altered by establishment of such conditioned reward systems during a critical developmental period. This sensitization process might parallel the induction of drug addiction in animal models[4], being facilitated in the human situation by depression, anxiety and stress.

The addictive quality of anorexia nervosa has been noted with the endorphin system implicated, and the association between self-starvation and overexercise in female rats was described almost three decades ago[5]. It has been suggested from more recent work using this paradigm, that hyperactivity does not result from reward mechanisms but represents hypothalamic compensation for starvation-induced reduction of noradrenergic turnover[6]. In anorexia nervosa, not all patients demonstrate increased exercise salience, although overexercise may precede self-starvation and in severe emaciation may be reduced to aimless restlessness[7].

Anorexia nervosa cannot be regarded simply as synonymous with self-starvation, although this component is critical, and conditioning to perverse rewards underlies the ethical challenge of treatment[8].

JANICE D. RUSSELL & GLENN E. HUNT
Department of Psychological Medicine
The University of Sydney
Sydney, NWS, 2006, Australia

Bergh & Södersten reply — The prognosis of anorexia nervosa is extremely poor (see table), and no effective treatment has been reported[2]. This alarming situation calls for new ways of looking at the disorder. Our Commentary offers a start by pointing out two factors known to precede the onset of the illness: reduced intake of food and enhanced physical activity. Russell and Hunt agree that reduced feeding is a risk factor but neglect the other one, which is equally important[9].

Beumont and colleagues have provided a long list of physical complications that result from starvation[2]. We suggest that "psychopathology" also results from starvation. The reason is simple: no one has shown that there is a psychopathology that causes the illness. If there were, one would have to conclude that psychopathology is several times more common among female athletes because the illness is several times more common among these[10]. This conclusion would be absurd. If, on the other hand, enhanced physical activity is a risk factor, the high incidence of anorexia among athletes is not surprising.

Prognosis for patients with anorexia nervosa

Years after onset	Recovered	Improved	Ill	Dead
10 (ref. 13)	25%	–	68%	6.6%
20 (ref. 14)	30%	33%	22%	15%

By stating that "eating disorders are associated with a range of conditions resulting from abnormal reward-seeking behaviors [for example, kleptomania] that might be seen to arise when hunger is not satisfied by food ingestion," Russell and Hunt put psychiatric labels on, rather than giving a scientific analysis to, the fact that bulimics (the condition to which they refer) steal things. An alternative and simple explanation is that it is expensive to be bulimic. Between 1985 and 1987, we interviewed 42 women and 3 men fulfilling DSM-III criteria of bulimia nervosa, and 62% considered economic problems the worst aspect of bulimia; the median cost of binge eating was $50 per day (today's dollars); 55% of the patients had stolen (food, money or clothes) at least once; and 27% had stolen more often. These data were validated against official records. Twenty-six percent of the patients had been given a psychiatric diagnosis (neurosis, schizophrenia, pathological personality type, affective psychosis) as their main diagnosis[11]. Psychiatric labels can obviously obscure the situation for bulimic patients and therefore possibly misdirect treatment.

Anorexia nervosa is considered a multifactorial disorder. This means that it can be caused by almost anything, including factors such as "abusive antecedents,"[8] although there is no scientific support for this[12]. How can a clinician be expected to treat a patient with this perspective? We suggest that the lack of a clear theoretical starting point is one reason for the poor results of most treatment programs.

We consider treatment, founded on a simple neurobiological hypothesis, to be an advantage. On the basis of our hypothesis we have developed a method of treatment and have treated eight self-starving patients, all of whom have recovered after a median of seven months (manuscript submitted), a major improvement over earlier reports[2].

CECILIA BERGH & PER SÖDERSTEN
Department of Clinical Neuroscience
Division of Applied Neuroendocrinology and Center for Eating Disorders
Karolinska Institute
Novum, S-141 57 Huddinge, Sweden

1. Bergh, C. & Södersten, P. Anorexia nervosa, self-starvation and the reward of stress. *Nature Med.* **2**, 21–22 (1996).
2. Beumont, P.J.V., Russell, J.D. & Touyz, S.W. Treatment of anorexia nervosa. *Lancet* **341**, 1635–1640 (1993).
3. McElroy, S., Keck, P. & Phillips, K. Kleptomania, compulsive buying and binge-eating disorder. *J. Clin. Psych.* **5** (suppl. 5), 614–626 (1994).
4. Robertson, T.E. & Berridge, K.C. The neuronal basis of drug craving: An incentive-sensitization theory of addiction. *Brain Res. Rev.* **18**, 247–291 (1993).
5. Routtenberg, A. Self starvation of rats living in activity wheels: Adaptation effects. *J. Comp. Psychol. Physiol.* **66**, 234–238 (1968).
6. Broocks, A., Liu, J. & Pirke, K.M. Semistarvation-induced hyperactivity compensates for decreased norepinephrine and dopamine turnover in the mediobasal hypothalamus of the rat. *J. Neurol. Trans.* **79**, 113–124 (1990).
7. Beumont, P.J.V., Arthur, B., Russell, J.D. & Touyz, S.W. Excessive physical activity in dieting disorder patients: Proposals for a supervised exercise program. *Int. J. Eating Disord.* **15**, 21–36 (1994).
8. Russell, J. Treating anorexia nervosa. *Br. Med. J.* **311**, 584 (1995).
9. Davis, C. *et al.* The role of physical activity in the development and maintenance of eating disorders. *Psychol. Med.* **24**, 957–967 (1994).
10. Sundgot-Borgen, J. Prevalence of eating disorders in elite female athletes. *Int. J. Sport Nutr.* **3**, 29–40 (1993).
11. Bergh, C. An explorative study of bulimia and other excessive behaviours. thesis, Karolinska Institute (1988).
12. Pope, H.G. & Hudson, J.I. Is childhood sexual abuse a risk factor for bulimia nervosa? *Am. J. Psychiatr.* **149**, 455–463 (1992).
13. Eckert, E.D. *et al.* Ten year follow-up of anorexia nervosa: Clinical course and outcome. *Psychol. Med.* **25**, 143–156 (1995).
14. Ratnasuriya, R.H. *et al.* Anorexia nervosa: Outcome and prognosis factors after 20 years. *Br. J. Psychiatr.* **158**, 495–502 (1991).