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LETTER TO THE EDITORS

Serotonin transporter binding in eating disorders

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A recent report suggested that patients who are in remission from an eating disorder have "divergent 5-HTT (serotonin transporter, *our addition*) activity" in some brain areas and raised the possibility that "this is a premorbid vulnerability for developing ED (eating disorder, *our addition*) symptoms" (Bailer et al. 2007). However, the patients also had increased levels of perfectionism and anxiety, and the alteration in 5-HTT activity may, therefore, be related to these symptoms rather than the previous eating disorder.

With one exception (Bergh et al. 2002), there are no effective treatments for patients with eating disorders, such as anorexia and bulimia nervosa (Berkman et al. 2007). As a consequence, patients who are discharged from treatment are never free of symptoms. While they may be in remission from their eating disorder, they typically display symptoms such as perfectionism and anxiety (Bailer et al. 2007), and most often, they relapse within a year or less (Berkman et al. 2007).

There are two ways to interpret this situation. Firstly, there may be something wrong with a patient, e.g., "divergent 5-HTT activity" in the brain, which puts her/him at risk of developing an eating disorder and persists at remission. This is the common interpretation (e.g., Bailer et al. 2007). Secondly, it is possible that there is something wrong with the treatments offered to patients with eating disorders (Södersten et al. 2006). Patients who are treated to a full remission, i.e., display no symptoms at discharge, run a less than 10% risk of relapse during a 5-year period of follow-up (Bergh et al. 2002).

The recent report by Bailer et al. (2007) provides an example of the first interpretation. Thus, as the patients in that report showed increases in perfectionism and anxiety, some patients had even elevated levels of obsessive compulsive disorder, it cannot be excluded that the "divergent 5-HTT activity" that Bailer et al. (2007) reported is an epiphenomenon to these symptoms and unrelated to the eating disorder. This is a problem with all studies on biological changes among eating disorder patients (Södersten et al. 2006). Most of these changes, as well as their psychological concomitants are reversible consequences of starvation, and unless the patient is properly treated, some of the symptoms persist, and the patient merely goes into partial remission and remains at risk of relapse (Södersten et al. 2006).

We do not know, therefore, whether the "divergent 5-HTT activity" in the brain reported by Bailer et al. (2007) is a cause or a consequence of an eating disorder.

There is another issue that needs clarification in the report of Bailer et al. (2007). Thus, the standard deviations in their Table 1 are greater than the associated mean values in 13 instances. For example, recovered anorexic women, restricting type (REC RAN) are reported to have a blood level of estradiol of 19.56 (26.68) pg/ml. This implies that in some women, blood levels of estradiol were negative, which is impossible.

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Conflict of interest statement Drs. Bergh (27.5%) and Södersten (27.5%) are owners of the Mandometer Clinics together with Investor AB (40%) and Dr. M Leon (5%).

References

Bailer UF, Frank GK, Henry SE, Price JC, Meltzer CC, Becker C, Ziolko SK, Mathis CA, Wagner A, Barbarich-Marsteller NC, Putnam K, Kaye WH (2007) Serotonin transporter binding after recovery from eating disorders. Psychopharmacology (Berl) 195:315–324

Bergh C, Brodin U, Lindberg G, Södersten P (2002) Randomized controlled trial of a treatment for anorexia and bulimia nervosa. Proc Natl Acad Sci U S A 99:9486–9491

Berkman ND, Lohr KN, Bulik CM (2007) Outcomes of eating disorders: a systematic review of the literature. Int J Eat Disord 40:293–309

Södersten P, Bergh C, Zandian M (2006) Psychoneuroendocrinology of anorexia nervosa. Psychoneuroendocrinology 31:1149–1153