

Finally, a major challenge for research is to develop alternative assessment forms aligned with the revised transfer concept. Traditional static, standardized tests of achievement should be replaced by dynamic assessments that allow students to demonstrate how their past learning activities and experiences have equipped them to approach new learning tasks.

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Cognition in Schizophrenia: Does Working Memory Work?

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Abstract

Recent research suggests that disturbances in social and occupational functioning in individuals with schizophrenia may be more influenced by the severity of cognitive deficits than by the severity of symptoms such as hallucinations and delusions. In this article, I review evidence that one component of cognitive dysfunction in schizophrenia is a deficit in working memory, associated with disturbances in the dopamine system in dorsolateral prefrontal cortex. I suggest that although the cognitive deficits

in schizophrenia include working memory dysfunction, because they arise from a disturbance in executive control processes (e.g., the representation and maintenance of context), they extend to a range of cognitive domains. Finally, I discuss the need for further research on the ways in which contextual processing deficits may influence other aspects of this illness, including emotional processing.

Keywords

schizophrenia; working memory; prefrontal cortex; cognition

Lay conceptions of schizophrenia typically focus on readily observable symptoms such as hallucinations, delusions, and disorganized speech. However, individuals with schizophrenia also commonly have disturbances in memory and cognition. Interestingly, recent research suggests that disturbances in social and occupational functioning in individuals with schizophrenia may be more influenced by the severity of their cognitive deficits than by the severity of symptoms such as hallucinations and delusions (Green, Kern, Braff, & Mintz, 2000). Such findings have led to a resurgence of interest in understanding the kinds of cognitive deficits present in this illness. In particular, a large body of research has focused on the idea that one of the primary cognitive functions disturbed in schizophrenia is working memory (WM). Although individuals with schizophrenia likely have disturbances in a number of different cognitive func-

tions, I focus on WM for the same reasons that have driven much of the research on WM in schizophrenia: First, research with humans suggests that WM is crucial for a wide range of real-world outcomes and abilities. Second, a wealth of animal data indicates that the dopamine system (one of the brain's neurotransmitter systems) and prefrontal cortex (PFC; a region of the brain), both of which are impaired in schizophrenia, play a role in WM.

WM is often defined as the ability to maintain and manipulate information over short periods of time. Although this is a relatively straightforward definition, research has shown that WM involves several different processes. For example, Baddeley's (1986) influential theory of WM distinguishes among three sub-components, short-term holding areas (i.e., buffers) for visual information (the visual-spatial scratch pad) and verbal information (the phonological loop) and a central executive component that guides the manipulation and transformation of information held within the storage buffers. Here I briefly review the literature suggesting that individuals with schizophrenia have deficits in WM, and that these deficits are associated with disturbances in the function of the dopamine system in PFC. I argue that WM deficits in schizophrenia are more likely to reflect a problem with processes engaged by the central executive than a problem with the buffer systems. Further, I argue that such executive control deficits also influence many cognitive domains other than WM, including inhibition (the suppression of unwanted or irrelevant information), selective attention (the selection of task-relevant information), and episodic memory (learning and retrieval of new information), all of which may then contribute to disturbances in higher cognitive processes such as problem solving, reasoning, and language production.

WORKING MEMORY IMPAIRMENTS IN SCHIZOPHRENIA

Early research by Park demonstrated that individuals with schizophrenia are impaired on WM tasks that require remembering spatial locations over a delay period (Park & Holzman, 1992). Other studies have shown that individuals with schizophrenia are also impaired on tasks that require a relatively large amount of information to be maintained in WM, even when this information only needs to be maintained for a short time (Barch, Csernansky, Conturo, Snyder, & Ollinger, 2002). Even individuals who do not have schizophrenia but are at risk for the disorder for a variety of reasons have impaired WM function. For example, the first-degree relatives of individuals with schizophrenia demonstrate WM deficits (Park, Holzman, & Goldman-Rakic, 1995). In addition, individuals who score high on measures of proneness to psychosis and individuals diagnosed with schizo-

typal personality disorder (a personality disorder thought to share genetic liability with schizophrenia; Farmer et al., 2000) display WM deficits. These results suggest that deficits in WM may represent an aspect of vulnerability for the development of schizophrenia. Moreover, such studies with at-risk populations control for alternative interpretations of the cognitive dysfunction associated with schizophrenia, such as those having to do with medication, hospitalization, or social factors associated with being diagnosed with a psychiatric disorder.

THE NEUROBIOLOGY OF WORKING MEMORY IN SCHIZOPHRENIA

The cognitive neuroscience literature points to an important role for dopamine (a neurotransmitter) in the PFC in WM function (see Fig. 1). For example, nonhuman primate studies suggest that PFC neurons show sustained activity dur-

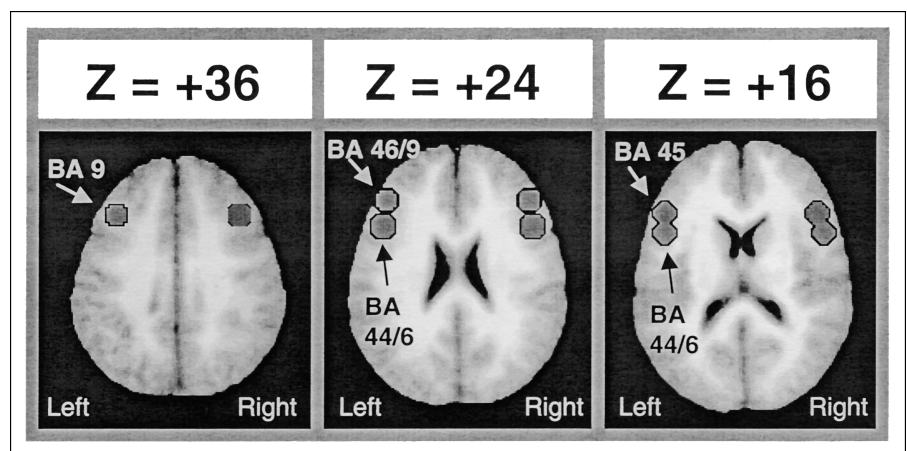


Fig. 1. Images of the brain highlighting regions of prefrontal cortex thought to be particularly important for working memory function. BA refers to Brodmann's areas, which are different areas of the brain defined by differences in the nature and organization of neurons in those areas. The regions labeled as BA 46 and BA 9 (on both the right and left sides of the brain) are often referred to as dorsolateral prefrontal cortex. The regions labeled as BA 44, 6, and 45 (on both the right and left sides of the brain) are often referred to as inferior or posterior prefrontal cortex. The Z numbers at the top of the figure refer to how many millimeters above a midpoint of the brain (the anterior and posterior commissures) each image is located.

ing delay periods when monkeys are remembering spatial locations and objects (Goldman-Rakic, 1987). The nonhuman primate literature also suggests that optimal dopamine function in PFC is important for good WM performance. Numerous human neuroimaging studies have shown activation of two different areas of the PFC in WM tasks. One of these areas is toward the top and toward the outer surface of the brain (see Fig. 1) and is referred to as dorsolateral prefrontal cortex. The other area is more toward the middle of the PFC (also toward the outer surface) and is referred to as inferior PFC (see Fig. 1).

Numerous studies of schizophrenia have shown abnormalities in PFC function during performance of WM tasks. Typically, these abnormalities involve reduced PFC activity in the individuals with schizophrenia, though a few studies have found increased PFC activity. There is also recent evidence that abnormal WM performance and PFC activity in schizophrenia reflect a disturbance in dopamine transmission (Abi-Dargham et al., 2002), which may be associated with mutations in genes governing dopamine metabolism (Egan et al., 2001).

WHAT ASPECT OF WORKING MEMORY IS IMPAIRED?

As I described earlier, Baddeley's (1986) model of WM distinguishes between buffer systems and a central executive that guides the manipulation of information held within the buffers. In this section, I discuss evidence indicating that schizophrenia is characterized by a disturbance in central executive processes, rather than in a particular buffer system.

A number of studies have shown that individuals with schizophrenia

tend to perform well when required to remember only a few items over a short period of time, especially when there are no other tasks to be done at the same time that could interfere with maintaining the information (Cohen, Barch, Carter, & Servan-Schreiber, 1999; Park & Holzman, 1992). Such WM tasks may primarily depend on short-term storage buffers and require little manipulation of information. However, when individuals with schizophrenia need to remember a larger number of items (which may require them to use strategies such as grouping different items together to remember the information) or when distracting information is introduced during the task (e.g., they have to remember information while performing an intervening task), their performance declines (Goldberg, Patterson, Taquq, & Wilder, 1998; Park & Holzman, 1992). WM theorists argue that central executive functions are particularly important when information in the buffers needs to be transformed in some way, or protected from interference. Thus, the conditions under which individuals with schizophrenia are particularly impaired suggest that they have a central executive deficit rather than a disturbance in the buffer systems.

Moreover, individuals with schizophrenia tend to show equal difficulty with WM tasks involving verbal (phonological loop), spatial (visual-spatial scratch pad), and object (visual-spatial scratch pad) information (Barch et al., 2002; Coleman et al., 2002). If WM deficits in schizophrenia were due to a disturbance in a specific buffer system, then one might expect to see greater problems for tasks that rely more on that system than the other. The fact that the data do not show this pattern suggests that the performance difficulties of individuals with schizophrenia result from poor central executive processes that are needed to support the ma-

nipulation of information or its protection from interference.

In addition, neurobiological evidence supports the hypothesis that the WM deficits in schizophrenia arise from the central executive. A number of theorists have argued that dorsolateral regions of PFC are critical for processes involved in the manipulation and transformation of information necessary to guide behavior (e.g., executive processes), whereas more inferior and posterior regions of PFC are more involved in the maintenance of information in WM through processes such as verbal rehearsal. In neuroimaging work, my colleagues and I have shown that schizophrenia is associated with deficits in dorsolateral PFC activity, but intact activity in more inferior and posterior regions of PFC (Barch et al., 2001, 2002). In addition, postmortem studies suggest that neuronal pathology in schizophrenia is selective to dorsolateral PFC and is not found in inferior PFC (Selemon, Mirzljak, Kleinman, Herman, & Goldman-Rakic, 2003). Such results indicate that the WM deficits associated with schizophrenia are due to a disturbance in central executive function supported by dorsolateral regions of PFC, rather than a disturbance in the kinds of rehearsal mechanisms that may be supported by more inferior PFC regions.

THE NATURE OF THE CENTRAL EXECUTIVE DEFICIT IN SCHIZOPHRENIA

Although I have argued that individuals with schizophrenia have a deficit in central executive function, it is informative to look more closely and consider what particular executive processes are disturbed. My colleagues and I have focused our work on a set of functions that we argue are critically

important to executive control, namely, the ability to represent and maintain context information (Braver, Barch, & Cohen, 1999; Cohen et al., 1999). By context information, we mean information relevant to the task at hand that is represented in a form that can bias selection of the appropriate behavioral response. For example, suppose you were reading the sentence "In order for the farmer to keep chickens, she needed a pen." In this example, the first part of the sentence (referring to the farmer and chickens) serves as context that biases you toward interpreting "pen" as referring to a fenced enclosure, rather than a writing instrument (the more common interpretation of "pen"). We view context as a subset of representations within WM that govern how other representations are used. Further, we have argued that context processing is one of the specific components of executive control supported by dopamine function in dorsolateral PFC, and that it is impaired in schizophrenia. These hypotheses have been implemented in formal computational models that can simulate behavior in WM tasks, and the results indicate that a deficit in context processing can account for the performance of individuals with schizophrenia (Barch et al., 2001; Braver et al., 1999; Cohen et al., 1999).

ARE THE COGNITIVE IMPAIRMENTS IN SCHIZOPHRENIA UNIQUE TO WORKING MEMORY?

Another important question is whether cognitive deficits in schizophrenia are limited to the domain of WM. The answer to this question is clearly no, because individuals with schizophrenia show disturbances in a variety of do-

main, such as inhibition, selective attention, episodic memory, problem solving, language, and some sensory and perceptual processes. This pattern makes sense if executive processes such as the processing of context are important for a range of cognitive functions. In fact, my colleagues and I have found that deficits across a number of task domains (i.e., inhibition, WM, selective attention, episodic memory) are correlated in schizophrenia (Barch et al., 2002; Cohen et al., 1999). We have also found that individuals with schizophrenia show impaired activity in the same regions of dorsolateral PFC for both WM and episodic memory tasks. Taken together, such results suggest that a common underlying cognitive function (or set of functions) that is impaired in schizophrenia gives rise to performance deficits in a range of task domains.

FUTURE DIRECTIONS

Despite the growing evidence for WM deficits in schizophrenia and their relationship to the dopamine system in PFC, a number of critical questions remain unanswered. First, it is not yet clear what role WM deficits play in the development or maintenance of schizophrenia. For example, one possibility is that deficits in WM directly contribute to the development of specific symptoms in schizophrenia. In particular, a number of studies have focused on the role that WM may play in the disturbances of language production displayed by individuals with schizophrenia (often referred to as formal thought disorder). This research has suggested that formal thought disorder may reflect, at least in part, an inability to maintain prior discourse context in WM, and to use this information to constrain ongoing language produc-

tion. Alternatively, however, WM deficits and symptoms in schizophrenia might have no direct causal relationship, and instead could be independent manifestations of one or more pathophysiological mechanisms.

A second major question is the extent to which deficits in WM (as well as other cognitive functions) are changeable and potentially amenable to treatment. Improving WM function in schizophrenia could have broad benefits given the data suggesting that functional outcome in schizophrenia is driven, at least in part, by the severity of cognitive dysfunction. Much of the existing longitudinal research on cognitive function in schizophrenia suggests that deficits in WM and other related cognitive functions tend to stay fairly stable, even when clinical symptoms change. In addition, initial research suggested that antipsychotic medications do not significantly alter WM function in schizophrenia. However, more recent research is more promising, with a number of studies demonstrating WM improvements following administration of the newer generation of antipsychotics. A growing area of research is focused on cognitive rehabilitation in schizophrenia. Results have been mixed, though some studies have found improvements in cognitive function after intervention, and one study showed improved PFC activity as well. Clearly, there is a need for more research on both psychological and pharmacological approaches to improving WM function in schizophrenia.

A third arena for future work on cognitive function in schizophrenia is the association between the impairment in WM and other aspects of this illness. Although researchers currently have little understanding of how deficits in emotion and cognition are related in schizophrenia, a growing literature suggests there are important links

between the two kinds of impairment. For example, the language production of individuals with schizophrenia is more impaired when they discuss negative topics than when they discuss neutral or positive topics (a phenomena referred to as affective reactivity of language). Furthermore, the severity of affective reactivity of language in schizophrenia is related to the severity of specific kinds of cognitive deficits, such as context-processing disturbances. At the same time, the literature suggests that the PFC and another brain region, the hippocampus, play important roles in regulating emotional processing. There is evidence that individuals with schizophrenia have disturbances in the hippocampus, as well as the PFC. Thus, it is possible that at least a subset of the emotional disturbances in schizophrenia reflect deficits in the ability to use context information to regulate emotional processing.

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Acknowledgments—This work was supported by Grants MH45156, MH47073, MH60887, and MH62130 from the U.S. National Institute of Mental Health and by a Young Investigators Award from the National Alliance for Research on Schizophrenia and Depression (NARSAD).

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