

## Early visual information processing in schizophrenia compared to recurrent depression

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Received 25 March 2003; accepted 28 June 2003

### Abstract

Patients with schizophrenia have repeatedly shown deficits in early visual processing using backward masking (VBM) tasks. Whether this represents a specific dysfunction in schizophrenia is an unsolved question. Patients with recurrent unipolar depression represent an interesting comparison group to examine the question of specificity, but have never previously been assessed on VBM. In addition to comparing VBM performance in patients with schizophrenia and patients with depression, we wanted to examine the relations between VBM and clinical symptoms. Fifty-one patients with schizophrenia were compared to 49 patients with recurrent unipolar depression and 47 healthy controls. All subjects were administered a two-digit identification task in a no-masking and four masking conditions. Patients with schizophrenia performed significantly worse than normal controls on four of the five conditions. No significant difference was found between depression patients and normal controls. The effect of masking stimuli had no differential effects on the three groups. VBM correlated strongly with positive symptoms in the schizophrenia group.

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*Keywords:* Schizophrenia; Depression; Information processing; Backward masking; Neuropsychology; Symptoms

### 1. Introduction

Patients with schizophrenia experience difficulties in a broad spectrum of neurocognitive domains, including attention and pre-attentional processes (Rund and Borg, 1998). Such basic information pro-

cessing deficits can be measured using visual backward masking (VBM) tasks. In VBM, a briefly presented initial (target) stimulus, usually letters or digits, is quickly replaced by a non-informative mask.

Two potential cognitive processes have been proposed to be involved in the masking phenomenon: integration and interruption (Rund, 1993; Turvey, 1973). Integration occurs when the target fuses with the mask and produces a confused image. Interruption is when processing of the target is disrupted by

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the mask so that further processing of the target does not occur.

Schizophrenic patients have repeatedly exhibited a deficit in such tasks (for overviews, see Green and Nuechterlein, 1999; Rund, 1993; Rund and Borg, 1998). Visual backward masking deficit in schizophrenia has been interpreted as slow information processing (Saccuzzo and Braff, 1981), as resulting from disrupted transient channels, disrupted sustained channels, or disruption in or between both of them (Balogh and Merritt, 1987), as abnormalities in transient channels, and that schizophrenic individuals have difficulties with interruptive mechanisms (Green et al., 1994), as dysfunction of lower-level visual pathways, most prominent for magnocellular biased stimuli (Butler et al., 2001), as a failure to decode the target stimuli, not simply a function of abnormalities due to overactive transient channel system (Weiss et al., 1992), and as reflecting susceptibility to sensorial interference (Suslow and Arolt, 1997).

Backward masking is considered to be a possible indicator of vulnerability to schizophrenia (Green and Nuechterlein, 1999) and as a candidate for a specific dysfunction in schizophrenia (Rund and Landrø, 1990). However, the question of specificity in schizophrenia remains unsolved. Dysfunctions at the earliest stages of information processing may play a prominent pathophysiological role and may provide important clues to the underlying etiology of schizophrenia (Butler et al., 2001).

There is also a general consensus that patients with mood disorders show cognitive deficits (Zakzanis et al., 1998). As to backward masking deficits, patients with mood disorder have shown inconsistent results. Saccuzzo and Braff (1981) failed to uncover deficits in this patient group. However, in a later report (Saccuzzo and Braff, 1986), they uncovered VBM deficits in schizo-affective and bipolar patients. Green and Walker (1986) found a non-significant trend for bipolar patients to perform worse than normal controls, and Fleming and Green (1995) reported that bipolar patients performed significantly worse than normal controls. Green et al. (1994) revealed that manic patients performed significantly worse than normal controls and comparatively with schizophrenic patients. MacQueen et al. (2001) found that euthymic bipolar disorder patients were significantly impaired compared to non-psychiatric controls on VBM tasks

as assessed by both speed and accuracy. Rund (1993) reported that affectively disturbed patients performed even poorer than schizophrenic patients. In addition, two recent studies (Green et al., 1997; Keri et al., 2001) have shown that unaffected relatives of schizophrenia patients also demonstrate a VMB deficit, although this was not found in relatives of patients with bipolar disorder (Keri et al., 2001).

To examine specificity for a VBM deficit, a relevant comparison group to schizophrenia would be a homogeneous group with recurrent unipolar major depression. There is some empirical evidence that some of the same brain areas, i.e. temporal lobe areas and primarily the hippocampus, are affected in schizophrenia and the recurrent type of depressive disorder (Bremner, 1999; Kirschbaum et al., 1996; Nelson et al., 1998). The two disorders are characterized by different symptomatology, however. On the VBM task, patients with unipolar depression previously only once have been compared to patients with schizophrenia (Suslow and Arolt, 1997, 1998). Results showed that paranoid schizophrenic patients made significantly more detection errors compared to depressive patients and non-psychiatric controls when stimulus intervals longer than 14 ms were used. However, in this study the group of depressive patients consisted of mainly single episode patients. In addition, Schubert et al. (1985) compared three groups of psychotic patients (manic, schizo-affective and major depressive) to borderline patients. All three psychotic groups performed poorer than borderline patients and normal controls. Among the psychotic groups, the schizo-affective patients were most impaired. Patient groups were extremely small, however, with only eight subjects in each group. Small sub-groups of patients with non-psychotic depression have also been included in some studies with larger samples of affective disorders, e.g. Saccuzzo and Braff (1981, 1986). These sub-samples have consistently shown no backward masking deficit. To our knowledge, patients with recurrent unipolar major depression have never been examined with the VBM paradigm.

Another association that has not been clarified is how VMB deficit is related to clinical symptoms. Suslow et al. (1998) examined the relation between VBM performance and clinical symptomatology in patients with schizophrenia. They found that a VBM deficit was related to the negative symptoms of affect-

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