



## NAG level differences in panic disorder and agoraphobia

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### Abstract

It is not clear if panic disorder (PD) and agoraphobia are variants of the same disorder or distinct diseases. A laboratory test could help resolve this issue. Research has shown that levels of the urinary lysosomal enzyme *N*-acetyl- $\beta$ -glucosaminidase (NAG) differ between patients with various psychiatric disorders. This study examined whether NAG levels would be similar in PD and agoraphobia, suggesting the two disorders may be the same disorder, or different, suggesting they may be distinct diseases. Differences found could suggest either qualitative or quantitative distinctions between these disorders. Ninety-one agoraphobics were compared to 24 patients with panic disorder. NAG levels were significantly lower in panic patients compared to agoraphobic patients  $9.7 \pm 8$  versus  $22 \pm 21$ ;  $P < .005$ . These data provide limited support for the hypothesis that PD and agoraphobia may be distinct diseases.

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It is not clear whether agoraphobia is a variant of panic disorder (PD) or a discrete illness. Several articles related to this issue have been reviewed (McNally, 1994; Noyes & Hoehn-Saric, 1998). Evidence that agoraphobia and PD are variants of the same illness is as follows. Most agoraphobic patients recall the occurrence of panic attacks before the onset of their agoraphobia (Aronson & Logue, 1987; Breier, Charney, & Heninger, 1986; Lelliott, Marks, McN

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amee, & Tobena, 1989; Starcevic, Kellner, Uhlenhuth, & Pathak, 1993; Thyer & Himle, 1985; Uhde et al., 1985). Several investigators have found few demographic differences between groups (Garvey & Tuason, 1984; Noyes, Clancy, Garvey, & Anderson, 1987; Thyer, Himle, Curtis, Cameron, & Nesse, 1985). For example the age of onset and sex ratio is similar for both groups of patients. Agoraphobia and PD respond to the same medications, a finding consistent with the postulate that these disorders may have similar biological abnormalities (Noyes et al., 1987).

Some researchers espousing a unitary view of panic disorder and agoraphobia suggest that agoraphobia may be a more severe variant of panic disorder (McNally, 1994). For example, PD is more likely to show remissions or improvement over time, than is agoraphobia.

Frequency of agoraphobia and PD within families could help illuminate the possible relationship of these two disorders. A family interview study showed that first degree relatives of agoraphobic probands, when compared to controls, had an increased age adjusted morbidity risk for agoraphobia (12%) and panic disorder (8%) (Noyes et al., 1986). Panic disorder probands had an increased risk for PD (17%) but not for agoraphobia. A parsimonious explanation for this data is that agoraphobia is a more severe variant of panic disorder.

Alternatively, there is some support for the idea that agoraphobia and PD are discrete disorders. One group of investigators reported that agoraphobia occurred before panic attacks in as many as 90% of patients (Fava, Grandi, & Canestrari, 1988; Fava, Grandi, Rafanelli, & Canestrari, 1992). ECA interviews of community samples indicate that agoraphobia without panic was more common than agoraphobia with panic (McNally, 1994). However, a follow-up of ECA patients at one site suggested that agoraphobia without panic attacks was incorrectly diagnosed in many cases (Horwath, Lish, Johnson, Hornig, & Weissman, 1993). In some patients the apparent existence of agoraphobia without panic may be the result of diagnostic confusion between agoraphobia and specific phobias. A review of several hundred cases of agoraphobia suggested that agoraphobia without panic attacks was very uncommon (McNally, 1994). At the present it appears that the weight of evidence favors the hypothesis that PD and agoraphobia are most likely variants of the same disorder. Laboratory testing could help clarify this issue. In many fields of medicine validation of diagnostic categories is based on laboratory tests.

There is a variety of evidence that serotonin may be involved in the pathogenesis of anxiety disorders (Kahn, Kalus, Wetzler, & van Praag, 1991; Stein & Stahl, 2000) In previous studies we found that *N*-acetyl- $\beta$ -glucosaminidase (NAG) an cellular enzyme was associated with urinary levels of the serotonin metabolite 5-hydroxyindole acetic acid (5-HIAA) (Garvey, Noyes, Woodman, & Laukes, 1995). In another investigation NAG was associated with the density of imipramine binding sites (Garvey & Black, 1993). These

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