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A Meta-Analysis on the Anxiety-Reducing Effects of Acute and Chronic Exercise

Outcomes and Mechanisms

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Contents

Summary The relationship between exercise and anxiety has been extensively examined over the last 15 years. Three separate meta-analyses were conducted to quantitatively review the exercise-

anxiety literature for state anxiety, trait anxiety and psychophysiological correlates of anxiety. Such a procedure allows tendencies of the research to be characterised.

The results substantiate the claim that exercise is associated with reductions in anxiety, but only for aerobic forms of exercise. These effects were generally independent of both subject (i.e. age and health status) and descriptive characteristics. Numerous design characteristics were different, but these differences were not uniform across the 3 meta-analyses. For state anxiety, exercise was associated with reduced anxiety, but had effects similar to other known anxiety-reducing treatments (e.g. relaxation). The trait anxiety mcta-analysis revealed that random assignment was important for achieving larger effects when compared to the use of intact groups. Training programmes also need to exceed 10 weeks before significant changes in trait anxiety occur. For psychophysiological correlates, cardiovascular measures of anxiety (e.g. blood pressure, heart rate) yielded significantly smaller effects than did other measures (e.g. EMG, EEG).

The only variable that was significant across all 3 meta-analyses was exercise duration. Exercise of at least 21 minutes seems necessary to achieve reductions in state and trait anxiety, but there were variables confounding this relationship. As such, it remains to be seen what the minimum duration is necessary for anxiety reduction. Although exercise offers therapeutic benefits for reducing anxiety without the dangers or costs of drug therapy or psychotherapy, it remains to be determined precisely why exercise is associated with reductions in anxiety. Since several mechanisms may be operating simultaneously, future research should be designed with the idea of testing interactions between these mechanisms.

The pervasiveness of mental health disturbances presents a significant social problem. For example, according to a recent report, approximately 30 million Americans suffer from anxiety and depression to the extent that a 'normal' lifestyle is interrupted (Dishman 1982). Additionally, estimates contained within earlier reports indicated that 10 million Americans manifest symptoms of anxiety neurosis (Morgan 1979) and that 30 to 70% of those patients seen by general practitioners and internists suffer from unrelieved stress.

Although a number of psychotherapeutic interventions (e.g. meditation, biofeedback, relaxation, hypnosis) exist for the alleviation of these conditions, substantial interest has centered, and continues to focus, on the mental health consequences resulting from physical activity. Many health professionals believe that exercise can prevent the onset of emotional problems and also serve as an effective treatment modality once such problems have developed (Morgan & Goldston 1987). A survey of 1756 primary care physicians (Ryan 1983) revealed that exercise was routinely prescribed for treatment of emotional disorders [depression (85%), anxiety (60%), and chemical dependence (43%)]. Primary care providers, however, are also prescribing psychotropic drugs for mental health problems (Dishman 1986). Since there are numerous adverse effects that can occur with psychotropic drug administration (e.g. potential withdrawal when treatment is stopped, requirement for prolonged use), and since exercise can potentially offer similar benefits, it would be of significant interest to determine the anxiolytic (i.e. anxiety-reducing) effects of exercise. The National Institutes of Mental Health have also identified the topic of anxiety and exercise as being of immediate concern (Morgan & Goldston 1987). Unfortunately, the scientific validation of these effects has been clouded by an abundance of testimonials and anecdotal reports touting unsubstantiated and extravagant claims. As Michael (1957) stated 'the feeling is subjective and the report is not accepted by many as reliable data' (p. 50).

During the 1960s a number of scientific investigations implicated physical fitness in the amelioration of various mental illnesses (Hodgdon & Reimer 1960; Morgan 1968, 1969, 1970a,b). In general, results from these studies revealed that various aspects of aerobic and musculoskeletal fitness were lowered in patients evidencing more severe clinical disturbance. Some reviews also ap-

peared which addressed the relationship between physical fitness and selected personality variables as well as mental health concerns (Cureton 1963; Layman 1974; Tillman 1965).

Early research results appeared quite intriguing; exercise seemed to lead to both physiological and psychological benefits, and was more economical and nonthreatening than traditional forms of psychotherapy. However, the majority of the early studies were correlational or cross-sectional (Hodgdon & Reimer 1960; Morgan 1968, 1969, 1970a,b) or lacked adequate motivation (i.e. expectancy) control groups (Cureton 1963), such that the conclusions drawn from them were generally limited by the failure to manipulate exercise. Accordingly, a number of alternative explanations could have accounted for the mental health profiles observed in the fit and unfit populations. Despite these concerns, the results were provocative and stimulated a number of investigators to actively control exercise environments while carefully examining psychological responses and adaptations to acute and chronic exercise stress.

Numerous authors have attempted to synthesise the research literature related to the anxiolytic effects of exercise (deVries 1981; Dishman 1982, 1985, 1986; Doan & Scherman 1987; Folkins & Sime 1981; Goff & Dimsdale 1985; Hughes 1984; Ledwidge 1980; Mihevic 1982; Morgan 1981, 1983, 1985; Phelps 1987; Raglin & Morgan 1985; Van Andel & Austin 1984), with some focusing on specific methodological approaches (e.g. psychophysiological, Hatfield & Landers 1987). While these 'narrative' reviews have been helpful to a certain extent, they remain problematic for a number of reasons. First of all, these reviews tend to be very selective, often neglecting research done in allied fields, unpublished findings (e.g. theses, dissertations), and studies deemed 'methodologically weak' in the reviewer's eyes. These studies are often eliminated *a priori* or their findings are minimised in interpreting the results of the review. Glass (1978) has argued that whether study quality makes a difference in the findings is a question that should be examined statistically *a posteriori.* Glass et al. (1981) further argue that respect for parsimony demands acceptance of the idea that flawed research can converge on a true conclusion. Only by including all studies, flawed or not, can it be determined whether study quality affected the outcome. This is not to say that if quality does not affect the findings that it is alright to conduct poorly designed studies. This simply allows an empirical determination of whether study quality has affected the results of research done so far. An interrelated issue concerns the scope of narrative reviews, which are often very small (i.e. use a small number of the total studies available).

These 2, issues lead to 2 criticisms of narrative or qualitative reviews: (a) these practices potentially allow a great deal of the author's personal bias to colour the findings; and (b) the results may not accurately reflect the entire area under review. A larger problem with the typical narrative review involves an inability to quantify the magnitude of the effect in question. Reviewers often say that the results are contradictory, with more research being necessary to clarify the findings. If there is an effect, it is impossible for the reviewer to determine the magnitude of the effect, unless the effect is very large. In the latter cases, however, the relationship is probably so well known that a review is rather meaningless.

One solution to the problems outlined above is the technique of meta-analysis (Glass et al. 1981), a set of procedures used to quantify, integrate and analyse findings from a large number of research studies. Meta-analysis allows for systematic investigation of specific moderating variables that typically can only be speculated about in narrative reviews. For example, Dishman (1986) notes that 'Changes in trait anxiety following chronic exercise training are much less reliable [than changes in state anxiety following acute exercise]. An equal number of studies show decreases and no change, while a few show increases' (p. 310). Meta-analysis would allow the trait anxiety-chronic exercise relationship to be examined more carefully. This could include examination of how variables like exercise mode, frequency, intensity, duration and length of training affect the relationship.

Knowing that the typical review is fraught with

problems, and that there is an attractive alternative, this review examines the exercise/mental health relationship by including a larger database and utilising quantifiable, integrative procedures (i.e. meta-analysis). In addition to examining the overall effect, variables that potentially moderate this relationship are examined. Both cross-sectional and longitudinal designs are included in the review. While previous reviews have been largely confined to the outcomes associated with training, "very little attention [has been] paid to the psychological effects, or covariates, of acute physical activity' (Morgan 1979, p. 141). Although the self-reported outcomes of exercise involvement provide evidence of change, they are limited in their ability to explain these changes. As such, this literature provides preliminary evidence to substantiate the phenomenon, but additional work examining moderating variables (e.g. psychophysiological) is necessary to understand underlying mechanisms.

The present review examines these moderating variables and attempts to synthesise them with the outcome variables. Although the basis for exercise-induced psychological alterations in humans is speculative, a number of tenable hypotheses do exist. We have chosen to discuss a number of these proposed mechanisms to explain the findings of the meta-analysis.

1. Meta-Analytic Review of the Literature

1.1 Overview of the Meta-Analytic Technique

Glass (1978) has defined meta-analysis as a methodology for combining the results of independent studies with the purpose of integrating their findings. Procedurally, meta-analysis involves the statistical analysis of the summary findings of many empirical investigations (Glass et al. 1981). The procedure also allows tendencies of the research to be characterised and yields information regarding the magnitude of differences between levels of independent variables. Meta-analysis makes single findings, or the failure to reproduce such findings, less important. Single studies may find relationships that are not real or they may fail to reveal relationships that are real (Friedman & Booth-Kewley 1987). Since meta-analysis involves averaging results from widely heterogeneous studies, interpretation may be problematic. Provided a large pool of studies is available, this problem is easily solved by blocking studies into categories on the basis of relevant moderating variables (Friedman & Booth-Kewley 1987).

1.2 Selection of Studies

Data were collected from studies reported between 1960 and January, 1989 reporting the effects of exercise on either self-reported state (i.e. acute) or trait (i.e. chronic) anxiety and/or psychophysiological correlates of anxiety (e.g. EMG, EEG α, blood pressure, Galvanic skin response). Studies were located through computer searches of ERIC and Psych INFO, and through hand searches of relevant journals (e.g. *Medicine and Science in Sports and Exercise, Journal of Sports Medicine and Physical Fitness, Research Quarterly for Exercise and Sport, Journal of Sport and Exercise Psychology,* etc.). Hand searches were also conducted through unpublished sources (e.g. *Dissertation Abstracts International)* and reference lists obtained from articles. Studies were included if the exercise-mental health relationship was examined in humans who were not interacting with experiment-imposed psychosocial stressors [see Crews & Landers (1987) for a review of the exercise-psychosocial stress literature]. The literature search yielded 124 studies which met inclusion criteria. The studies used in the meta-analyses are listed in the Appendix (pages 168 to 182). Compared to other reviews, this number represents an increase of almost 300% in the number of studies reviewed. 20 studies were excluded because basic information for the calculation of an effect size was not reported in the article and could not be obtained from the author(s).

1.3 Coding Characteristics of the Studies

Studies were coded on several characteristics which reflected *a priori* decisions regarding potential moderating variables for the exercise-mental

health relationship. These characteristics were divided into 5 main areas: subject characteristics, design characteristics, exercise characteristics, anxiety measure used and descriptive characteristics. The variables investigated in each of these areas are listed in table I.

1.3.1 Subject Characteristics

Few studies have examined the effects exercise may have in reducing anxiety across gender and various age groups. As such, separate effect sizes were calculated for males and females (where possible) and for subjects of varying ages.

Effect sizes were also coded for the subjects' mental health. This allowed comparisons to be made between subjects who were considered 'normal' from a mental health perspective and those considered mentally unhealthy (e.g. high and low anxious, psychiatric), as well as those who were involved in cardiac rehabilitation or weight-control programmes.

1.3.2 Design Characteristics

Glass (1978) argued for examination of study design issues in an *a posteriori* fashion. As such, studies were coded as having either a pre-post or multiple assessment design, a no treatment control group, a motivational control group or a control group which received some type of known anxiety-reducing treatment (e.g. relaxation, quiet rest, meditation).

Motivational control groups included such ma-

Table I. Coding categories for study characteristics

Subject characteristics	**Design characteristics**
Age	Comparison group
Health status	no treatment control
normal	motivational control
cardiac rehabilitation	other anxiety-reducing treatments (e.g. relaxation)
high/low anxious	same subjects: pre-post or multiple assessments
psychiatric patients	Subject assignment
obese	random assignment
Gender	matching
	intact groups (convenience sample)
Exercise characteristics	single group repeated
Type	other
aerobic	Exercise paradigm
nonaerobic	acute exercise
Frequency (no. sessions/week)	chronic exercise
Intensity	
$\%HR_{max}$	**Anxiety measures**
$\%\dot{V}O_{2max}$	State anxiety
freely chosen	STAI (A-state)
Duration (min/session)	POMS (tension)
Length of training (in weeks; chronic studies only)	MAACL (anxiety)
Time anxiety measured postexercise (min)	other
	Trait anxiety
Descriptive characteristics	STAI (A-trait)
Publication form	other
journal article	Psychophysiological measures
journal abstract	diastolic blood pressure
unpublished	systolic blood pressure
Author background	heart rate
exercise science	skin measures
psychology	electromyography (EMG)
medicine	central nervous system measures

nipulations as: (a) using intact lecture classes that met for the same duration and frequency as the exercise group; (b) assigning subjects to treatments which emphasised the fun and enjoyment aspects of motor activities while avoiding any aerobic involvement (e.g. stretching classes, hobby classes, social activity programmes); and (c) waiting list control groups.

To further assess how design might influence the anxiety-reducing effects of exercise, the way subjects were assigned to groups was examined, e.g. random assignment, matching, intact groups, or a single group where subjects served as their own controls in a repeated assessments design.

Finally, studies were coded for the type of exercise paradigm used. This allowed examination of how the anxiety-reducing effect of exercise was related to acute exercise paradigms and for chronic designs (i.e. training studies).

1.3.3 Exercise Characteristics

In many respects, the characteristics of the exercise itself may be the most important variables to examine since such analysis may suggest minimal requirements (e.g. duration, frequency, intensity) for achieving anxiety reduction. Type of exercise (aerobic *vs* nonaerobic). as well as specific modes of aerobic exercise (e.g. walking, jogging, running, swimming, cycling), frequency (sessions/wk), duration, intensity (as $\%\dot{V}O_{2max}$, $\%HR_{max}$ or self-selected), and length of training (for chronic studies only) were coded and examined. In addition, Morgan and O'Connor (1988) have speculated on the time course of the postexercise anxiety-reducing effect. To further examine this temporal effect, studies were coded for the time following exercise when anxiety was assessed.

1.3.4 Anxiety Measures

The various types of state, trait and physiological measures of anxiety used were coded. To provide a suitable N for the statistical analysis of the physiological measures, the 10 different measures were grouped into the following 6 categories: systolic blood pressure, diastolic blood pressure, heart rate, skin response (Galvanic, palmar sweating and skin temperature), CNS measures (EEG α, Hoffman reflex), and electromyography (EMG). Since there has been some discussion in the literature on the advisability of using frontalis EMG as an index of anxiety (deVries 1968), the frontalis (n = 7) was compared to all other reported EMG sites (n = 8) [an additional 8 sites were not specified].

The 15 measures used to examine state anxiety were combined into the following 4 categories: State-Trait Anxiety Inventory (A-state) [STAI], Profile of Mood States (tension subscale) [POMS], Multiple Affect Adjective Check List (anxiety subscale), and 'Other'. The 9 measures used to index trait anxiety were divided into State-Trait Anxiety Inventory' (A-trait) and 'Other'. When studies used a trait anxiety measure (e.g. STAI) immediately after exercise this was recoded as a state anxiety measure. The studies used in the meta-analyses and their various characteristics are presented in the Appendix.

7.3.5 *Descriptive Characteristics*

Since published studies tend to be biased toward statistical significance, publication status was examined. This allowed a determination of whether this bias may yield overly optimistic estimates of the anxiety-reducing properties of exercise. It was also of interest to examine whether psychologists, with their extensive background in psychology, or exercise scientists, with their backgrounds in exercise, conduct studies which yield larger/smaller effect sizes.

1.4 Estimation and Interpretation of Effect Sizes

Bangert-Drowns (1986) has suggested that when different dependent measures represent different constructs, separate analyses should be conducted. As such, 3 separate analyses were performed on the present data, one each for self-reported state and trait anxiety, and one for psychophysiological correlates of anxiety. Since lower anxiety is considered desirable, the treatment (i.e. exercise) group mean ($M_{treatment}$) was subtracted from the comparison group mean ($M_{comparison}$). However, when the an-

xiety measure would be expected to be greater because of exercise (e.g. EEC a), the comparison mean was subtracted from the treatment mean. As such, a positive effect size indicates a reduction in anxiety. For each analysis, the primary variable of interest was the effect size (ES) which was calculated using the following standard formula (Thomas & French 1986):

$$\text{Effect size} = \frac{M_{comparison} - M_{treatment}}{SD_{pooled}}$$

Due to the information available in the various studies used in this meta-analysis, there were 2 types of effect sizes: (a) those calculated by comparing an exercise group with a comparison group after the test only; and (b) those calculated by comparing an exercise group before and after the test.' The pooled standard deviation was used since it provides a more precise estimate of the population variance (Hedges 1981). When means and standard deviations were unavailable, effect sizes were estimated from F, t, r or p values using procedures outlined by Hedges and Olkin (1985). Since small sample sizes tend to positively bias the effect size, the calculated effect size was corrected for sample size (Thomas & French 1986).

Many studies measured self-reported and/or psychophysiological anxiety in multiple ways, resulting in multiple effect sizes for these studies. This offers the potential of nonindependent data points (Bangert-Drowns 1986) since effect sizes may be correlated, and as such may pose a threat to the assumption of statistical independence. However, combining all effect sizes from a single study into one would conceal many interesting relationships. For example, effect sizes were calculated for the length of time following exercise when anxiety was measured. Some studies had multiple measurements fallowing exercise, and combining all measures would not allow an examination of the time course of the postexercise anxiolytic effect. The multiple effect size issue has also been criticised because studies contributing many effect sizes may have a greater influence on the findings than studies contributing only a single effect size (Bangert-Drowns 1986). The potential impact of this problem was examined by excluding such studies from the analyses to determine if they exerted undue influence. In no case did this occur. As such, this does not seem to be an important problem for the present findings.

Once effect sizes were calculated for each study, an overall average was calculated. Average effect sizes were also calculated for each level of the moderator variables listed in table I. Four tests were used to determine the significance of effect sizes. First, an unweighted Stouffer meta-analytic Z-test (Rosenthal 1978) was used to determine if the effect was significantly different from zero, Second, to determine if various groupings of effect sizes were different, the homogeneity (H) statistic (Hedges & Olkin 1985) was calculated. Third, if the H-statistic was significant (i.e. indicating that effect sizes were not all similar), further analysis of the various moderator variables was carried out using 1-way analyses of variance (ANOVAs) to determine where there were differences between levels of moderator variables. Finally, when a significant omnibus F resulted, Tukey *post hoc* tests were calculated to further delineate the differences.

If significant differences were found among effect sizes, interactions with other moderator variables were examined. When such interactions did occur, the data were checked for very large positive or negative effect sizes to insure that a small number of effect sizes were not unduly influencing the results. These effect sizes were also double-checked against the original source to determine possible reasons why they were so unusual.

1.5 Results and Discussion

From the 104 studies included in the meta-analyses, 408 effect sizes (ES) were calculated based on a population of 3048 subjects [age (mean ± SD)

1 All statistical tests were performed using the two types of calculated effect sizes as an interaction variable. In no case were there any significant interactions with any of the moderator variables. Thus, while the two types of effect sizes are derived from different sources (i.e. pre-post *vs* post only comparisons), they yield similar quantitative information.

= 34.16 ± 13.95 years; range = 10.6 to 73.4]. Separate effect sizes were calculated for males and females (where possible) to examine potential gender differences. Unfortunately, most studies involving a combination of males and females failed to specify the number of males and females in the various groups. As such, there were not enough effect sizes available to make meaningful comparisons. Overall mean effect size, n, and SEs are presented in tables II, III and IV for state anxiety, trait anxiety and psychophysiological correlates of anxiety, respectively.

1.5.1 Self-Reported State Anxiety

The overall mean effect size for self-reported state anxiety was 0.24 (SE = 0.04), indicating that exercise was associated with a small reduction in state anxiety of slightly less than 0.25 standard deviation units (sec table II). The H-statistic was significant, x^2 (206) = 469.70, $p < 0.001$, warranting further *post hoc* examination among moderator variables. One-way ANOVAs indicated that there were no differences among types of state anxiety measures, acute and chronic exercise (effect size = 0.23 and 0.25, respectively), different exercise intensities, or for when state anxiety was assessed immediately or up to 30 minutes after exercise. Characteristics that were coded which did not yield any significant findings included subject assignment, age, and health status.

A significant difference emerged for publication form, F (2,204) = 3.27, p = 0.04. *Post hoc* analysis revealed that journal abstracts yielded the largest effect sizes. These were significantly larger than effect sizes from unpublished sources (i.e. theses, dissertations, unpublished papers). Since abstracts that were eventually published were not included in this category, it may be that unpublished studies cither had weaknesses or the weaker findings discouraged authors from attempting to publish their research (i.e. file drawer problem).

The effect of author background was significant, F (3,203) - 2.78, p = 0.04. The more conservative *post hoc* analysis, however, failed to confirm the significant effect.

There was a significant effect for comparison group, F (3,203) = 9.84, $p < 0.0001$. Without any control group(s), the before *vs* after within-subjects design can only yield an absolute charge in state anxiety which may be due to exercise or quite possibly other uncontrolled factors. The effect size for this type of design was 0.47, representing a change of almost 0.5 standard deviation units below pre-exercise levels. Contrasting this to the no treatment control group design, the effect size was diminished to 0.22. Thus, when factors of internal validity are controlled (e.g. testing effect, maturation, instrumentation; Campbell & Stanley 1963), the effect of exercise on state anxiety was diminished by approximately one-half. When a motivational control group (e.g. intact lecture classes, waiting list control group) was used to control for a Hawthorne effect, the effect size was 0.26. Thus, even in comparison with control groups designed to control subjects' movitation, exercise was still associated with lower anxiety.

A question often addressed in the literature is whether exercise reduces anxiety as well as, or better than, other therapeutic modalities (e.g. meditation, relaxation, quiet rest). Examination of studies using control groups receiving other anxiety-reducing treatments revealed that exercise was no better at reducing anxiety than these treatments, effect size = -0.04. This effect size was not significantly different from zero. Besides the physiological benefits that exercise offers, the finding that exercise is as effective in reducing anxiety as more traditional therapeutic modalities may explain the high incidence of physicians who prescribe exercise as a treatment for anxiety (Ryan 1983).

Various types of aerobic (e.g. walking, jogging, running, swimming, cycling) and nonaerobic (e.g. weight-training) exercise were also examined. Differences among the various types of aerobic exercise were assessed and since there were no significant differences among them ($p > 0.05$), they were collapsed into the larger category of aerobic exercise. The results revealed a significant difference between aerobic and nonaerobic exercise, F (1,184) = 4.18, $p < 0.04$. Aerobic exercise yielded an effect size of 0.26, whereas the effect size for nonaerobic exercise was -0.05, not significantly different from

Table II. Moderator variables for self-reported state anxiety

Moderator variable	n	Effect size	SE	p[a]
Overall	207	0.24	0.04	< 0.0001
Anxiety measure				
STAI (A-state)	94	0.23	0.05	< 0.0001
POMS (tension)	66	0.25	0.07	< 0.0001
MAACL (anxiety)	13	0.47	0.17	0.0006
Other	34	0.17	0.09	0.0480
Exercise paradigm				
Acute	119	0.23	0.05	< 0.0001
Chronic	83	0.25	0.05	< 0.0001
Comparison group[b]				
No-treatment control	67	0.22[c,e]	0.07	< 0.0001
Motivation control	25	0.26	0.10	0.011
Other anxiety-reducing treatments	49	-0.04[c,d]	0.05	0.4472
Pre-post within subjects design	66	0.47[d,e]	0.06	< 0.0001
Type of exercise[b]				
Aerobic	173	0.26	0.04	< 0.0001
Nonaerobic	13	-0.05	0.19	0.7948
Intensity of exercise stimulus (% HR_{max} or % $\dot{V}O_{2max}$)				
40-59	4	0.19	0.31	0.5418
60-69	39	0.27	0.10	0.0070
70-79	51	0.18	0.08	0.0244
> 80	20	0.33	0.12	0.0006
Duration of exercise session (min)[b]				
0-20	61	0.04=	0.07	0.5686
21-30	57	0.41[c]	0.05	< 0.0001
31-40	24	0.25	0.10	0.0132
> 40	40	0.19	0.09	0.0434
Time anxiety measured postexercise (min)[b]				
0-5	143	0.21	0.04	< 0.0001
5	14	0.36	0.15	0.0136
10	17	0.27	0.13	0.0376
20	12	0.49	0.09	< 0.0001
> 20	9	0.27	0.04	< 0.0001
Publication format[b]				
Journal article	121	0.23	0.05	< 0.0001
Journal abstract	24	0.49[c]	0.08	<0.0001
Unpublished	62	0.17[c]	0.07	0.0150
Author background[b]				
Psychology	42	0.40	0.09	< 0.0001
Exercise science	123	0.18	0.05	< 0.0001
Medicine	13	0.46	0.15	0.0022
Other	29	0.18	0.08	0.0244
Subject assignment				
Random	73	0.16	0.06	0.0008
Matching	5	0.63	0.16	< 0.0001
Intact groups	60	0.24	0.08	0.0026
Single group repeated	65	0.32	0.06	< 0.0001
Other	4	0.16	0.13	0.2186
Age (years)				
< 18	21	0.20	0.12	0.0950
18-30	97	0.19	0.06	< 0.0001
31-45	72	0.36	0.05	< 0.0001
> 45	11	0.08	0.19	0.6628
Health status				
Normal	177	0.26	0.04	< 0.0001
Cardiac rehabilitation	3	0.48	0.33	0.1470
Psychiatric	4	0.21	0.10	0.0358
High anxious	17	0.17	0.13	0.1902
Obese	2	0.26	0.04	< 0.0001

a p-value indicates significance of unweighted Stouffer meta-analytic Z-test, i.e. whether effect size is significantly different from zero.

b Significant F-test for moderator variable.

c, d, e Means with the same superscript are significantly different from each other as determined by Tukey post *hoc* tests.

zero. Although there were many more studies employing aerobic exercise, the results are strikingly different with the anxiolytic effect of exercise only occurring for aerobic activities. It may be that duration of continuous exercise is an important factor in this relationship, a point which is discussed next

Finally, there was a significant difference between levels of duration of exercise, F (3,178) =

5.39, p = 0.0014. This can be seen in figure 1 where each point represents the length of the exercise session. Specifically, *post hoc* tests revealed that exercise lasting 0 to 20 minutes (ES = 0.04) resulted in significantly lower effect sizes than exercise lasting 21 to 30 minutes (effect size = 0.41). There were no differences between the other time categories.

Closer inspection is necessary before concluding that exercise needs to occur for at least 20 minutes to produce anxiety-reducing effects. Of the effect sizes in the 0- to 20-minute duration category, 48% were derived by comparing exercise with known anxiety-reducing treatments (e.g. relaxation, meditation). Only 18% of the effect sizes in the other duration categories were derived from such comparisons. When effect sizes derived from such comparisons were eliminated, the effect size for 0 to 20 minutes' duration was substantially increased (effect size = 0.22). This effect size was significantly different from zero and was no longer different from the other exercise durations. At least within the categories established in this study, the anxiety-reducing effect that accrues from exercise seems to be present regardless of the duration of the exercise.

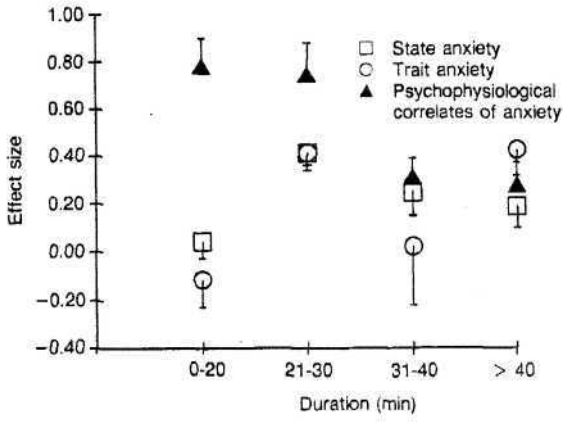

Fig. 1. Effect size (mean ± SE) as a function of exercise duration. A positive ES indicates a reduction in anxiety.

7.5.2 *Self-Reported Trait Anxiety*

The overall mean effect size for self-reported trait anxiety was 0.34 (SE = 0.06), indicating that chronic exercise was. associated with a reduction in trait anxiety of more than 0.30 standard deviation units below comparison groups. The H-statistic was significant, x^2 (61) = 103.76, p < 0.001, indicating heterogeneity of effect sizes.

Post hoc comparisons among the different moderator variables are contained in table III. There were no significant differences found for type of trait anxiety measure, publication form, exercise intensity, age, health status, or type of comparison group. Since all studies examining changes in trait anxiety used a longitudinal design, comparisons were not made between acute and chronic exercise. There were also no effects for the time anxiety was measured postexercise since trait anxiety was not assessed immediately following exercise sessions. While effect size means for aerobic and nonaerobic exercise were quite discrepant (0.36 and -0.16, respectively), these differences were not significant.

Author background was significant, F (3,58) = 4.43, p = 0,007, with studies from psychology departments yielding larger effect sizes than studies from exercise science, medicine and other areas. There were no apparent differences between studies conducted in psychology departments and those conducted in other fields in terms of the subject, design and exercise characteristics coded for in this meta-analysis.

For subject assignment, F (4,57) = 5.03, p = 0.0015, *post hoc* comparisons revealed that studies using random assignment yielded significantly larger effect sizes than studies using intact groups (0.54 *vs* 0.05, respectively). Use of intact groups may include confounding variables which lessen the anxiety-reducing effect of exercise.

Length of training was significant, F (4,55) = 3.45, p < 0.014. As shown in table III and figure 2, the length of the exercise training programme had much stronger effects when the programme exceeded 9 weeks. The strongest effects were seen for programmes greater than 16 weeks (effect size = 0.90), which were significantly different than programmes lasting 9 weeks or less. One of the outliers

Table III. Moderator variables for self-reported trait anxiety

Moderator variable	n	Effect size	SE	p[a]
Overall	62	0.34	0.06	< 0.0001
Anxiety measure				
STAI (A-trait)	48	0.28	0.06	< 0.0001
Other	14	0.55	0.13	< 0.0001
Comparison group				
No-treatment control	22	0.29	0.12	0.0156
Motivation control	10	0.42	0.18	00182
Other anxiety-reducing treatments	15	0.31	0.12	0.0086
Pre-post within subjects design	15	0.40	0.06	< 0.0001
Type of exercise				
Aerobic	51	0.36	0.06	< 0.0001
Nonaerobic	2	-0.16	0.20	0.4238
Intensity of exercise stimulus (% HR_{max} or % $\dot{V}O_{2max}$)				
70-79	15	0.29	0.14	0.0376
> 80	4	0.45	0.09	< 0.0001
Duration of exercise session (min)[b]				
0-20	9	-0.12[c,d]	0.11	0.2758
21-30	13	0.41=	0.07	< 0.0001
31-40	4	0.02	0.24	0.9362
> 40	24	0.42[d]	0.10	< 0.0001
Length of training programme (weeks)[b]				
4-6	8	0.14[C]	0.09	0.1236
7-9	19	0.17[d]	0.09	0.0466
10-12	19	0.50	0.12	< 0.0001
13-15	10	0.36	0.13	0.0048
> 15	4	0.90[c,d]	0.29	0.0016
Publication format				
Journal article	34	0.45	0.07	< 0.0001
Journal abstract	2	0.26	0.31	0.1142
Unpublished	26	0.21	0.10	0.0198
Author background[b]				
Psychology	15	0.57[c]	0.15	< 0.0001
Exercise science	24	0.36	0.07	< 0.0001
Medicine	8	0.44	0.09	< 0.0001
Other	15	0.02[c]	0.10	0.9840
Subject assignment[b]				
Random	25	0.54°	0.10	< 0.0001
Matching	3	0.43	0.16	0.0072
Intact groups	23	0.05°	0.08	0.5286
Single group repeated	10	0.43	0.08	< 0.0001
Other	1	0.82		
Age (years)				
< 18	3	0.47	0.66	0.4778
18-30	14	0.09	0.17	0.5962
31-45	34	0.45	0.05	< 0.0001
> 45	6	0.41	0.13	0.0016
Health status				
Normal	46	0.30	0.06	< 0.0001
Cardiac rehabilitation	4	0.44	0.17	0.0096
Psychiatric	1	0.55		
High anxious	11	0.47	0.06	< 0.0001

a p-value indicates significance of unweighted Stouffer meta-analytic Z-test, i.e. whether affect size is significantly different from zero.

b Significant F-test for moderator variable.

c, d Means with the same superscript are significantly different from each other as determined by Tukey *post hoc* tests.

(effect size = 1.70; Hilyer et al. 1982; was in the > 16 weeks' category. Even with this very large effect size (1.70) eliminated, the > 16-week category continued to have a significantly larger effect size (0.63) than the 4- to 6-week and 7- to 9-week categories. These findings suggest that for reductions in trait anxiety to occur, exercise needs to be done for more than 10 weeks. It may be that in order to change a relatively stable personality disposition like trait anxiety, exercise must be done over a longer time.

Finally, duration of exercise was significant, $F(3,46) = 4.96$, $p < 0.05$. Exercise of less than 20 minutes showed a negative effect (-0.12), but was not significantly different from other durations. The studies using 0 to 20 minutes' duration were fur-

ther examined to make sure that other moderating variables were not unduly influencing the effect of duration. As with the state anxiety results, another moderating variable was found to be influencing the effect size for this group. Eight of the 9 effect sizes were in the 7- to 9-week category (effect size = -0.20) and 1 was in the 13- to 15-week category (effect size = 0.52). Given the existence of confounding variables, it may be premature to conclude that exercise for longer than 20 minutes is necessary in order to achieve anxiety-reducing effects (e.g. Dishman 1986). To determine the minimum duration needed to reduce anxiety, experimental studies are needed so that duration of exercise can be manipulated within the 0- to 20-minute time frame (e.g. 5, 10, 15 or 20 minutes).

1.5.3 Psychophysiological Correlates of Anxiety

The overall mean effect size for psychophysiological correlates of anxiety was 0.56 (SE = 0.06), indicating that exercise was associated with a change in anxiety of more than 0.50 standard deviation units below comparison groups. Further examination of moderator variables was undertaken because of the significant H-statistic, x^2 (137) = 379.3, $p < 0.001$.

For the studies using psychophysiological measures of anxiety, no nonaerobic studies were located negating comparison of this moderator variable. No significant differences were found for author background, publication form, health status, intensity, time anxiety was measured postexercise or length of training.

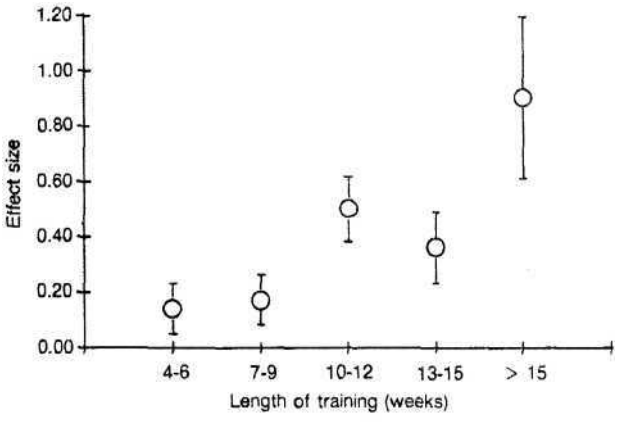

Fig. 2. Effect size (mean ± SE) of trait anxiety as a function of length of training. A positive ES indicates a reduction in anxiety.

To examine whether the various physiological measures used in these studies yielded different effect sizes, a 1-way ANOVA was computed for the various measures coded for in this study (see table IV). There was a significant effect for type of physiological measure used to index anxiety, F (5,132) = 3.97, p = 0.002. *Post hoc* comparisons revealed that the effect sizes for blood pressure and heart rate measures were significantly lower than the effect sizes for CNS measures (EEG α, Hoffman reflex), EMG and skin measures. It is interesting to note that the majority of studies (61%) used heart rate and blood pressure measures, which produced the weakest effect sizes. Although EMG measures showed large effect sizes, it should be pointed out that the; effect sizes derived from frontalis EMG (n = 7, effect size = 0.16) were considerably smaller than those derived from other sites (n = 8, effect size = 0.60).

Since heart rate and blood pressure measures produced the smallest effect sizes, the other moderating variables (table IV) were examined with only the heart rate and blood pressure measures included to determine if there were differential effects depending on the type of measure used. When these ANOVAs were recalculated, all of the previously significant moderating variables were still significant.

Significant differences emerged for subject assignment, F(4,133) = 4.99, p = 0.0009. Studies using random assignment had significantly lower ESs (-0.13) than those using intact groups (0.70) or single group pre-post designs (0.59), the latter not being different from each other. Further inspection of the characteristics of studies using random assignment revealed that there were 2 studies which were outliers (Bahrke 1977; Emery & Gatz 1985), with average effects sizes of -0.33 and -0.52, respectively. The other studies using random assignment had an average effect size of 0.21.

Table IV. Moderator variables for psychophyslological correlates of anxiety

Moderator variable	n	Effect size	SE	p[a]
Overall	138	0.56	0.06	< 0.0001
Anxiety measure[b]				
Systolic blood pressure	28	0.38[c,f,i]	0.11	0.0006
Diastolic blood pressure	28	0.33[d,g,j]	0.12	0.0060
Heart rate	20	0.31[e,h,k]	0.11	0.0048
Skin measures	5	1.14[c,d,e]	0.19	< 0.0001
EMG	23	0.80[f,g,h]	0.19	< 0.0001
CNS measures	34	0.77[i,j,k]	0.11	< 0.0001
Exercise paradigm[b]				
Acute	85	0.65[c]	0.08	< 0.0001
Chronic	53	0.40[c]	0.07	< 0.0001
Comparison group[b]				
No-treatment control	16	0.52	0.14	< 0.0001
Motivation control	12	0.10[c]	0.20	0.6170
Other anxiety-reducing treatments	20	0.15[d]	0.14	0.2938
Pre-post within subjects design	90	0.71[c,d]	0.07	< 0.0001
Intensity of exercise stimulus (% HR_{max} or % $\dot{V}O_{2max}$)[b]				
40-59	13	1.06[c]	0.30	0.0004
60-69	31	0.74	0.09	< 0.0001
70-79	24	0.41[c]	0.13	0.0020
> 80	20	0.68	0.15	< 0.0001
Duration of exercise session (min)[b]				
0-20	50	0.78[c]	0.12	< 0.0001
21-30	22	0.75	0.13	< 0.0001
31-40	22	0.31[c]	0.08	< 0.0001
> 40	32	0.28	0.09	0.0022
Length of training programme (weeks)				
4-6	5	0.65	0.21	0.0016
7-9	14	0.39	0.12	0.0006
10-12	13	0.29	0.17	0.0872
13-15	12	0.61	0.12	< 0.0001
> 15	9	0.15	0.14	0.2984
Time anxiety measured postexercise (min)				
0-5	95	0.50	0.07	< 0.0001
5	13	0.60	0.17	0.0006
10	5	1.02	0.13	< 0.0001
20	4	0.29	0.08	< 0.0001
>30	14	0.48	0.11	< 0.0001
Publication format[b]				
Journal article	90	0.55	0.07	< 0.0001
Journal abstract	20	0.85[c]	0.15	< 0.0001
Unpublished	28	0.36[c]	0.12	0.0026
Author background				
Psychology	30	0.53	0.08	< 0.0001
Exercise science	72	0.61	0.09	< 0.0001
Medicine	24	0.65	0.16	< 0.0001
Other	12	0.11	0.21	0.6030
Subject assignment[b]				
Random	15	-0.13[c,d]	0.15	0.5028
Matching	3	0.87	0.10	< 0.0001
Intact groups	39	0.70[c]	0.09	< 0.0001
Single group repeated	76	0.59[d]	0.08	< 0.0001
Other	5	0.73	0:09	< 0.0001
Age (years)[b]				
<18	8	0.38	0.12	0.0016
18-30	45	0.69[c]	0.08	< 0.0001
31-45	42	0.55	0.12	< 0.0001
>45	26	0.14[c]	0.12	0.2420
Health status				
Normal	115	0.54	0.06	< 0.0001
Cardiac rehabilitation	5	0.51	0.09	< 0.0001
Psychiatric	3	0.87	0.10	< 0.0001
High anxious	15	0.65	0.22	0.0016

a p-value indicates significance of unweighted Stouffer meta-analytic Z-test, i.e. whether effect size Is significantly different from zero.

b Significant F-test for moderator variable.

c-k Means with the same superscript are significantly different from each other as determined fay Tukey *post hoc* tests.

A closer examination of these 2 studies revealed possible reasons why the effect sizes may have been so different. Bahrke (1977) reported differences for systolic and diastolic blood pressure pre- to post-exercise. However, the quiet rest control group had pretest systolic and diastolic blood pressure values

that were 10.14 and 7.1 mm Hg lower than the exercise and meditation groups. For systolic blood pressure, both the exercise and meditation groups showed a greater pre-post reduction (2.54mm Hg) than the control group (1.04mm Hg). Because of the way effect sizes were calculated (i.e. control - treatment), the initially lower values of the control group misrepresent the actual pre-post changes occurring in the treatment groups.

In the Emery and Gatz (1985) study, men and women (ages 61 to 86 years) were randomly assigned to either a social activity group, a waiting list control group or a 12-week training programme. The training programme consisted of rapid walking (5 to 7 minutes), stretching exercises, leg kicking, heel and toe lifts, repetitive sitting and standing, side and knee bends and a dance routine. There was no measure of aerobic capacity used in this study and the heart rate and blood pressure measures did not show a training effect. It appears that the very low intensity exercise employed in this study was of no aerobic benefit and therefore should not be expected to produce significant pre-post physiological or psychological changes.

Considering the problems in these outlying studies, the moderating variables for all psychophysiological measures were reanalysed with these 2 studies eliminated. Without these 2 studies in the analysis, only duration of exercise remained significant, $F(3,l\,12) = 5.27$, $p < 0.002$. *Post hoc* tests revealed that exercise lasting from 0 to 20 minutes had significantly larger effect sizes (0.78) than exercise lasting 31 to 40 minutes (0.31) and > 40 min (0.28). With less than 20 minutes of exercise at the low to moderate intensities used in these studies, only a modest increase in sympathetic nervous system activity above pre-exercise levels would be expected. Compared to longer durations of exercise, physiological recovery postexercise occurs more quickly and may be more likely to show a much larger overcorrection (i.e. activation levels below the pre-exercise baseline). It must be emphasised, however, that effect sizes were significantly greater than zero regardless of the duration of exercise. Therefore, even though longer durations do not produce effects of the same magnitude as shorter bouts of exercise, anxiety is significantly changed for all exercise durations,

2. General Discussion

The results of the 3 meta-analyses are summarised and integrated in the next section. Following this, these findings are discussed in light of the various mechanisms proposed for the anxiolytic effect of exercise.

2.1 Summary of Findings

Across all 3 meta-analyses, the overall effect sizes were significantly greater than zero. This indicates that no matter how anxiety is assessed (i.e. state, trait or a psychophysiological measure), exercise is associated with a reduction in anxiety. Although there are consistent relationships, the results of the meta-analyses do not suggest a causal effect for exercise (or something associated with exercise) in alleviating anxiety.

Examinatian of moderating variables was done to establish where relationships might be strongest. Such knowledge may have implications for the design of future research and, more importantly, may suggest which of the proposed explanations or mechanisms may be the most viable and worthy of further investigation.

When the confounding influences due to outliers were controlled, the anxiety reduction associated with exercise was generally independent of subject (i.e. age, health status) and descriptive (i.e. publication form, author training) characteristics. There were only 2 exceptions to this: (a) for state anxiety measures, journal abstracts showed a larger effect size than unpublished studies; and (b) for trait anxiety, the studies of authors from psychology departments contained larger effect sizes than authors from departments other than exercise science/ physical education or medicine. Although present, these effects were not very meaningful since they were limited and provided no information on design and exercise characteristics that may have suggested methodological reasons why larger effects were found.

Several design characteristics were statistically different. Most of these, however, did not generalise across the state, trait and psychophysiological measures employed. Because studies using trait and physiological measures either did not examine nonaerobic exercise or had too few effect sizes for meaningful comparisons to be made, the aerobic/nonaerobic variable was only significant in the state anxiety meta-analysis. In this case, aerobic exercise produced larger effect sizes than nonaerobic exercise. The other design characteristics not generalising across conditions were type of comparison group for state anxiety and subject assignment for trait anxiety. The former showed that the effect size (0.47) for studies using pre-post within-subjects designs was reduced when no treatment and motivational control groups (0.22, 0.26, respectively) were added.

Furthermore, if the control group used consisted of an anxiety-reducing intervention other than exercise (i.e. meditation, relaxation, quiet rest), there was no difference between exercise and other anxiety-reducing treatments. The latter finding for subject assignment showed that studies employing random assignment had larger effect sizes than studies using intact groups.

The only difference among anxiety measures was found in comparisons of physiological correlates. In this case, the effect sizes for the skin, muscle, and central nervous system measures were higher than effect sizes derived from measures of heart rate and systolic and diastolic blood pressure.

For the exercise characteristics, length of training was a significant moderating variable, but only for the trait anxiety measure. Subjects training for 16 weeks or longer had larger effect sizes than subjects training 9 weeks or less. The only moderating variable to show significant differences across all types of anxiety measures (state, trait, physiological) was duration of training. For state and trait anxiety, the results indicated that at least 21 minutes of exercise was necessary in order to achieve significant effect sizes. However, caution is needed in interpreting these findings since in both cases duration was confounded with other design or exercise variables. The duration findings for the physiological correlates of anxiety were different from the state and trait anxiety findings. In this case, the effect size was significantly larger for 0 to 20 minutes than it was for over 31 minutes.

2.2 Suggested Mechanisms of Change Underlying Response to Exercise

The outcomes associated with exercise involvement have been attributed to a number of mechanisms ranging from psychological to neurophysiological. For example, some (Benight 1989; Ismail & Trachtman 1973) have advanced the notion that positive consequences follow from exercise because of mastery feelings or the sense of accomplishment that comes from successful completion of an exerci.se workload. Others have considered exercise to be a distraction (or 'time out') from the daily work routine (Bahrke & Morgan 1978). Still others have discussed neurophysiological models of central affective change (deVries 1968).

2.2.1 Thermogenic Model

It has been proposed that elevations in body temperature will produce therapeutic benefits. For example, research has shown that whole-body warming (e.g. sauna bathing, warm shower or fever therapy) reduces muscle tension (i.e. somatic anxiety) [deVries et al. 1968] and self-reported state anxiety levels (Raglin & Morgan 1985), reduces γ neuron motor activity (von Euler & Soderberg 1957), and increases EEG α wave frequency (Bennett et al. 1941).

The hypothesis with regard to anxiety reduction is that core temperature, increased in proportion to the intensity of exercise, is responsible for the observed anxiolytic effect. Studies examining only exercise and temperature have shown that with moderate exercise (20-minute duration), elevations in body temperature have been fairly small (0.70°C) and short-lived. Temperature returns to normal levels within approximately 90 minutes following exercise (Hori et al., 1978). These small elevations in temperature have also been seen with prolonged exercise (9-hour duration), but in this case the tem-

perature remained elevated above resting levels for 11 hours (Haight & Keatings 1973).

A derivative of the thermogenic hypothesis is the hyperthermic model by von Euler and Soderberg (1957). Based on their research with animals, von Euler and Soderberg hypothesised that hyperthermia would promote reductions in γ ncuronal activation. They found that while a moderate increase in hypothalamic temperature (up to 41°C) reduced muscle spindle activity (i.e. reduced reflexive action), further heating (41 to 43°C) generally increased muscle spindle activity. A similar pattern was observed for EEG in that moderate increases in temperature resulted in increased α activity, but further increases showed an arousal reaction (i.e. increased EEG β activity °C) have also been seen in humans. Since further heating would be considered a stressor, there should be an increase in the stress response. Thus, it appears that an elevation in temperature may cause both anxiety reductions and increases, depending on the degree of temperature increase.

Even if we simply assume that temperature changes are associated with exercise, only the work of Bulbulian and Darabos (1986), deVries et al. (1982) and deVries et al. (1981) have measured neuronal activation. These investigators employed an alternate measure of spinal motor neuron excitability, the H/M ratio derived from evocation of the Hoffman reflex, as a more general index of anterior motor horn activation. For the deVries et al. (1981, 1982) studies, a mild exercise stimulus was employed which consisted of 20 minutes of cycle ergometry at an intensity of 40% HR_{max}. Bulbulian and Darabos (1986) used the same duration, but varied the intensity of exercise (40 and 70% $\dot{V}O_{2max}$) while subjects did a walk/run. In these studies, equivalent nonexercise control periods consisted of three 20-minute sessions during which the subjects simply sat and read. Across all exercise sessions the subjects consistently reduced the magnitude of the H/M ratio from baseline. Whereas von Euler and Soderberg (1957) proposed that exercise-induced hyperthermia would reduce γ-neuronal activation, the work of deVries and his colleagues seems to suggest that peripheral neurological change is restricted to α motoneuron activity (Hatfield & Landers 1987).

The meta-analysis showed that duration of exercise was an important factor in the amount of anxiety reduction experienced by the subjects. With anxiety measured physiologically, exercise of shorter duration (<30 minutes) showed larger effect sizes than exercise of longer duration (>31 minutes). With state and trait anxiety, the effect sizes increased from 0 to 20 minutes to 21 minutes of exercise, but levelled off from 21 to 40 minutes and longer. While these results do not support the predicted positive linear relationship (i.e. between duration of exercise/temperature increase and reduction in anxiety) of the thermogenic hypothesis, they are more in line with the hyperthermic model's curvilinear relationship (von Euler & Soderberg 1957).

It is possible that exercise is different from passive body heating in producing anxiolytic effects since body heat is more easily dissipated with exercise. While the meta-analytic results do not support the thermogenic hypothesis, this is obviously only indirect evidence since temperature was not directly measured. However, a report by Reeves et al. (1985) did measure body temperature in conjunction with exercise and their results also refute the thermogenic hypothesis. Using 20 minutes of callisthenics (jumping jacks, push-ups, running in place, stationary stair stepping) along with vapour-barrier and insulative clothing, increased temperature (+1.2°C) resulted in increased anxiety. A control group (callisthenics without insulative clothing) showed no temperature change (± 0.1 °C) and no change in anxiety. Additionally, Reeves et al. (1985) found the relationship between temperature and anxiety to be strong (Φ coefficient = 0.80, $p < 0.01$). Although this is the only report found which has examined exercise, anxiety and temperature concurrently, it is possible that increased anxiety was due to the excessive clothing and not the increased temperature. Whether or not temperature affects anxiety needs to be examined further in future research.

2.2.2 Cardiac Influence Model: Visceral-Afferent Feedback

An additional conceptual basis for changes in central activation is provided by the visceral-afferent feedback model. In fact, the effects of changes in the cardiovascular system and its somatic afferents upon the CNS (i.e. arising from muscular and autonomic activity during exercise) seem to complement those of central hyperthermia.

Neurophysiological evidence (Bonvallet & Bloch 1961; Iwamoto & Kaufman 1987) has shown that afferent impulses transmitted from the working muscles are eventually received by a number of brainstem collateral neurons. This results in increased stimulation of the ascending reticular activating system. Indirect support for this stimulation effect has been shown as an increase in state anxiety following the initiation of exercise (Morgan et al. 1980). Bonvallet and Bloch (1961) note, however, that cortical excitation may eventually reach a point at which an inhibitory mechanism is stimulated in the bulbar region of the brainstem. This stimulation may have an arresting influence upon the reticular formation so that somatic afferent stimulation to the cortex is reduced. As such, cortical excitation may be decreased, with this dampening effect lasting substantially beyond the initiating stimulus to promote a prolonged poststimulus effect (Bonvallet & Bloch 1961).

Support for the visceral afferent-feedback model is evident in EEG studies of cortical activation following various forms of aerobic exercise. Based on the traditional belief that the presence of EEG α reflects decreased cortical activation, investigators have interpreted the increase in EEG α activity following exercise as indicative of a relaxation response or anxiety reduction (Boutcher 1986; Boutcher & Landers 1988; Daugherty et al. 1987; Farmer et al. 1978; Kamp & Troost 1978; Pineda & Adkisson 1961; Wiese et al. 1983). According to Wiese et al. (1983), 'the increased alpha power after exercise could contribute to an altered state of consciousness and could help to explain the psychological benefits, including reductions in anxiety and depression, that have been reported with regular exercise' (p. 117).

These EEG studies are limited, however, in that some have failed to statistically analyse EEG pre/post differences and, with the exception of the Boutcher (1986) study, they have failed to obtain concurrent assessment of self-reported anxiety. In addition, none of the studies report data on EEG frequencies; beyond the α range (i.e. 8 to 12Hz). This may be problematic since research by Ray and Cole (1985) suggests that affect or emotion is more appropriately indexed by β activity (i.e. 13 to 30Hz). In light of Ray and Cole's (1985) findings, examination of the full frequency spectrum to index affective states during/following exercise seems to be warranted in future EEG research.

Some investigators (Davidson et al. 1981; Walker & Sandman 1979, 1982) maintain that the effect of autonomic activation upon the cerebral hemispheres is lateralised. Using reaction-time tasks, these investigators found that increased autonomic activity (i.e. heart rate and blood pressure) was associated with right-hemisphere electrocortical deactivation. Tucker (1981) has also noted that behavioural, psychophysiological and neuroanatomical evidence indicates that the right hemisphere may be more closely involved with somatic processes (e.g. those seen during exercise), In the exercise science literature, only 3 studies (Boutcher & Landers 1988; Pineda & Adkisson 1961; Wiese et al. 1983) have examined bilateral EEG differences. Of these, Pineda and Adkisson did not statistically examine hemispheric differences and Boutcher and Landers collapsed across hemispheres because their statistical analysis showed no significant hemispheric difference before or following 20 minutes of exercise.

The only study to show statistically significant hemisphenc differences following exercise (25 minutes of cycling at 40% $\dot{V}O_{2max}$ and 15 minutes of cycling at 60% $\dot{V}O_{2max}$) was done by Wiese et al. (1983). Unfortunately, the significant shift they noted in the right/left ratio toward equality was not accompanied by data for the mean change for each hemisphere. Thus, it was not possible to discern from the report how this shift was obtained (e.g. increase in right α, decrease in left α or both). However, based on the results, they speculated that the

'right-to-left [hemispheric] changes suggest a decrease in hemispherisation in the cortex during exercise which could facilitate an atmosphere of psychological change' (p. 117). This conclusion is further supported by research (Sackeim & Weber 1982; Tucker et al. 1981) which suggests that deactivation of the right hemisphere, either through drugs, structural damage (i.e. lesions), or possibly exercise, results in the expression of positive emotional responses (e.g. decreased anxiety).

Whether the autonomic changes accompanying exercise produce a deactivation in the right hemisphere remains to be determined. Both the cerebral lateralisation hypothesis (Hatfield & Landers 1987) and the visceral afferent-feedback hypothesis are tenable; however, due to the paucity of research in this area, their confirmation (or refutation) awaits future research.

2.2.3 Opponent-Process Model

Another conceptual explanation for exercise-induced affect is Solomon's opponent-process theory of acquired motivation (Solomon 1980; Solomon & Corbit 1973, 1974). This is a model which utilises a physiological mechanism (e.g. endorphins) to explain psychological change. Solomon's theoretical position basically assumes that the brain is organised to oppose pleasurable or aversive emotional processes. This is accomplished by countering an arousing stimulus with an opposite or 'opponent' reaction. Stimulus onset causes an increase in sympathetic nervous system (SNS) activity, what Solomon has called the 'a process'. The opponent, or 'b process', is aroused by the a process and acts to return the organism to homeostasis. While not specified by Solomon, this process could theoretically be produced via activity of the parasympathetic nervous system (PNS).

An important component of the model is that with long term exposure to a stimulus (e.g. exercise), the a process and its associated affective state (e.g. the anxiety associated with exercise) remains constant, while the opponent process and its affective state (e.g. relaxation) becomes increasingly stronger. In essence, this developmental occurrence represents a shift in the amplitude and duration of the b process.

Boutcher and Landers (1988) tested this model in relation to the mental health effects of exercise. Self-reported state anxiety of trained runners was compared with anxiety responses of untrained runners after 20 minutes of treadmill running at an intensity of 80 to 85% HR_{max}. Trained runners (i.e. subjects who have adapted to the exercise stimulus) experienced a significant decline in anxiety after exercise, at both 5 and 13 minutes after exercise. No such change was observed for the untrained runners. These results are in agreement with those predicted by opponent-process theory; that is, trained runners had a much larger opponent affective reaction following cessation of the stimulus, in this case exercise.

The opponent-process model is also supported by the results of the meta-analysis. Using exercise as the stimulus, the theory' would predict reduced anxiety postexercise. By examining when anxiety was measured following exercise (see table II), it can be seen that at most of the time intervals coded (e.g. 0 to 5, 5, 20 and >30 minutes) anxiety was significantly reduced. Unfortunately, it is difficult to adequately evaluate Solomon's model by examining only the acute effects of exercise represented in the state anxiety mcta-analysis.

A more direct test of Solomon's model would involve the examination of postexercise anxiety over the course of an exercise training programme. Solomon's model predicts that as subjects became more physically fit (i.e. they adapted to the exercise stimulus), they would exhibit larger reductions in postexercise anxiety. In addition, anxiety responses during the exercise bout, assessed in a manner similar to Morgan et al. (1980), would be reduced as the exercise stimulus became less aversive. Within the present research, Solomon's opponent-process theory remains an attractive explanation for exercise-induced anxiety reduction, but further testing of the model is necessary, particularly within a training study paradigm.

Opponent-Process Mechanisms: Endorphins and Nervous System Alterations

Solomon (1980) has proposed that endorphins, the body's natural opioid peptides (i.e. endogenously produced), may be important mediators of the opponent-process sequence. Specifically, Solomon proposes that endorphins could be the major substrate for the opponent process, with their presence being responsible for development of affective habituation to aversive stimuli (e.g. feeling better following repeated exposure to exercise).

Numerous studies have examined the relationship between exercise and endorphins [see Sforzo (1988) for a review]. It remains a debatable issue whether or not endorphins actually mediate affective change centrally (Catlin et al. 1980; Gerner et al. 1980; Kline et al. 1977), peripherally (Farrell et al. 1982; Janal et al. 1984), or not at all (Farrell et al. 1982; Goldfarb et al. 1987; Hatfield et al. 1987; Markoff et al. 1982). Some researchers have suggested that 'peripherally administered B-endorphin may enter the CNS' (Catlin et al. 1980, p. 470). McArthur (1985) cited unpublished evidence that the permeability of the blood-brain barrier may actually be altered during exercise. Recent work by Farrell (1989), however, has shown that exercise does not alter the blood-brain barrier such that peripheral endorphins can act directly upon the brain. Farrell's data support the contention that endorphin action is limited to the periphery (e.g. heart), and until it can be shown unequivocally that exercise alters the blood-brain barrier, central effects are at best speculative.

In light of the preceding discussion, it is possible that the primary role of endorphins is to promote physiological economy during exercise (i.e. as opposed to a primary role of affective change). Harber and Sutton (1984) and Santiago and Edelman (1985) have discussed the role of circulating endorphins in promoting reduction of ventilatory activity during exercise. In addition, Morgan (1985) has postulated that the peripheral effects of endorphins, such as adipolysis and ventilatory restraint, may promote a central psychological effect by way of reduced discomfort during exercise. Another hypothesis put forth by Gillman and Katzeff (1988) proposes that the antistress effect of endorphins is possibly activated through inhibited peripheral sympathetic activity. This inhibition may be due to an increased dominance of the parasympathetic nervous system. While problems exist with the use of endorphins as an explanation for exercise-induced affective change, it does remain a tenable hypothesis.

Endorphins offer an appealing explanation for affective change associated with exercise, but they are not the only mechanism that can explain how anxiety may be changed in a manner consistent with the opponent-process model. Dienstbier (1989) has proposed a 'physiological toughness' model which offers a framework for explaining how exposure to stressors (e.g. electric shock, exercise) can lead to psychological coping and emotional stability (i.e. reduced anxiety) through alterations in sympathetic nervous system functioning. The basic premise is that intermittent exposure to stressors reduces basal SNS arousal. In a challenge/threat situation, however, SNS-adrenal-medullary arousal onset in physiologically tough individuals is fast and strong; arousal declines fast with stressor offset. Across repeated episodes, brain catecholamine depletion and pituitary adrenal-cortical responses are suppressed.

With respect to the anxiety reduction (i.e. emotional stability) component of Dienstbier's model (1989, p. 90), results from the trait anxiety meta-analysis seem most relevant. Chronic exercise was associated with reductions in trait anxiety, especially when training programmes exceeded 9 weeks in length (see figure 2). Dienstbier's model proposes this link between active toughening (e.g. exercise) and emotional stability as causative, and while the meta-analysis cannot directly confirm this, it certainly offers support for a causal link.

To explain the links between active physiological toughening and emotional stability, Dienstbier suggested the following 4 mediating physiological factors: (a) central catecholamines [i.e. noradrenaline (norepinephrine), dopamine]; (b) peripheral catecholamines [i.e. adrenaline (epinephrine), noradrenaline], implying an increase in catecholamine availability to 'challenge/threat' situations; (c) SNS (β) sensitivity, implying enhanced response asso-

ciated with β-adrenergic receptors; and (d) cortisol suppression, suggesting an inhibition of cortisol release from the adrenal cortex. While the meta-analyses did not examine catecholamine or cortisol responses to chronic training, Dienstbier's model offers a number of hypothetical pathways that remain to be tested.

While Dienstbier's model deals with the SNS, other investigators have focused on the PNS and whether exercise increases its influence relative to the SNS. PNS activity is usually measured by assessing respiratory sinus arrhythmia (RSA) as an index of vagal tone. RSA refers to the amount of the variation in the interbeat interval of the heart that is associated with respiration. RSA is typically assessed noninvasively by measuring either heart rate variability (HRV), peak to trough differences in the interbeat interval, or using power spectral analytic techniques to analyse the ECG. These estimates of RSA have been demonstrated to be highly correlated with more invasive measures of vagal tone (e.g. cooling the vagus nerve to block its activity, or pharmacologically blocking/enhancing vagal activity) [Eckberg 1983; Fouad et al. 1984; Katona & Jih 1975; Porges et al. 1982].

The opponent-process model would predict that repetition of an arousing stimulus (e.g. chronic exercise) leads to an increase in the magnitude of the b process. As such, PNS activity may become more influential over time, with a resultant increase in relaxation after an exercise bout following training. Kenney (1985) found a significant correlation between fitness (i.e. $\dot{V}O_{2max}$) and PNS activity using HRV as an index of PNS tone. However, Chase et al. (1988), using HRV, and Hatfield et al. (1989), using power spectral analysis of the ECG, did not find significant differences in PNS tone between fit and unfit subjects. These conflicting findings could be due to methodological differences between the studies. Both Kenney (1985) and Hatfield et al. (1989) used quasiexperimental designs (i.e. one was correlational, one was cross-sectional in nature). Chase et al. (1988) did conduct a longitudinal training study, but failed to control for respiration. Measuring HRV without controlling for respiration may reflect the impact of both PNS and SNS activity (Andreassi 1980). Again the idea of increased PNS influence and an enhanced relaxation response as a function of exercise is a plausible hypothesis, but needs further investigation.

2.2.4 Distraction Hypothesis

While the previous explanations for the reduction in anxiety associated with exercise all utilise physiological mechanisms, Bahrke and Morgan's (1978) distraction hypothesis proposes a psychological mechanism. This hypothesis maintains that being distracted from stressful stimuli, or taking 'time-out' from the daily routine, is responsible for the anxiety reduction seen with exercise. As Morgan and O'Connor (1988) have stated, this hypothesis does not dispute the influence of physiological mechanisms (e.g. EEG alterations, temperature effects). It is possible, however, that changes that occur with exercise may also occur with distraction therapies (e.g. quiet rest, meditation, relaxation).

The results of the state and trait anxiety meta-analyses both supported and refuted the distraction hypothesis. For state anxiety, exercise and cognitively-based distraction therapies were equally effective in reducing anxiety (effect size = -0.04). For trait anxiety, however, exercise had superior anxiolytic effects compared to cognitive strategies. This finding fits quite well with Dienstbier's physiological toughness hypothesis (see above). Dienstbier (1989) postulated that cognitively based anxiety reduction strategies provide only a short-term solution to anxiety reduction. Active toughening procedures, like aerobic exercise, lead to long term adaptation.

It seems then, that exercise and cognitively-based distraction therapies provide similar quantitative benefits (i.e. short term anxiety reduction). This effect may be qualitatively different (Morgan & O'Connor 1989), however, in at least 2 ways. First, it appears that anxiety reduction following exercise lasts for a longer period of time than it does for other anxiety-reducing strategies (Raglin & Morgan 1987). Second, the long term effects of exercise as an anxiolytic therapy are greater than for cognitively based anxiety reduction strategies based on

the results of the trait anxiety meta-analysis presented above. Thus, while the distraction hypothesis remains tenable, it appears that the anxiety reduction seen following exercise is due to more than simply taking time out from one's daily routine.

2.2.5 Other Psychological Explanations

Although no research was found suggesting this, it is possible that the 'feel better' phenomenon is simply due to cognitive dissonance (Festinger 1957). Cognitive dissonance is a drive-like state that exists whenever an individual holds incompatible cognitions. For example, the affect associated with exercise may not be enjoyable initially and many people simply quit. However, for those who continue to exercise (which includes all subjects used in the meta-analyses presented here), they must find a way to justify the time and effort expended to do something that others believe to be stressful. In this case, cognitive dissonance theory would predict that the person's attitude (and mood) would shift in a more positive direction. The more effort and hard work the individual must put forth, the greater the dissonance, and the greater the likelihood that the person will feel better about the activity.

The results from the trait anxiety meta-analysis fit nicely within the cognitive dissonance framework. The longer the training programme, the larger the reduction in anxiety. It would be expected that if anxiety reduction was due to cognitive dissonance, both exercise and a motivational control should have yielded similar results. This is not the case, however, since exercise was associated with greater anxiety reduction. It is entirely possible, indeed likely, that those involved in exercise were required to exert a larger degree of effort and experienced more discomfort than motivational control subjects. If this is indeed the case, it would be expected that subjects involved in some sort of exercise programme may overestimate the value of the activity (i.e. report feeling better after completing the programme).

Alternatively, people may report feeling better following exercise because they expect such a change. With all of the media attention related to the positive psychological consequences of exercise (e.g. 'runner's high', endorphins), people may report feeling better simply because they have been told they should feel this way. This is certainly not refuted by the findings from the meta-analyses. This expectation may even be reinforced as the person begins to lose weight, discovers that she or he can exercise for a longer time, can run greater distances, or is not as fatigued following an exercise session. This could be tested in future research by having subjects assess their expectations of psychological states/traits with respect to exercise.

Another potential psychological explanation could be described from more of an information processing perspective. It has been proposed that the affective change associated with exercise is due to monitoring of ongoing physiological processes (e.g. Rejeski, personal communication). Subjects are not reporting anxiety as much as they are reporting physiological changes. During exercise, autonomic activity increases well above baseline levels; subjects also report increased anxiety (Morgan et al. 1980). Upon cessation of exercise, autonomic activity decreases and self-reported anxiety is also reduced.

This information processing idea could help explain the state anxiety findings. It is possible that when subjects arrive at the laboratory for testing, they may be experiencing pretest apprehension or may reflect higher anxiety as a result of anticipating the exercise session. This could easily be manifested by increased autonomic activity. As such, the 'baseline' anxiety measures that are obtained may not be true baselines (Farha & Sher 1989). Future research needs to address this issue by using adaptation sessions in the laboratory to allow subjects to familiarise themselves with experimental procedures. If this pre-exercise apprehension is artificially inflating baseline anxiety levels, lower postexercise anxiety may occur because: (a) the uncertainty of the experimental procedures is no longer having an influence; and (b) there is also the reduction in physiological arousal after exercise.

While the information processing model is interesting, it does not account for the changes seen in trait anxiety since these measures are taken outside of an exercise context (i.e. not following an

exercise bout) and not in conjunction with any exercise-induced increases in autonomic activity. As such, this information processing explanation could fit the acute situation (i.e. state anxiety), but it would be difficult to see how this would explain the trait anxiety results obtained from the meta-analysis.

Numerous 'self variables have been suggested as potentially mediating the exercise-affect relationship. Self-efficacy (Bandura 1977), self-mastery (Ismail & Trachtman 1973), perceived self-competence (Harter 1978) and self-esteem (Sonstroem 1984) may all be related to the change in affect associated with exercise. The basic idea of these intertwined concepts is that completion of an effortful task (e.g. exercise), which is important for one's goals and motives, can promote a sense of self-competence, a sense of self-mastery, or a good feeling from having achieved a goal.

Self-efficacy, the strength of the belief that one can successfully execute a behaviour, has been examined a great deal in the exercise science literature. Bandura (1977) has postulated self-efficacy as a common cognitive mechanism for mediating motivation and behaviour. The perception of one's capabilities to perform in demanding situations (e.g. exercise) affects that individual's emotional reactions. Self-efficacy is derived from numerous sources of information (e.g. performance accomplishments, vicarious experiences, verbal persuasion), including physiological arousal. Individuals assess their level of physical self-efficacy in strength or endurance activities, by using levels of fatigue, fitness, and sensations of pain. As an individual's fitness increases through training, feelings of fatigue and pain will be reduced and self-efficacy should be increased. Since the individual perceives that his or her capabilities have improved, anxiety should decrease as well. Previous self-efficacy research, however, has shown that in repeated assessments of the same situation (e.g. diving, weightlifting), self-efficacy is less directly influential on performance. Other variables, like previous performance and one's perception of that performance, may be more influential than self-efficacy (Feltz 1982).

Sonstroem and Morgan (1989) have proposed a model based on some of these 'self variables (e.g. physical self-efficacy, physical competence, physical acceptance, self-esteem) which may offer utility in future examination of the exercise-affect relationship. Trait anxiety may decrease over time because as an individual continues through an exercise programme self-efficacy statements (i.e. competency judgements or mastery) would most likely become stronger. Anxiety decreases as the certainty that one can successfully complete a demanding activity increases.

This idea is indirectly supported by the results from the trait anxiety meta-analysis. If physical activity is important to an individual (i.e. the individual has a high degree of physical self-esteem), there should be a greater anxiety reduction effect for exercise when compared to some type of motivational control. While this did happen, self-esteem was not a factor that was examined in the meta-analysis. Future research might assess these 'self variables to determine if any might be mediating the anxiolytic effects of exercise.

2.3 Conclusions

A number of potential mechanisms have been advanced in an attempt to explain the exercise-anxiety relationship. Hopefully, the mechanisms presented will contribute to the generation of testable hypotheses in future investigations. Given the present level of knowledge concerning this complex relationship, reliance upon a single mechanism to explain the observed consequences may be too simplistic. Such outcomes may be determined by a number of factors acting and interacting concurrently or in a series. As such, resultant anxiety states may be explained by a number of simultaneous causal agents. While a number of basic descriptive questions remain to be answered, future research must begin to examine causes rather than continue to describe what has come to be a fairly solid relationship.

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The studies used in the meta-analyses are listed in the Appendix *which follows*

Appendix. Characteristics of the studies used in the meta-analyses. * indicates published journal article

Study	Acute/ chronic exercise	Length of training (weeks)	n	Subject assignment	Health status	Sex	Mean age (years)	Type of exercise	Duration (min)	Intensity	Frequency (times/ wk)	Comparison group	Anxiety category: measure
Abood (1981)	Acute		42	Random	HA	F	18-22	Aerobic	5	nr		No trtmt	State: STAI
			43	Random	LA	F	18-22	Aerobic	5	nr		No trtmt	State: STAI
Allen & Coen (1987)*	Acute		12	W/in Ss design	N	M	21.0	Aerobic	45	Graded		Pre-post	State: POMS-ten
Andres et al. (1978)	Acute		16	W/in Ss design	N	M	18-32	Aerobic	20	70% HR_{max}		Pre-post	State: STAI
Bahrke (1981)*	Acute		30	Random	N	M,F	40.1	Aerobic	20	75% HR_{max}		Quiet rest	State: STAI
Bahrke & Morgan (1978)*	Acute		75	Random	N	M	51.9	Aerobic	20	70% HR_{max}		Meditation	State: STAI Physiol: SBP, DBP
												Quiet rest	State: STAI Physiol: SBP, DBP
Bahrke & Smith (1985)*	Acute		43	Random	N	M,F	10.6	Aerobic	15	Self-selected		Busy work	State: STAI
			44			M,F						Quiet rest	State: STAI
Balog (1983)*	Acute		20	W/in Ss design	N	M, F	18-30	Aerobic	30	Graduated		Pre-post	Physiol: EMG
Berger & Owen 1983)*	Chronic	14	28	Intact	N	M	22.3	Aerobic	40	Self-selected	2	Lecture class	State: POMS-ten
			39			F							
			31										
			44			F							
Berger & Owen (1987)*	Chronic	14	52	Intact	N	M, F	22.3	Aerobic	40	nr	2	Pre-post	Trait: STAI State: STAI
	Chronic	5	48	Intact	N	M, F	25.3	Aerobic	55	nr	4	Pre-post	Trait: STAI State: STAI
Berger et al. (1988)*	Chronic	4	61	Intact	N	M	19.5	Aerobic	20	65-80% HR_{max}	3	No trtmt	State: POMS-ten
		8										Relaxation	
		4	59	Intact								Discussion	
		8											
		4	53	Intact									
		8											

		4 8	92	Intact	N	F	19.5	Aerobic	20	65-80% HRmax	3	No trtmt Relaxation Discussion	State: POMS-ten
		4 8	86	Intact									
		4 8	86	Intact									
Blumethal et al. (1980)*	Chronic	10	46	W/in Ss design	N	M, F	42.6	Aerobic	30-45	70-85% HR_{max}	3	Pre-post	Physiol: SBP, DBP
Blumenthal et at. (1982a)*	Chronic	10	32	Matching	N	M, F	45.1	Aerobic	45	70-85% HR_{max}	3	No trtmt	Trait: STAI State: STAI, POMS-ten
Blumenthal et al. (1982b)*	Chronic	11	24	W/in Ss design	N	M, F	69.3	Aerobic	25	70-85% HR_{max}	3	Pie-post	State: POMS-ten Physiol: SBP, DBP, HR
Boutcher (1986)	Acute		15	Intact (runners)	N	M	27.8	Aerobic	20	80-85% HR_{max}		Quiet rest	State: STAI, POMS-ten Physiol: EEG α
												Pre-post	State: STAI. POMS-ten Physiol: EEG α
			15	Intact (nonrunners)	N	M	27.8	Aerobic	20	80-85% HR_{max}		Quiet rest	State: STAI. POMS-ten Physiol: EEG α
												Pre-post	State: STAI, POMS-ten Physiol: EEG α
Boyll (1985)	Chronic	6	14	Intact	N	M, F	18-30	Aerobic	50	nr	2	No trtmt	State: STAI
Buccola & Stone (1975)*	Chronic	14	20	Intact	N		60-79	Aerobic	10-40	nr	3	Pre-post	Trait 16PF-Q4 Physiol: SBP, DBP, HR
			16	Intact	N	M	60-79	Aerobic	10-40	nr	3	Pre-post	Trait: 16PF-Q4 Physiol: SBP, DBP, HR
Bulbulian & Darabos (1986)*	Acute		10	W/in Ss design	N	M, F	28.7	Aerobic	20	40% $\dot{V}O_{2max}$ 70% $\dot{V}O_{2max}$		Rest	Physiol: H/M reflex

continued over

Appendix. Contd

Study	Acute/ chronic exercise	Length of training (weeks)	n	Subject assignment	Health status	Sex	Mean age (years)	Type of exercise	Duration (min)	Intensity	Frequency (times/ wk)	Comparison group	Anxiety category: measure
Bulbulian & Ebert (1987)	Acute		11 10	Intact	Type A Type B	F F	nr	Unspecific	20	60% $\dot{V}O_{2max}$		Pre-post	Physiol: EMG, SBP, DBP
Bursten (1982)	Acute		130	W/in Ss design	N	M, F	38.5	Aerobic	nr	Self-selected		Pre-post	State: POMS-ten
Cherico (1987)	Acute Acute		54 46	Random Random	N N	F F	34.5 34.5	Aerobic Aerobic	30 30	Self-selected Self-selected		Pre-post Pre-post	State: POMS-ten State: POMS-ten
Daugherty et al. (1987)	Acute		20	Intact	N	M	nr	Aerobic	nr nr nr	Maximum 65% $\dot{V}O_{2max}$ 50% $\dot{V}O_{2max}$		Pre-post Pre-post Pre-post	Physiol: EEG α Physiol: EEG α Physiol: EEG α
deVries (1968)*	Acute		29	W/in Ss design	N	M, F	22.2	Aerobic	5	nr		Exercise vs control Pre-post	Physiol: EMG
	Chronic	6	18	Intact	N	M	40.4	Aerobic	60	nr	3	No trtmt	Physiol: EMG
deVries et al. (1972)*	Acute		10	W/in Ss design	N	M, F	62.6	Aerobic	15	60% HR_{max}		Quiet rest	Physiol: EMG
deVries et al. (1981)*	Acute		10	W/in Ss design	N	M, F	36.4	Aerobic	20	40% HR_{max}		Pre-post	Physiol: H/M reflex
deVries et al. (1982)*	Acute		6	W/in Ss design	N	M, F	25.5	Aerobic	20	40% HR_{max}		Quiet rest	Physiol: H/M reflex
Dishman & Gettman (1981)	Chronic	20	33	Random	N	M	31.0	Nonaerobic	nr	nr	3	No trtmt	State: POMS-ten
Duncan & Farr (1987)	Chronic	15	64	Random	HT	M	21-37	Aerobic	35	nr	3	No trtmt	Trait: STAI
Dyer & Crouch (1987*)	Acute Acute		39 39	Intact (beg run) Intact (adv run)	N N	M, F M, F	18-24 18-24	Aerobic Aerobic	nr nr	Self-selected Self-selected		Lecture class Lecture class	State: POMS-ten State: POMS-ten

Eby(1984)	Chronic	6	21	Random	N	M, F	19-31	Aerobic	60-90	Self-selected	3	Delayed treatment	Trait: STAI
			20					Nonaerobic	60-90	Self-selected	3	Delayed treatment	Trait: STAI
Eickoff et al. (1983)*	Chronic	10	39	Random	N	F	19-36	Aerobic	30	80% HR_{max}	3	No trtmt	Physiol: HR_{rest}
Einhaus (1984)	Chronic	5	48	W/in Ss design	N	M, F	11.3	Aerobic	15	nr	3	Pre-post	State: STAI
El-Naggar (1986)*	Chronic	16	30	W/in Ss design	N	M	25-65	Aerobic	90	nr	3	Pre-post	Trait: 16PF-Q4 Physiol: SBP, DBP, HR
Emery & Gatz (1985)	Chronic	12	25	Random	N	M, F	71.6	Nonaerobic	60	nr	3	Social activities	Trait: CES-anx Physiol: SBP, DBP, HR
												Waiting list	Trait: CES-anx Physiol: SBP, DSP, HR
	Chronic	24	29	Random	N	M, F	71.6	Nonaerobic	60	nr	3	Social activities	Trait: CES-anx Physiol: SBP, DBP, HR
Farmer et al. (1978)	Acute		6	Intact	Type A		18-30	Aerobic	6	'Submax' 'Max'		Pre-post	Physiol: EMG, EEG
			6		Type B	M	18-30	Aerobic	6	'Submax' 'Max'			
Farrell et al. (1983)*	Acute		14	W/in Ss design	N	M, F	35.0	Aerobic	10km race	Self-selected		Pre-post	State: POMS-ten
Farrell et al. (1986)*	Acute		8	W/in SS design	N	M	24.2	Aerobic	30	70% $\dot{V}O_{2max}$		Pre-post	State: POMS-ten Physiol: SBP, DBP
Farrell et al. (1987)*	Acute		7	W/in Ss design	N	M	27.4	Aerobic	80 60 40	40% $\dot{V}O_{2max}$ 60% $\dot{V}O_{2max}$ 80% $\dot{V}O_{2max}$		Pre-post	State: POMS-ten
Fasting & Gronnings-aeter (1986)*	Chronic	12	34	Matching	N	M, F	24-45	Aerobic	120	Self-selected	2	Study group	Trait STAI
			33	Matching	N	M, F	24-45	Aerobic	120	Self-selected	2	No trtmt	Trait STAI

continued over

Appendix. Contd

Study	Acute/ chronic exercise	Length of training (weeks)	n	Subject assignment	Health status	Sex	Mean age (years)	Type of exercise	Duration (min)	Intensity	Frequency (times/ wk)	Comparison group	Anxiety category: measure
Folkins (1976)*	Chronic	12	18	W/in Ss design	CR	M	40-58	Unspecific	nr	nr	3	Pre-post	Trait MAACL-anx
Folkins et al. (1972)*	Chronic	16	21	Intact	N		18-30	Aerobic	30	nr	2	Pre-post	Trait: MAACL-anx
Fort (1982)	Acute		40	W/in Ss design	N	M, F	22.8	Aerobic	30	60% HR_{max}		Pre-post	State: STAI Physiol: PSI
			20				22.4						State: STAI Physiol: PSI
			20			F	23.2						State: STAI Physiol: PSI
Frazier & Nagy (1989)*	Chronic	15	31	W/in Ss design	N	F	21.4	Aerobic	30	65-85% HR_{max}	3	Pre-post	State: POMS-ten
			55	W/in Ss design	N	F	21.4	Aerobic	30	65-85% HR_{max}	3	Pre-post	State: POMS-ten
Frye (1982)	Chronic	10	47	Intact	N	M, F	nr	Aerobic	45	Self-selected	4	No trtmt	State: STAI, POMS-ten Trait: STAI
			33	Intact	N	M, F	nr	Aerobic	75	Self-selected	2.5	No trtmt	State: STAI, POMS-ten Trait: STAI
			30	Intact		M, F		Aerobic	75	Self-selected	2.5	No trtmt	State: STAI, POMS tan Trait: STAI
Gillespie et al. (1982)	Chronic	11	65	W/in Ss design	N	M	45.4	Aerobic	30-40	70-85% HR_{max}	3	Pre-post	State: POMS-ten Physiol: HR, SBP, DBP
Goldfarb & Hatfield (1987)*	Acute		9	W/in Ss design	N	M	24.0	Aerobic	Vol. fat.	Graded		Pre-post	State: MAACL-anx
Goldwater & Collis (1985)*	Chronic	6	32	nr	N	M	19-30	Aerobic	90	Self-selected	5	Recreation (nonaerobic)	Trait: Taylor Manifest Anxiety

Griffith (1982)	Chronic	9	12	Intact	HA	F	18-25	Aerobic	20	75% HR_{max}	4	No trtmt	State: STAI Trait: STAI
			13									Quiet rest	State: STAI Trait: STAI
			15									No trtmt	State: STAI Trait: STAI
			18									Quiet rest	State: STAI Trait: STAI
			19	Intact	LA	F	18-25	Aerobic	20	75% HR_{max}	4	No trtmt	State: STAI Trait STAI
			19									Quiet rest	State: STAI Trait: STAI
			17									No trtmt	Stale: STAI Trait: STAI
			14									Quiet rest	State: STAI Trait: STAI
Hannaford et al. (1988)*	Chronic	8	18	Random	PSY	M	25-60	Aerobic	30	60% HR_{max}	3	Corrective therapy	State: STAI Physiol: Skin temp, EMG
												Waiting list	State: STAI
Hatfield et al. (1987)*	Acute		9	W/in Ss design	N	M	26.1	Aerobic	nr	Graduated		Pro-post	State: MAACL-anx
			7	W/in Ss design	N	M	66.0	Aerobic	nr	nr		Pre-post	State: MAACL-anx
Hatfield et al. (1988)	Acute		12	W/in Ss design	N	M	27.0	Aerobic	30	60% $\dot{V}O_{2max}$		Pre-post	State: POMS-ten, STAI, MAACL-anx
										70% $\dot{V}O_{2max}$		Pre-post	State: POMS-ten, STAI, MAACL-anx
										80% $\dot{V}O_{2max}$		Pre-post	State: POMS-ten, STAI, MAACL-anx
Hill (1981)*	Chronic	16	12	W/in Ss design	CR	M	nr	Aerobic	nr	nr	nr	Notrtmt	Trait: IPAT
			24	Random								Motivation control	Trait: IPAT
Hilyer et al. (1982)*	Chronic	20	43	Random	N	M	15-18	Aerobic	65	Self-selected	3	Counseling	State: STAI, POMS-ten Trait: STAI

continued over

Appendix. Contd

Study	Acute/ chronic exercise	Length of training (weeks)	n	Subject assignment	Health status	Sex	Mean age (years)	Type of exercise	Duration (min)	Intensity	Frequency (times/ wk)	Comparison group	Anxiety category: measure
Hughes et al. (1986)*	Chronic	12	28	Random			32.6	Aerobic	45	Self-selected		No trtmt	State: POMS-ten
Johnston & Allen (1988)	Chronic	12	96	Intact	N	M, F	39.8	Aerobic	nr	nr	2.5	Adult education	Trait: STAI
Jones (1981)	Chronic	10	51	Intact	N	M, F	15-35	Aerobic	30	Self-selected	2	Meditation	State: STAI CSAQ-C,-S Trait: STAI
			52	Intact	N	M, F		Nonaerobic	40	Self-selected	2	Meditation	State: STAI CSAQ-C, -S Trait: STAI
Jones & Weinhouse (1979)*	Chronic	56	12	W/in Ss design	N	M. F	30.3	Aerobic	45	75% $\dot{V}O_{2max}$	3	Pre-post	Trait: 16PF-Q4, Self-rating Physiol: SBP, DBP, HR
Klein et al. (1985)*	Chronic	12	28	Random	PSY	M, F	30.3	Aerobic	30	Self-selected	3	Group therapy	Trait SCL-90, CMI-ten
			22	Random	PSY	M, F	30.3	Aerobic	30	Self-selected	2	Meditation	Trait: SCL-90, CMI-ten
			14	W/in Ss design	PSY	M, F	30.3	Aerobic	30	Self-selected	3	Pre-post	Trait: SCL-90, CMI-ten
Koriath et al. (1987)*	Acute		15	W/in Ss design	N	M	18-25	Aerobic	20	48% HR_{max}		Rest	Physiol: EEG, EEG
										62% HR_{max}		Rest	Physiol: EEG, EEG
Kowal etal. (1978)*	Chronic	6	177	Random	N	M	19.6	Basic training	nr	nr	nr	No trtmt	State: STAI, POMS-ten Trait: STAI
	Chronic	6	187	Random	N	F	20.9	Basic training	nr	nr	nr	No trtmt	State: STAI, POMS-ten Trait: STAI
Kowal et al. (1979)	Chronic	12	20	Intact	N	F	10-22	Aerobic	nr	nr	nr	Pre-post	State: STAI, POMS-ten

Kuehne & Perrigo (1980)	Chronic	12	35	Intact	CR	M, F	nr	Aerobic	nr	nr	3	No trtmt	Trait: STAI
Levin & Gimino (1982)	Chronic	10	14	Intact	PSY	M, F	nr	Aerobic	40	nr	3	Pre-post	State: POMS-ten Trait: STAI
Lichtman & Poser (1983)*	Acute		32	Intact	M	M, F	25.6	Aerobic	45	nr		Pre-post	State: POMS-ten, Nowlis-anx
Lion (1978)*	Chronic	8	6	Random	PSY	M, F	nr	Aerobic	Self-selected	Self-selected	3	No trtmt	Trait: STAI
Lobitz et al. (1983)*	Chronic	7	11	Random	Type A	M, F	36.5	Aerobic	60	75-95% HR_{max}	3	No trtmt	State: STAI Trait: STAI Physiol: SBP, DBP
												Anxiety management training	State: STAI Trait: STAI
Long (1984)*	Chronic	10	38	Random	N	F	39.9	Aerobic	15-60	60-90% HR_{max} 50-85%	3-5	Waiting list	State: STAI, Ten therm Trait: STAI
			36	Random	N	F	39.9	Aerobic	15-60	60-90% HR_{max} 50-85%	3-5	Stress inoculation	State: STAI, Ten therm Trait: STAI
			25	Random	N	F	39.9	Aerobic	15-60	60-90% HR_{max} 50-85%	3-5	Stress inoculation	State: STAI, Ten therm Trait: STAI
Long & Haney (1988a)*	Chronic	8	39	Random	N	F	40.0	Aerobic	nr	nr	2	Relaxation	Trait: STAI
Long & Haney (1988b)*	Chronic	8	50	Random	N	F	39.8	Aerobic	nr	Self-selected	3	Relaxation	Trait: STAI

continued over

Appendix. Contd

Study	Acute/ chronic exercise	Length of training (weeks)	n	Subject assignment	Health status	Sex	Mean age (years)	Type of exercise	Duration (min)	Intensity	Frequency (times/ wk)	Comparison group	Anxiety category: measure
Maloney et al. (1986)*	Chronic	5	75 21 54	W/in Ss design	N	M, F M F	26.4	Aerobic	30	Self-selected	3	Pre-post	Trait: STAI
	Chronic	16	43 15 28	W/in Ss design	N	M, F M F	31.0	Aerobic	30	Self-selected	3	Pre-post	Trait: STAI
Markoff et al. (1982)*	Acute		15	W/in Ss design	N	M, F	36.8	Aerobic	60	Self-selected		Pre-post	State: POMS-ten
Massie & Shephard (1971)*	Chronic	28	27	Intact	N	M	36.4	Aerobic	40-45	Sell-selected	3	Pre-post	Trait: Taylor Manifest Anxiety
McGlynn et al. (1983)*	Chronic	14	30	Intact	N	M, F	18-26	Aerobic	35-40	Self-selected	2	Lecture class	State: STAI Trait: STAI Physiol: SBP, EMG
McGowan et al. (1985)*	Acute		12	W/in Ss design	N	M	21-29	Aerobic	15	40% PWC 55% PWC 70% PWC		Exercise vs baseline	Physiol: EMG
Mendez (1985)	Chronic	7	41	Intact	CR	M	57.6	Aerobic	45	nr	3	No trtmt	State: SCL-90
Mitchum (1976)	Acute		40 20 20	W/in Ss design	N	M, F F	21.5	Nonaerobic	15	Self-selected		Pre-post	State: STAI State: STAI State: STAI
Moran (1984)	Chronic	7	46	Intact	N	M, F	14-18	Aerobic	15	60-90% HR_{max}	4	Lecture class	State: MAACL-anx Physiol: HR_{rest}, rec
Morgan et al. (1971)*	Acute		12	Random	N	M	18-30	Aerobic	17	63% HR_{max} 72% HR_{max}		Rest	State: IPAT-anx
			12	Random	N	F	18-30	Aerobic	17	55% HR_{max} 63% HR_{max}		Rest	State: IPAT-anx
Nowlis & Greenberg (1979)*	Acute		18	W/in Ss design	N	M, F	17-55	Aerobic	12.5mi	Self-selected		Pre-post	State: MACL-anx, MACL-relax, STAI (used trait)

Pauly et al. (1982)*	Chronic	14	73	W/in Ss design	N	M, F	36.0	Aerobic	20	60-85% HR_{max}	3	Pre-post	Trait: STAI Physiol: SBP, DBP, HR
Perri (1980)	Chronic	14	42	Matching	N	M, F	65.6	Aerobic	30	45-50% max	3	No trtmt	State: MAACL-anx Physiol: HR_{rest}
Pineda & Adkisson (1961)*	Acute		16	W/in Ss design	N	M, F	22-36	Aerobic	50	Graduated		Pre-post	Physiol: EEG
Popejoy (1967)	Chronic	20	45	Intact	N	F	20-54	Aerobic	40	Graduated	4	No trtmt	Trait: IPAT, NSQ
Porcari et al. (1986)	Acute		36	W/in Ss design	N	M, F	37.4	Aerobic	40	50% $\dot{V}O_{2max}$		Pre-post	State: STAI Physiol: SBP. DBP
Prosser et al. (1981)*	Chronic	15	23	Random	CR	M	50.1	Aerobic	45	nr	2	No trtmt	Trait: Freedom from anxiety
Raglin & Morgan (1987)*	Acute		15	W/in Ss design	N	M	34.2	Aerobic	40	Self-selected		Quiet rest	State: STAI Physiol: SBP, DBP
Raglin & Morgan (1987)*	Acute		15	W/in Ss design	HT	M	60.2	Aerobic	40	Self-selected		Quiet rest	State: STAI Physiol: SBP, DBP
Reiter (1981)	Chronic	5	128	Intact	N	F	73.4	Aerobic	10-15	Self-selected	2	Alternate activities	State: STAI
Roviaro et al. (1984)*	Chronic	12	48	Intact	CR	M	56.0	Aerobic	60	nr	3	Routine care	Trait: STAI Physiol: SBP, DBP
Rozenek (1985)	Acute		10	Intact		M	27.2	Aerobic	47	62.5% $\dot{V}O_{2max}$		No trtmt	State: POMS-ten
			10	Intact	N	M	27.6	Nonaerobic	47	62.5% of 1RM		No trtmt	State: POMS-ten
Schwartz et al. (1978)*	Chronic (cross-sectional)	24	77	Intact	N	M,F	27.4	Aerobic	60	nr	3.6	Meditation	Trait: CSAQ
Setaro (1985)	Chronic	10	50	Random	PSY	M, F	18-35	Aerobic	nr	nr	2	No trtmt	Trait: MMPI-anx
			50									Nonaerobic activity group	Trait: MMPI-anx
			50									Counselling	Trait: MMPI-anx

continued over

Appendix. Contd

Study	Acute/ chronic exercise	Length of training (weeks)	n	Subject assignment	Health status	Sex	Mean age (years)	Type of exercise	Duration (min)	Intensity	Frequency (times/ wk)	Comparison group	Anxiety category: measure
Shephard et al. (1985)*	Chronic	56	317	Intact	CR	M	50.5	Aerobic	nr	nr	nr	Pre-post	State: POMS-ten Physiol: HR, SBP, DBP
Silvestri (1985)	Chronic	4	46	Intact	N	F	15.1	Aerobic	50	nr	4	No treatment	State: STAI Trait: STAI Physiol: SBP, DBP MR_{rest}, rec
Steptoe S Cox (1988)*	Acute		32	W/in Ss design	N	F	20.0	Aerobic	8	51% HR_{max} 78% HR_{max}		Pre-post	State: POMS-ten
Sweeney et al. (1978)*	Acute		24	Alternated	PSY	F	40.4	Aerobic	45	Sell-selected		No trtmt	State: STAI
Thaxton (1982)*	Acute		18	Random	N	M, F	36.0	Aerobic	30	Sell-selected		No trtmt	State: POMS-ten Physiol: GSR
Thayer (1987)*	Acute		18	W/in Ss design	N	M, F	28.5	Aerobic	10	Self-selected		Pre-post	State: ADACL
Thomas (1983)	Chronic	4	14	Random	OB	F	43.0	Aerobic	24	60% HR_{max}	3	Nonaerobic activity	Trait MACL-anx
Weinberg et al. (1988)*	Acute		97	Intact	N	M, F	18-30	Aerobic	30	60% HR_{max}		No trtmt	State: STAI, ADACL, POMS-ten
			89					Aerobic				No trtrtrt	State: STAI, ADACL. POMS-ten
			102					Nonaerobic				No trtmt	State: STAI. ADACL, POMS-ten
Weiser (1982)	Chronic	8	20	W/in Ss design	N	M, F	35.2	Aerobic	nr	Self-selected	3	Pre-post	State: STAI Trait: STAI Physiol: SBP, DBP, HR
			22	W/in Ss design	N	M, F	35.3	Aerobic	nr	Self-selected	3	Pre-post	State: STAI Trait STAI Physiol: SBP, DBP, HR

Wiese et al. (1983)	Acute		20	W/in Ss	N	M, F	nr	Aerobic	40	25 min at 40% $\dot{V}O_{2max}$, 15 min at 60% $\dot{V}O_{2max}$		Pre-post	Physiol: EEG α
Wilfley & Kunce (1986)*	Chronic	8	49	W/in Ss design	N	M, F	43.0	Aerobic	60	nr	3	Pre-post	State: POMS-ten Physiol: HR_{rest}
Williams & Getty (1986)*	Chronic	10	306	Intact	N	M. F	18-30	Aerobic	50	60-70% HR_{max}	3	Class drop-outs	State: POMS-ten Physiol: HR_{rest}
			390	Intact	N	M, F	18-30	Aerobic	50	60-70% HR_{max}	3	Recreation, games	State: POMS-ten Physiol: HR_{rest}
Wilson (1985)	Chronic	16	34	Random	N	F	32.1	Aerobic	20-30	60-80%	3	No treatment	State: STAI Trait: STAI Physiol: HR_{rest}
Zertner (1981)	Acute		25	Random	N	M, F	42.0	Aerobic	40	nr		No trtmt	State: POMS-ten

Abbreviations and symbols: nr = not reported: W/in Ss = within subjects; N = normal: CR = cardiac rehabilitation or risk; PSY = psychiatric; HA = high anxious: LA = low anxious; OB = obese; PSI = Palmer Sweat Index; Type A = Type A personality; Type B = Type B personality; HT = hypertensive; STAI = State Trait Anxiety Inventory; POMS-ten = Profile of Mood States-tension; SBP = systolic blood pressure; DBP = diastolic blood pressure; EMG = electromyography; HR = heart rate; rec = recovery; 16PF-Q4 = Cattell's 16 Personality Factor (relaxed-tense): H/M reflex = Hoffman reflex; ADACL = Activation Oeactivation Adjective Check List; CES-anx = Centers for Epidemiological Studies-anxiety; EEG = electroencephalogram; MAACL-anx = Multiple Affect Adjective Check List-anxiety; MACL-anx (- relax) = Mood Adjective Check List-anxiety (- relaxation); CSAQ-c = Cognitive Somatic Anxiety Questionnaire-cognitive; CSAQ-s = Cognitive Somatic Anxiety Questionnaire-somatic; CMI-ten = Cornell Medical Index-tension; SCL-90 = Symptom Check List-90; Ten therm = Tension thermometer; IPAT = Institute for Personality & Ability Testing-anxiety; MMPI-anx = Minnesota Multi-Phasic Personality Inventory; NSQ = Neuroticism Scale Questionnaire; GSR = Galvanic skin response; vol. fat. = volitional fatigue; beg (adv) run = beginner (advanced) runners.

References for the Appendix

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