European Eating Disorders Review
Eur. Eat. Disorders Rev. **12**, 333–336 (2004)

Viewpoint
Mandometer Treatment of Eating Disorders; A Reply

Cecilia Bergh and Per Södersten
Karolinska Institutet, Centre for Eating Disorders, Novum, Huddinge, Sweden

In a recent Viewpoint paper, Schmidt (2003) offers her views on our method of treating eating disorders after hearing a presentation by Bergh at a meeting in Helsinki. We would like to correct some misunderstandings that she has regarding the treatment that we have developed.

In our framework, reduced food intake and enhanced physical activity are the principle risk factors for eating disorders, rather than an unspecified mental disorder. In support of this notion are data collected by Keys and collaborators, who decreased the food available to mentally and physically healthy men. These men developed most of the symptoms of eating disorders, including the psychiatric symptoms (Keys, Brozek, Henschel, Mickelsen, & Taylor, 1950), indicating that an eating disorder can be developed without an antecedent mental disorder.

Dr Schmidt wonders how our treatment is related to this model. Specifically, on the basis of this information, we suggested that the behavioural, physiological and psychological changes in eating disorders are the result of eating too little, rather than its cause. To treat this problem, we retrain affected individuals to eat normally via continual feedback about their food intake during meals, and as they learn to eat normally, the symptoms of their mental disorders disappear (Bergh, Brodin, Lindberg, & Södersten, 2002). We facilitate this process by keeping the patients warm, since warmth reduces hyperactivity and facilitates eating in animal models of anorexia (Morrow et al., 1997), while conserving calories otherwise used to maintain thermal homeostasis in a body with a relatively small biomass accompanied by a relatively normal surface area that allows disruptive heat loss. We also include procedures for reducing physical activity to restrict the use of calories for that purpose. Patients also are trained to return to their ordinary living conditions by maintaining their schooling, their social life, and their physical appearance. We treat patients with anorexia or bulimia similarly both because we find the symptoms of the patients are similar and a similar treatment is equally successful for both disorders. Dr Schmidt sees elements of different treatment approaches which have either not been used in eating disorders, or have been used ineffectively in that regard. It should be even more interesting that these elements work together in our treatment. She also notes that we had used cisapride to facilitate intestinal flow in those patients who required such treatment, but we of course stopped such treatment at the same time as all others when concerns over its safety were voiced. We also withdrew patients from selective serotonin reuptake inhibitors (SSRIs), as these drugs suppress food intake (Bergh, Eriksson, Lindberg, & Södersten, 1996).

Dr Schmidt also indicated a desire to know more about the population served by our clinic and the method for recruitment in the study, suggesting that our patients were unusual in that those on the waiting list remained there even as their condition did not improve. All patients were from the Stockholm area and all had failed in treatment in at least two other clinics that had been using standard of care treatments. Rather than being mildly affected, as Dr Schmidt suggests, these were very ill individuals. She also notes that an unusually large proportion of our subjects were inpatients; that situation existed because the patients were very seriously affected by their eating disorder and had to start their treatment as inpatients. It seems more than odd that Dr Schmidt would refer to our study patients as having a'. . . a short duration of illness and relatively mild anorexia nervosa and bulimic patients whose symptom severity is not adequately described'.

*Correspondence to: C. Bergh, Karolinska Institutet, Centre for Eating Disorders, Novum, S-141-57 Huddinge, Sweden. E-mail: cecilia.bergh@neurotec.ki.se

Published online in Wiley InterScience (www.interscience.wiley.com). **DOI**: 10.1002/erv.610

The anorexics in the study had a median body mass index (BMI) of 15, ranging as low as 10.8; to regard these individuals as mildly affected is simply incomprehensible. Moreover, their abnormal depression, anxiety, obsession and eating disorder inventory status was also reported, along with their age, duration of disorder, BMI, number of previous treatments, daily binge eating, daily vomiting, attempted/considered suicide, use of psychopharmacological drugs, alcohol consumption, smoking, presence of headache, nausea, fatigue, insomnia, dyspepsia/constipation, lanugo hair, menstruation, as well as heart rate and blood pressure. It is not clear what other measures would have made her more familiar with the characteristics of the subjects in our study.

Dr Schmidt's notion that this treatment was effective only for young patients is undermined by that fact that our patients ranged in age from 10–33 years old. The likely reason that they refrained from seeking out alternative forms of therapy as they waited for our treatment is that they all had been through those other forms of treatments with no improvement. The reason that no additional details were given about the treatment in the long-term follow-up study is that the treatment was identical to that reported in the randomized clinical trial. Finally, Dr Schmidt does not seem to understand how a rapid and long-lasting remission of seriously affected eating disorder patients would be more cost effective than the chronic treatment of such patients as they repeatedly become hospitalized over many years.

Dr Schmidt refers to our randomized clinical trial as 'very small'. A power calculation was done using our preliminary results to estimate the appropriate number of patients needed to define a statistically valid conclusion and the effect of our treatment was statistically significant (Bergh et al., 2002). However, the statistical significance of the effect would not have been more impressive had it been obtained on a larger group of patients. If an experimenter injected five experimental rats with arsenic and five control rats with the vehicle and found that all the arsenic-treated rats died but that none of the controls died, one should not inject more rats to find out if arsenic is lethal. Instead, one should conclude that arsenic has a strong effect on rats ($p = 0.004$ using a one-sided Mann–Whitney U-test). More importantly than the size of the groups, our study used a minimum of exclusion criteria, and it is therefore likely that the results are applicable to a broader range of patients than would have been the case had the patients been more narrowly defined. We have now successfully treated over 200 patients and we continue to show significant improvement in about 90% of even the most severely affected patients and complete remission in about 75% of those serious cases. As important, we find that only about 10% of those in remission relapse over a 5-year period.

That Dr Schmidt regards these results as being typical of what others report is astonishing; there are no published reports of success rates anywhere near these. Dr Schmidt is incorrect in arguing that family-based interventions have good long-term effects on outcome in young anorexic patients. The best studies on this topic are the randomized clinical trial by Russell, Szmukler, Dare, and Eisler (1987) and the follow-up by Eisler, Dare, Russell, Szmukler, & Dodge (1997). These authors concluded that much of the improvement at follow-up can be 'attributed to the natural outcome of the illness' (Eisler et al., 1997). In other words, the treatment did not have a major effect on the outcome of the disorder.

The subsequently published studies on the same topic are, if anything, even less convincing that there is an effective standard of care treatment (Eisler et al., 2000; Robin et al., 1999). Indeed, the only study showing an effect of treatment in adult anorexic patients reported that cognitive behavioural therapy reduced the rate of relapse from 53 to 22% compared to nutritional counselling during only a 1-year period (Pike, Walsh, Vitousek, Wilson, & Bauer, 2003). These rates of relapse are much higher than those that we reported, the patients were followed for a fraction of our follow-up period, and the authors report no improvement in the rate of remission.

The evidence for the claim that cognitive behavioural therapy is effective in bulimia (Fairburn & Harrison, 2003) also is weak. A recent series of studies showed that only 27 (19%) out of 194 patients who entered such therapy for bulimia were in remission 4 months after treatment (Agras et al., 2000; Halmi et al., 2002; Mitchell et al., 2002). Clearly, outcome in both anorexia (Steinhausen, 2002) and bulimia (Quadflieg & Fichter, 2003) remains poor using the treatments that are commonly in use.

The other issues regarding our data that Dr Schmidt raises also are somewhat puzzling. She suggests that the anorexics are underweight at remission, but they are clearly not underweight. Indeed, the only case where a patient had a BMI at remission as low as 15.5 was 10 years old, a normal BMI for that age. She further notes that we did not restore menstruation in all of our patients. However, we explain in our report that some of our patients were prepubertal (such as that 10-year-old), and it therefore

would have been inappropriate to include menstruation as a condition of remission. Our remission criteria did include having normal blood tests, having normal eating patterns, having a BMI in the normal range, having normal psychiatric profiles (including a lack of compensatory behaviours in bulimics), being back in school or work and having re-established their normal social interactions. The patients also must have been able to say that they did not consider eating to be a problem for them. These high standards for remission probably form the basis for our low relapse rate.

Our study also includes a description of the outcome of all the patients that we had treated through October 31, 2000. Dr Schmidt's review of this part of our study constitutes a series of misunderstandings regarding our data. We have noted that many clinical researchers have insufficient knowledge of survival analysis and methods of statistical estimation. Space does not permit extensive discussion of these topics here. Suffice it to mention that all patients in a clinical population should be included is such estimates, independent of how long they have been in the treatment programme and that the Kaplan–Meier plot indicates the estimated probability that the patients will remain 'not in remission' at each time point (Pocock, 1998).

Dr Schmidt notes that 'In the absence of data, feelings tend to rule', to which we would respond that in this case, feelings tend to rule even in the presence of data. Indeed, it is interesting that Dr Schmidt's comments follow the classic response to new treatments in medicine. When the data are published, even in a rigorously reviewed journal such as the *Proceeding of the National Academy of Sciences*, the findings are ignored, trivialized or attacked. There are typically expressions of scepticism or disbelief, along with a careful misreading of the data and conclusions, followed by a dismissal of the new findings as no better than current ineffective treatments (Thagard, 2000). One can only hope that our approach to the treatment of eating disorders will be accepted in a shorter period of time than it took for British physicians to treat scurvy properly (50 years after its cause had been discovered; Brown, 2004), or for American physicians to begin using antibiotics to treat ulcers (only 25% of such cases were thus treated more than a decade after its bacterial cause was found; Munnangi & Sonnenberg, 1997). One despairs at having evidence-based medicine play a significant role in the shaping of modern medical practice.

As Dr Schmidt proposes, our treatment should be evaluated further with a randomized clinical trial against the standard of care. However, from a statistical point of view, the standard of care should have some demonstrable effect before it would be appropriate to be used as a point of comparison. There is no compelling evidence that any of the standard treatments that are presently used to treat eating disorders are effective, let alone as effective as the treatment that we have described.

REFERENCES

Agras, W. S., Crow, S. J., Halmi, K. A., Mitchell, J. E., Wilson, G. T., & Kraemer, H. C. (2000). Outcome predictors for the cognitive behavior treatment of bulimia nervosa: Data from a multisite study. *American Journal of Psychiatry, 157*, 1302–1308.

Bergh, C., Brodin, U., Lindberg, G., & Södersten, P. (2002). Randomized controlled trial of a treatment for anorexia and bulimia nervosa. *Proceedings of the National Academy of Sciences, U.S.A., 99*, 9486–9491.

Bergh, C., Eriksson, M., Lindberg, G., & Södersten, P. (1996). Selective serotonin reuptake inhibitors in Anorexia. *The Lancet, 348*, 1458–1459.

Brown, S. R. (2004). *Scurvy: How a surgeon, a mariner, and a gentlemen solved the greatest medical mystery of the age of sail*. London: Summersdale Publishers.

Eisler, I., Dare, C., Russell, G. F. M., Szmukler, G., & Dodge, E. (1997). Family and individual therapy in anorexia nervosa. *Archives of General Psychiatry, 54*, 1025–1030.

Eisler, I., Dare, C., Hodes, M., Russell, G., Dodge, E., & le Grange, D. (2000). Family therapy for adolescent anorexia nervosa: The result of a controlled comparison for two family interventions. *Journal of Child Psychology and Psychiatry, 41*, 727–736.

Fairburn, C. G., & Harrison, P. J. (2003). Eating disorders. *Lancet, 361*, 407–416.

Halmi, K., Agras, W. S., Mitchell, J., Wilson, G. T., Crow, S., Bryson, S. W., & Kraemer, H. (2002). Relapse predictors of patients with bulimia nervosa who achieved abstinence through cognitive behavioral therapy. *Archives of General Psychiatry, 59*, 1105–1109.

Keys, A., Brozek, J., Henschel, A., Mickelsen, O., & Taylor, H. L. (1950). *The biology of human starvation*. Minneapolis, MN: The University of Minnesota Press.

Mitchell, J. E., Halmi, K., Wilson, G. T., Agras, W. S., Kraemer, H., & Crow, S. (2002). A randomized secondary treatment study of women with bulimia nervosa who fail to respond to CBT. *International Journal of Eating Disorders, 32*, 271–281.

Morrow, N. S., Schall, M., Grijalva, C. V., Geiselman, P. J., Garrick, T., Nuccion, S., & Novin, D. (1997). Body temperature and wheel running predict survival times in rats exposed to activity-stress. *Physiology and Behavior, 62*, 815–825.

Munnangi, S., & Sonnenberg, A. (1997). Time trends of physician visits and treatment patterns of peptic ulcer disease in the United States. *Archives of Internal Medicine, 175*, 1489–1494.

Pike, K. M., Walsh, B. T., Vitousek, K., Wilson, G. T., & Bauer, J. (2003). Cognitive behavior therapy in the

posthospitalization treatment of anorexia nervosa. *American Journal of Psychiatry, 160*, 2046–2049.
Pocock, S. J. (1998). *Clinical Trials*. New York: Wiley.
Quadflieg, N., & Fichter, M. M. (2003). The course and outcome of bulimia nervosa. *European Child and Adolescent Psychiatry, 12*(Supplement 1), 199–109.
Robin, A. L., Siegel, P. T., Moye, A. W., Gilroy, M., Dennis, A. B., & Sikand, A. (1999). A controlled comparison of family versus individual therapy for adolescents with anorexia nervosa. *Journal of the American Academy of Child and Adolescent Psychiatry, 38*, 1482–1489.
Russel, G. F. M., Szmukler, G. I., Dare, C., & Eisler, I. (1987). An evaluation of family therapy in anorexia nervosa and bulimia nervosa. *Archives of General Psychiatry, 44*, 1047–1056.
Schmidt, U. (2003). Mandometer musings. *European Eating Disorder Reviews, 11*, 1–6.
Steinhausen, H.-C. (2002). The outcome of anorexia nervosa in the 20th century. *American Journal of Psychiatry, 159*, 1284–1293.
Thagard, P. (2000). *How scientists explain disease*. Princeton, NJ: Princeton University Press.

