



Review

Gray colored glasses: Is major depression partially a sensory perceptual disorder?



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ABSTRACT

Background: Major depression is a neuropsychiatric disorder that can involve profound dysregulation of mood. While depression is associated with additional abnormalities besides reduced mood, such as cognitive dysfunction, it is not well established that sensory perception is also altered in this disorder (aside from in psychotic depression). Recent studies have shown that visual processing, in as early a stage as the retina, is impaired in depression. This paper examines the hypothesis that major depression can involve alterations in sensory perception.

Methods: A Pubmed literature search investigated several lines of evidence: innervation of sensory cortex by serotonin and norepinephrine; antidepressant drugs and depression itself affecting processing of facial expressions of emotion; electroencephalography (EEG) studies of depressed persons and antidepressant drugs; involvement of the serotonergic 5HT2A receptor in both depression and hallucinogenic drug action; psychotic depression involving sensory distortions; dopamine possibly playing a role in depression; and the antidepressant effect of blocking the NMDA receptor with ketamine.

Results: Data from each of these lines of evidence support the hypothesis that major depression can involve sensory perceptual alterations.

Conclusions: Loss of interest in one's daily activities and inability to experience pleasure, also known as anhedonia, in major depression may in part be mediated by sensory abnormalities, whereby normal sensory perceptions are no longer present to activate reward circuitry.

Limitations: The data supporting the hypothesis tend to be associative, so further confirmation of the hypothesis awaits additional controlled experiments.

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1. Introduction

Major depression is a neuropsychiatric disorder that can be characterized by profound dysregulation of mood. Depression is also associated with other abnormalities, such as cognitive dysfunction, sleep and appetite disturbance, and fatigue (American Psychiatric Association, 2000). While the psychotic subtype of depression can be associated with hallucinations, it is not well established that sensory perception is altered in non-psychotic depression.

Another symptom in many cases of major depression is “markedly diminished interest or pleasure in all, or almost all, activities” (American Psychiatric Association, 2000). It is not clear how such loss of interest (which may be closely related to anhedonia) is mediated, including the brain mechanisms involved. A fairly recent study by Bubl and colleagues put forth the finding that in persons experiencing a major depressive episode, visual processing in as early a stage as the retina shows diminished contrast gain (Bubl et al., 2010). This adds to a previous finding by this group that visual contrast discrimination performance is behaviorally impaired in persons with major depression (Bubl et al., 2009). This group also recently found that reduced retinal contrast gain in persons with depression was normalized after these subjects achieved remission on antidepressant therapy (Bubl et al., 2012).

One possibility is that visual perception is altered in persons experiencing depression, and that recovery from depression is associated with normalization of visual perception. Likewise, other sensory modalities, such as hearing (Schwenzer et al., 2012) and touch (Freedman, 1994; Adler and Gattaz, 1993), may also be altered by major depression. Disease-related alterations in sensory perception may contribute to loss of interest or anhedonia by negatively impacting the production of “positive” or “hedonic” emotional responses that would have been produced by unaltered, non-pathological sensory perception that could more effectively activate brain reward circuits.

This paper puts forth the hypothesis that some cases of non-psychotic major depression are characterized by sensory perceptual alterations, in addition to more well-established symptomatology such as cognitive dysfunction or sleep disturbance. It is hypothesized that these sensory disturbances involve the visual system, but may also extend to hearing, touch, smell, and taste. While these putative sensory disturbances may include depression-related abnormalities in the periphery, such as the retina and the cochlea, a principal site of action is also assumed to be the brain, including its sensory cortical circuits. The proposed sensory alterations may involve changes in sensory acuity, such as decrease or increase in sensitivity, contrast, or gain. And they may include more complex transformations of the sensory stimulus, such that it less closely resembles its original form.

2. Literature search details

In what follows, I briefly describe several lines of evidence related to this hypothesis. These findings include: (1) innervation of sensory cortex by serotonin (5-hydroxytryptamine; 5-HT) and norepinephrine (NE); (2) antidepressant drugs affecting processing of facial expressions of emotion; (3) depression itself affecting processing of facial expressions; (4) electroencephalography (EEG) studies of depressed persons and antidepressant drugs; (5) involvement of the serotonergic 5HT2A receptor in both depression and hallucinogenic drug action; (6) psychotic depression involving sensory distortions; (7) dopamine possibly playing a role in depression, where this molecule is associated with hallucinations and antipsychotic drug action; and (8) the antidepressant effect of

blocking the NMDA receptor with dissociative drugs such as ketamine.

Pubmed searches, conducted as recently as June 2, 2013, included terms such as: depression tactile/auditory/visual perception, antidepressant/drug facial emotions, facial recognition emotions depression, visual N1 depression/antidepressant, LSD 5HT2A, hallucinations psychotic depression, schizophrenia dopamine D2 hallucinations, dopamine/antipsychotic depression, NMDA receptor/ketamine depression.

3. Innervation of sensory cortex by serotonin and norepinephrine

5-HT and NE, two neurotransmitters that have been extensively associated with major depression, innervate sensory cortex (Brown et al., 1979), and thereby may have the ability to modulate sensory perception. A neuroanatomical study of monkeys found that the visual portion of inferotemporal cortex, which is specialized for processing complex stimuli such as faces, is very densely innervated by serotonergic fibers and very lightly innervated by noradrenergic ones (Morrison and Foote, 1986). Ionophoretic application of 5-HT or NE to somatosensory cortex affects response properties of individual neurons (Bassant et al., 1990). Also, a more recent study (Maya Vetencourt et al., 2008) showed that the serotonergic antidepressant fluoxetine affects neuronal plasticity in adult rat visual cortex. A recent study suggested that whereas magnetic resonance spectroscopy data have consistently shown reduced brain concentrations of the inhibitory neurotransmitter GABA in acute depression, measures of this transmitter were normal in remitted depression (Shaw et al., 2013).

4. Antidepressants affecting processing of facial expressions

Acute or subchronic administration of serotonergic or noradrenergic antidepressants modulates behavioral recognition of facial expressions of particular emotions, both in healthy controls alone (Harmer et al., 2003; Harmer et al., 2004), and in depressed individuals relative to healthy controls (Harmer et al., 2009). These three studies were double blind and placebo controlled. In particular, healthy female volunteers given an acute infusion of the 5-HT boosting drug, citalopram, detected a higher number of facial expressions of fear and happiness relative to other expressions (Harmer et al., 2003). Subchronic administration of citalopram or the NE boosting drug, reboxetine, to healthy male and female volunteers, resulted in reduced identification of facial expressions of anger and fear (Harmer et al., 2004). In depressed subjects, acute administration of reboxetine increased recognition of positive facial expressions (Harmer et al., 2009). In another study by this group, the beta blocking drug propranolol, which antagonizes NE receptors, selectively impaired recognition of facial expressions of sadness when given to healthy volunteers (Harmer et al., 2001).

5. Depression affecting processing of facial expressions

Depression itself, which may involve alterations in the endogenous serotonergic and noradrenergic systems, affects processing of facial expressions of emotion. Consistent with major depression being associated with alteration of visual perception, persons with this disorder exhibit changes in processing of facial expressions, including sadness and happiness (Rubinow and Post, 1992). A recent study found that major depression alters speed of access to awareness of facial expressions of sadness and happiness, providing direct support for the main hypothesis of this paper (Sterzer et al., 2011). In persons whose task was to identify facial

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