Exercise Dependence

D. M. W. DE COVERLEY VEALE M.B., B.S., B.Sc.

Registrar, Academic Department of Psychiatry, Royal Free Hospital, London NW3 2QG, U.K.

Summary
Exercise can become a compulsive behaviour and harmful to an individual. This review proposes diagnostic criteria for ‘exercise dependence’ to facilitate recognition in Sports clinics and further research. The importance of diagnosing exercise dependence lies in the prevention of morbidity and rarely mortality if exercise is continued in the presence of illness or injury. There is insufficient evidence to postulate opioid peptides as a physiological basis of dependence. A distinction is made between a primary form of exercise dependence and that which is secondary to an eating disorder.

Introduction
Regular exercise results in physiological benefits in the prevention of coronary heart disease and obesity and is widely enjoyed by the majority of the population. Exercise can occasionally become a compulsive behaviour and the following terms have been used to describe this phenomenon: ‘running addiction’ (Glasser, 1976; Sachs & Pargman, 1984), ‘negative addiction’ (Morgan, 1979), ‘obligatory runners’ (Yates et al., 1983), ‘running anorexics’ (Norval, 1980), and ‘morbid exercising’ (Chalmers et al., 1984). The term ‘exercise dependence’ is preferred in this article because it does not refer to any particular sport and classifies the phenomenon with other compulsive behaviours. The proposed diagnostic criteria (Table 1) are based on the core features of a dependence syndrome (Edwards et al., 1977) which are to be incorporated into the revised DSMIII (Rounsaville et al., 1986). The same caveat to the alcohol dependence syndrome applies, that is, it does not imply any particular aetiology and there exist degrees of dependence with many gradations. Impairment in the levels of functioning at either a physical or social level are essentially epiphenomena that are a consequence of dependence (Russell, 1976) and are listed in Table 1 as associated features.

Background
Russell (1976) has proposed a broad definition of dependence, of which the crucial feature is a negative affect experienced in the absence of a drug, object or activity. The degree of dependence is equated with the amount of this negative affect, which may range from mild discomfort to extreme distress, or it may be equated with the amount of difficulty or effort required to do without the drug, object, or activity.

Exercise is a stimulant which leads to physiological arousal of the brain. The mood is altered in the short-term and may occasionally reach a state of euphoria (the so-called ‘runner’s high’) (Wagemaker & Goldstein, 1980; Pargman & Baker, 1980). Glasser (1976) first described the negative affect that occurs when such runners are forced to stop because of injury, illness, or circumstance. The symptoms consisted of minor mood disorders (for example depressed mood, irritability, fatigue, anxiety, impaired concentration, and sleep disturbance). The withdrawal symptoms were further documented in runners by Thaxton (1982), who found that after one day’s absence there were both significant increases in depression (but not in other moods) on the Profile of Mood States and in the galvanic skin response. Morgan (1979) has reported
Table 1. Proposed Diagnostic Criteria for 'Exercise Dependence'

(A) Narrowing of repertoire leading to a stereotyped pattern of exercise with a regular schedule once or more daily.
(B) Salience with the individual giving increasing priority over other activities to maintaining the pattern of exercise.
(C) Increased tolerance to the amount of exercise performed over the years.
(D) Withdrawal symptoms related to a disorder of mood following the cessation of the exercise schedule.
(E) Relief or avoidance of withdrawal symptoms by further exercise.
(F) Subjective awareness of a compulsion to exercise.
(G) Rapid reinstatement of the previous pattern of exercise and withdrawal symptoms after a period of abstinence.

Associated Features
(H) Either the individual continues to exercise despite a serious physical disorder known to be caused, aggravated or prolonged by exercise and is advised as such by a health professional, or the individual has arguments or difficulties with his partner, family, friends, or occupation.
(I) Self-inflicted loss of weight by dieting as a means towards improving performance.

case histories and described 'negative addiction' as a process that evolves gradually with different stages of dependence.

Yates et al. (1983) reported three cases from a group of 60 marathon runners who were identified as 'obligatory runners' by a screening questionnaire. They were predominantly male between the ages of 30 and 50. Yates argued that obligatory runners have many characteristics in common with patients with Anorexia Nervosa, such as inhibition of anger, high self-expectation, and tolerance of physical discomfort. In addition it was noted that subjects feared physical ineffectiveness and this fear did not diminish as their strength grew: they were diet-conscious and placed undue emphasis on weight reduction to improve speed. Yates speculated that running provided an identity for the individuals and a feeling of bodily control.

Blumenthal et al. (1984) selected 43 obligatory runners on the basis of the screening questionnaire used by Yates. The runners were compared to 24 patients with Anorexia Nervosa by the response to the Minnesota Multiphasic Personality Inventory (MMPI). The former scored within the normal range while the latter had elevated scores for depression, hostility and anxiety. This study was criticized by Dresser (1985) because of bias in the selection of obligatory runners. The patients with Anorexia Nervosa were hospitalized and diagnosed according to DSM III, whereas none of the runners had even been referred for treatment. This suggested that the group selected as 'obligatory runners' had no serious impairment in their level of functioning. Blumenthal et al. (1985) re-examined their original data and found that even the most severe obligatory runners who indicated by questionnaire that they exercised even when they were sick or injured did not exhibit significant psychopathology as measured by the MMPI. They provided no details of the severity of the dependence, in terms of the seriousness of the physical disorders or conflicts with their families, when the runners continued to exercise. They argued that a psychopathological or 'disease' model of obligatory runners was "misleading and unnecessarily pejorative". These issues recall similar arguments over whether alcoholism is a disease or a learned behaviour. Blumenthal et al., also propose that running is best viewed as a coping strategy for the regulation of affect. Evidence for this is the high prevalence of a past history of affective disorders in runners (Colt et al., 1981a) and the increasing interest in the use of exercise in an adjunctive treatment of depression (Simons et al., 1985). But this hypothesis does not address the impairment in physical or social functioning that may occur. The runners may well have experienced an improvement in their psychological functioning but at a cost of endangering their physical health or disrupting their family and social life, and may require help to reduce the degree of dependence on a single activity such as exercise.

Exercise dependence is most likely to present as a persistent injury at a sports clinic. Thus the importance of diagnosing exercise dependence lies in the prevention of morbidity and even mortality if exercise is continued in the presence of physical illness or injury. The physical complications of exercise dependence have been documented most comprehensively in long-distance runners. They include repeated soft tissue injuries and stress fractures (Stanish, 1984; Colt & Spyropoulos, 1979); pressure-sores (Liberman & Palek, 1984);
gastro-intestinal blood loss and anaemia (Liberman & Palek, 1984; Patel et al., 1983; Lee, 1986); myocardial infarction and death (Noakes et al., 1977). Jim Fixx (1981) is the best known example of a runner who was able to tolerate the physical symptoms of ischaemic heart disease and as a result died from a myocardial infarct while running. (It should be pointed out however that statistically an individual is at far greater risk of dying from coronary heart disease if he or she does not regularly exercise).

**Epidemiology**

The prevalence of exercise dependence is not known but only a very small percentage of sportsmen and women are likely to have a severe dependence. Some may have a partial syndrome characterized by a pre-occupation with physical fitness. This may be manifest by excessive exercise; feelings of guilt when they are unable to exercise; diet to improve performance, and exercising in spite of minor injuries which might not seriously endanger their health.

**Psychophysiology**

The physiological basis for dependence on exercise and the high tolerance of pain in such individuals has been linked to opioid peptide activity in the central nervous system. A number of investigators (Fraioli et al., 1980; Colt et al., 1981b; Carr et al., 1981; Howlett et al., 1984) have demonstrated marked elevations of plasma opioid peptides during exercise, which appears however to be a relatively non-specific response to acute stress. The peripheral opioid peptides are relatively impermeable to the blood-brain barrier and cannot therefore be implicated in the acute changes of mood, the high tolerance of pain, or as a reinforcer for dependence on exercise. There may well be central changes in opioid peptides with exercise but the evidence for this could only be obtained directly from the CSF. Even then it would be difficult to know how altered lumbar CSF levels reflect changes in discrete brain areas and altered mood states. Animal studies have however demonstrated acute increases in beta-endorphin and leu-enkephalin in the limbic system immediately after a fatiguing bout of exercise. (Blake et al., 1984; Pert et al., 1979). Indirect evidence for central opioid activity in humans can at present be gained from the use of antagonist drugs or a PET Scan with an opiate labelled ligand (Jones et al., 1985). To date only antagonist drugs such as Naloxone and Naltrexone have been used which have preferential affinity for mu-receptors and an analgesic function. For example Janal et al. (1984) studied 12 normal distance runners on two occasions in a double-blind crossover trial. On one day they received 0.8 mg Naloxone at 20 min intervals following a 6 mile run. On the other day they were given two equal volumes of normal saline. Ischaemic pain and discriminability of thermal stimuli were significantly reduced after the run with normal saline compared to Naloxone. The hypoalgesic effect was equivalent to 10 mg Morphine and lasted about 25 min after the run.

It has been suggested that regular exercise enhances the central release of opioid peptides which provides the physiological basis of exercise dependence (Yates et al., 1983). Evidence for enhanced secretion of peripheral beta-endorphins after an exercise programme was demonstrated by Carr et al. (1981), although this study was criticized for retesting subjects after their exercise programme at a higher physical workload. Howlett et al. (1984) studied 15 normal women during an intensive 8-week exercise programme and failed to replicate the findings of Carr. Animal studies have also failed to demonstrate any enhancement in central opioid peptide levels after an 8-week training programme (Blake et al., 1984).

In summary, experimental evidence exists for acute but not chronic central changes in opioid peptides with exercise. Opiate receptor function is still best confined to analgesia and the role of opioid peptides in altered mood states and exercise dependence can only be satisfactorily tested when there are more specific opiate receptor antagonists or a PET Scan becomes more widely available.

**Primary and Secondary Exercise dependence**

A distinction should be made between primary exercise dependence and exercise dependence which is secondary to an eating disorder. A diagnostic hierarchy occurs in a case of exercise dependence, whereby the diagnosis of Anorexia Nervosa should be first excluded, followed by Bulimia Nervosa. A diagnosis of primary exercise dependence should only then be made.

The diagnosis of Anorexia Nervosa includes three fundamental features which must all be present (Garfinkel & Garner, 1982). The first criterion is: "Self-inflicted loss of weight, using one or more of the following: (a) avoidance of foods considered to be ‘fattening’ (especially carbohydrate-containing
foods); (b) self-induced vomiting; (c) abuse of purgatives; and (d) excessive exercise". Some criteria such as DSMIII stipulate that the weight loss must be at least 25% of the original body weight coupled with a refusal to maintain body weight over a minimal normal weight for age. The excessive exercise is regarded as a secondary feature as a means towards losing weight or balancing the intake of ‘calories’, and occurs to varying degrees in most anorexics. Occasionally the excessive exercise and the subsequent physical complications are the presenting feature of Anorexia Nervosa (Chalmers et al., 1985; Waldstreicher, 1985; Katz, 1986). The exercise may reach a state of dependence but it is still regarded as secondary to the eating disorder.

Self-inflicted loss of weight by dieting is also a feature of primary exercise dependence and is common in many sportsmen and women. The weight loss is, however, used as a means of improving performance. In general they do not have a morbid pre-occupation with their weight which does not fall below 25% of the original body weight—if it is too low their performance will deteriorate. The social pressure to reduce body fat in young athletes (Smith, 1980) and ballet-dancers (Garner & Garfinkel, 1980; Szmukler et al., 1985) may however be an important factor in the development of Anorexia Nervosa.

The second criterion for a diagnosis of Anorexia Nervosa is:

“A secondary endocrine disorder of the hypothalamic-pituitary-gonadal axis manifest in the female as amenorrhoea and in the male by a diminution of sexual interest and activity.” A secondary endocrine disorder cannot be used to differentiate an eating disorder from primary exercise dependence. A delayed menarche and amenorrhoea is extremely common among female athletes (Frisch et al., 1981). A common factor between Anorexia Nervosa and male athletes is the low body fat but there may be additional psychological factors in both populations. Diminished sexual interest has been reported in a male 'obligatory runner' (Patel et al., 1983), and serum testosterone and prolactin were significantly lower in high mileage runners compared to non-running controls (Wheeler et al., 1986).

The third criterion for a diagnosis of Anorexia Nervosa is: “A psychological disorder that has as its central theme a morbid fear of being unable to control eating and hence becoming too fat.” Normal runners do not appear to exhibit any of the characteristic psychopathology of Anorexia Nervosa, namely the morbid fear of losing control of one’s eating and the morbid fear of fatness. The evidence for this comes from two studies. In the first, (Goldfarb & Plante, 1984) a ‘fear of fat’ questionnaire was administered to 200 normal distance runners who were competing in a 10 km road-race and were found to score in the low-normal range. In the second, Wheeler et al. (1986) studied 31 high-mileage runners, 18 low-mileage runners and 18 non-running controls. There was some evidence of a distortion of body-image as high-mileage runners significantly over-estimated their waist-width compared to the control group. In addition, there were small but significant differences in the EAT scores between the runners and controls, and 20% of the runners had EAT scores beyond two standard deviations above the mean of non-running controls. However no runners scored above the score of 30 which is usually indicative of Anorexia Nervosa, and no such clinical diagnosis was made. A study on the eating attitudes and dietary behaviour of subjects diagnosed as primary exercise dependent remains to be performed. However if a subject in such a study were found to have abnormal attitudes towards eating with a morbid fear of fatness, then by definition he or she would be fulfilling at least one of the criteria for a diagnosis of Anorexia Nervosa and should not be included in a sample of primary exercise dependent subjects.

Bulimia Nervosa has a similar psychopathology to Anorexia Nervosa with a morbid fear of fatness, but in the context of recurrent episodes of binge eating and devices aimed at counteracting the ‘fattening’ effect of the food ingested (starvation; self-induced vomiting; abuse of purgatives or diuretics; excessive exercise.) The body weight is usually within the normal range, but some may be slightly underweight and others may be overweight. Again the excessive exercise may reach a state of dependence but it is still regarded as secondary to the Bulimia Nervosa.

In summary a diagnosis of primary exercise dependence can be differentiated from an eating disorder by clarifying the ultimate aim of the exercise. In primary exercise dependence, the exercise is an end in itself and the dieting and weight loss is used to improve performance. Other devices to lose weight such as self-induced vomiting, purgative or diuretic abuse is indicative of an eating disorder. In Anorexia Nervosa the exercise is used as a means towards losing weight or balancing ‘calories’, and is associated with a morbid fear of fatness.


