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Review

The effect of regular aerobic exercise on positive-activated affect: A meta-analysis

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ABSTRACT

Objective: The purpose of this meta-analysis was to examine the effect of regular aerobic exercise on self-reported positive-activated affect (PAA). Samples from 105 studies (1980–2008) were included yielding 370 effect sizes (ESs) and 9840 participants.

Method: Studies were coded for the following moderators: baseline affect, exercise frequency, intensity, time, program duration, exercise dose, study quality, and study source. The analysis employed multiple measures of affect and corrected for statistical artifacts using the meta-analytical methods of Hunter and Schmidt (1990, 2004).

Results: The overall mean corrected ES ($\bar{d}_{corr}$) and standard deviation (SD_{corr}) were .57 and .48, respectively. Two clear moderator effects were found: the inverse association between baseline PAA and ES and the positive association between study quality and ES. The effect also varied with exercise frequency (positive relation) and exercise intensity (negative relation). Exercise dose was only a weak moderator, but the results indicate the following aerobic exercise program as optimal for improving PAA: low intensity (~30% VO_2R), 30–35 min, 3–5 days/wk for 10–12 weeks. Similar effects were found for published and unpublished studies (source). Control conditions produced little change in PAA ($\bar{d}_{corr} = .03$, $SD_{corr} = .11$).

Conclusion: Regular aerobic exercise results in moderate increases in self-reported PAA, but the effects vary by baseline affect and study quality. Exercise-related variables produced weaker moderating effects. PAA was unchanged for control conditions. A more comprehensive understanding of exercise-related affect will emerge when researchers examine the interaction of acute and chronic responses.

The field of positive psychology considers well-being an important topic of scientific inquiry. Well-being, defined as engagement, life satisfaction, and positive affect (Lyubomirsky, King, & Diener, 2005) correlates with job satisfaction (Miner, 2001), marital satisfaction (Rogers & May, 2003), mortality (Fiscella & Franks, 1997), longevity (Kubzansky, Sparrow, Vokonas, & Kawachi, 2001), and immune function (Cohen, Doyle, Turner, Alper, & Skoner, 2003). And while physical inactivity is related to fatigue and low energy (Puetz, 2006), those who exercise more report higher levels of well-being (Lox, Burns, Treasure, & Wasley, 1999).

Positive affect may be the component of well-being associated with physical (Rozanski & Kubzansky, 2005) and psychological health (Lyubomirsky et al., 2005). Steptoe, Wardle, and Marmot (2005), for example, found an inverse relationship between positive affect and heart rate, cortisol, and plasma fibrinogen. Findings were independent of age, gender, body mass and psychological distress and were confirmed at 3-year follow-up (Steptoe & Wardle, 2005). And cross-sectional, longitudinal, and experimental studies show that positive affect is related to success in several life domains, including work and personal relationships (Lyubomirsky et al.).

A key theoretical feature relevant to the improvement and maintenance of well-being is the repeated experience of positive affect (Lyubomirsky et al., 2005). Exercise appears to be a viable strategy for improving affect and several lines of research support this including qualitative (Jeng, Yang, Chang, & Tsao, 2004), cross-sectional (Audrain, Schwartz, Herrera, Goldman, & Bush, 2001), epidemiological (Kritz-Silverstein, Barrett-Connor, & Corbeau, 2001), and experimental (Ekkekakis, Hall, VanLanduyt, & Petruzzello, 2000). In fact, although exercise appears effective for regulating affect (Thayer, Newman, & McClain, 1994), the benefits are best obtained with regular exercise and are lost if the behavior is discontinued (Kritz-Silverstein et al., 2001). Therefore, an examination of the effect of regular exercise on positive affect is

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theoretically and practically important for the well-being literature. The focus will be on aerobic exercise as the majority of the research on the exercise-affect relation has examined this type of activity.

Affect will be defined for this paper according to the concept of core affect (Russell, 2003). Core affect describes the quality of subjective experience along the valence (positive–negative) and activation (activated–deactivated) dimensions of the affect circumplex (Posner, Russell, & Peterson, 2005). Our focus will be aerobic exercise-induced changes in affective states of the upper right quadrant of the circumplex (see Fig. 1). We refer to this quadrant as *positive-activated affect* (PAA).

Aerobic exercise-related PAA changes may be important for several reasons including, neurobehavioral systems that promote goal-directed behavior (Depue & Collins, 1999; Tomarken & Keener, 1998), dopamine pathways related to self-reported physical activity (Simonen et al., 2003), effective psychological and behavioral coping (Lyubomirsky et al., 2005), and exercise adherence (Thayer, 1996). In fact, studies show that improvements in exercise-related changes in PAA are more consistent than changes in depression and anxiety (Gauvin, Rejeski, & Norris, 1996; Watson, 2000).

The purpose of this meta-analysis is to estimate the magnitude and variability of the effect of regular aerobic exercise on PAA and test potential participant, exercise, and research- related moderators. Using the advantages of meta-analysis this study aims to improve and extend current knowledge and provide quantitative answers not found in prior reviews.

Potential moderators

Moderators were selected from the results and theoretical implications of descriptive studies, experimental studies, and quantitative and narrative reviews of the acute and chronic exercise-affect literature.

Baseline PAA

Quantitative reviews have found regular exercise associated with greater reductions in negative affect for participants with initially higher levels of anxiety and depression (e.g., North, McCullagh, & Tran, 1990; Petruzzello, Landers, Hatfield, Kubitz, & Salazar, 1991). Subsequent prospective studies confirm these findings (Mobily, Rubenstein, Lemke, O'Hara, & Wallace, 1996). The PAA literature appears more mixed. Some studies show no relation between baseline and post-exercise PAA (e.g., Brown, Goldstein-Shirley, Robinson, & Casey, 2001; Gitlin et al., 1992; Lox, McAuley, & Tucker, 1995) while others show an association (Blumenthal, Emery, & Rejeski, 1988; Gauvin et al., 1996; Simons & Birkimer, 1988) with low baseline PAA resulting in higher post-exercise PAA (e.g., Annesi & Unruh, 2008) and high baseline PAA associated with small to negligible effects (e.g., Mack, Huddleston, & Dutler, 2000; Nagy & Frazier, 1988). Methodological factors, however, may be partly responsible for the mixed literature. For example, some studies tested the relation by dichotomizing responses using a median split (e.g., Annesi, 2002a; Brown et al., 2001) resulting in a loss of statistical power and an underestimation of the effect (Cohen, 1983; MacCallum, Zhang, Preacher, & Rucker, 2002). Reed and Ones (2006) found an inverse relation between initial scores and post-exercise PAA change for acute studies, a result supporting the law of initial value (LIV; Wilder, 1967). Thus, the literature leads to a tentative hypothesis of an inverse relation between baseline PAA and the magnitude of improvement from an aerobic exercise program.

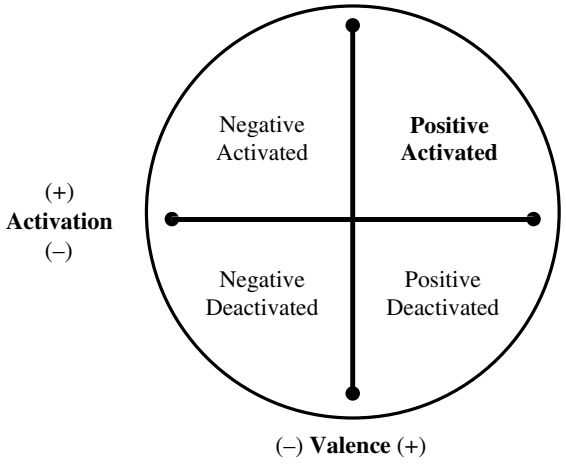

Fig. 1. A circumplex model of self-reported affect. Adapted from Yik et al. (1999).

Exercise frequency

In quantitative reviews of self-reported depression neither North et al. (1990) nor Craft and Landers (1998) found a relation between the number of sessions per week and effect size. However, North et al. found a positive relation between the total number of sessions and the magnitude of decrease in reported depression suggesting an effect for program duration. For self-reported anxiety (Long & Van Stavel, 1995) and affect (Arent, Landers, & Etnier, 2000), exercise vs. control group results support greater affective improvement in studies with frequencies less than three compared to greater than three days per week, although for Long and Stavel this result was reversed for the within-group analyses. Individual studies of PAA also show mixed results. Vigor and energy often improve in studies examining frequencies of 1 or 2 days per week (e.g., Lox & Treasure, 2000; Moore & Bracegirdle, 1994), but in other studies, improvement is not different from controls (e.g., Simons & Birkimer, 1988). Similarly, at 5 days per week, positive affect has been shown to improve relative to controls (e.g., Osei-Tutu & Campagna, 2005), but others have found no differences (e.g., Cramer, Nieman, & Lee, 1991; Nieman, Custer, Butterworth, Utter, & Henson, 2000). Given the equivocal state of the literature, we tentatively suggest that frequency per week will not moderate the effect of aerobic exercise on PAA.

Exercise intensity

Narrative and quantitative reviews of acute exercise support an inverse relation between intensity and post-exercise PAA (Ekkekakis & Petruzzello, 1999; Reed & Ones, 2006). Strenuous exercise generally decreases (e.g., Pronk, Crouse, & Rohack, 1995) and light to moderate exercise increases PAA (e.g., Ekkekakis et al., 2000; Thayer, 1996). However, strenuous exercise may increase PAA by way of a post-exercise rebound (Bixby, Spalding, & Hatfield, 2001; Solomon, 1980). In addition, fitness (Ekkekakis, 2003; Ekkekakis & Petruzzello, 1999) and exercise experience (Hsiao & Thayer, 1998) appear to interact with higher intensity exercise such that greater fitness and experience increase the magnitude and duration of post-exercise PAA compared to less fit participants. Greater fitness and aerobic capacity imply workload adjustments and assuming adjustments are part of a training study one would expect positive changes in post-exercise PAA. Most studies use a relative gauge of intensity, such as percent maximum heart rate, so workloads are adjusted to maintain the same relative intensity as fitness

improves. However, the question with respect to intensity as a moderator of post-exercise PAA in exercise training studies is whether a comparison of studies employing different relative intensities result in appreciable differences in PAA from baseline to post-program. For example, given the results of the acute literature, one might expect an inverse relation between intensity and PAA. On the other hand, higher relative intensities, because of the greater effect on fitness, may impart PAA improvements over time equal to or greater than those for lower intensities. Because of the difficulty in understanding how the interaction of exercise intensity and fitness influence post-exercise PAA *over time*, we hypothesize that different intensity levels will produce differences in PAA but we cannot predict the direction of this change.

Exercise time

Individual studies (e.g., Petruzzello & Landers, 1994) and quantitative reviews of negative (e.g., North et al., 1990; Petruzzello et al., 1991) and positive affect (Reed & Ones, 2006) show little moderating effect of exercise time on pre to post-affective change. Although Berger and Motl (2000) proposed 20-min as a threshold duration for PAA improvement, the bulk of the literature does not support exercise time as a consistent moderator, because mixed results are found across the range of typical session times of 15–45 min. For example, small to negligible effects are found for less than 30 min (e.g., Polis, 1989), 30 min (e.g., Nagy & Frazier, 1988) and greater than 30 min (e.g., Lesniak, 1990). Likewise, many studies show large effects for the same range: less than 30 min (e.g., Steptoe, Edwards, Moses, & Mathews, 1989), 30 min (e.g., Annesi, 2002a), and greater than 30 min (Tsai et al., 2004). Given the mixed literature, we hypothesize that exercise time within the context of a regular exercise program will not moderate PAA.

Program duration

The literature supports program duration as a moderator of the effect of regular exercise on self-reported anxiety (Petruzzello et al., 1991) and depression (Craft & Landers, 1998; North et al., 1990) with larger effects for programs at least nine or ten weeks in length. However, the effect of program duration on changes in PAA is less clear. A quantitative review of regular exercise on feelings of energy and fatigue found no association between program duration and effect size (Puetz, O'Connor, & Dishman, 2006) although effect sizes in this analysis reflected changes in energy and fatigue so the effect of program duration on PAA independent of fatigue remains untested. Cross-sectional data indicate differential affective responses between beginners and experienced exercisers (Hsiao & Thayer, 1998), implicating program duration as a moderator, but the results of experimental studies are mixed. For example, in programs less than 10 weeks, some studies show effects (e.g., Partonen, Leppamaki, Hurme, & Lonnqvist, 1998) while others do not (e.g., Steptoe, Kimbell, & Basford, 1998). Longer programs show a similar pattern: some studies show changes (e.g., Annesi, 2002a; DiLorenzo et al., 1999), but others do not (e.g., Nagy & Frazier, 1988). Additionally, Steinberg et al. (1998) assessed the acute effects of exercise once a week for six weeks and found that exercise reliably increased positive affect, but the magnitude of the pre to post-exercise change remained the same from week-to-week. These results are important because they point to the notion that exercise improves PAA by "reinstating" positive affect from week-to-week and suggest that the pre to post-program change would be similar regardless of the length of the program. Thus, we hypothesize that studies of different durations will not reliably alter the magnitude of the effect of aerobic exercise on PAA.

Exercise dose

Acute studies employing lower doses (e.g., Ekkekakis et al., 2000) generally find affective benefits while those testing extreme bouts do not (e.g., Acevedo, Gill, Goldfarb, & Boyer, 1996) and meta-analytical evidence from the acute literature supports dose as a moderator of PAA when dose is evaluated as a function of intensity and exercise time (Reed & Ones, 2006). Conversely, in one of the few studies to examine the chronic effects of dose, Blanchard, Rodgers, Wilson, and Bell (2004) compared two equal-dose exercise prescriptions each with a different blend of intensity and duration and found improved affect in both protocols but no difference between them. These results indicate that equal duration programs (e.g., 12 weeks) with equal doses produce similar improvement regardless of acute exercise-related variables. There are no data comparing programs of different durations employing various combinations of exercise frequency, intensity and time. Given the results of Blanchard et al., however, and our expectation regarding program duration effects, we tentatively hypothesize that dose will not moderate post-exercise PAA when examined as a function of acute variables and program duration.

Study quality and source

Several authors have argued that methodologically inferior studies should not be included in quantitative reviews (Eysenck, 1994; Slavin, 1986), but others are not of this opinion (Glass, 1983; Hunter & Schmidt, 2004) noting that journal editors tend to accept methodologically superior studies and in turn exclude poorer quality studies which can produce biased meta-analytical results (Cook et al., 1992; Greenland, 1998). Meta-analyses of exercise and negative affect have found either larger effects for studies with moderate levels of internal validity (North et al., 1990), or have failed to find differences between validity levels (Long & Van-Stavel, 1995; Petruzzello et al., 1991). The PAA literature appears more consistent in that generally studies with low internal validity show smaller PAA changes (e.g., Mack et al., 2000; Simons & Birkimer, 1988) compared to studies controlling more threats to validity (e.g., Brown et al., 2001; Cramer et al., 1991) and the acute exercise and PAA literature shows a similar pattern (Reed & Ones, 2006). Therefore, we hypothesize a positive association between study quality and effect size (ES).

Source is often analyzed relative to publication bias defined as the inclination for editors and reviewers to favor studies with positive or "significant" results and reject "non-significant" studies. The result is a biased literature producing inaccurate meta-analytical results, even when a relatively small number of unpublished studies are excluded from the analysis (Scargle, 2000). Publication bias has been found in several fields including medicine (Sutton, Duval, Tweedie Abrams, & Jones, 2000) and psychology (Baldwin, 2007). Within the exercise-affect literature, some authors have found evidence for publication bias (Craft & Landers, 1998; North et al., 1990; Petruzzello et al., 1991), while others have not (Arent et al., 2000; Long & Van-Stavel, 1995). The PAA literature appears more consistent with published research typically showing improvement (e.g., Osei-Tutu & Campagna, 2005) and unpublished studies often yielding small changes (e.g., Jewell, 1987; Lesniak, 1990). Reed and Ones (2006) also provide good evidence for a source bias specific to the acute PAA literature as they found larger effects for published studies. We therefore hypothesize published studies will yield larger effects.

Method

Description of the database

Searches were performed for studies on aerobic exercise and mood or affect to include activities such as walking, jogging, running, swimming, and cycling. Relevant studies from January 1980 to December 2008 were identified using computer databases (*Dissertation Abstracts International*, ERIC, *Medline*, *PsychINFO*, Pub Med, *SPORTDiscus*, and *World Cat*), searches of narrative and quantitative reviews, and books (e.g., Seraganian, 1993). Reference lists from published articles, theses, and colleagues were examined for additional studies. Non-English studies were translated if possible. Authors of studies with missing data were contacted. Twenty authors were contacted and twelve responded, the others either did not respond or could not be located. Of those who responded, four provided the requested information.

Studies assessing affect with scales representative of PAA were included. The majority of scales employed a "right now" or "one week" time set: 62.25% and 16.85% of the samples, respectively. Time set was not reported in 20.90% of the samples. To avoid confounding separate constructs, we did not include scales assessing positive "deactivated" affect (lower right quadrant in Fig. 1). Table 1 displays the instruments and subscales in the meta-analysis.

Table 1
Instruments, subscales and effect sizes (ESs) contributing to the meta-analysis.

Instrument (reference)	Subscale	ESs
Affect balance (Bradburn, 1969)	Positive affect	4
Affectometer (Kammann & Flett, 1983)	Positive affect	4
Benfindlichkeitsskalen (BFS) (Abele & Brehm, 1986)	Liveliness	1
Emotional Assessment Scale (EAS) (Carlson et al., 1989)	Happiness	4
Exercise-Induced Feeling Inventory (EFI) (Gauvin & Rejeski, 1993)	Positive engagement revitalization	7
General Well-Being Scale (GWBS) (McDowell & Newell, 1996)	Positive well-being	24
Medical Outcomes Study Short Form (MOS SF-36) (Ware et al., 1994)	Vitality	31
Mood Adjective Checklist (MACL) (Nowlis, 1965)	Pleasantness activation	6
Physical Activity Affect Scale (PAAS) (Lox et al., 2000)	Positive affect	1
Positive and Negative Affect Schedule (PANAS) (Watson et al., 1988)	Positive affect	26
Profile of Mood States Bipolar (POMS-BI) (Lorr & McNair, 1988)	Energetic-tired	4
Profile of Mood States Long Form (POMS-LF) (McNair et al., 1992)	Vigor	185
Profile of Mood States Short Form (POMS-SF) (McNair et al., 1992)	Vigor	29
RAND 36-Item Health Survey (Hays et al., 1993)	Energy	13
Subjective Exercise Experiences Scale (SEES) (McAuley & Courneya, 1994)	Positive well-being	4
Thoughts and Real-Life Experiences Scale (THARL) (Dua & Price, 1992)	Thought-related positive affect	2

Note. Affect balance, affectometer, and POMS-Bi were scored using bipolar format. We assumed that bipolar terms acted as reciprocal pairs such that a decrease in unpleasant deactivated states resulted in a corresponding increase in pleasant activated states producing comparable ESs. Bipolar scored ESs comprised 3.2% of the ESs in the database.

The MOS 36-Item Health Survey and the RAND 36-Item Health Survey are very similar, but scored differently. Therefore, separate ES totals and instrument listings are shown.

There were seven affect scales developed by the researcher specifically for the affective aspect of their study. In each case, we examined and deemed the scale content appropriate to assessing to PAA. Researcher developed scales comprised 6.75% of the ESs in the database.

The psychometric studies of Yik, Russell, and Feldman Barrett (1999) provide an empirical backdrop for the inclusion of the affect subscales in Table 1. Yik et al., with structural equation modeling found that Feldman Barrett and Russell's Activated (Feldman Barrett & Russell, 1998), Watson, Clark, and Tellegen's Positive Affect (Watson, Clark, & Tellegen, 1988), Thayer's Energy (Thayer, 1986), and Larsen and Diener's Activated Pleasant (Larsen & Diener, 1992), all fell within the quadrant of the affect circumplex we call PAA (see Yik et al., Fig. 6). We determined the number of affect terms from the subscales in Yik et al. analysis that matched words from the subscales in Table 1: Affect Balance (Bradburn, 1969), 4 of 6, Benfindlichkeitsskalen (BFS; Abele & Brehm, 1986), 3 of 5, Emotional Assessment Scale (EAS; Carlson et al., 1989), 2 of 3, Exercise-Induced Feeling Inventory (EFI; Gauvin & Rejeski, 1993), 3 of 6, General Well-Being Schedule (GWBS; McDowell & Newell, 1996), 2 of 3, Medical Outcomes Study Short Form (MOS SF-36; Ware, Kosinski, & Keller, 1994), 4 of 4, Mood Adjective Checklist (MACL; Nowlis, 1965), 7 of 8, Physical Activity Affect Scale (PAAS; Lox, Jackson, Tuholski, Wasley, & Treasure, 2000), 2 of 3, Positive and Negative Affect Schedule (PANAS; Watson et al., 1988), 10 of 10, Profile of Mood States Bipolar (POMS-Bi; Lorr & McNair, 1988), 5 of 7, Profile of Mood States Long Form (POMS-LF; McNair, Lorr, & Droppleman, 1992), 6 of 8, Profile of Mood States Short Form (POMS-SF; McNair et al., 1992), 5 of 5, RAND 36-Item Health Survey (Hays, Sherbourne, & Mazel, 1993), 4 of 4, Subjective Exercise Experiences Scale (SEES; McAuley & Courneya, 1994), 1 of 4, Thoughts and Real-Life Experiences Scale (THARL; Dua & Price, 1992), 2 of 3.

Two scales require comment: EFI and SEES. First, the EFI terms *upbeat*, *refreshed*, and *revived* were not matches with our criterion word set, but they appear to have PAA qualities because the Positive Engagement (PE) and Revitalization subscales correlate with PANAS Positive Affect (PA): $r = .69$ ($p < .001$) and $r = .58$ ($p < .001$), respectively (see Gauvin & Rejeski, 1993). In addition, Rejeski, Reboussin, Dunn, King, and Sallis (1999) argue for the inclusion of these subscales in the PAA quadrant (see Rejeski et al., 1999, p. 98). Second, although the SEES terms *great*, *positive*, and *terrific* were not matches, we believe they qualify as positively activated because McAuley and Courneya (1994) reported a significant correlation between SEES Positive Well-Being (PWB) and PANAS PA: $r = .71$, ($p < .01$). Finally, Lox et al. (2000) found significant correlations between the SEES PWB and EFI Revitalization ($r = .81$, $p < .01$), SEES PWB and EFI PE ($r = .78$, $p < .01$), and between the EFI Revitalization and PE ($r = .86$, $p < .01$) indicating a considerable amount of common variability between these subscales and suggesting a common underlying construct. Thus, based on logical, semantic, and statistical grounds, we believe the subscales in Table 1 are representative of PAA.

We excluded studies with potential confounders such as counseling (e.g., Oldridge, Streiner, Hoffmann, & Guyatt, 1995), or psychotherapy (e.g., Soloff, 1978) either in conjunction with exercise sessions or before post-program affect assessment. Only the aerobic exercise groups were included for studies with different exercise interventions. Studies with similar authors were evaluated for sample overlap and when overlap occurred, studies with more complete data were included. Duplicate articles were excluded. Table 2 provides a summary of the article elimination process.

The database contained 105 studies, 370 ESs, and 9840 participants. Study year ranged from 1980 to 2008 ($M = 1994$, SD = 6.98). Gender was reported in 97.50% of the studies. Males comprised 13.5%, females, 42.75%, and mixed gender 43.75% (mixed defined as <75% of either gender). Age was reported in 97.32% of the studies ($M = 42.41$, SD = 15.93). Samples included participants from the community (58.03%), clinical groups (21.42%), college students (12.50%), mixed samples of faculty, staff, and students (4.46%) and

Table 2
Summary of article elimination procedure.

Procedure/item	Removed	Remaining
Initial search	0	757
Exclusion item		
Title or abstract (e.g., acute study or athlete profiling)	518	239
Could not determine exercise type	9	230
Could not separate acute and chronic results	1	229
Dependent *t* test (no pre–post-correlation)	2	227
Confounded exercise treatment	8	219
PAA not reported	50	169
Exercise combination (aerobic + another activity)	17	152
Resistance training only	11	141
Redundant with another publication	11	130
Lack of data for ES calculation	25	105

athletes (1.8%). Most of the research was conducted in the U.S. (78.57%).

Study coding

Baseline PAA

Because researchers have utilized a number of different affect scales, we computed *z*-scores for all pre-study affect values as a method of standardizing this variable for the moderator analysis. Details of this procedure can be found in Reed and Ones (2006).

Exercise characteristics

Frequency was coded as days per week (days/wk). Intensity was coded using percent oxygen uptake reserve (%VO_2R), a relative index that allows consistent coding across different indices such as percent maximum oxygen uptake (%VO_{2max}), percent heart rate maximum (% HR_{max}), or perceived exertion (Howley, 2001). The data allowed for categories of low (~30% VO_2R), moderate (~50% VO_2R), and high (~70% VO_2R). We converted %VO_{2max} to %VO_2R when needed using appropriate equations (Swain & Leutholtz, 1997; Swain, Leutholtz, King, Haas, & Branch, 1998). We coded exercise time in minutes, excluding warm-up and cool-down. Program duration was coded as weeks (wks) resulting in groups of 4–9, 10–12, and 13–32 wks. We quantified dose using the product of acute (intensity, time) and chronic (frequency, program duration) variables. That is, dose = intensity × time × frequency × program duration. Gaps in dose values produced three levels with the following average characteristics: (a) low: 2 days/wk, 20 min, moderate intensity, 8 wks, (b) moderate: 3 days/wk, 30 min, moderate intensity, 11 wks, (c) high, 4 days/wk, 40 min, moderate intensity, 13 wks.

Study quality and source

Threats to internal validity that authors attempted to control were coded with a higher number indicating greater internal validity and study quality. We considered: history, maturation, testing, statistical regression, selection bias, experimental mortality, compensatory rivalry, resentful demoralization, Hawthorne effect, demand characteristics, halo effect, and expectancy effects. Source (publication bias) was coded as an unpublished master's thesis or doctoral dissertation, or as a published journal article or abstract.

Calculation of ESs

The pooled standard deviation (SD_p) was used to calculate ESs, resulting in Cohen's *d* statistic (Cohen, 1977). Each ES (*d*-value) was weighted by study sample size and corrected for affect scale measurement error using reliability data from the study, a validation study, or test manual. Because the data contained studies ranging from 4 to 32 weeks, we developed a formula using a test–retest reliability decay curve (Kammann & Flett, 1983) to estimate the test–retest decay for alpha coefficients ranging from .70 to .99. For example, we entered a test–retest reliability of .64 for a 12-week study that used a scale with an alpha of .96. Further corrections were made for small sample bias, unequal samples, and treatment by subject interaction (Hunter & Schmidt, 2004, pp. 266, 279, 282).

The following four types of ESs were calculated: a) pre-study exercise vs. pre-study control, b) pre–post-exercise, c) pre–post-control, and d) post-study exercise vs. post-study control. A separate meta-analysis was performed for each type of ES. Descriptive statistics for the test–retest reliabilities used in these four meta-analyses were: pre-exercise vs. pre-control (Mr_{yy} = .89, SDr_{yy} = .03); pre–post-control (Mr_{yy} = .62, SDr_{yy} = .05); pre–post-exercise (Mr_{yy} = .62, SDr_{yy} = .04); post-exercise vs. post-control (Mr_{yy} = .63, SDr_{yy} = .06) where Mr_{yy} = average reliability and SDr_{yy} = reliability standard deviation, respectively. ESs based on paired *t*-tests without appropriate *r*-values were not included because paired-sample data without corresponding correlations results in an upward bias of the effect (Dunlap, Cortina, Vaslow, & Burke, 1996).

An important feature of meta-analysis is the calculation of the sampling error of the *d* statistic, in particular, if the literature contains within and between-group study designs (as is the case with the PAA and exercise literature), a different sampling error formula must be used for each design. Hunter and Schmidt provide the appropriate within and between-group sampling error formulas (Hunter & Schmidt, 2004, p. 305, 370). Therefore, for each meta-analysis, the sampling error was calculated separately for within and between-group ESs.

Coder reliability

To estimate coder drift (Orwin, 1994), the first author recoded 10 randomly selected studies 2 weeks after the coding phase. A per-item agreement rate was calculated (number of items coded the same divided by the total number of items coded). To avoid overestimating coder reliability, items prone to error such as moderators, samples sizes, and ESs were coded while information not prone to error such as publication dates was not included (Kuncel, Hezlett, & Ones, 2001). The agreement rate was 98.93% (925 agreements for 935 items). The categories and number of discrepancies were: study design level (3), fitness level (4), exercise intensity (1), sample size (1), and reliability (1). Thus, coding errors were negligible.

Analysis

We used a random effects model (Hunter & Schmidt, 1990, 2004) and SAS software (Version 9.1.3) to analyze the data. Random effects models are preferred to fixed-effects because varying population effects appear to be the rule rather than the exception for most real-world data (Field, 2003). The SD of population ESs (SD_{corr}) is an important statistic in random effects models (Hunter & Schmidt, 1990, p. 453). However, outliers inflate the ES variance above that predicted by sampling error and other artifacts (Schmidt et al., 1993) resulting in overestimation of SD_{corr}. Therefore, we employed Tukey's (1977) method using a SAS macro (Cody, 1999) to identify and omit outlier ESs prior to each meta-analysis.

Meta-analyses

The following were computed for all meta-analyses: total sample size (N), number of ESs (K), mean sample-size weighted observed ES ($\overline{d}_{obs}$), $\overline{d}_{obs}$ 95% confidence interval (95% CI), mean sample-size weighted corrected ES ($\overline{d}_{corr}$), corrected standard deviation (SD_{corr}), residual standard deviation (SD_{res}), percent of d_{obs} variance due to sampling error (%Var_e), 90% credibility interval (90% CrI), and fail-safe N (N_{fs}). The 95% CI approximates the sampling error in $\overline{d}_{obs}$ while $\overline{d}_{corr}$ and SD_{corr} are the population parameter estimates. When the 90% CrI includes zero $\overline{d}_{corr}$ is the mean of several parameters indicating the presence of moderators (Whitener, 1990); otherwise, ESs generalize across settings (Ones, Viswesvaran, & Schmidt, 1993). The N_{fs} equation is based on a random effects model and estimates the number of unlocated studies with null results needed to reduce $\overline{d}_{corr}$ to the lowest ES considered practically or theoretically important (Hunter & Schmidt, 2004, p. 500; Orwin, 1983). This lowest meaningful ES is the $\overline{d}_{critical}$ value in the N_{fs} formula. We set $\overline{d}_{critical} = .20$ based on the scale of magnitudes for r-values developed by Cohen (1988) and Hopkins (2002). Cohen and Hopkins classified an r of .10 as a logical benchmark for the smallest meaningful association between two variables. The correlation can be converted to a d-value with Friedman's (1968) formula: $d = 2r/[(1 - r^2)^{1/2}]$ and when $r = .10$, $d = .20$. Therefore, we set $\overline{d}_{critical} = .20$ for the N_{fs} analysis.

Overall meta-analyses

These meta-analyses are based on the four ES types. The first compared the pre-study exercise and control groups where positive ESs indicated greater PAA in exercise samples. We tested pre-study equivalence because large group differences may confound conclusions about exercise-related moderators. The second tested pre–post-changes for control samples with positive ESs reflecting increased PAA relative to baseline. Attention controls (e.g., wait list) comprised 83% and activity controls (e.g., flexibility) were 17% of the control ESs, respectively. The third examined pre–post-changes for exercise samples with positive ESs reflecting increased PAA relative to baseline. The fourth compared post-study exercise and post-study control group values to examine the potential effect of demand characteristics on exercise-related PAA (Long & Van-Stavel, 1995). For example, pre-study equivalence and no control effect should produce similar results for pre–post-exercise effects and post-study exercise vs. control group effects thus strengthening the notion that increased PAA is largely due to exercise interventions. Alternatively, improved pre–post-control PAA should result in smaller post-study exercise vs. post-study control effects relative to pre–post-exercise changes, a result suggesting that pre–post-exercise effects are in part due to demand characteristics or some other confounding factor. Finally, six studies gathered follow-up data ranging from three to six months post-intervention. Unfortunately, post-intervention exercise was monitored in only two studies and therefore we elected not to analyze follow-up results.

Moderator analyses

These analyses were performed using the pre–post-exercise database. Given an overall pre–post-exercise 90% CrI that included zero (or a sufficiently wide 90% CrI) moderators and moderator subgroups were formed. First, we examined moderator correlations for unexpectedly high relationships that might confound interpretation of the results. Next, each moderator was explored by examining $\overline{d}_{corr}$ subgroup differences and changes in the SD_{corr} across subgroups (Hunter & Schmidt, 2004, p. 293). However, Hunter and Schmidt (2004) do not provide guidelines on the size of subgroup differences with respect to the strength of the moderator. They consider a .20-subgroup difference "wide" (p. 298) and characteristic of a moderator effect although we consider a .20 difference less convincing. Therefore, we developed the following $\overline{d}_{corr}$ subgroup difference scale to assess moderator strength: <.10, trivial; .10–.19, weak; .20–.39, moderate; .40–.59, strong; ≥.60, very strong. For analyses with more than 2 subgroups, moderator strength was judged using the largest subgroup difference. Finally, we used 90% CrI values to determine the generalizability of a subgroup effect or to suggest the presence of additional unexamined moderators.

Results

Overall meta-analyses

Pre-study exercise vs. control group differences were negligible, $\overline{d}_{corr} = -.02$ ($SD_{corr} = .20$). The pre–post-control analysis showed little change, $\overline{d}_{corr} = .03$ ($SD_{corr} = .11$) and since %$Var_e \sim 90\%$, the remaining variance is likely due to additional uncorrected artifacts (Hunter & Schmidt, 2004), leaving the control group effect hovering around zero. Pre–post-exercise changes were moderate and positive $\overline{d}_{corr} = .57$ ($SD_{corr} = .48$), but SD_{corr} and 90% CrI suggest the presence of moderators. The post-study exercise vs. post-study control effect was in line with the negligible control group effect and the magnitude of exercise-related change. That is, given no pre-study differences and little change in control group responses, one would expect similar pre–post-exercise and post-study exercise vs. control effects. Fail-safe Ns suggest good tolerance to availability bias. For example, 213 ESs with $\overline{d}_{corr} = .00$ need to be included to reduce the pre–post-exercise $\overline{d}_{corr}$ to .20. In sum, relative to control groups, aerobic exercise programs produce moderate improvements in self-reported PAA. Results are presented in Table 3.

Moderator analyses

Moderator correlations

Nearly all inter-moderator correlations ranged from −.15 to .15, indicating moderators were unrelated. As expected, however, exercise dose was moderately correlated with the variables comprising it: exercise frequency, intensity, time, and program duration. Only baseline PAA and threats controlled had adequate associations with d_{corr} (−.43 and .32, respectively), suggesting moderator effects. Table 4 displays results for baseline PAA and exercise characteristics and Table 5 shows results for study quality and source.

Baseline PAA

Results support the hypothesis of larger effects for lower baseline PAA. Participant groups in the lower third of the baseline distribution recorded a $\overline{d}_{corr} = .81$ ($SD_{corr} = .40$), nearly twice the magnitude of the middle category (−.5z to .5z) and three times higher than groups in the upper third of the baseline distribution ($\overline{d}_{corr} = .26$, $SD_{corr} = .02$). The large subgroup difference of .55 indicates a strong moderator effect. None of the 90% CrI included zero, indicating generalizable increases in PAA, although the lower bound estimate for the middle subgroup is close to zero, suggesting that effects may be negligible in some instances. The findings show that when baseline affect is below average, aerobic exercise programs result in consistent, robust, generalizable increases in self-reported PAA.

Exercise characteristics

Contrary to expectation, frequency produced a subscale difference of .27 between 3 days/wk and >3 days/wk and therefore appears to moderate PAA. This subscale difference, however, should

Table 3
Overall meta-analyses.

Analysis	*N*	*K*	$\overline{d}_{obs}$	95% CI	$\overline{d}_{corr}$	SD_{corr}	SD_{res}	$\%Var_e$	90% CrI	N_{fs}
Pre-study: exercise vs. control	4508	75	−.02	−.10 to .06	**−.02**	.20	.19	69.32	−.27 to.23	83
Pre–post-control	1767	67	.03	−.05 to .10	**.03**	.11	.10	89.08	−.11 to.17	55
Pre–post-exercise	3374	115	.49	.40 to .58	**.57**	.48	.41	38.37	−.04 to 1.18	213
Post-study: exercise vs. control	9542	103	.51	.43 to .59	**.60**	.39	.33	42.42	.10 to 1.09	206

Note. *N* = total sample size; *K* = number of ESs; $\overline{d}_{obs}$ = mean sample-size weighted observed ES; 95% CI = $\overline{d}_{obs}$ 95% confidence interval; $\overline{d}_{corr}$ = mean sample-size weighted corrected ES; SD_{corr} = sample-sized weighted corrected standard deviation; SD_{res} = residual standard deviation; $\%Var_e$ = percent of d_{obs} variance due to sampling and measurement error; 90% CrI = 90% credibility interval; N_{fs} = $\overline{d}_{corr}$ fail-safe *N* with $\overline{d}_{critical}$ = .20,$\overline{d}_{unlocated}$ = .00 for $\overline{d}_{corr}$ values above .20 and $\overline{d}_{unlocated}$ = .40 for $\overline{d}_{corr}$ values below .20. Boldface entries are best estimates of the population mean ES.

be interpreted with caution as *K*-values of 20 and 18 are smaller than optimal and may result in greater sampling error and less stable ES estimates. Frequencies > 3 days/wk produced the largest increases ($\overline{d}_{corr}$ = .79, SD_{corr} = .34), with effects generalizing across settings (90% CrI: .35–1.23). Intensity produced moderate-size $\overline{d}_{corr}$ differences across subcategories, partially supporting our expectation. Low intensities resulted in the greatest increases ($\overline{d}_{corr}$ = .72, SD_{corr} = .00) although the ES estimates associated with *K*-values of 16 and 20 merit caution. Exercise time appears to be a stable, but weak moderator with fail-safe (N_{fs}) values of 50–87 and a maximum subgroup difference of .19. However, all subcategory effects were moderate and generalize across settings. Program duration was a weak, but stable moderator, results contrary to our hypothesis. While 10–12-week programs yielded the greatest improvements ($\overline{d}_{corr}$ = .63, SD_{corr} = .42), even 4–6-week programs appear to produce moderate increases. Exercise dose was a trivial moderator in line with the hypothesis (subgroup differences < .10). Dose subgroup effects were moderate, generalizable, and stable with respect to N_{fs}. Finally, the moderate dose lower 90% CrI value was close to zero, indicating that while not likely to be negative, effects may be small and dependent on unexamined moderators.

Study quality and source

A positive association appeared for threats controlled indicating greater effects in studies having higher internal validity, supporting our expectation. The $\overline{d}_{corr}$ subgroup differences show a strong moderating effect and a threshold for greater effects starting with studies having more than 5 threats controlled. Results were stable except for the first subgroup (N_{fs} = 8) and only the second subcategory (4–5) was not clearly generalizable. Contrary to the hypothesis, the $\overline{d}_{corr}$ for published studies of .56 (SD_{corr} = .36) was

Table 4
Moderator analyses for baseline Paa and exercise characteristics.

Analysis	*N*	*K*	$\overline{d}_{obs}$	95% CI	$\overline{d}_{corr}$	SD_{corr}	SD_{res}	$\%Var_e$	90% CrI	N_{fs}
Baseline PAA										
<−.5*z*	1171	35	.68	.54–.84	**.81**	.40	.34	43.34	.29–1.32	106
−.5*z* to .5*z*	1125	38	.38	.26–.51	**.45**	.34	.29	48.45	.01–.88	47
> .5*z*	592	24	.21	.11– .32	**.26**	.02	.02	99.32	.23–.29	8
Not reported	268	8	.39	.23–.55	**.47**	.01	.01	99.52	.46–.48	11
Exercise frequency										
<3 days/wk	713	20	.49	.33–.65	**.57**	.29	.25	51.89	.20–.94	37
3 days/wk	1776	61	.43	.32–.54	**.52**	.37	.31	49.80	.05–.99	97
>3 days/wk	415	18	.68	.47–.88	**.79**	.34	.29	56.41	.35–1.23	53
Not reported	200	3	.07	−.02–.15	**.08**	.00	.00	100.00	.08–.08	2
Exercise intensity[a]										
Low	483	16	.61	.48–.73	**.72**	.00	.00	100.00	.72–.72	42
Moderate	1455	56	.42	.29–.55	**.50**	.46	.39	40.20	−.09 to 1.09	84
High	645	20	.58	.40–.76	**.68**	.34	.29	49.87	.24–1.12	48
Not reported	618	17	.34	.17–.51	**.40**	.30	.26	48.00	.01–.79	17
Exercise time										
15–25 min	504	30	.47	.28–.66	**.55**	.38	.33	60.80	.06–1.04	53
30–35 min	1051	36	.57	.42–.72	**.68**	.43	.36	41.70	.13–1.23	87
40–60 min	1327	34	.42	.28–.55	**.49**	.37	.31	38.33	.02–.97	50
Not reported	256	6	.31	.16–.46	**.36**	.00	.00	100.00	.36–.36	5
Program duration										
4–9 wks	995	30	.45	.33–.58	**.51**	.28	.25	49.07	.15–.88	47
10–12 wks	1208	45	.54	.40–.69	**.63**	.42	.36	44.67	.10–1.17	97
13–32 wks	879	27	.35	.21–.49	**.45**	.28	.22	65.98	.09–.81	34
Exercise dose										
Low	688	31	.52	.37–.66	**.60**	.28	.25	63.98	.23–.96	61
Moderate	808	28	.49	.30–.68	**.56**	.40	.35	41.91	.05–1.07	50
High	997	29	.52	.37–.68	**.65**	.38	.31	46.26	.16–1.14	66
Not reported	645	18	.31	.14–.49	**.36**	.33	.29	42.70	−.06–.78	14

Note. *N* = total sample size; *K* = number of ESs; $\overline{d}_{obs}$ = mean sample-size weighted observed ES; 95% CI = $\overline{d}_{obs}$ 95% confidence interval; $\overline{d}_{corr}$ = mean sample-size weighted corrected ES; SD_{corr} = sample-sized weighted corrected standard deviation; SD_{res} = residual standard deviation; $\%Var_e$ = percent of d_{obs} variance due to sampling and measurement error; 90% CrI = 90% credibility interval; N_{fs} = $\overline{d}_{corr}$ fail-safe *N* with $\overline{d}_{critical}$ = .20, $\overline{d}_{unlocated}$ = .00 for $\overline{d}_{corr}$ values above .20 and $\overline{d}_{unlocated}$ = .40 for $\overline{d}_{corr}$ values below .20. Boldface entries are best estimates of the population mean ES.

[a] Exercise intensity: low ~ 30% VO_2R, moderate ~ 50% VO_2R, High ~ 70% VO_2R.

Table 5
Moderator analyses for study quality and source.

Analysis	*N*	*K*	$\overline{d}_{obs}$	95% CI	$\overline{d}_{corr}$	SD_{corr}	SD_{res}	$\%Var_e$	90% CrI	N_{fs}
Threats controlled										
1–3	650	23	.22	.11–.33	**.27**	.00	.00	100.00	.27–.27	8
4–5	1249	33	.37	.23–.51	**.43**	.37	.31	39.74	−.04–.90	38
6–7	951	38	.68	.54–.83	**.80**	.37	.31	53.19	.33–1.27	114
8–9	202	10	.62	.44–.81	**.77**	.00	.00	100.00	.77–.77	29
Source										
Unpublished	1035	45	.47	.33–.62	**.54**	.43	.34	45.74	.04–1.04	76
Published	2103	61	.47	.36–.57	**.56**	.36	.31	43.25	.09–1.03	109

Note. *N* = total sample size; *K* = number of ESs; $\overline{d}_{obs}$ = mean sample-size weighted observed ES; 95% CI = $\overline{d}_{obs}$ 95% confidence interval; $\overline{d}_{corr}$ = mean sample-size weighted corrected ES; SD_{corr} = sample-sized weighted corrected standard deviation; SD_{res} = residual standard deviation; $\%Var_e$ = percent of d_{obs} variance due to sampling and measurement error; 90% CrI = 90% credibility interval; N_{fs} = $\overline{d}_{corr}$ fail-safe *N* with $\overline{d}_{critical}$ = .20, $\overline{d}_{unlocated}$ = .00 for $\overline{d}_{corr}$ values above .20 and $\overline{d}_{unlocated}$ = .40 for $\overline{d}_{corr}$ values below .20. Boldface entries are best estimates of the population mean ES.

only .02 SDs greater than for unpublished studies ($\overline{d}_{corr}$ = .54, SD_{corr} = .43). Source effects were stable and generalizable, although the 90% CrI approached zero for both subgroups indicating that effects are trivial in some settings. The weak correlation between threats controlled and source (r = −.09) shows that published and unpublished samples each contained studies that controlled a range of threats to internal validity.

Discussion

The overall results indicate a) pre-study samples report similar PAA, b) control conditions leave PAA unchanged, c) aerobic exercise programs increase PAA, and d) post-study exercise vs. post-study control differences are similar to pre–post-exercise effects. The findings provide support for the favorable effect of regular aerobic exercise on PAA and are comparable to those found in meta-analyses for acute exercise (Reed & Ones, 2006).

Baseline PAA

Studies with participants having pre-study PAA in the lower third of the distribution were associated with larger increases in PAA than those in the other groups, supporting our hypothesis and in line with the literature (North et al., 1990; Petruzzello et al., 1991; Puetz et al., 2006; Reed & Ones, 2006). An implication of this finding is the use of exercise as a strategy to improve feelings of energy and positive affect (Thayer, 1996). However, it is unclear whether such a strategy can prevent affective disturbances as recent data on identical twins indicate that increased exercise behavior does not predict changes in anxiety and depression (De Moor, Boomsma, Stubbe, Willemsen, & deGeus, 2008), casting doubt on the causal effect of exercise on affective change. Similar population-based studies are needed on PAA.

Exercise characteristics

Frequency

Despite the equivocal literature and our tentative hypothesis, the results showed that frequency moderated post-program PAA. Such discrepancies are likely to occur in particular when the literature is mixed. Hunter and Schmidt (2004) discuss the pitfalls associated with narrative-review generated hypotheses, and when in doubt, the results of meta-analyses are preferred. In this case, >3 sessions/wk was associated with greater post-program PAA.

Intensity

The analysis found the largest effects for the lowest and highest intensities in contrast to Reed and Ones (2006) who found an inverse relation between intensity and ES in a meta-analysis of acute aerobic exercise and PAA. To speculate, one possible explanation for this difference is that repeated higher intensity aerobic exercise may produce physical and psychological adaptations that shift post-exercise PAA to a level similar in magnitude to the often greater and more consistently observed increases in PAA following lower intensity aerobic exercise (Ekkekakis et al., 2000; Reed & Ones, 2006; Thayer, 1986).

Time

Similar to the acute PAA literature (e.g., Reed & Ones, 2006), the current analysis did not find compelling evidence for a threshold effect for exercise time (session duration) on post-exercise PAA. Although 30–35 min appears optimal, this elevated effect did not escape sampling error overlap from the other time categories. The acute literature mentions that fatigue may be a factor around 40 min (Reed & Ones, 2006), suggesting that fitness may play a role in a threshold exercise time effect.

Program duration

Programs of 10–12 weeks produced the largest improvements but, again, this elevated effect did not completely escape sampling error overlap. Effects appear to diminish for programs > 13 weeks. Certainly, as with other psychological phenomena, habituation may be playing a role in the attenuated responses of longer exercise programs.

Exercise dose

Dose was a trivial moderator. To speculate, one possibility for this result may be chance occurrence. Intensity was labeled as "moderate" for all subgroups because the computed average for each subgroup corresponded to the code value for moderate intensity (see *Study coding* in the Method section). Inspection of the data revealed that the similar subgroup averages were the result of an even distribution of intensity levels within subgroups. Therefore, subgroup ESs were not as different as they might have been because intensity was controlled for in the dose calculations. Another speculative explanation is that dose differences were apparent after the first few exercise sessions, but faded after several weeks. For example, ESs may have remained similar from pre to post-program in low and moderate dose studies, while ESs for high dose studies may have been small initially then increased over time, the result being the lack of a dose moderator effect. Certainly, a combination of these two possibilities may have operated to attenuate dose subgroup differences. Finally, although dose did not provide insight as a moderator, the results indicate the following aerobic exercise program as optimal for improving PAA: low intensity (∼30% VO_2R), 30–35 min, 3–5 days/wk for 10–12 weeks.

Study quality and source

Studies with higher internal validity produced larger effects and this result was independent of study source. To speculate, one potential explanation for this result is the known tendency for methodological weaknesses to reduce ES magnitude (Hunter & Schmidt, 2004) and therefore one might expect smaller effects in studies with weaker internal validity. For source, no difference was found in ES magnitude between published and unpublished studies indicating that factors other than internal validity are likely related to whether studies were published.

Theoretical and practical implications

Researchers have proposed several explanations for the affective benefits of regular aerobic exercise. Haskell (1987) contends that affective benefits develop from the "accumulation" of repeated acute exercise-affective changes. Tuson and Sinyor (1993) suggest different mechanisms for acute and chronic effects: acute exercise influences transient mood and chronic exercise influences affect. Salmon (2001) proposed that repeated exercise results in stress adaptation, which gradually improves affect.

Another possibility is that acute aerobic exercise improves PAA and the observed changes with regular exercise simply reflect the repeated reinstatement of the acute affective improvement. We call this a "maintenance" explanation. Several convergent lines of evidence appear to support this idea. First, exercise-related PAA improvements do not depend on changes in fitness (e.g., Ekkekakis et al., 2000). That is, the acute PAA effects of aerobic exercise are observed with little or no training response. Second, the magnitude of the overall effect of the present meta-analysis differs by only one-tenth of a SD from the overall effect of an acute exercise PAA meta-analysis conducted using identical methods (Reed & Ones, 2006). Third, Steinberg et al. (1998) assessed affect once a week for seven weeks in four different exercise groups and found consistent and similar affective improvement week after week for all groups with no signs of adaptation or tolerance. More studies that examine week-to-week PAA changes are needed to gain a better understanding of the interaction between acute and regular aerobic exercise responses.

The dual-mode model (Ekkekakis, 2003) may offer theoretical clarity to the intensity results as the model proposes that low, moderate, and high intensity aerobic exercise can result in post-exercise-affective change: at lower intensities due to cognitive factors and increases in activation perceived as pleasant, and at higher intensities due to an affective "opponent process" (Solomon, 1980), possibly related to endorphins that rapidly reverse the negative affective valence reported during exercise. Such a proposal was recently tested and verified (Ekkekakis, Hall, & Petruzzello, 2008).

Although the dual-mode model focuses on acute exercise, our results support the model for the intensity moderator, as pre–post-PAA increased for all intensity levels. Two aspects of these results are of interest. One is the lower intensity subcategory. These results strongly suggest that lower intensity aerobic exercise produces robust, consistent, and generalizable post-exercise increases in PAA. The other is the PAA increase in the high intensity category for which there are a few plausible, albeit speculative explanations. First, we may be observing consistent affective improvement akin to a "maintenance" hypothesis. Second, improvements may be related to an adaptive process whereby PAA changes are initially small and increase over time supporting either an opponent process (Solomon, 1980) or stress adaptation (Salmon, 2001) model that predict the gradual increase of PAA with exercise training. The high intensity improvements may also be the result of a combination of maintenance, opponent process and stress adaptation phenomena.

Puetz et al. (2006) discuss the minimum program duration necessary to improve PAA as a relevant practical issue. We agree and the data shed some light on the issue. Further analysis of the program duration data indicated that $\overline{d}_{corr}$ for 4- and 5-week programs equaled .47 (95% CI = .29–.65, $K = 11$), quite close to the $\overline{d}_{corr}$ of .51 for the 4–9-week moderator subcategory. Although these data lack week-to-week effects, we submit that little change will be found when more data become available. The reason is twofold: first, meta-analytical results of acute PAA effects (Reed & Ones, 2006) mirror program duration effects, and second, studies examining week-to-week responses do not appear to show large week-to-week variations (e.g., Steinberg et al., 1998). It appears reasonable, therefore, to recommend 4–5 weeks of regular aerobic exercise as sufficient to impart noticeable PAA benefits.

Strengths and limitations

While meta-analysis has numerous strengths (Egger & Smith, 1997; Howard, Maxwell, & Fleming, 2000), this analysis has several specific strong points. First, the database included 105 studies and nearly 400 ESs, which improves the accuracy of population estimates (Hunter & Schmidt, 2004). Second, our theoretical approach allowed us to examine responses from a single quadrant of the affect circumplex and thereby eliminate the confounding and interpretational difficulties associated with aggregating responses from more than one quadrant (Posner et al., 2005). Third, adjusting for methodological and statistical artifacts is important to understanding the true magnitude of effect and some but not all of these were considered and corrected for in this meta-analysis (Hunter & Schmidt, 2004). We should note however, that the number of ESs was small enough to warrant caution for several moderator analyses, the results of which should be considered preliminary. Also, data for affective responses during exercise were not included. As more data become available, these responses should be part of future quantitative reviews.

Summary

The results show that aerobic exercise programs produce favorable increases in PAA. The effect, however, is moderated by pre-program affective state and the degree of internal validity of the study design. Although exercise dose was a weak moderator, the results indicate that an optimal combination of exercise characteristics for enhancing PAA includes low intensity for 30–35 min, 3–5 days/wk for 10–12 weeks. We continue the call for theory-driven research with one goal being the introduction of a new breed of meta-analysis consisting only of studies based on a single or a few closely related theoretical approaches. Finally, the results of this meta-analysis and those of the acute PAA literature indicate that a more complete understanding of exercise-related affect will emerge when researchers examine the interaction of acute and chronic responses.

Acknowledgements

We thank Patrick O'Connor and colleagues for providing references used in this meta-analysis.

Appendix

Characteristics of included studies.

Authors and year	*N*	Publication status	Study[a] quality	Exercise characteristics			
				Frequency (sessions/wk)	Intensity[b] (% HRR)	Time (min)	Duration (weeks)
Puetz, Flowers, and O'Connor (2008)	36	Published	6	3	40–59	30	6
Annesi and Unruh (2008)	102	Published	6	3	40–59	30	24
Hoffman and Hoffman (2008)	48	Published	4	NR	40–59	NR	NR
Elavsky (2006)	163	Unpublished	6	3	40–59	35	16
Osei-Tutu and Campagna (2005)	40	Published	7	5	40–59	30	8
Cluphf, Schroeder, and Lox (2004)	25	Published	5	NR	NR	NR	NR
Tsai et al. (2004)	102	Published	5	3	60–84	50	10
Oken et al. (2004)	35	Published	7	4	<20	32	24
Wenneberg, Gunnarsson, and Ahlstrom (2004)	36	Published	5	NR	NR	NR	12
Yu, Li, Ho, and Lau (2003)	112	Published	5	2	60–84	60	8
Valim et al. (2003)	32	Published	7	3	NR	45	20
Annesi (2002b)	36	Published	4	2	40–59	30	14
Annesi (2002a)	69	Published	4	3	40–59	25	12
Burnham and Wilcox (2002)	18	Published	6	3	40–59	20	10
Blanchard, Rodgers, Courneya, Daub, and Black (2002)	81	Published	4	2	40–59	45	6
Tsai et al. (2002)	53	Published	5	3	NR	50	12
Mostert and Kesselring (2002)	26	Published	6	5	40–59	25	4
Peters, Stanley, Rose, Kaney, and Salmon (2002)	228	Published	9	2	40–59	40	10
Watanabe, Takeshima, Okada, and Inomata (2001)	33	Published	1	3	40–59	40	12
Sutherland, Andersen, and Stoove (2001)	11	Published	6	3	NR	45	10
Segal et al. (2001)	123	Published	6	5	40–59	NR	26
Brown et al. (2001)	112	Published	9	4	20–39	20	8
Lee et al. (2001)	102	Published	6	NR	40–59	NR	7
Mack et al. (2000)	62	Published	2	NR	NR	NR	7
Gracia and Marco (2000)	107	Published	4	3	20–39	45	32
Nieman et al. (2000)	91	Published	6	5	40–59	45	12
Sledge, Ragsdale, Tabb, and Jarmukl (2000)	87	Published	4	3	40–59	45	8
Dimeo, Stieglitz, Novelli-Fischer, Fetscher, and Keul (1999)	59	Published	5	7	40–59	15	NR
Shin (1999)	27	Published	3	3	40–59	55	8
DiLorenzo et al. (1999)	110	Published	3	4	60–84	45	12
Lee (1999)	10	Unpublished	3	2.5	NR	45	8
Hall and Petruzzello (1999)	41	Published	4	4	40–59	60	NR
Quittan, Sturm, Wiesinger, Pacher, and Fialka-Moser (1999)	27	Published	6	2	40–59	50	12
Steptoe et al. (1998)	73	Published	5	3	NR	NR	2
Partonen et al. (1998)	98	Published	6	2.5	60–84	50	8
Engels, Drouin, Zhu, and Kazmierski (1998)	23	Published	5	3	40–59	60	10
Vilvens (1998)	126	Unpublished	4	NR	NR	40	6
Lochbaum (1998)	53	Unpublished	4	3	60–84	30	20
Slaven and Lee (1997)	220	Published	4	2	40–59	35	12
Hassmen and Koivula (1997)	40	Published	6	3	20–39	NR	12
Hargraves (1997)	20	Unpublished	6	5	60–84	45	12
Glisky (1997)	30	Unpublished	3	3	NR	45	16
Pinto et al. (1997)	60	Published	6	3	20–39	30	12
Annesi and Mazas (1997)	39	Published	8	3	NR	25	14
Holloway (1997)	94	Unpublished	7	3	40–59	30	12
Stanton and Arroll (1996)	177	Published	5	3	40–59	40	24
Gauvin et al. (1996)	86	Published	1	3	NR	NR	6
Petajan et al. (1996)	46	Published	5	3	40–59	30	5
Skultety (1996)	146	Unpublished	4	3	50–70	35	12
Hayes (1996)	20	Unpublished	1	2	<20	30	10
McGee and Horgan (1996)	20	Published	1	NR	NR	NR	7
Brown et al. (1995)	135	Published	5	3	60–84	35	16
Lox et al. (1995)	33	Published	6	3	60–84	24	12
Slaven and Lee (1994)	60	Published	4	NR	NR	NR	NR
Tsai (1994)	110	Unpublished	1	2	60–84	30	12
Prince (1994)	9	Unpublished	1	3	NR	NR	5
Anshel and Russell (1994)	28	Published	6	4	50–70	30	12
Moore (1993)	189	Published	4	NR	NR	NR	NR
Takenaka (1993)	24	Published	6	3	NR	30	4
Nieman, Warren, Dotson, Butterworth, and Henson (1993)	32	Published	9	5	40–59	35	5
Wormington, Cockerill, and Nevill (1992)	57	Published	5	NR	NR	NR	NR
Gitlin et al. (1992)	267	Published	6	3	60–84	30	16
Stoughton (1992)	87	Unpublished	6	4	40–59	40	12
Dua and Hargreaves (1992)	47	Published	4	2	NR	NR	NR
Cramer et al. (1991)	35	Published	8	5	60–84	45	15
McAuley (1991)	80	Published	1	NR	NR	NR	10
Brown (1991)	27	Published	5	3	NR	20	9
Alton-Stonebrook (1991)	26	Unpublished	2	NR	NR	NR	4
Perlman et al. (1990)	43	Published	2	2	40–59	25	16
Emery and Gatz (1990)	38	Published	5	3	40–59	25	12
Lesniak (1990)	34	Unpublished	6	6	≥85	60	10

Appendix (*continued*).

Authors and year	*N*	Publication status	Study[a] quality	Exercise characteristics			
				Frequency (sessions/wk)	Intensity[b] (% HRR)	Time (min)	Duration (weeks)
Deivert (1990)	40	Unpublished	6	3	40–59	20	8
Steptoe et al. (1989)	47	Published	6	4	40–59	20	10
Gauvin (1989)	122	Published	4	3	NR	NR	NR
Polis (1989)	29	Unpublished	5	3	40–59	25	10
Simons and Birkimer (1988)	53	Published	3	2	20–39	30	8
Nagy and Frazier (1988)	85	Published	1	3	40–59	30	15
Segebartt, Nieman, Pover, Arabatzis, and Johnson (1988)	25	Published	4	3	NR	15	NR
Soutter (1988)	55	Unpublished	1	1	NR	30	12
Aghekian (1988)	84	Unpublished	4	3	NR	20	NR
Pelham (1988)	5	Unpublished	9	2	40–59	25	12
Agnew and Levin (1987)[c]	2210	Published	3	NR	NR	NR	NR
Johnston (1987)	59	Unpublished	4	NR	NR	NR	15
Ferretti (1987)	35	Unpublished	5	3	40–59	35	6
Jewell (1987)	64	Unpublished	3	2	40–59	20	16
Lennox (1987)	44	Unpublished	8	3	60–84	40	13
Sheales (1987)	49	Unpublished	3	2	60–84	20	13
Dyer (1987)	70	Unpublished	4	3	NR	45	6
Williams and Getty (1986)	430	Published	5	3	40–59	40	10
Wilfley and Kunce (1986)	49	Published	1	3	NR	NR	8
Hughes, Casal, and Leon (1986)	14	Published	5	5	40–59	45	12
Abele and Brehm (1986)	186	Published	3	NR	NR	NR	NR
Schneider (1986)	94	Unpublished	5	4	60–84	30	6
Goldwater and Collis (1985)	51	Published	5	5	NR	NR	6
Cherico (1985)	100	Unpublished	4	3	40–59	30	12
Cavalieri (1985)	57	Unpublished	6	3	NR	40	10
Gondola and Tuckman (1983)	464	Published	1	NR	NR	NR	NR
Thomas (1983)	14	Unpublished	6	3	40–59	25	8
Campbell (1983)	12	Unpublished	4	3	40–59	25	10
Johnson (1983)	20	Unpublished	2	3	60–84	27	12
Blumenthal, Williams, Needels, and Wallace (1982)	16	Published	6	3	60–84	45	10
Hilyer et al. (1982)	43	Published	5	NR	NR	NR	NR
Zentner (1981)	80	Unpublished	5	3	NR	50	10
Reiter (1981)	128	Unpublished	6	2	<20	55	5
Wilson, Morley, and Bird (1980)	30	Published	4	4	NR	NR	NR

Note. Each study occupies a single row in the table; however, many studies employed more than one experimental or control group and these groups were included in the meta-analysis if appropriate. NR indicates "Not Reported". This designation refers to study information that was either not reported or not reported clearly enough to be coded accurately.

[a] Study quality is a non-weighted index of the number internal validity threats the researcher attempted to control in the study design.

[b] Intensity is based the classification system outline by Howley (2001). %HRR = percent heart rate reserve.

[c] Due to the large sample size, this study was entered only for the post-study exercise vs. post-study control meta-analysis.

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