



# EFFECTS OF PHYSICAL EXERCISE ON ANXIETY, DEPRESSION, AND SENSITIVITY TO STRESS: A UNIFYING THEORY

Peter Salmon

University of Liverpool

**ABSTRACT.** *Until recently, claims for the psychological benefits of physical exercise have tended to precede supportive evidence. Acutely, emotional effects of exercise remain confusing, both positive and negative effects being reported. Results of cross-sectional and longitudinal studies are more consistent in indicating that aerobic exercise training has antidepressant and anxiolytic effects and protects against harmful consequences of stress. Details of each of these effects remain unclear. Antidepressant and anxiolytic effects have been demonstrated most clearly in subclinical disorder, and clinical applications remain to be exploited. Cross-sectional studies link exercise habits to protection from harmful effects of stress on physical and mental health, but causality is not clear. Nevertheless, the pattern of evidence suggests the theory that exercise training recruits a process which confers enduring resilience to stress. This view allows the effects of exercise to be understood in terms of existing psychobiological knowledge, and it can thereby provide the theoretical base that is needed to guide future research in this area. Clinically, exercise training continues to offer clinical psychologists a vehicle for nonspecific therapeutic social and psychological processes. It also offers a specific psychological treatment that may be particularly effective for patients for whom more conventional psychological interventions are less acceptable.* © 2000 Elsevier Science Ltd.

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BENEFITS OF PHYSICAL exercise are well established in the cardiovascular system and are becoming clear in a range of physical disorders including diabetes, renal disease, and osteoporosis (Fentem, 1994). Increased physical activity therefore reduces premature mortality (Paffenbarger & Hyde, 1988), and the establishment and maintenance of exercise habits has become a target for clinical and health psychologists on these grounds alone (Dubbart, 1992). However, physical exercise is relevant to clinical psychology as a possible psychological intervention in its own right.

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Correspondence should be addressed to Peter Salmon, Department of Clinical Psychology, University of Liverpool, Whelan Building, Liverpool L69 3GB, United Kingdom. E-mail: psalmon@liv.ac.uk

## THE EXERCISE LITERATURE

Literature on psychological effects of exercise has burgeoned to the extent that even reviews of reviews are now available (Scully, Kremer, Meade, Graham, & Dudgeon, 1998). The present article addresses several limitations of the existing review literature. First, previous reviews have tended to focus on specific effects, particularly depression. In the present article, an account of relevant evidence across related areas will allow the development of a theory in which the effects of physical exercise can be linked to processes and interventions that are more familiar in clinical and experimental psychology. Secondly, theoretical development has been constrained by overreliance on meta-analytic reviews which categorize studies according to pre-existing ideas. The approach here will be to review related areas of literature in a way that is exhaustive of substantive findings and of significant theoretical and clinical issues in each area. This will provide a sound empirical basis for a novel, integrated account of emotional effects of exercise. Relevant empirical articles linking physical exercise and fitness to mood, anxiety, depression, and psychological stress, published in English-language scientific journals during 1990–1998, were identified from Science and Social Science Citation Indices, and supplemented by tracking relevant citations. Reports excluded from the review are those that make no significant contribution to the argument, typically because of methodological limitations or because they merely repeat designs and findings which are the subject of numerous previous reports.

## EXERCISE, FITNESS AND EXERTION

Physical *exercise* implies a regular, structured, leisure-time pursuit, whereas physical *activity* also arises in domestic or occupational tasks. Although physical activity has benefits for cardiovascular health (Paffenbarger & Hyde, 1988), its possible psychological benefits have been neglected because research has focused on formal exercise programs. In general, prior evidence of the cardiovascular benefits of exercise has shaped research into its psychological effects. For instance, the typical duration of training programs in psychological literature (around 10–12 weeks) reflects the minimum period necessary for demonstrable cardiovascular conditioning. Similarly, the overwhelming emphasis on aerobic exercise, which involves prolonged activity of large muscle groups, such as in running, swimming, or aerobic dancing, and which is integral to cardiovascular conditioning programs, has outweighed the attention given to anaerobic exercise, in which muscular activity is intense, brief, and unsustainable, such as in weight lifting. The usual measure of fitness in psychological research has, accordingly, been aerobic fitness: the body's capacity for aerobic work. This is operationalized by oxygen uptake at maximal exertion ( $VO_2\text{max}$ ) which, although universally adopted, has limitations. It is influenced by many factors, such as genetic inheritance, so fitness and exercise history are not synonymous. Furthermore, maximal exertion is not a purely physiological limit; even when exercising "to exhaustion," the offer of financial reward further increases its intensity (Felig, Cherif, Minagawa, & Wahren, 1982). In practice, because of obvious ethical and technical difficulties with maximal exercise,  $VO_2\text{max}$  is usually estimated by extrapolation from heart rate at submaximal workloads.

Choosing control procedures for exercise is not straightforward. Nonstrenuous procedures such as relaxation and flexibility training have been designed to be compar-

ble with exercise for skill mastery, distraction from normal activities, or social interaction. However, expectations of health, fitness, and well-being surround exercise in Western society, and the emotional effects of exercise training are influenced by such expectations, not only in the exerciser (Desharnais, Jobin, Cote, Levesque, & Godin, 1993), but also in the exerciser's reference groups (Heaps, 1978; Hilyer & Mitchell, 1979; Ransford & Palisi, 1996). Control for expectations is limited by the impossibility of blinding participants to the fact that they are exercising (Ojanen, 1994).

### **HEDONIC PROPERTIES OF EXERCISE**

Despite popular awareness that regular and relatively strenuous exercise improves physical health, few people exercise (Brawley & Rodgers, 1993): only around 30% of Western populations engage in significant amounts of exercise weekly and, once initiated, attrition is high (around 50% of participants being lost within 3–6 months). The exercise literature has tended to present this as paradoxical, reflecting an assumption that, as well as being beneficial, exercise is enjoyable. Accordingly, published attempts to explain reluctance to exercise continue to emphasize psychological deficits in the individual (see Dishman, 1994), such as deficient self-motivation or self-efficacy, inappropriate health beliefs, or lack of an internal locus of control.

The clearest evidence that physical exercise is enjoyable has emerged when mood has been measured immediately before and after regular exercisers undertake strenuous exercise at a level with which they are familiar. Although discrepant results exist, the overwhelming evidence confirms mood improvement (Step toe, Kimbell, & Basford, 1998; see Yeung, 1996). Where exercise is competitive, effects obviously depend on the degree of success (Clingman & Hilliard, 1994). Even where general measures of positive or negative mood are unaffected, specific moods, such as vitality, can be improved (Rejeski, Gauvin, Hobson, & Norris, 1995). Effects are clearest where mood is poor before exercise (Gauvin, Rejeski, & Norris, 1996).

Sedentary samples have been much less commonly studied. In these reports, exercise has most clearly been a positive experience where relatively mild or moderate exercise has been voluntarily performed in the course of a normal day or contrived in an experimental study. Such exercise has been followed by more positive mood and, less clearly, by less negative mood (McIntyre, Watson, & Cunningham, 1990; Raglin & Wilson, 1996; Step toe, Kearsley, & Walters, 1993a; Thayer, 1987a; Watson, 1988). Where prior mood was examined, mood improvement was confined to those who were relatively unhappy initially (Tuson, Sinyor, & Pelletier, 1995).

Exercise that is more intense than participants' habitual level is less likely to improve mood and, indeed, is liable to worsen it. Exercise at competitive levels can worsen mood in habitual exercisers (see Yeung, 1996), and strenuous exercise in people who are not selected for having intense exercise habits has commonly, although not invariably, been unpleasant; that is, it increased negative mood or decreased positive mood (Petruzzello, Jones, & Tate, 1997; Raglin & Wilson, 1996; Step toe & Bolton, 1988; Step toe & Cox, 1988). Comparing responses of sedentary or exercising subjects between different studies carried out under different conditions will never lead to definitive conclusions. Comparisons within studies have generally shown that mood has improved after strenuous exercise selectively in fitter or more active subjects (Boutcher & Landers, 1988; Dishman, Farquhar, & Cureton, 1994; Kraemer, Dziewaltowski, Blair, Rinehardt, & Castracane, 1990; Parfitt, Markland, & Holmes, 1994; Petruzzello et al., 1997) or in those with

more confidence in their exercise capacity (Bozoian, Rejeski, & McAuley, 1994) which could, in turn, reflect greater experience of exercise.

The unpleasant effects of exercise are likely to be heavily underestimated because of selection bias: subjects with negative experiences of exercise would be unlikely to volunteer for the studies reviewed here. Moreover, generalizations must be cautious because the measurement of mood is complex and different negative moods can be affected differently (Petruzzello et al., 1997; Pronk, Crouse, & Rohack, 1995). Effects change over time, too, so that initial mood-worsening during exercise can change into mood-improvement 30 or more minutes later (Raglin & Wilson, 1996; Tate & Petruzzello, 1995). Comparability between different forms of exercise cannot be assumed. Anaerobic exercise has had less clear effects than aerobic exercise. Comparisons, although complicated by the difficulty of matching the two forms of exercise for factors such as exertion and skill, have shown smaller anaerobic effects (Garvin, Koltyn, & Morgan, 1997; McGowan, Pierce, & Jordan, 1991; O'Connor, Bryant, Veltri, & Gebhardt, 1993; Raglin, Turner, & Eksten, 1993). Effects are not specific to exercise; whereas effects of aerobic exercise may persist for longer (Garvin et al., 1997), much of the initial effect, at least, is shared by diverse "control" activities, including relaxation or simply doing nothing (see Yeung, 1996; Youngstedt, O'Connor, Crabbe, & Dishman, 1998). Generalization from these findings is fraught also because, although exercise is regarded in most research as a purely physiological stimulus, its emotional effects depend intimately on social and other environmental cues, and on participants' expectations and concurrent activity (Breus & O'Connor, 1998; Meyer, Kroner-Herwig, & Sporkel, 1990; Turner, Rejeski, & Brawley, 1997; White & Knight, 1984; Zillman, Katcher, & Milavsky, 1972). Even physiological effects of exertion are influenced by environmental stimuli (Harte & Eifert, 1995; Voigt, Ziegler, Grunert-Fuchs, Bickel, & Fehm-Wolfsdorf, 1990). Nevertheless, most available data are accounted for by the generalization that aerobic exercise is a positive experience when performed at the individual's habitual level, and that, although strenuous exercise improves mood in regular exercisers, it can worsen mood, particularly in nonexercisers.

This suggestion offers a simple explanation for why strenuous exercise is adopted much less than its advocates wish: nonexercisers find it unpleasant. This view has important implications for attempts to increase exercise habits, and it sits uncomfortably with the assumption that enjoyment is necessary for both adherence and psychological benefits (Wankel, 1993). It is possible that people who exercise do so *because* they experience exertion positively, although this explanation would beg the question as to why they do. For the present, it is more parsimonious to suppose that the hedonic nature of exercise reverses in the course of training, an hypothesis which can readily be tested. This paradoxical property of exercise will prove central to a way of understanding, below, the long-term effects of exercise training.

## EFFECTS OF EXERCISE TRAINING ON MOOD AND EMOTIONAL DISORDER

More important clinically than the short-term effects of single sessions of exercise are the enduring effects of long-term training. Although systematic research into mental health benefits has continued since Morgan's (1969) demonstration that physically unfit psychiatric patients were more depressed than their fit counterparts, claims for an antidepressive effect have tended to anticipate rather than reflect the accumulation of strong evidence (Folkins & Sime, 1981; Kostrubala, 1976).

### ***Cross-sectional and Longitudinal Surveys***

Recently, however, cross-sectional studies have consistently associated high self-reported levels of habitual physical activity with better mental health. The correlation of habitual exercise level with low depression (but not anxiety) in adolescents (Norris, Carroll, & Cochrane, 1992) and elderly subjects (Ruuskanen & Ruoppila, 1995) is hard to interpret because control variables were omitted. Using a similar, but large ( $N = 5,061$ ) cohort, Steptoe and Butler (1996) showed that vigorous exercise participation was related to lower emotional distress, after controlling for social class and health status. Similarly, in 16,483 university undergraduates, reported exercise correlated with lower depression, after controlling for age and sex only (Steptoe et al., 1997); in 1,536 adults (Weyerer, 1992) and, in separate samples totalling 55,000 (Stephens, 1988), self-reported level of recreational physical activity correlated with better mental health, including fewer symptoms of both anxiety and depression, after controlling for confounding variables including sex, age, socioeconomic status, and physical illness. The association of exercise with well-being may be greater in older than young people (Stephens, 1988; Ransford & Palisi, 1996). Large samples are necessary for positive results and a negative report in a small sample ( $N = 62$ ) should be discounted (de Geus, van Doornen, & Orleibenke, 1993).

Fitness was not directly assessed in these studies. However, Thirlaway and Benton (1992) found that fitness interacted with exercise habits such that highly fit people who did not exercise were in poorer spirits than all others. Fit nonexercisers may have been temporarily prevented from exercise, which worsens mood (Morris, Steinberg, Sykes, & Salmon, 1990). Whatever the reason, emotional correlates of regular exercise cannot simply be attributed to fitness.

Relationships between exercise habits and mood measured simultaneously in such cross-sectional surveys are inherently ambiguous about cause and effect. One methodological improvement is to use structural equation modeling to test causal models, as by Krause, Goldenhar, Liang, Jay, and Maeda (1993) who related exercise habits to low depression in a Japanese elderly sample. Better still, longitudinal surveys have now shown definitely that exercise habits do predict later freedom from depression. Paffenbarger, Lee, and Leung (1994) found that physical activity negatively correlated with depression approximately 25 years later in a sample of 10,201 men. In a sample of 4,848 (Camacho, Roberts, Lazarus, Kaplan, & Cohen, 1991), absence of exercise habits was linked to later depression across two successive 9-year periods. However, despite statistical control for many demographic variables, there was no control for depression at the time that exercise was assessed. Without this, the results might reflect merely the restriction of activity by enduring depression. Farmer et al. (1988) stratified 1,900 adults for preexisting depression. Habitual physical activity predicted freedom from depression at 8-year follow-up, after controlling for demographic and medical variables, although the strength of the effect depended on sex and initial depression. In 2,084 elderly people, stratified into low and high depression groups, daily walking predicted improved depression in each group 3 years later (Mobily, Rubenstein, Lemke, O'Hara, & Wallace, 1996). Similarly, in 1,758 adults with a variety of (mainly physical and chronic) health problems, self-reported time spent exercising predicted a range of positive health outcomes 2 years later including wellbeing and low anxiety, depression and fatigue after allowing for baseline demographic and health indicators, including depression (Stewart et al., 1994). Control variables can be problematic. The lack of relationship of walking habits to emotional distress 7 years

later might reflect the choice of control variables collinear with exercise: body mass index and self-rated health (Emery, Huppert, & Schein, 1996). In a report in which control variables were not included, 679 nondepressed elderly people's exercise habits failed to predict depression 5 years later, but the small proportion of depressed subjects (< 10%) compromised statistical power (Kivela, Kongas-Saviaro, Kimmo, Kesti, & Laippala, 1996).

Another exception to the trend is a report of a male cohort, recruited as medical students, in which strenuous exercise habits were unrelated to depression over successive periods of 15, and 2 years (Cooper-Patrick, Ford, Mead, Chang, & Klag, 1997). This negative result from a sociodemographically homogeneous sample carries a warning: previous positive findings might have arisen because exercise habits correlated with other uncontrolled sociodemographic influences on emotional state. Therefore, causal inferences must still be qualified by failure to measure other key variables. These might include engagement in sedentary activities. In adults over 55 years old, lower depression and greater well-being were associated cross-sectionally with a physical activity (swimming), but similar relationships with sedentary hobbies and visiting friends suggest that physical exercise might merely have been a marker of engagement (Dupuis & Smale, 1995). However, the mediating role of sedentary activities cannot be assumed. In adolescents, participation in such activities was related to *greater* psychological and somatic symptoms, which contrasted with the apparent protective effect of vigorous activity (Steptoe & Butler, 1996).

### ***Exercise Training as an Intervention in Depression***

Whereas cross-sectional studies have related spontaneous exercise habits to lower depression, experimental studies have tested whether formal exercise training programs reduce depression. In an early series of single-case studies of depressed patients, stationary cycling improved mood by comparison with a prior spurious "subliminal" task which controlled for attention and expectations of improvement, although not for skill mastery (Doyne, Chambless, & Beutler, 1983). However, the opportunity for controlled trials of exercise training in people who are clinically depressed is limited because it is implausible that such patients can readily be motivated to exercise. Exercise in such patients is likely to depend on persuasive or therapeutic maneuvers of the kind that are integral to conventional psychological treatment (Beck, Rush, Shaw, & Emery, 1979). That is, the institution of exercise habits could be the *evidence* rather than the basis of successful treatment.

Therefore, most controlled trials that were stimulated by the early case-reports involved subclinically depressed people. They are therefore vulnerable to "floor" effects, where the sample is insufficiently depressed to show improvement. This might explain why depressed mood was unaffected by aerobic training in a well-controlled (but non-randomized) study of unselected adolescents (Norris et al., 1992). Nevertheless, meta-analyses have estimated that depression scores decrease by between 0.3 and 1.3 of a standard deviation after exercise training by comparison with a variety of control conditions, depending on various features of study design (Craft & Landers, 1998; McDonald & Hogdon, 1991; North, McCullagh, & Tran, 1990). However, this generalization masks important methodological problems, particularly with the choice of control procedure. For instance, little can be concluded about specific effects of exercise from comparisons in which controls were untreated (e.g., Doyne et al., 1987), continued with only routine treatment (e.g., Veale et al., 1992), were unsupervised

(McCann & Holmes, 1984), or received a very different psychological treatment (Fremont & Craighead, 1987). Exercisers have sometimes had greater contact than have controls with their therapists or, when exercised in groups, with each other (Griest et al., 1979). In other studies, the very different nature of control and exercise activities is likely to have led to different amounts or types of social interaction (Bosscher, 1993). In other trials, including a recent demonstration that anaerobic training relieved depression (Singh, Clements, & Fiatarone, 1997), control activities were less interesting and engaging than exercise, with less opportunity for skill mastery and social interaction. The use of "occupational therapy" as a control therefore detracts from the finding that depression in psychiatric inpatients was reduced by a program of jogging, cycling, skiing, and swimming (Martinsen, 1987; Martinsen, Medhus, & Sandvik, 1985). A more engaging control activity (meditation and relaxation) produced similar improvement in self-rated depression as did exercise training, each being compared with a psychotherapy group (Klein et al., 1985). Increased social activity is likely to have been a critical feature of exercise in many early studies: solitary exercise did not improve depression (Hughes, Casal, & Leon, 1986).

Exercise training therefore clearly provides a vehicle for nonspecific therapeutic processes. Nevertheless, aerobic training has now been shown specifically to reduce depression in two well-controlled studies of 10–11 weeks of walking and running in subjects selected for subclinical emotional disturbance or exposure to stress. In one, comparison was with relaxation in undergraduates selected for high recent life stress (Roth & Holmes, 1987); the second comparison was with strength and flexibility training in subjects selected for high anxiety (Step toe, Edwards, Moses, & Mathews, 1989). Follow-up showed a maintained effect at 3 months (Step toe et al.) and a nonsignificant effect at 2 months (Roth & Holmes).

Clinically, depression is not defined by high scores on a depression questionnaire, but by patients who are severely demotivated and seek help. Exercise training, which emphasizes patients' motivation and responsibility, does not obviously meet the immediate needs of such patients. It remains for clinical researchers to show that exercise participation is a treatment for severe depression, rather than evidence that it has been treated. Meanwhile, it is unfortunate that the procedures that have been used to motivate exercise in existing studies have only rarely been described (Crook et al., 1998; Friedrich, Gittler, Halberstadt, Cermak, & Heiller, 1998), because they may contain critical treatment components. Furthermore, comparisons are needed between exercise and effective psychological and pharmacological treatments.

### ***Exercise as an Intervention for Anxiety***

Early, uncontrolled reports in which phobic patients were successfully treated by exposure to the phobic stimulus after exhaustive exercise (Driscoll, 1976; Muller & Armstrong, 1975; Orwin, 1973) were explained in terms similar to systematic desensitization; the conditioning to the phobic stimulus of a physiological response (exhaustion) incompatible with anxiety. Current cognitive accounts of anxiety suggest an alternative explanation: Exercise might have facilitated a benign attribution of the arousal produced by the phobic stimulus and thereby prevented the fear-induced element of panic (Clark, 1986). The same reasoning could explain why anxiety responses to adrenaline infusion in undergraduates were least in fit subjects, who might have been more familiar with exercise-induced exertion (van Zijderveld et al., 1992). Panic patients tolerate aerobic exercise, showing physiological responses no greater than in

controls (Rief & Hermanutz, 1996; Stein et al., 1992), even though subjective anxiety may be increased more than in other people (Cameron & Hudson, 1986). In a randomized controlled trial in panic anxiety, dropout from 10 weeks of group and individual strenuous exercise treatment was no greater than from placebo drug treatment (around 30%; Broocks et al., 1998).

Meta-analyses have indicated an anxiolytic effect of aerobic exercise training (Long & van Stavel, 1995; McDonald & Hogdon, 1991; Petruzzello, Landers, Hatfield, Kubitz, & Salazar, 1991). However, the evidence resembles that for depression. Many positive reports were uncontrolled or inadequately controlled by procedures which were less involving (e.g., Goldwater & Collis, 1985) or less plausible than exercise (e.g., Fasting & Gronningsaeter, 1986). Many controlled trials have shown benefits which have proved nonspecific to exercise. Anxiety was reduced similarly by a jogging program as by stress-inoculation (Long, 1984), relaxation (Long & Haney, 1988) or even regular social eating (Wilson, Berger, & Bird, 1981). The nonspecific benefits of exercise clearly help to reduce anxiety, as they do depression. However, exercise training specifically has reduced anxious mood (by comparison with strength and flexibility training) both in subjects selected for high anxiety (Steptoe et al., 1989; in which the effect remained at 3-month follow-up) and in normal subjects (Moses, Steptoe, Mathews, & Edwards, 1989; Norris et al., 1992). The expectation that exercise training would preferentially improve somatic over cognitive anxiety (Schwartz, Davidson, & Goleman, 1978) has not been confirmed (Long, 1984).

Although the clearest evidence of anxiolytic and antidepressant effects of exercise training is therefore from relatively mildly, nonclinically impaired subjects, there are indications, in some of these studies, of greater effects in the more disturbed subjects (Fasting & Gronningsaeter, 1986; Roth & Holmes, 1987; Simons & Birkimer, 1988; Williams & Lord, 1997).

Clinically, severe anxiety is not characterized primarily by patients complaining of high trait-anxiety, but by patients who panic. Therefore, as with studies concerning depression, future research will have more clinical purchase if it addresses the clinical reality of panic anxiety. Broocks et al. (1998) reported clinical improvement in panic anxiety after exercise training by comparison with placebo drug treatment (although less than with clomipramine treatment), but the design did not dissociate exercise effects from nonspecific influences of the therapist or fellow patients.

### ***Emotional Effects of Exercise Training in Physical Conditions***

Where anxiety and depression arise in connection with physical disorders, similar relationships with exercise have been seen. Mood deterioration premenstrually is less in regular exercisers than nonexercisers (Choi & Salmon, 1995a), and there is some evidence that exercise training causes this difference (Israel, Sutton, & O'Brien, 1985; Prior, Vigma, Sciarretta, Alojado, & Schulzer, 1987; Steege & Blumenthal, 1993). The suggestion that exercise might be particularly valuable in pregnancy or postpartum has not been pursued systematically (Koltyn & Schultes, 1997). In substance abuse, despite early positive uncontrolled findings (Sinyor, Brown, Rostant, & Seraganian, 1982), adequately controlled evidence is awaited. Palmer, Palmer, Michiels, and Thigpen (1995) reported that a body-building (i.e., anaerobic) program reduced depression in drug detoxification inpatients, whereas aerobic training did not. However, training was for only 4 weeks and the anaerobic and aerobic programs were apparently social and solitary, respectively.



Exercise training has long been part of rehabilitation programs for coronary patients. A recent meta-analysis has shown significant improvement in anxiety and depression in such studies (Kugler, Seelbach, & Kruskemper, 1994) although, because primarily physiological outcomes have been targeted, control procedures have been psychologically limited. Exercise has also been employed with other disabled or diseased groups. Depression, anger, and fatigue were improved by aerobic exercise in multiple sclerosis patients (Petajan et al., 1996) but comparison was with a no-treatment control.

Syndromes which consist of persistent physical symptoms in the absence of physical pathology are of particular interest because, although patients often seek somatic treatment, their needs are more likely to be psychological. Indeed, in primary care, depression commonly presents somatically (Katon, Kleinman, & Rosen, 1982) and depression has been implicated in major "functional" conditions, in particular chronic fatigue (Wessely & Powell, 1989). These syndromes may therefore include depressed patients who, while rejecting conventional psychological treatment, would be receptive to the somatic orientation of treatment by physical exercise. Although exercise has often been included in rehabilitation and mobilization packages, it has only rarely been isolated for evaluation. The history of therapeutic failure in this type of patient can reduce take-up and retention (Norregaard, Lykkegaard, Mehlsen, & Danneskiold-Samsøe, 1997). Nevertheless, there are preliminary positive reports. The (uncontrolled) addition of brief (4–6 week) aerobic and other exercise training to educational interventions ameliorated low back pain and disability and increased self-efficacy in fibromyalgia (Burckhardt, Mannerkorpi, Hedenberg, & Bjelle, 1994; Frost, Moffett, Moser, & Fairbank, 1995). Also in fibromyalgia, Wigers, Stiles, and Vogel (1996) found improvement in pain and energy after a 14-week aerobic program in comparison to routinely treated controls, but exercised patients rated the social content as a key component of treatment. Although showing that aspects of exercise training can help in mobilizing such patients, these results do not confirm specific effects of exercise. However, by using relaxation and flexibility training as a control, Fulcher and White (1997) have shown that aerobic training reduced fatigue in patients with chronic fatigue syndrome. It remains to determine how generally applicable are the specific benefits of exercise in patients with "functional" conditions. In a study of primary care patients with persistent unexplained symptoms of diverse kinds, we have found that self-rated depression improved comparably after aerobic exercise training and a relaxation and stretching control (Peters, Stanley, Rose, Kaney, & Salmon, 2000).

### **PSYCHOPATHOLOGY IN EXERCISERS**

If exercise is a way of improving emotional state, it might be expected that adherents include many who take up exercise because of emotional problems. Reliable evidence is obviously hard to obtain although, from retrospective interviews with runners, Colt, Dunner, Hall, and Fieve (1981) reported such a finding. The gradual increase in symptoms of depression and anxiety over 2 weeks after cessation of regular running is consistent with recovery of preexisting emotional disorder (Morris et al., 1990).

In clinical literature, however, intense exercise has commonly been seen as an expression or cause of pathology rather than a way of coping with it, a view which would militate against encouraging exercise for clinical reasons. There is little support for the views that intense commitment to exercise represents a narcissistic concern with

the body (Sacks, 1987) or, conversely, a form of masochism (Cooper, 1981). Notwithstanding evidence that weight preoccupation and excessive exercise occur in largely separate groups of women (Davis & Fox, 1993), and that intense runners and anorexia nervosa patients have different physiological and personality profiles (Powers, Schocken, & Boyd, 1998), preoccupation with diet, pathological attitudes to exercise, and obsessive-compulsiveness are all associated in anorexic patients (Davis et al., 1995). This is consistent with the clinically based hypothesis that excessive exercise is homologous with anorexia nervosa (Yates, 1991; Yates, Leehey, & Shisslak, 1983). There is little support for the suggestion that excessive exercise leads to dieting and weight preoccupation (Davis, Fox, Cowles, Hastings, & Schwass, 1990).

Excessive exercise has been viewed as giving rise to physiological dependence (Loumidis & Wells, 1998; Veale, 1987) although this view is supported mainly by anecdotal and single-case evidence (e.g., Griffiths, 1997). Interruption of exercise leads, within one week, to physical symptoms, somatic anxiety and feelings of inability to cope, but the intensity of these feelings does not approach the intensity of withdrawal from opiates (Gauvin & Szabo, 1992; Morris et al., 1990).

### EXPLAINING EMOTIONAL EFFECTS OF EXERCISE TRAINING

Changes in aerobic fitness are probably unimportant to the effects on mood. First, although anaerobic exercise has received very little attention, the evidence that exists indicates an antidepressant effect comparable to that of aerobic exercise. However comparisons have been uncontrolled (Martinsen, Hoffart, & Solberg, 1989) or controlled by untreated subjects (Doyle et al., 1987; Norvell & Belles, 1993) or groups have differed in therapist supervision (Anshel & Russell, 1994). Secondly, after aerobic training, reduction in anxiety or depression has generally not correlated with physiological indices of fitness (Fasting & Gronningsaeter, 1986; Martinsen et al., 1989; Simons & Birkimer, 1988; Steptoe et al., 1989). Thirdly, anxious mood is reduced by mild exercise training, insufficient to increase fitness, whereas training which is sufficiently intense to increase fitness is less effective at relieving anxiety (Moses et al., 1989). Furthermore, in Roth and Holmes' (1987) and McCann and Holmes' (1984) studies, depression declined within 5 weeks from the start of training, before fitness would have been expected to change. Conversely,  $VO_2$ max can be improved by exercise training, but without improvement in depression (Swoap, Norvell, Graves, & Pollock, 1994).

Explanations for emotional effects of exercise training should therefore be considered in which aerobic fitness does not feature. Diverse suggestions have included changes in body temperature or cerebral blood flow (see Dishman, 1995; Martinsen, 1987), improvement in self-esteem (Folkins & Sime, 1981), distraction from negative thoughts (Morgan, 1985, 1987), or improved retrieval of positive thoughts (Clark, Milberg, & Ross, 1983). However, it is premature to pursue such specific explanations until more general questions have been addressed.

Broadly, there are two possible types of explanation. One is that emotional benefits arise from the accumulation of acute mood improvement caused by the individual sessions of exercise. Accumulation of acute effects has been suggested by mainly anecdotal, single-case, or uncontrolled reports that have suggested that mood deteriorates rapidly when exercise regimes are interrupted (Baekeland, 1970; Conboy, 1994; Mondin et al., 1996; Sime, 1987; Szabo, Frenkl, Janek, Kalman, & Laszay, 1998; Thaxton,

1982). Even reduction in intensity of training has been reported to worsen mood (Wittig, McConell, Costill, & Schurr, 1992). However, a theory based entirely on acute emotional effects is implausible because, as was argued above, exercise is likely to be aversive to many people, particularly at the start of training. Moreover, one controlled report of relatively prolonged deprivation is available which suggests a more complex picture (Morris et al., 1990). This showed that, despite a relatively rapid increase in physical symptoms and feelings of being unable to cope, depression and anxiety increased only after 1–2 weeks of deprivation. The relatively long-term appearance of anxiety and depression suggests a gradual loss of a long-term effect of exercise training, and is consistent with an alternative explanation that repeated exercise recruits an enduring process which gradually improves mood. This will be pursued below.

### EXERCISE TRAINING AND RESISTANCE TO STRESS

A hitherto separate research area has concerned the effect of exercise training to reduce vulnerability to stress. Reports can be distinguished according to whether differences in exercise experience have been studied cross-sectionally or experimentally, whether stress has been studied in real life or modelled in the laboratory and, finally, according to the types of stress and stress response that have been examined.

#### ***Cross-sectional Studies of Controlled Laboratory Stressors***

This refers to studies in which groups have been selected on the basis of preexisting differences in exercise history (or physical fitness) and then exposed to a contrived stressor. Index responses have typically been cardiovascular. A meta-analysis is available, summarizing mainly cross-sectional studies, which found an association of fitness with smaller stress responses (Crews & Landers, 1987). This conclusion masks a large degree of inconsistency out of which, nevertheless, some patterns emerge.

Most negative results accrued from attempts to contrast physiological responses (typically heart rate and systolic and diastolic blood pressure) to mental arithmetic or psychomotor tasks between fit and unfit people drawn from the normal population (Clayton, Cox, Howley, Lawler, & Lawler, 1988; de Geus et al., 1993; Hollander & Seraganian, 1984; Hull, Young, & Ziegler, 1984; Keller & Seraganian, 1984; Plante & Karpowitz, 1987; Seraganian, Roskies, Hanley, Oseasohu, & Collu, 1987; Sinyor, Schwartz, Peronnet, Brisson, & Seraganian, 1983; Zimmerman & Fulton, 1981). Significant contrasts have been more likely when this procedure has been modified in one of three ways. First, use of more subtle measurements of cardiovascular function to indicate sympathetic activity has yielded effects in some studies (van Doornen & de Geus, 1989; de Geus, van Doornen, de Visser, & Orlebeke, 1990; Shulhan, Scher, & Furedy, 1986) but not all (de Geus et al., 1996). A second approach has been to contrast extreme groups finding, in response to stress, less electrodermal lability in marathon runners than sedentary subjects (Keller & Seraganian, 1984), smaller heart rate responses in very fit than in very unfit undergraduates (Holmes & Roth, 1985; Light, Obrist, James, & Strogatz, 1987) and smaller increases in heart rate, diastolic blood pressure, and total peripheral resistance in athletes than in normal controls (van Doornen & de Geus, 1989). A similar comparison, but with negative results (Clayton et al., 1988), was based on very small samples ( $Ns = 8$ ).

The third approach to demonstrating differences between fit and unfit groups has been to select them from populations known to display greater than normal cardiovascular lability in response to psychological stress. Thus, in subjects with a family history of hypertension, being fit protected against blood pressure responses to a color-word conflict task (Holmes & Cappo, 1987; c.f. O'Brien, Hayes, & Mumby, 1998). Age may be a further moderator of the effects of fitness. In an isolated report, Hull, Young, and Ziegler (1984) found no association of fitness with smaller hemodynamic responses to stress, except in a subgroup aged over 40 years.

### ***Experimental Studies of Controlled Laboratory Stressors***

Truly experimental studies, in which exercise training has been controlled, have been fewer than the cross-sectional ones. Here, also, the emphasis has been on cardiovascular responses. Despite positive findings in an early nonrandomized comparison (Holmes & McGilley, 1987), many negative reports have since accumulated (Blumenthal et al., 1991; Sinyor, Golden, Steinert, & Seraganian, 1986; Steptoe, Kearsley, & Walters, 1993b; Steptoe, Moses, Mathews, & Edwards, 1990), even after extended training for 4–6 months (Albright, King, Taylor, & Haskell, 1992; de Geus et al., 1993). In other studies, the familiar effect of exercise training to reduce baseline heart rate and blood pressure has obfuscated differences in response to stress (Plante & Karpowitz, 1987; Holmes & Roth, 1988). In a recent randomized comparison, heart rate during recovery from stress was lower after exercise training (which included aerobic and anaerobic components) than a control activity (but this was merely group seminars; Calvo, Szabo, & Capafons, 1996).

As with cross-sectional studies, positive results have been more likely where samples have been selected for cardiovascular sensitivity to stress. In two studies of Type A men, a 12-week walking and jogging program reduced heart rate and blood pressure responses to mental arithmetic by comparison with a strength and flexibility control (Blumenthal et al., 1988, 1990). Using a similar design, Sherwood, Light, and Blumenthal (1989) found a similar result, but only in those Type A men who also were borderline hypertensive. One study of Type A men found no effect of exercise training, but subjects with exaggerated psychophysiological activity had been excluded (Seraganian et al., 1987). In uncontrolled studies in borderline hypertensive subjects, low- or moderate-intensity training has reduced blood pressure responses to the Stroop color-word conflict test (Rogers, Probst, Gruber, Berger, & Boone, 1996) or a video game (Cleroux, Peronnet, & de Champlain, 1985).

### ***Validity of Laboratory Stressors and Responses***

One conclusion from the inconsistency of this evidence is that although, on balance, exercise training bestows some protection against stress responses, its effect depends on subject variables or procedural details. First, however, the validity of the mental stress tasks that have featured in this work should be questioned. There has been a tendency to regard different stressors as interchangeable. However, stress is not unitary, and different demands have different physiological effects: in particular, whereas tasks that demand effortful coping responses preferentially stimulate noradrenergic responses, novelty, lack of control, or the need for adaptation are features to which the pituitary-adrenal system is more sensitive (Steptoe, 1983). These distinctions have not been systematically related to effects of exercise train-

ing. Nevertheless, it has been suggested that fitness effects on cardiovascular or sympathoadrenal responses are seen preferentially in well-learned tasks rather than novel, threatening ones (Blaney, Sothmann, Raff, Hart, & Horn, 1990). A separate consideration is the ecological validity, or realism, of the stressors. A report in which exercise training did reduce blood pressure and heart rate stress responses in an unselected male group used a more life-like stressor than has been typical: losing a motor task to a female (Anshel, 1996).

The validity of the cardiovascular responses which have usually been measured must also be questioned. Their predominance in the literature reflects an assumption that, because exercise training reduces cardiovascular responses to physical stress, it should have a similar effect in psychological stress. However, this assumption is negated by the different physiological mechanisms that underlie superficially similar cardiovascular responses to physical and psychological challenge (van Doornen, de Geus, & Orlebeke, 1988). Moreover, conclusions cannot be simply generalized from laboratory stressors to ambulatory conditions (Steptoe & Vogele, 1991). Neither can cardiovascular effects be generalized to other responses—even physiological ones. The pituitary-adrenal axis has received little attention in this context, but the few studies in which cortisol or ACTH have been measured have shown no difference between fit and unfit subjects in responses to a variety of tasks (Blaney et al., 1990; Brooke & Long, 1987; Sinyor et al., 1983; Sothmann, Hart, & Horn, 1991). Furthermore, cardiovascular responses do not correlate with mood changes (Steptoe, Moses, Edwards, & Mathews, 1993). Behavioral indices of resistance to stress have been well-developed in animal experiments which focus on persistence, that is, continuing an activity that stress normally disrupts (Amsel, 1972; Gray, 1975). This approach has not been used in human studies.

One approach to choosing an index behavioral response is according to its ecological validity. In a complex design, Zillman, Johnson, and Day (1974) found that fitter subjects retaliated least to a provocative stooge. However, interpretation is complicated by the unconventional measure of fitness (recovery in blood pressure after cycling) and the use of exercise to stimulate arousal shortly before exposure to the stooge. In Anshel's (1996) simpler design, which exposed males to the stress of losing a motor task to a female, mood was said to be better preserved in exercise-trained than control subjects, but the report is unclear in this. Clearer evidence is from Calvo et al. (1996) who used self-reported and behavioral observations to show that anxiety associated with evaluative stress was lower after exercise training than in untrained controls.

### ***Cross-sectional Studies of Responses to Real-life Stress***

Some instances of real-life stress can be studied in a controlled way although generalizability of findings to more routine stressors cannot be assumed. Thus, Brooke and Long (1987) found that subjective anxiety and plasma noradrenaline levels recovered faster from abseiling in fit than in unfit subjects.

Questionnaires can be used to quantify more mundane, spontaneously occurring stressors, although the findings are inherently ambiguous concerning the direction of cause and effect, as in a report that people who habitually exercise find their lives less stressful (Norris et al., 1992). More recently, Aldana, Sutton, Jacobson, and Quirk (1996) correlated perceived life stress with low levels of physical activity, after controlling for major life change and self-ratings of physical health. Kobasa, Maddi,

Puccetti, and Zola (1985) selected business executives for a high level of recent life event stress, and found fewest symptoms of physical and psychiatric illness in those who exercised most. There is no reason to suppose that these symptoms were an effect of stress, but other studies have confirmed that the statistical relationship of recent life event scores to illness is weaker in fit than in unfit subjects (Brown, 1991; Brown & Lawton, 1986; Roth & Holmes, 1985) or in exercisers than nonexercisers (Brown & Siegel, 1988). Although Roth, Wiebe, Fillingim, and Shay (1989) could not replicate this, they categorized subjects according to their own subjective estimates of fitness.

Given the correlational design, this pattern of findings is open to different interpretations. An unmeasured constitutional or environmental variable might lead both to resilience and to readiness to exercise, or people who are less disturbed by stress might simply be more ready to take up exercise training. Alternatively, physical exercise training might confer protection from deleterious effects of stress. Consistent with this, Steptoe, Kimbell, et al. (1998) found that exercise was related to lower perceived stress in day-to-day, within-subjects variation, although only in a subgroup who were low in anxiety.

### ***Experimental Studies of Real-life Stress***

To overcome this ambiguity, controlled trials of exercise training are required in which responses to stress are studied prospectively. Cramer, Nieman, and Lee (1991) found that reported daily hassles were reduced 6 weeks into a walking program, compared to an untreated control group (although not after 15 weeks). Unfortunately, a second report that perceived life stress was reduced by exercise training (Norris et al., 1992) is compromised by nonrandom allocation to exercise and control groups. Focusing on responses to a specific stressor (being diagnosed HIV positive) LaPerriere et al. (1990) found that men who had trained aerobically for 5 weeks were protected from the increase in emotional distress and impairment of immune function (decline in natural killer cell number) shown by untrained controls.

In real life, physical activity is decreased by the stress of academic examinations (Steptoe, Wardle, Pollard, Canaan, & Davies, 1996) or treatment for cancer (Courneya & Friedenreich, 1997)—although not by unpredictable hassles (Steptoe, Lipsey, & Wardle, 1998). The resulting loss of the protective benefit of exercise could further intensify the response to those stressors.

## **EXPLAINING EFFECTS ON STRESS RESPONSES**

The details of, and constraints upon, the effect of exercise on stress responses remain to be clarified. Nevertheless, the balance of the evidence indicates that sensitivity to stress is reduced after exercise training. As with antidepressive and anxiolytic effects, two broad explanations should be considered. The first is the accumulation of acute effects of individual exercise sessions. In turn, two types of acute effect can be envisaged. One is to palliate responses to concurrent or recent stress. Although cardiovascular responses to mental stress are clearly increased by concurrent exercise (Rousselle, Blascovich, & Kelsey, 1995), an inhibitory effect of exercise on concurrent *emotional* stress responses (Girodo & Pellegrini, 1976) is consistent with popular be-

lief that exercise can help one to cope with stress and other problems (Choi & Salmon, 1995b; King & Brassington, 1997; Long, 1993) and with evidence, in animals, that wheel-running exercise reduces sympathoadrenal or pituitary-adrenal responses to prior stress (Mills & Ward, 1986; Starzec, Berger, & Hesse, 1983).

The second possible acute effect would be to attenuate responses to stressors experienced shortly *afterwards*. Despite its well-attested acute hypotensive effect, evidence is mixed as to whether cardiovascular or sympathoadrenal responses to psychological stress are reduced by prior exercise. It is not clear what distinguishes studies in which one or more variable has shown positive results (Anshel, 1996; Boone, Probst, Rogers, & Berger, 1993; Ebbesen, Prkachin, Mills, & Green, 1992; Fillingim, Roth, & Cook, 1992; Hobson & Rejeski, 1993; Peronnet, Massicotte, Paquet, Brisson, & deChamplain, 1989; Probst, Bulbulian, & Knapp, 1997; Rejeski, Thompson, Brubaker, & Miller, 1992; Roy & Steptoe, 1991; Steptoe et al., 1993b) from those without effect (Flory & Holmes, 1991; McGowan, Robertson, & Epstein, 1985; Roth, 1989; Roth, Bachtler, & Fillingim, 1990). Emotional responses to stress have been both reduced or increased, depending on the conditions. Increase (Meyer et al., 1990; White & Knight, 1984; Zillman et al., 1972) has been explained by subjects misattributing to the emotional challenge the physiological arousal produced by exercise; where the experiment was not designed to promote such misattribution, prior exercise reduced anxiety associated with threatening tasks (Roth, 1989), although this effect was no greater than that of prior relaxation (Doan, Plante, Digregio, & Manuel, 1995; Rejeski et al., 1992). Subjective stressfulness of mental stress was unaffected by prior exercise in one report (Ebbesen et al., 1992), whereas personal problems felt less serious after a moderate walk (Thayer, 1987b).

The alternative to attributing the stress-reducing effects of exercise to the accumulation of acute effects is to suppose that a long-term process is recruited. One way to distinguish short-term from long-term effects is to study the effects of interruption of regular exercise. Whereas an acute effect should dissipate rapidly, a long-term effect would be expected to persist. There is one uncontrolled report that cardiovascular responses to mental stress did not change after 1 week of exercise interruption, (Szabo & Gauvin, 1992). Any long-term process is unlikely to involve aerobic fitness. In a cross-sectional study in which subjects were selected to be similar in exercise habits, although varying in fitness, cardiovascular reactivity was *greater* in the fitter subjects (de Geus et al., 1993). Moreover, low-intensity training, which did not increase  $\text{VO}_2\text{max}$ , has more effectively reduced cardiovascular stress-responses than a high-intensity program which did improve fitness (Rogers et al., 1996).

## EXERCISE TRAINING AS STRESS ADAPTATION

Diverse explanations have been proposed for one or other psychological effects of exercise training, but many have been inconsistent with understanding of the mechanisms that control emotional state or stress responses (see Dishman, 1995), or have focused on one effect only. By contrast, the overall pattern of effects is an invitation to a broader, unifying theory. Such a theory should accommodate key features of the evidence reviewed here:

1. exercise can be aversive, but also has positive hedonic properties, most clearly after extended training;

2. exercise training has antidepressive and anxiolytic effects;
3. exercise training reduces sensitivity to stress.

In setting out his theory of opponent processes, Solomon (1980) cited exercise as an instance of a class of stimuli which, upon repetition, lost their negative hedonic tone: that is, produced tolerance. This tolerance was attributed to the gradual recruitment of a counter-regulatory process which ultimately leads to a positive hedonic response to such stimuli. Although Solomon thought that the opponent process was automatically elicited, there is evidence to attribute it to classical conditioning (Schull, 1979). The limitation of Solomon's theory for present purposes is that it cannot explain how repeated exercise could change the hedonic response to stimuli other than exercise. Lees and Dygdon (1988) drew on a separate conditioning-based theory of opponent-type processes: counterconditioning. This explains how stimuli that are aversive can acquire positive motivational properties by Pavlovian association with stimuli which are themselves positive. Lees and Dygdon (1988) argued that exercise, although initially unpleasant, could acquire positive tone by its contingent relationship with positive reinforcers, particularly those arising from the social interaction that characterizes exercise. Counterconditioning is, however, a more powerful explanatory construct than Lees and Dygdon envisaged. In animal experiments, effects extend beyond the specific aversive stimulus that was employed in conditioning. For instance, animals that have learned to tolerate fear of electric shock through its association with food reward turn out to tolerate stimuli predicting a very different aversive event, also: frustrative nonreward (Gray, 1975). In theories that differ in the detailed conditioning mechanisms that they assume, both Amsel (1972) and Gray (1975, 1982) have explained how resistance to stress or disruptive events in general can be acquired through exposure to one type of aversive event in a counterconditioning paradigm. In people, of course, Pavlovian conditioning, including counterconditioning, does not require formal contingencies. Verbally transmitted information about the health benefits or social approval of exercise can substitute for these (Lees & Dygdon, 1988).

Counterconditioning provides an instance of a more general phenomenon of generalized stress tolerance—or “toughening up” (Gray, 1982)—which occurs across a variety of paradigms in animal research. For example, after repeated exposure to cold water, animals are protected from disruptive behavioral effects of uncontrollable electric shock, and vice versa (Weiss, Glazer, & Miller, 1975). Exercise has sometimes been a component of the stressful procedures employed in this research, such as in cold-water swimming, above. A few experiments have attempted to isolate effects of exertion from the stressors with which it has been confounded in such paradigms, showing that animals with extensive prior experience of running in a wheel, or of swimming, show reduced behavioral disruption when tested in an open field (a large open arena in which sensitivity to stress is indicated by reduced mobility: Dishman et al., 1996; Tharp & Carson, 1975; Weber & Lee, 1968) or when tested for escape learning after uncontrollable electrical shock (Dishman et al., 1997).

The importance of these paradigms to understanding the range of effects of exercise training is not just that they model sensitivity or resilience to stress, but that they have also been regarded as models of anxiety and depression, their validity being argued theoretically and empirically from effects of anxiolytic and antidepressant drugs (Gray, 1982; Willner, 1985). Therefore, the present thesis is that stress-adaptation provides a theoretical framework for understanding the effects of exercise training on anxiety and depression, and on resistance to stress.



## NEUROCHEMICAL CORRELATES OF EFFECTS OF EXERCISE

In parallel with behavioral adaptation, stress causes physiological adaptation. Michael (1957) suggested that adaptation of the adrenal glands underlay stress-adaptation by exercise. In the intervening decades it has been appreciated that the physiological adaptations that underlie behavioral adaptation to stress are to be found, not peripherally, but in the central nervous system. Changes in several neurotransmitter systems have been causally implicated in behavioral adaptation. Review of these is beyond the scope of the present article. However, noradrenergic and opioid effects of exercise have particular implications for understanding clinical effects. Previously, each has been invoked as an explanation for psychological effects of exercise: noradrenergic systems have been suggested to subserve antidepressant effects, and opioid activation has been invoked to explain mood improvement. Rather than using neurochemical arguments in this reductionist way, the approach here is to support the emerging behavioral theory by showing parallels with, and links to, neurochemical adaptation to exercise.

### *Central Catecholamine Systems in Exercise*

In general, stressors activate brain norepinephrine systems in animals and acutely deplete brain levels of norepinephrine. When stress is chronic, synthesis of norepinephrine is increased so that brain concentrations are preserved. In some theories of resistance to stress, these changes have been regarded as causal. Effects of exercise resemble those of other forms of stress. Brain norepinephrine turnover is increased by swimming or wheel-running (Chaouloff, 1989) and norepinephrine levels are depleted by swimming (Barchas & Friedman, 1963) and forced running (Gordon, Spector, Sjoerdsma, & Udenfriend, 1966). Effects of long-term exercise training also parallel repeated exposure to other stressors. Long-term regimes of swimming (Ostman & Nyback, 1976) or running (whether compelled by a treadmill, induced by shock-avoidance, or spontaneous) preserve or increase brain norepinephrine levels (Brown & van Huss, 1973; Brown et al., 1979; Dishman et al., 1997).

### *Opioid Mechanisms in Effects of Exercise*

Stress also activates central (and peripheral) opioid systems and this accounts for some instances of the analgesia which is caused by stress. Spontaneous exercise shares these effects, increasing endogenous opioid activity in the peripheral and central nervous system (Harber & Sutton, 1984); Thoren, Floras, Hoffman, & Seals, 1990). There is indirect evidence that such release is psychologically important. Strenuous aerobic exercise is analgesic in man, and opioid antagonists can reverse some instances of exercise-induced analgesia (Fuller & Robinson, 1993; Haier, Quaid, & Mills, 1981; Janal, Colt, Clark, & Glusman, 1984; Koltyn, Garvin, Gardiner, & Nelson, 1996). Opioid mechanisms have also been implicated in mood improvement by running in regular runners; the opioid antagonists, naloxone, attenuated this effect in two reports (Allen & Coen, 1987; Janal et al., 1984), although not in a third (Markoff, Ryan, & Young, 1982). In animals, the repeated activation of endogenous opioid systems by exercise leads to tolerance and withdrawal phenomena that are similar to, and cross-tolerant with, those caused by repeated administration of exogenous opiates (Christie &

Chesher, 1982; Christie, Chesher, & Bird, 1981; Christie, Trisdikoon, & Chesher, 1982).

In the present context, the functional importance of these opioid responses arises from their inhibitory control of stress responses. Opioid antagonists increase cardiovascular stress responses to both physical and psychological challenge (Grossman & Moretti, 1986; Morris, Salmon, et al., 1990), increase the intrinsically smaller stress reactivity in certain individuals (McCubbin, Kaplan, Manuck, & Adams, 1993), and reverse the effect of relaxation training to reduce blood pressure responses to psychological stress (McCubbin et al., 1996). In the central nervous system, also, catecholaminergic stress responses are under opioid inhibition (Tanaka et al., 1983). The key to the role that opioid mechanisms might play in effects of exercise is their dependence on exercise history. For instance, the plasma beta-endorphin response to exercise increases with training (Carr et al., 1981), and there is evidence that the potentiation of opioid inhibition accounts for the reduction in cardiovascular stress responses after exercise training (McCubbin, Cheung, Montgomery, Bulbulian, & Wilson, 1992).

There are paradoxes in the view that regular exercise recruits opioid activation, and the popular belief that attributes many of the effects of exercise to a release of endorphins is certainly an oversimplification. For instance, exogenous opiate is not necessarily experienced as pleasant, particularly in regular users (O'Brien, Ehrman, & Ternes, 1986). There are also important gaps in the picture; for instance regarding the interaction of increased opioid activation by exercise training with the opioid tolerance that develops through training. Nevertheless, available data are consistent with a theory in which adaptive changes in opioid systems link regular exercise to reduced stress responses, particularly those controlled by noradrenergic systems.

### IMPLICATIONS FOR FUTURE RESEARCH

The function of proposing that exercise is a human analogue of stress adaptation is not to provide answers, but to offer a way of asking questions about effects of exercise in future that are better integrated into psychobiological theory than hitherto. In reality, no single theory can account for the effects of such a complex stimulus as exercise. Nevertheless, although processes such as social integration, self-mastery, and distraction will, in practice, influence the effects of exercise, the present theory leads to predictions that depend specifically on the stressfulness, or aversiveness, of exercise.

The first prediction is that development of the positive hedonic tone of exercise, and the long-term protective effects of exercise against emotional disorder and stress, depend on its initial unpleasantness. To confirm this would contrast with the usual assumption that enjoyment of exercise is a prerequisite for adherence and psychological benefits (Wankel, 1993). It would, for instance, have implications for the expectation that is commonly provided to novices that exercise should be pleasant from the start.

Different sets of predictions arise from the different explanations that have been offered for stress tolerance (Gray, 1982). From a counterconditioning view, it would be predicted that social or other rewards which are conventionally associated with exercise are crucial to its benefits. These would, however, be unimportant according to the view that stress adaptation is essentially a function of the repetition of exercise. Although repeated exposure to uncontrollable stressors eventually produces resistance to stress, exposure to controllable stress achieves this more quickly (Maier & Selig-

man, 1976; Weiss & Glazer, 1975). The particular value of exercise might therefore be that it is a controllable stressor. On this basis, to maximize clinical benefit, participants' perception of being in control of the exercise regime should be maximized. Correlated with stressor controllability is predictability and this may be the more important property for stress adaptation. Indeed, a paradigm of unpredictable stress is used as a model for sensitization to stress (Willner, 1985). On this reasoning, the routine and predictable nature of exercise would prove critical.

## CONCLUSION

Claims for the emotional benefits of exercise are rooted in philosophical and religious ideas that date from at least 2,500 years ago (Dishman, 1986) and evidence is now catching up with these claims. Undoubtedly, exercise provides a vehicle for many nonspecific therapeutic processes, including physiological benefits of mobilization and psychological benefits of self-mastery and social integration. Effects related specifically to exertion include anxiolytic and antidepressant action, but also resistance to physiological and emotional consequences of psychological stressors.

There is a need for greater clinical realism in evaluating emotional effects of exercise. Too many studies demonstrate antidepressant, anxiolytic, or stress-reducing effects in people who have not asked for these benefits. In particular, future research should explore effects in panic anxiety and clinical depression. In addition to providing a novel approach to familiar clinical problems, exercise permits intervention in new areas. Whereas treatments in clinical psychology routinely aim to alleviate the emotional effects of stressors that have already occurred, exercise training provides a way to ameliorate effects of stressors yet to occur.

The potential value of physical exercise to the clinical psychologist derives not merely from its empirical and theoretical base, but from its popularity and face validity as a way of improving well-being. In this respect, for many individuals, it is likely to contrast with cognitive and behavioral approaches that are more common in the psychologist's armamentarium but appear less accessible to the general population. For instance, exercise might prove to be of particular use where patients with emotional problems reject ostensibly psychological diagnoses and treatments.

Physical exercise is potentially important to clinical research also, because it may allow the experimental manipulation of resilience in a way that has, hitherto, been largely confined to the animal laboratory. Nevertheless, exercise is a complex psychological stimulus, which changes as its cultural significance changes. Therefore the challenge for future research is to be grounded in psychobiological theory, while also being sensitive to the social and cultural context in which exercise occurs.

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