What is the evidence basis for existing treatments of eating disorders?

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Most existing treatments of eating disorders (ED) produce a period of remission that is short lived and expressed in fewer than 50% of the patients. Antidepressants (*eg*, selective serotonin reuptake inhibitors [SSRI]) have a small effect in bulimia nervosa and they are not recommended in anorexia nervosa (AN) because serotonin inhibits food intake. In a randomized, controlled trial, training of eating behavior and satiety, supply of warmth, reduction of physical hyperactivity, and restoration of social activities brought 75% of patients with ED into remission, and 93% remained in remission during follow-up. Further randomized, controlled trials comparing presently used interventions will provide the evidence needed to improve the treatment of ED. Curr Opin Pediatr 2003, 15:344–345

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Abbreviations

AN	anorexia nervosa
ED	eating disorders
SSRI	selective serotonin reuptake inhibitors

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The effect of most treatments of ED is unknown [1]. However, evidence from randomized, controlled trials supports the use of family therapy in young, mildly ill patients with AN, although the effect is small and short lived [2]. Randomized, controlled trials also support the use of cognitive behavioral therapy in bulimia nervosa, but 20% of the patients drop out of the treatment, fewer than 50% respond, and more than 40% relapse within 4 months after treatment [3].

Most drugs have no effect on ED [4]. However, indirect serotonergic agonists (*eg*, SSRI), may be useful in bulimia nervosa, although their effect is smaller than that of cognitive behavioral therapy [4]. Because serotonin can inhibit food intake [5], gonadotropin secretion [6], and sexual behavior [7], the widespread use of SSRI in underweight, peripubertal women with AN, often with primary amenorrhea, is surprising. Moreover, SSRI have no effect on the psychopathology of AN [8] and can reduce body weight [9]. Accordingly, the American Psychiatric Association does not support the use of SSRI in the management of underweight patients with AN [10].

Thus, the use of SSRI in AN is inconsistent with the role of serotonin in neuroendocrine regulation as is their use in preventing relapse in patients with AN who are in remission. This was tried in a widely cited study, which, however, reported a marked and rapid relapse among placebo-treated controls (84% relapsed in 4 months), which was more conspicuous than the slight reduction of that among the SSRI-treated patients (37% relapsed in 8 months—also a rapid and high rate of relapse) [11].

Perhaps because of the modest success of most treatment methods, ED are considered multifactorial disorders, of unknown etiology and with many different causes.

As an alternative, we have suggested that both AN and bulimia nervosa are caused by the same two risk factors: reduced food intake and enhanced physical activity, and that they are maintained by activation of the neural networks of reward and attention [12]. On this framework, we have developed a method by which all ED are similarly treated. Training of eating behavior and satiety by computer support, reduction of physical hyperactivity, supply of warmth, and restoration of social function are its central features [13]. Psychopharmacologic drugs are withdrawn within the first months of treatment. The treatment was found effective in a randomized trial using

an untreated control group. The estimated rate of remission was 75% and the rate of relapse was 7% in a large group of patients [13].

The importance of reducing physical hyperactivity and supplying external heat in the management AN was emphasized in Gull's original description of the disorder [14]. Recent animal experiments [15], clinical case reports [16], and our study [13] suggest a causal link between warmth, reduced physical activity, and recovery from ED. As has been pointed out [1], however, randomized, controlled trials are needed to determine more precisely which interventions are effective in the management of ED and which are not.

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