Objective: To review the eating disorder research investigating the psychobiological connections between self-starvation and high-level exercising, including both animal experimentation and clinical field studies. In recent years it has been proposed that physical activity plays a central role in the pathogenesis and progression of the eating disorders—in particular, anorexia nervosa.

Method: A review of research from animal experimentation and from clinical field studies investigating the biological and psychological implications of physical activity and starvation in the pathogenesis of eating disorders.

Results: Animal research indicates that physical activity and starvation seem to potentiate one another and that alterations in the serotonergic system may underlie this process. Similar behavioural results have been found in recent clinical studies with eating-disordered patients, which suggests that physical activity plays a more central role in the development and maintenance of the eating disorders than had previously been thought.

Conclusions: The emerging picture is that psychosocial factors seem to provide the most compelling factors in the etiology and onset of the disorder, while biological factors—in most cases induced by severe malnutrition and strenuous overexercising—predominate in the maintenance of the disorder.

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Key Words: eating disorders, hyperactivity, psychobiology

There are distinctive features of the eating disorders that set them apart in a special way from all other psychiatric disorders. They are, in a manner that other disorders are not to the same extent, or even at all, truly psychobiological. I use this term in its broadest sense, meaning that a complete understanding of anorexia nervosa (AN) and bulimia nervosa (BN) requires information about complex psychological as well as biological functioning. In fact, like psychobiological models in general, it is probably more precise and accurate to say that the span of necessary knowledge is really “biosocial” and therefore even broader in scope (1).

At a psychological level, authorities in the field of eating disorders recognize the etiological significance of certain personality traits—albeit themselves partly biologically and genetically determined—in conjunction with environmental influences, particularly those pertaining to family dynamics. There is solid evidence that characteristics such as anxiety (2), pronounced perfectionism (what some have termed “neurotic perfectionism” [3,4]), obsessive compulsiveness, borderline tendencies, and narcissism (5) describe core features of the premorbid profile of many eating-disordered patients. We also know, however, that a plethora of psychological disturbances occur as a consequence of starvation. These include depressed mood, lowered self-esteem, reduced libido, and aspects of cognitive impairment (6).

Similarly, research confirms that aspects of biological functioning affect both the etiology and the progression of eating disorders (7,8). Concerning the former, there is growing evidence that developmental factors, in particular early childhood traumas, play a crucial role in the organizational and functional status of the mature brain (9). Specifically, trauma induces neurophysiological hyperarousal and dissociation, which seem to function, in the short term at least, as adaptive biological responses but can, when they persist, become maladaptive traits (10). It is also believed that these alterations in physiology increase susceptibility, probably at several levels, to a number of psychological disturbances.
Indeed, correlational evidence has shown links among disso-
liation, trauma, and eating psychopathology (11).

The biological sequelae of starvation and/or the binge–
purge cycle have also been well researched and are numerous. Disturbances have been found in virtually every endocrine system, as well as abnormal regulation in many neurotrans-
mitter systems (8). Furthermore, in conjunction with and
perhaps as a consequence of these biological changes, a
number of medical complications typically occur such as
electrolyte abnormalities, dehydration, and gastrointestinal
complications (12).

What especially distinguishes the eating disorders from
other psychiatric disturbances, however, is a set of unique
psychobiological characteristics. For a start, the ultimate de-
ing symptoms of the eating disorders are somatic ones of
a kind that directly translate the psychology into an immedi-
ately relevant biology. Moreover, AN is the only psychiatric
condition that requires, for its clinical diagnosis (13), a bio-
logical abnormality (amenorrhea). Finally, the eating disor-
der are probably the only psychiatric disorders that carry, as
an intrinsic (as distinct from secondary) feature, the risk of
fatal outcome.

In this paper I will take the position that physical activity—
in the most general sense, which includes competitive sport
and recreational exercise as well as relentless and ritualized
hyperactivity—also has a pivotal psychobiological function
in the longitudinal dynamics of the eating disorders. I say this
because a converging body of research, both from animal
experimentation and from clinical field studies, demonstrates
in a convincing way that together the psychology and the
biology of physical activity can play a causal role in the
development and maintenance of some eating disorders.
Moreover, these factors tend to operate in a mutually reinforc-
ing manner, at least in our present culture, with its emphasis
on ultraslenderness and hyperfitness as putative social mark-
ers of health and female sexual attractiveness. Finally and
importantly, we see that these influences can occur at all
stages of the disorders: during their etiology, their progress-
on, and perhaps especially in their maintenance.

The Prevalence of Hyperactivity in the Eating Disorders

From the earliest case reports and since that time, clini-
cians have recognized that generalized hyperactivity and/or
excessive exercising are characteristic of many patients with
eating disorders, even in the face of severe emaciation
(14–16). Historically, this behaviour has been viewed merely
as a consequence of an extreme desire for thinness—in other
words, as an analogue of purgation and an adjunct to calorie
restriction.

Unfortunately, there have been no population estimates of
the prevalence of hyperactivity and excessive exercising
among eating-disordered women; the rate of occurrence
across diagnostic categories has also not been established. In
fact, I am aware of only a few studies that have addressed this
issue at all, and the findings are limited in generalizability by
their lack of representativeness. In these cases, the study data
were obtained from patients under the care of a major treat-
ment centre in Canada or the United States. It is probable,
therefore, that the samples comprised a greater proportion of
those who were treatment-resistant and/or had more severe
symptomatology than is typically found in the general popu-
lation. Nevertheless, since these are the only data available,
they provide a starting point and reference for future compar-
sions with more representative data.

Based on a classification criterion we have used in pre-
vious research (17), Brewerton and his associates (18) re-
cently reported that 28% of a patient sample (AN = 39, BN =
71) were excessive or compulsive exercisers and that there
was a trend for a higher proportion of exercisers in those
patients with AN (39% versus 23%) \( (P < 0.06) \). In an earlier
study, we found a much higher rate of 78% among hospital-
ized AN patients (19). This discrepancy between findings
may be explained by the fact that Brewerton and others seem
to have based their classification on the patients’ exercise
status at the time of diagnosis (or treatment onset), whereas
our study assessed lifetime occurrence of excessive exercising.

We now have additional information that sheds more light
on this subject. Preliminary analyses have been carried out on
data from 2 ongoing studies, one at The Toronto Hospital
(Davis, Kaplan, Olmsted, and Woodside) and one at the
Hospital for Sick Children (Davis, Katzman, and Kirsh). These
studies provide current and historical physical activity data,
as well as data for adult and adolescent patients. In the
adult sample, 75% of those with AN \( (n = 36) \) had exercised
excessively at some point in their disorder, and this rate
increased to 84% when we included patients who, at the time
of assessment, met criteria for BN but who had had an earlier
diagnosis of AN \( (n = 56) \). These results essentially
replicated our earlier findings (19). Among patients with BN
and no history of AN, however, the rate of excessive exercis-
ing was only 54%. A comparison of group frequencies indi-
cated that the AN–BN differences were statistically
significant \( (\chi^2 = 8.833, P = 0.003) \).

By contrast, when the data were analyzed according to the
criterion used by Brewerton and others (18)—that is, weekly
hours of exercise (in our case based on data averaged over the
12 months prior to assessment)—we found that only 51% of
AN patients and 31% of BN patients were exercising exces-
sively \( (\chi^2 = 3.39, P = 0.06) \). While these rates are still
somewhat higher than those reported by Brewerton and
others, the differences are not substantial. Importantly, how-
ever, both studies found a similar trend towards higher preva-
ience rates in patients with AN.
The adolescent sample offers another point of comparison. We found that 82% of AN patients aged 13 to 16 were exercising excessively at the time of their admission (n = 28). In most cases, these were first-time admissions, and in almost all cases, admission took place within 12 months of the onset of disorder. It is interesting to note that the prevalence of hyperactivity among these patients is virtually identical to the previously mentioned lifetime rates (84%) found in the adult AN patients.

At this point one can only speculate on the pathogenic implications of these findings. Nevertheless, based on the totality of evidence, it is not unreasonable to conclude that physical activity is a bona fide factor in the development of eating disorders, particularly among patients with AN. Furthermore, the likelihood of an occurrence of hyperactive behaviour at some stage in the symptom profile of a patient is extremely high. It also seems that the exercising–calorie-restriction dyad is most likely to occur during the early stages of an individual’s disorder and thus may serve a causal role in its progression. The finding in Brewerton and others’ (18) study that dieting onset began at a significantly earlier age in compulsively exercising patients with AN than in nonexercisers further substantiates this notion.

It is also worth noting that the average age of our adult sample was 28.1 years and included a considerable number of patients with a long duration of illness. The substantial diminution from the adolescent to adult patients in current exercising rates (82% to 51%), therefore, is most likely a function of chronicity. The probable explanation is that many of the adult patients had, over time, simply become too weak or too unwell medically and/or psychologically to continue their activity behaviour, at least at a strenuous and frenetic level.

**Psychological Implications of Physical Activity in the Eating Disorders**

The way in which contemporary women have come to view their bodies and the significance of physical activity in this formulation must be considered against the backdrop of our current preoccupation with matters of fitness and health. It has been said that the trappings of this industry, with its aggressive marketing of exercise facilities, equipment, and attire, are the collective hallmark of this decade (20). In the broader context, this meshes with Lasch’s (21) claim that post-World War II society has been characterized by a pervasive and increasing “cultural narcissism.” Elsewhere, I too have argued that perhaps there is no better testimony to this sociocultural phenomenon of self-absorption than the time-consuming, expensive, and often destructive efforts of many to achieve a semblance of bodily perfection (22).

We are frequently inundated with messages of the virtues of exercise as a guaranteed weight-loss strategy, sometimes under the insidious guise of health promotion and often containing fallacious information. Since the mid-1980s, for example, the number of articles in popular women’s magazines promoting exercise for slimming has surpassed the number recommending traditional calorie-reduced dieting (23). Not surprisingly, several studies have shown that exercising for weight loss is one of the principal reasons reported by men and women for beginning a fitness program (24–26).

It should go without saying that the relationship between body-image perceptions and exercise participation is complex and is undoubtedly a dynamic rather than a static one. In fact, the contradictory findings in the research literature bear witness to this viewpoint. Some studies have found that exercising is associated with better body image (27); others have found the reverse (28); and still others have found no relationship at all (29). We do, however, have more consistent evidence that body focus (that is, the degree to which general well-being is dependent on one’s physical appearance) is positively associated with exercise involvement, although the direction of causality is not clear and may even be bidirectional (27,30).

For many women, involvement in an exercise program may, at the onset, produce a number of personal and social reinforcers as a result of real (or perceived) changes in body tone, energy level, and weight. Assiduous exercise involvement can also have certain negative consequences, however, particularly among those who are prone to self-criticism and high perfectionism. Comparisons with video and magazine images of superfit models may foster in these individuals a fault-finding view of their own physical appearance and an increasing sense of disenchantment. Together, these influences can increase vulnerability and risk for the development of an eating disorder.

A second cultural influence, which is profound, although it affects a much smaller segment of the population, is the world of competitive athletics. A relatively high prevalence of eating pathologies, including potentially harmful weight-loss techniques, have been reported among female athletes and dancers, particularly those involved in high-level competition, despite the fact that most of them are, by objective standards, of normal weight or even underweight (31–34). Almost by definition, competitive athletes in any sport are heavily invested in a high-intensity, physically taxing training regimen. In addition, many female athletes are chronically subjected to strong pressures to maintain a very low level of body fat in order to achieve the ultraslender body shape that has become the ideal in many sports such as gymnastics, figure skating, and dance. It is therefore not surprising that we found in a recent study that 60% of a sample of hospitalized, eating-disordered patients had been high-level competitive athletes at some point in their lives (19).
Biological Implications of Physical Activity in the Eating Disorders

While the behavioural links between exercise and self-starvation are well established clinically, it is only relatively recently that some understanding of the biological connections has emerged. Interestingly, we have known for over 30 years that physical activity can be fatal for laboratory rats who are placed on restricted feeding schedules (35). In the intervening years, numerous studies have demonstrated that this phenomenon—sometimes known as starvation-induced hyperactivity, or conversely as activity-induced weight loss—is easily inducible and highly reliable (36,37). In the generic protocol, food-restricted experimental rats with free access to a running wheel will reduce their food intake and their body weight in almost direct proportion to an increase in physical activity. In fact, in a relatively short period of time, the animals can literally run themselves to death.

The practical relevance of these findings, however, has been a fairly recent consideration. Indeed, it was not without controversy when investigators first suggested that this exercise-starvation syndrome extrapolates nicely to the human condition and serves as a useful animal model of AN (38,39). It was argued that for many women, anorexia is not strictly a nervosa; instead, it is a physiologically mediated disorder that occurs when food restriction is paired with physical activity. Specifically, the case was made that strict calorie restriction and strenuous exercise are reciprocal behaviours which eventually become self-perpetuating and resistant to change.

Over the past decade, there has been substantial support for the utility of this syndrome as a psychobiological model for AN. One of its most significant experimental features is the ability to control for the general effects of weight loss by including in the testing protocol a nonexercising, weight-matched group. This affords the opportunity to tease out biological changes that cannot be attributed to the secondary consequences of weight loss (40). One of the weaknesses of this model, however, is the inability to control for the general effects of excessive exercise, since there is no suitable way to stimulate animals of normal weight to run freely to the same extent.

In recent years, several neurochemical mechanisms have been proposed to explain the activity-induced weight-loss syndrome. For example, there was some enthusiasm for the notion that a state of β-endorphin “autoaddiction” occurs during its development which serves to maintain the maladaptive behaviours of overactivity and self-starvation (41); however, it now seems that the evidence generally fails to support a significant role for the opioids (42).

Other research has focused on the role of the monoamines, in particular, serotonin (5-HT). This neurotransmitter is of special interest because it regulates hunger, satiety, activity, arousal, and pain sensitivity, all of which are disturbed in AN and are relevant to the activity-induced weight-loss syndrome (43,44). In this area, studies have shown that both physical exertion and starvation independently increase central 5-HT synthesis and turnover (43,45), but what is especially interesting is that an increase in the hypothalamic metabolism of 5-HT occurs to a greater extent in hyperactive, semistarved rats than in their sedentary, semistarved counterparts. These data support the hypothesis of a self-perpetuating biological feedback loop; activity-induced 5-HT stimulation or turnover leads to reduced food intake and body weight, which in turn provides a further stimulus for physical activity (43).

Others have also provided corroborating evidence of 5-HT dysfunction that is specific to the syndrome and is not a general consequence either of weight loss or of exercise on its own. Data from pharmacological challenges indicate that the syndrome is relevant both to the adverse effects of strenuous exercise on the body-weight regulation of otherwise healthy subjects as well as to the hyperactivity and weight loss often seen in AN patients (42,46).

The Interdependence of Obsessive Compulsiveness, Physical Activity, and the Eating Disorders

Research linking obsessive–compulsive disorder (OCD) and the eating disorders has also bourgeoned in the past 5 years (47–54). Based largely on the similarity of symptom profiles, there is now general agreement of a substantial comorbidity between the 2, although there is no consensus on whether the prevalence rates are higher among AN or BN patients (52,54–56).

Not only are there psychopathological similarities between the eating disorders and OCD, there also appears to be a marked neurochemical correspondence. In particular, a number of studies have found altered serotonergic function in OCD patients, although the specific mechanisms of dysfunction are still not entirely clear (57–61). While studies of 5-HT status in eating-disordered patients are not plentiful, the existing research also suggests there are functional alterations (18,50,51). It should be noted, however, that much of this evidence is indirect and has been inferred, in large part, from drug-response data (62,63).

The generalized hyperactivity or excessive exercising found in eating-disordered patients is of considerable relevance in its link with OCD. Altemus and her colleagues have proposed that the activity-induced weight-loss syndrome described earlier is not only a good animal model for AN but also a valid model for OCD (64). In many ways, the increasing, almost perpetual, running observed in this syndrome resembles the compulsive and ritualized behaviours seen in patients with OCD. For example, the animal’s behaviour is clearly repetitive and excessive and is obviously maladaptive. The soundness of this connection is also supported by the fact that laboratory rats pretreated with fluoxetine (a 5-HT
reuptake inhibitor used successfully in the treatment of OCD) showed a significant attenuation of hyperactivity and weight loss when subjected to the food-restriction procedures described earlier.

At a clinical level, excessive exercising and the eating disorders are both activities that share a strong relationship to obsessionality. For example, several studies report that high-level exercisers typically display a compulsive behaviour pattern as well as an obsessional and rigid personality profile (65–68). Furthermore, exercising has been positively related to dietary restraint (69) and weight preoccupation (30,70). There is also evidence that AN patients tend to have personality traits such as rigidity, restraint in emotional expression, and greater impulse control—factors which are highly compatible with obsessive–compulsive personality disorder (50,51).

In a recent study, we investigated, for the first time in a systematic way, claims that the activity-induced weight-loss syndrome is a valid analogue of eating disorders in the human condition (19). Our findings were overwhelmingly supportive. Among other things, we found that 75% of a hospitalized AN patient sample reported an inverse relationship between food intake and physical activity level during the acute weight-loss phase of the disorder. Moreover, in describing this behaviour pattern subjectively, exercising patients uniformly stated that their involvement in physical activity progressed from a voluntary and mostly enjoyable endeavour to a state where the behaviour became “obsessive” and “out of control”—something they often wanted to stop doing, but simply could not.

We have known for a long time that starvation increases generalized obsessionality in a state-dependent manner (6). It is less clear, however, to what extent obsessionality is also an antecedent of self-starvation in the case of eating-disordered patients. There has been little controlled research on which to draw conclusions. One study found that heightened trait obsessionality was not changed in a group of adolescents after weight restoration even though there was a diminution in obsessive–compulsive symptomatology (71), while another found no change in obsessional symptoms after refeeding (72).

The expanding body of animal experimentation and our first clinical findings naturally generated other questions concerning the associations among personality factors, activity, and underlying biological mechanisms. In particular, the evidence that neurochemical changes which mimic those found in OCD (specifically those related to 5-HT dysfunction) occur to a greater degree when strenuous exercise is combined with starvation than when starvation occurs alone gave rise to the hypothesis that obsessional symptomatology should be greater in AN patients who are hyperactive than in nonhyperactive AN patients.

In our initial investigation, we found, as predicted, a positive relationship between obsessive–compulsive symptomatology and exercise frequency in a group of high-level exercising women, as well as among hospitalized women diagnosed with AN (73). Furthermore, in the patient group, weight preoccupation was positively associated with the frequency of exercising, with obligatory attitudes to exercise (for example, feelings of guilt associated with missed exercise sessions), and with pathological attitudes to exercise (for example, exercising in the face of sickness and injury).

We then speculated that obsessive–compulsive personality traits should also be more common in AN exercisers than in nonexercisers because this personality type—characterized largely by perfectionism, perseverance, and rigidity—is temperamentally better suited to the rituals of an assiduous exercise and dieting regimen than less obsessive individuals. To extend our previous findings, we compared groups of exercising and nonexercising AN patients on obsessional symptomatology and obsessive personality traits. For the purpose of comparison, we also included an assessment of several other psychopathological factors that are typically associated with the eating disorders (for example, perfectionism, self-esteem, body dissatisfaction, weight preoccupation, and attitudes to exercise) (74). Our study sample comprised 50 AN patients who were classified as excessive exercisers (n = 22) or as moderate to nonexercisers (n = 28), according to the criterion described earlier (17,18) and based on data averaged over the 12 months prior to program admission. Supporting our predictions, we found that exercisers were significantly more obsessive and compulsive both with respect to their symptomatology and their personality characteristics. They also reported a greater degree of self-oriented perfectionism and pathological commitment to exercising. However, on all other psychological measures, and on Body Mass Index (that is, weight[kg]/height[m²]), there were no differences between the groups.

Unfortunately, the only other study that has compared exercising with nonexercising eating-disordered patients did not provide an assessment of obsessionality (18). Furthermore, although these investigators found that exercisers had significantly greater body dissatisfaction, that finding is seriously confounded by the fact that the samples comprised almost twice as many BN as AN patients, and there was no attempt to control for body size or partial out its influence statistically.

Up to this point, our primary interest had been in the starvation–exercise interaction, and so our analyses have focused on patients with AN. To expand the picture, we repeated the analyses described previously (using the same exercise classification criterion) with a group of BN patients who had no prior history of AN. Interestingly, there were no differences between exercising and nonexercising BN
patients on either measure of obsessionality or on any of the measurements, including Body Mass Index. Although these findings seem theoretically reasonable—mainly because BN is associated with impulsivity, which is seen as the behavioral opposite of obsessionality (75)—they should be interpreted cautiously because the overall sample was relatively small and the groups were not at all balanced in size (exercisers = 12; nonexercisers = 27).

Finally, we decided to compare the total BN sample with the exercising and with the nonexercising AN patients. We found that obsessive–compulsive symptomatology and personality were considerably higher among exercising AN patients than among those with BN, and these differences were highly statistically significant. In contrast, the BN group did not differ statistically from the AN nonexercisers on any measures. Taken together, our clinical findings map extremely well onto the animal research reviewed earlier, especially that pertaining to the activity-induced weight-loss syndrome.

Conclusion

In this paper, I have considered evidence from a variety of sources, both psychological and biological, that highlights the significance of physical activity in the development and progression of some eating disorders. The emerging picture is that psychosocial factors seem to offer the most fruitful explanatory concepts in etiological terms, while biological influences predominate in the maintenance of the disorder.

In many cases of AN, particularly those with origins in adolescence, the disorder may actually stem from attitudes and behaviours that are initially rather benign. Repeatedly, we have seen cases where a preoccupation with aspects of the physical self, in conjunction with peer and social influences, simply motivates a desire for a so-called healthier lifestyle. This typically means restricting high-fat foods and becoming physically active. Regrettably, what can (and by many accounts almost mysteriously) develop is an intractable disorder with serious medical complications and psychological difficulties.

The research I have presented and reviewed in this paper, both from animal experiments and clinical field studies, strengthens the psychobiological model of eating disorders that my colleagues and I proposed earlier (74). Our model integrates, in a dynamic and nonrecursive manner, obsessive compulsiveness, physical activity, and self-starvation in the pathogenesis of some eating disorders. In combination with certain salient personality characteristics and environmental influences, we believe that behaviours such as dieting and exercise tend to reinforce each other because of the many personal and social benefits that typically accrue.

“If a little bit is good, then a lot is better” becomes the credo. Higher levels of physical activity can lead to further food restriction because of its appetite-suppressing effects (76), and because it may encourage a greater focus on appearance and weight (77,78). Eventually—and here we extrapolate from a large body of animal research—neurochemical changes are likely to take place that increase obsessionality and thereby exacerbate the obligatory nature of the commitment to dieting and exercise (19,46,50,51,64). The result is an even greater increase in these behaviours. Attitudes and cognitions become more pathological as obsessionality increases, and eventually the condition seems to be maintained beyond a level of willful control.

In proposing this process as a model of AN, we acknowledge that the etiology of eating disorders is highly complex, multidimensional, and variable across cases. Clearly, our model attempts to explain only certain aspects of the pathogenesis of AN, and these for only some individuals. Our model says very little about personal motivations, and it does not address the complexity of factors that predispose some individuals and not others to become heavily invested in athletics and exercise.

Clinical Implications

- Physical activity and self-starvation tend to function synergistically in the maintenance of some eating disorders.
- The prevalence of hyperactivity in the eating disorders, particularly anorexia nervosa, is extremely high.
- Obsessionality may function as an antecedent as well as a consequence of excessive exercise and disordered eating.

Limitations

- At the present time, only a handful of studies have investigated the relationships between physical and clinical eating disorders in a systematic way.
- Further research, including biologically based studies, is needed to understand more fully the interconnections between caloric restriction and physical activity in patients with anorexia and bulimia nervosa.

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Résumé

Objectif : Passer en revue les recherches sur les troubles alimentaires ayant pour but d’examiner les liens psychobiologiques entre l’auto-inanition et l’activité physique pratiquée de façon intensive, incluant les expériences animales et les études cliniques sur le terrain. Au cours des dernières années, il a été proposé que l’activité physique jouait un rôle majeur dans la pathogenèse et la progression des troubles alimentaires, en particulier de l’anorexie mentale.

Méthode : Examen des expériences animales et des études cliniques menées sur le terrain dans le but d’étudier les incidences biologiques et psychologiques de l’activité physique et de l’inanition dans la pathogenèse des troubles alimentaires.

Résultats : Les recherches sur les animaux montrent que l’activité physique et l’inanition semblent exercer l’un sur l’autre un effet de potentialisation, un phénomène qui pourrait être attribuable à des altérations du système sérotoninergique. Des études cliniques récentes auprès de patients souffrant de troubles alimentaires font état de résultats comportementaux similaires, ce qui porte à croire que l’activité physique joue un rôle plus important qu’on le croyait jusqu’ici dans l’apparition et le maintien des troubles alimentaires.

Conclusions : Le tableau qui se dessine est le suivant : les facteurs psychosociaux semblent être les facteurs déterminants dans l’étiologie et l’apparition des troubles, alors que les facteurs biologiques—dans la plupart des cas provoqués par une inanition grave et un niveau intensif d’activité physique—influencent surtout sur le maintien des troubles.