



Recognition memory for faces in schizophrenia patients and their first-degree relatives[☆]

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Abstract

It has consistently been shown that schizophrenia patients are impaired in recognition memory for faces. However, studies have not examined the specificity of this deficit relative to other cognitive functions nor the relationship between this deficit and particular schizophrenia symptoms. In addition, no studies have examined recognition memory for faces in unaffected biological relatives of schizophrenia patients who likely share some of the genetic diathesis for this disorder without presenting the potential confounds of mentally ill study samples. The Faces subtests from the Wechsler Memory Scale—Third Edition were used to evaluate recognition memory for faces in 39 schizophrenia patients, 33 of their first-degree relatives and 56 normal controls. Both schizophrenia patients and their relatives were impaired, relative to control participants, in recognition memory for faces after partialing out group differences in spatial attention or verbal memory. Further, recognition memory for faces was associated with positive symptoms in the schizophrenia group and schizotypal personality traits in the relative group. These findings may have important implications for reducing etiological heterogeneity among schizophrenia populations, identifying disorder susceptibility among their relatives and furthering understanding of disorder etiology.

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1. Introduction

Ever since it was observed clinically that individuals, despite being otherwise cognitively intact, could fail to recognize familiar faces (i.e. prosopagnosia) there has been interest in face recognition as a discrete mnemonic ability. Evolutionarily, it would make sense for face memory to be functionally and neuroanatomically distinct from other memory abilities, as the discrimination between familiar or unfamiliar, friend or enemy, related or unrelated is critical for survival. Therefore, there may have been selective pressure that led to the development of unique neural mechanisms for face memory [11]. Support for neural “hardwiring” of face analysis and recognition comes from the cross-cultural recognition of typical facial expressions (e.g. [12]), as well as the early devel-

opmental capacity of infants to recognize familiar faces [20].

Three lines of evidence have contributed to the identification of discrete neural circuits subserving face recognition ability: case studies of humans with focal brain lesions, neuroimaging research with neurologically intact individuals and single-unit recordings with nonhuman primates. In brain damaged adults, a double dissociation has been identified whereby patients may demonstrate impaired face recognition but intact object recognition (e.g. [13]) or impaired object recognition but intact face recognition (e.g. [35]), indicating separate processing centers for faces and objects. An early debate emanating from neuropsychological studies surrounded the hemispheric laterality of face recognition ability. Both autopsy results and CT-scan studies of individuals with prosopagnosia now suggest that the disability can be produced by a right hemisphere lesion alone [3]. Reviews of the neuropsychological literature are consistent with a right hemisphere advantage for face recognition [44]. Clinical observation supports the finding that patients with posterior right hemispheric disease show a disability in recognition of *unfamiliar* faces [3], suggesting that failure to recognize

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familiar faces and unfamiliar faces share overlapping neural substrates. Taken together, neuropsychological findings suggest that patients with damage to the posterior right hemisphere, most commonly the ventral occipitotemporal cortex, demonstrate selective loss of the ability to recognize faces that appears to be irrespective of face familiarity.

Findings from studies of brain damaged individuals have been supplemented with functional neuroimaging research that allows for greater spatial localization of face recognition ability. Consistent with case studies of individuals with brain lesions, neuroimaging studies have revealed a predominantly right-sided pattern of activation for memory for unfamiliar faces [27,28,32]. More specifically, regions of the fusiform gyrus, located within the ventral occipitotemporal cortex, have been found to be more active during face than object recognition (e.g. [25,40,41,49]). For example, regions of the fusiform gyrus show greater activation during the viewing of whole faces than of scrambled faces [40], letterstrings [41] or textures [41]. In an effort to test alternative interpretations of neuroimaging findings suggestive of neural specificity for faces, Kanwisher et al. [25] conducted a systematic set of experiments. These investigators found a region in the right fusiform gyrus that was more active during face viewing than object viewing. In addition, this area responded more strongly to intact two-tone faces than scrambled versions of the same faces, photographs of faces than houses, and photographs of human faces than hands, thus ruling out alternative interpretations of this activation pattern that hinge on luminance differences, exemplars of the same category or presence of any body part [25]. In addition to activation in the fusiform gyrus, half of the subjects in these experiments also showed activation for faces, compared to objects, in the region of the middle temporal gyrus/superior temporal sulcus [25].

Most of the studies reviewed thus far demonstrate involvement of the fusiform gyrus in face-processing tasks independent of mnemonic events such as encoding and retrieval. In a study examining activation patterns for face and word recognition memory, Kim et al. [28] found face recognition to be lateralized to the right hemisphere and located in an area including the right lingual and fusiform gyri. Similarly, Simons et al. [48] found recognition memory for faces, in comparison to objects, led to activation of the anterior temporal lobes (more extensively on the right), a region of the right parahippocampal gyrus, the right posterior fusiform gyrus and the anterior prefrontal cortex. This finding of frontal involvement in face recognition ability has been reported by other researchers [27,28,32,33]. Neuroimaging studies of face recognition memory also suggest that areas of activation are virtually the same for familiar or unfamiliar faces [28].

Single-unit recordings in nonhuman primates allow for even greater spatial resolution than neuroimaging studies for identifying neural substrates involved with face processing and memory. Rolls [45] reported on a series of studies of single cell recordings in the rhesus macaque that indicated

populations of neurons in the inferior temporal lobe that are specialized to respond to faces, suggesting involvement of posterior visual association cortex. Subsequently, other research groups have located cells in the temporal cortex in the macaque brain that are selectively responsive to faces, including the lateral and ventral surfaces of the inferior temporal cortex and the upper bank, lower bank and fundus of the superior temporal sulcus [37]. These investigators also proposed that properties of some of these cells suggest a role in the recognition of familiar individuals [37].

While various methodological factors make it difficult to make direct comparisons among findings from neuropsychological studies of brain injured adults, neuroimaging studies with neurologically intact individuals and single-unit recording studies in nonhuman primates, all three areas of research converge in identifying a specific neural circuit associated with face recognition. This evidence suggests that the right inferior occipitotemporal area, believed to be an association area involved in the transmission of visual information to mesial-temporal areas involved with memory, is critical to the recognition of faces. Secondary areas (e.g. frontal) likely support this ability.

Neuropsychological deficits in schizophrenia, including memory dysfunction, may provide important clues to underlying pathophysiology. Schizophrenia is a severe, persistent form of psychopathology that is associated with brain abnormalities. Neuropsychological tests have been used both to elucidate cognitive deficits that may be secondary to these abnormalities, as well as to suggest hypotheses about corresponding sites of neuropathology. Schizophrenia patients have been found to be impaired on a range of neuropsychological tasks (e.g. [8,16,36]), with some investigators suggesting the breadth of deficits reflects pathology in most brain areas. A recent investigation of first-episode schizophrenia patients, using a comprehensive neuropsychological battery, found a large generalized deficit in schizophrenia patients with superimposed deficits in memory and executive functions [5]. This finding was interpreted as support for dysfunction in an integrated frontal-temporal system in schizophrenia.

It has consistently been shown that patients with schizophrenia, when presented with faces portraying different emotions, have greater difficulty identifying the displayed emotion than normal controls (e.g. [14]). This finding has led investigators to examine component processes of emotion identification, including the ways in which schizophrenia patients scan faces, match facial expressions with emotion labels and recognize faces they have seen previously. Subsequently, multiple studies have found that schizophrenia patients are impaired in recognition memory for faces (e.g. [19,22]). However, it is unclear whether this represents a specific deficit in face memory or a more generalized deficit in memory function. For example, Hellewell et al. [22] reported that schizophrenia patients were impaired on the Warrington Recognition Memory Test for Faces; however, these patients also demonstrated impair-

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