann (2017) discuss the difficulties in recognizing exemplars of visually homogenous categories in reported cases of prosopagnosia. This long-standing in-category recognition account doesn't hold prosopagnosia patients without basic-level object recognition impairments have been reported, such as PS, who is able to correctly and rapidly individualize visual exemplars of non-face categories. Moreover, these patients are relatively less impaired with physically similar than dissimilar faces. Unfortunately, this within-category account of prosopagnosia persists because of its evaluation with visual object agnosic patients, who make visual under specification errors when attempting to recognize non-face objects. This again supports the view that significant progress in the understanding of the nature of human face recognition requires restricting the definition of prosopagnosia to an acquired selective disorder of visual recognition, i.e., with no basic-level object recognition impairment, in line with its original definition.

Signs and Symptoms

In order to understand the difference between knowing a person's face and knowing their name is crucial. These two forms of personal knowledge readily dissociate. For example, one may recognize other commuters' faces but then never learn their names. On the other hand, one may remember the names of well-known authors but never see their faces. The important fact is that we must establish criteria for determining whether or not someone knows a person's face. The participant must be able to generate a clear mental picture of the face or (ii) believe they would identify the face when they see it for recall. Participants must be able to distinguish two distinct photographs of a known individual to be recognized. (Jenkins, 2018)

Prosopagnosia is not limited to human faces, but also emerges in connection to other visual stimuli whose recognition is dependent on the evocation of specific contextual features and associations, and which are visually 'ambiguous' which means different stimuli belonging to the same group but having similar physical structure. Prosopagnosia is the physiological effect of a failure to activate memories related to visual stimuli based on those stimuli.

Prosopagnosia can cause an individual to avoid social involvement and develop social anxiety disorder, which is marked by an overpowering fear of social interactions. They might also have trouble building relationships or dealing with challenges in their careers. Depression is a common occurrence.

Diagnosis

Prosopagnosia is characterized by the loss of recognition of previously recognized faces and an inability to learn a new faces. In the past, tests using renowned faces or case studies with examples of the subject's inability to recognize acquaintances or family members were widely used to demonstrate. Determining standard diagnostic criteria from such testing, in contrast, is difficult. Familiarity with known faces is affected by age, culture, education, and subject interests, for example, and carefully matched controls are vital in evaluating the outcomes of these reviews.

However, most neurodevelopmental disorders are polygenic combinations of allelic variants present in general population. Along these lines, recent studies of 24 subjects reported that common single-nucleotide polymorphisms in the oxytocin receptor

gene are associated with developmental prosopagnosia (Cattaneo, 2016).

Face perception tests, which assess the potential to recognize distinctions between faces, are insufficient to identify prosopagnosia. What they could do is show if prosopagnosia is driven by impaired facial structure encoding, indicating an apperceptive version, or whether such encoding is intact, indicating an associative form. The Cambridge Face Perception Test and a Glasgow Face Matching Test, which entail sorting or matching faces by their identities with low demands on memory, have been used to assess face perception deficits. Children can benefit from the Dartmouth Face Perception Test. (Dalrymple, 2014)

Treatment

It is unusual for acquired prosopagnosia to resolve spontaneously and development prosopagnosia is a condition that lasts a lifetime. As a result, clinically relevant methods for increasing facial recognition skills in these individuals are of interest.

Prognosis

Face processing could not be enhanced prosopagnosia, according to clinicians and researchers. Over the last fifty years, more than a dozen research attempted to improve face processing in prosopagnosics, but evidence for viable treatment techniques has only recently begun to surface. The present evidence on spontaneous recovery in acquired prosopagnosia (AP), as well as treatment attempts in acquired and developmental prosopagnosia (DP), with a distinction made between compensatory and remedial treatments. Strategic compensating training, such as verbalizing specific face traits, has proven to be the most successful way for AP, rather than remedial methods. Compensatory training has also been demonstrated to be useful in children with DP. Two recent large-scale investigations in adults with DP, one utilizing remedial instruction and the other using oxytocin, have shown group-level gains and signs of generalization (DeGutis 2014).

According to Dalrymple et al. (2012), DeGutis and colleagues attempted to teach 12-year-old T.M. to recognize his mother's face. When T.M. was shown a picture of his mother or age-matched females, he gave a "mom/not-mom" response, and he was given feedback following each response. T.M. showed no appreciable improvements on the mom/not-mom task after 47 sessions of training (10 - 15 minutes per session) over a 10-month period, nor did he indicate any improvements in his daily life.

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