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Meta-Analysis of Interventions to Increase Physical Activity among Cardiac Subjects

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Abstract

Background—Extensive primary research has tested interventions to increase physical activity (PA) among adults with cardiovascular disease. This meta-analysis integrates the extant research about how to increase PA in cardiac samples.

Methods—Extensive literature searching located published and unpublished intervention studies that measured PA outcomes. Results were coded from primary studies. Fixed-and random-effects meta-analytic procedures included moderator analyses.

Results—Data were synthesized across 11,877 subjects from 79 eligible research reports. The overall mean PA effect size for 2-group comparisons was 0.35 (higher mean for treatment than control), which is consistent with a difference of 1984 kilocalories/week for treatment subjects versus 1615 for control subjects. The fitness effect size for 2-group comparisons was .17. Other statistically significant positive 2-group effect sizes were .24 for quality of life and .23 for subsequent cardiac events. Effect sizes for anthropometric measures and blood pressure did not differ significantly from 0. Exploratory moderator analyses found large effect sizes for PA among studies that had (1) an exclusive focus on PA versus diverse health behaviors, (2) more contact between interventionists and subjects, (3) supervised exercise sessions, (4) fitness testing, (5) face-to-face encounters versus mediated intervention delivery, and (6) more minutes of activity per week. Effect sizes were unrelated to funding status, dissemination vehicle, gender distribution, or attrition rate.

Conclusions—These findings document that interventions can be effective in increasing PA among patients with cardiovascular diseases. Primary research should compare interventions in randomized trials to confirm causal relationships.

1. Introduction

Cardiovascular disease remains the most common cause of mortality and health care utilization in the United States [1]. William Heberden, the physician who first described angina pectoris more than two centuries ago, was also the first to observe the potential benefit of physical activity (PA) for people with cardiovascular disease [2]. Hellerstein dramatically changed

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cardiac rehabilitation by including exercise [3]. The emphasis on PA continues in modern treatment, and international cardiac rehabilitation guidelines consistently call for exercise as a central component of therapy [4,5]. Evidence that PA may reduce or retard the atherosclerotic process is especially exciting [6,7]. However, many adults fail to achieve therapeutic goals, despite extensive advances in medical treatment of cardiovascular disease. Numerous studies show that people with cardiovascular disease often do not follow prescribed medical regimens, including performing adequate PA [8–13]. The importance of PA has contributed to the proliferation of trials testing interventions to increase PA behavior. This study meets the pressing current need to integrate and synthesize this large body of research findings to guide future research and inform practice.

Several meta-analyses have dealt with the importance of PA among people with cardiovascular disease. The link between PA and subsequent cardiac and total mortality has been established [14–17]. PA can reduce triglycerides, obesity, insulin resistance, glucose intolerance, and blood pressure, as well as increase high-density lipoprotein cholesterol and quality of life [1,4,18–30]. Habitual PA reduces symptoms in people with established cardiovascular disease, even in those who are not candidates for revascularization [22]. PA also reduces the risk of other chronic illnesses, such as type 2 diabetes and obesity [22,31]. Although much of the existing research occurred before today's efficacious treatments (e.g. widespread use of acute thrombolytic therapy, primary angioplasty, aggressive revascularization, β -adrenergic blockers, aggressive lipid management, angiotensin-converting enzyme inhibitors), a recent meta-analysis found that PA benefits did not differ between studies reported prior to 1995 and those reported later [5]. Thompson et al. noted an urgent need for new knowledge about strategies to increase PA behavior [22]. None of the existing syntheses has examined subjects' PA behavior after completing the interventions, despite the previous meta-analytic findings regarding health consequences of PA. Dusseldorp et al. and Ketola et al. set out to integrate findings of studies attempting to increase PA, but they abandoned their projects after their limited search strategies retrieved only 8 and 10 studies, respectively [32,33]. This meta-analysis, with extensive search mechanisms, integrates studies designed to increase PA.

This synthesis furthers our understanding of primary studies designed to increase PA among adults with cardiovascular disease. The two primary research questions were: (1) What are the overall effects of interventions to increase PA on PA behavior after interventions? (2) Do interventions' effects on PA behavior vary depending the characteristics of the intervention, sample, or methodology? The secondary research questions were: (3) In studies that report PA outcomes, what are the overall effects of interventions on systolic and diastolic blood pressure, fitness, quality of life, anthropometric measures and subsequent cardiac events? (4) What are the effects of interventions on outcomes among studies comparing treatment subjects before versus after interventions? (5) Do control groups exhibit changes in outcomes between pre- and post-testing? (6) Is there evidence of publication bias?

2. Methods

We used standard quantitative review methods to identify and retrieve potential studies, assess eligibility, code data from research reports, meta-analyze primary study results, and interpret findings. The study was approved by the Institutional Review Board for the Protection of Human Subjects.

Primary study search strategies

We used multiple search strategies to ensure a comprehensive search, move beyond previous reviews, and limit the bias associated with narrow searches [34]. An experienced health sciences reference librarian performed searches in MEDLINE, PsychINFO, EMBASE, Cochrane Controlled Trials Register, Healthstar, Combined Health Information Database,

Sport Discus, Dissertation Abstracts International, Nursing and Allied Health Database, Educational Resources Information Center, and Database of Abstracts of Reviews of Effectiveness. Comprehensive searching with broad search terms was employed. Ancestry searches were conducted on eligible studies and previous narrative and quantitative reviews. We searched the National Institutes of Health Computer Retrieval of Information on Scientific Projects database of funded studies back to 1973. We also performed computerized database searches on all authors of retrieved studies that met the inclusion criteria and on principal investigators of funded studies. Hand searches were conducted in *American Heart Journal*, *American Journal of Cardiology*, *American Journal of Health Promotion*, *American Journal of Hypertension*, *Archives of Internal Medicine*, *Archives of Physical Medicine & Rehabilitation*, *British Medical Journal*, *British Heart Journal*, *British Journal of Sports Medicine*, *Cardiology*, *Circulation*, *European Heart Journal*, *Heart*, *Heart & Lung*, *International Journal of Cardiology*, *JAMA*, *Journal of Cardiopulmonary Rehabilitation*, *Journal of Hypertension*, *Journal of Psychosomatic Research*, *Journal of Sports Medicine & Physical Fitness*, *Journal of the American College of Cardiology*, *Journal of the American Geriatrics Society*, *Medicine & Science in Sports & Exercise*, *Patient Education & Counseling*, and *Sports Medicine*. Potential studies were identified using title and abstract visual heralds. These comprehensive search strategies yielded 4,798 reports that we examined to determine eligible primary studies.

Selection criteria

Inclusion criteria are found in Table 1. We included studies that had both an explicit intervention to increase subjects' PA and a measure of PA behavior after completion of the intervention. We defined PA as any bodily movement that results in elevated energy expenditure beyond basal levels [22]. Exercise, a subset of PA, was defined as planned, structured, and repetitive movement intended to increase fitness [22]. Most studies focused on exercise, and some addressed the broader PA construct.

We included small-sample studies that often lack statistical power to detect treatment effects. These studies may report on novel interventions or may include difficult-to-recruit subjects [35]. We weighted studies so that those with larger samples had proportionally more impact on aggregate findings. We included pre-experimental studies if they contained pre-intervention and post-intervention data to calculate effect sizes because some investigators find it unethical to withhold treatment in studies of patients for whom treatment is thought to be beneficial [32,36]. Randomization may not be feasible in some studies where contamination is likely [32]. We conducted separate analyses for single-group and 2-group comparisons.

We included both published and unpublished studies. Quantitative syntheses that include only published studies may overestimate the magnitude of the true population effect because the single largest difference between published and unpublished research is the statistical significance of the findings [35]. By using small-sample studies, unpublished reports, and pre-experimental research we were able to include a richer variety of interventions than if we had limited our project to large randomized controlled trials. We used the most distal data collection point available when reports presented outcomes at different intervals following the intervention because enduring PA behavior changes will most likely lead to health benefits.

Data evaluation and coding

We developed a coding frame for source, participant, and experimental characteristics as well as study findings. The coding frame was developed from elements coded in previous meta-analyses of health behaviors, suggestions from both meta-analysis and PA experts, intervention components reported in empirical literature, and findings from the research team's preliminary studies. Prior to implementation, we pilot tested the coding frame with 20 studies. Publication

year, presence of funding, and dissemination vehicle (e.g. journal article, dissertation) were coded as source characteristics. Participant characteristics included gender, age, and cardiac diagnoses, among others. We did not code New York Heart Association functional classification information because it was infrequently reported in the primary studies, the few studies that reported the information included varied combinations of categories, and none of the studies reported outcome data separately by functional classification.

We coded detailed intervention information, including content, format, interventionist, delivery medium, social setting, intensiveness, behavioral target (PA only vs. PA plus other behaviors), and recommendations about specific forms/intensities of PA. We developed explicit descriptions of intervention components that we subsequently piloted, revised, and finally implemented to fully describe interventions to increase PA (Table 2 lists the most prevalent interventions in the synthesized studies). We coded the number of days between completion of the intervention and measurement of outcome variables.

We developed a list of potential outcomes from the studies and subsequently generated a coding frame to capture them. In addition to PA behavior, we coded results for systolic and diastolic blood pressure, cardiovascular fitness, anthropometric measures (e.g. body mass index), subsequent cardiac events, and quality of life. A priori lists of measures were used to select among multiple possible dependent variable measures to avoid coder or author bias. Measures with high validity and reliability were selected over other measures. Two extensively trained coders extracted data independently. The senior author or another member of the research team resolved discrepancies between coders. We did not mask data extraction because evidence indicates it does not decrease bias [37]. Coded data were duplicate-entered to ensure accuracy. To ensure that only independent samples were analyzed, we cross checked author lists to locate research reports that might contain overlapping samples. When necessary, we contacted senior authors to clarify the uniqueness of samples.

Data analysis

We calculated a standardized mean difference (d) for each comparison as the difference between treatment group versus comparison group post-intervention means divided by the SD, with SD pooled between treatment and comparison groups when possible. For treatment pre-post comparisons, d was calculated as the difference between post- versus pre-intervention means divided by the pre-intervention SD when possible. Both types of ES were adjusted for small sample bias [38,39]. A positive d reflects more favorable outcome scores for treatment groups or at post-test. Larger samples were given more influence in the analyses by weighting each ES by the inverse of its sampling variance. We used normal-theory standard errors to construct 95% confidence intervals for the common or mean ES and to test whether the ES was zero. A conventional heterogeneity statistic (Q) was used to assess ES between-study homogeneity.

Studies with 2 or more treatment groups and 1 control group were included in the meta-analysis by accounting for dependence due to the common control group. This involved using generalized least-squares for fixed-effects analyses and, for random-effects analyses, a 2-stage approach whereby each study's dependent ESs were combined into a single independent ES then submitted to standard univariate random-effects analysis [40]. Whenever baseline data were available, we compared single treatment group pre- and post-intervention scores. Single-group studies were analyzed separately from 2-group comparisons. These single-group ESs were expressed in the metric of raw-score (vs. change-score) to facilitate comparisons with the 2-group ESs. Studies with multiple treatment groups but no control group were treated as single-group studies.

We created funnel plots of ES against sampling variance to explore potential publication bias [41]. Larger samples yield less sampling error in observed ESs. In the absence of publication bias, observed d s should be symmetric about the same overall average regardless of sample size. We also examined ESs graphically and statistically to identify potential outliers. Omitting each ES 1 at a time, we checked for large externally standardized residuals or substantially reduced measures of heterogeneity.³⁸ For PA, 4 independent group comparisons [42–45], 9 treatment group pre-post comparisons from 8 reports [42,44–60] and 2 control group pre-post comparisons [46,51], were excluded as outliers.

Single-group studies present challenges for meta-analyses. Pre- and post-intervention dependent variable scores are probably correlated in single-group design studies. Although not reported in the primary studies, some measure of this correlation is needed for meta-analysis of pre-post ESs [39]. Lacking empirical evidence, we conducted the analysis under 2 very different assumptions for all studies: strong positive correlation ($\rho_{12} = 0.80$) versus no correlation ($\rho_{12} = 0.00$) between pre- and post-test measures. The .80 assumption tends toward understating within study sampling error and overstating heterogeneity, whereas the 0.00 assumption tends to err in the opposite direction. Both analyses are presented for comparison. We also explored pre-post ESs from control subjects when these data were available from 2-group studies to partially address common concerns about single-group designs. Control group change information may be helpful for interpreting the findings from single-group intervention studies.

We conducted analyses using both fixed- and random-effects models [52]. The random-effects model assumes that observed ESs vary due to study-level sources of error (e.g., intervention variations) in addition to subject-level sampling error. This model treats any given implementation of a study as a random realization from a population of studies that could have been conducted. The random-effects model is more appropriate in situations where study implementation is heterogeneous, because it supports generalizations to studies whose features may differ from those included in the meta-analytic sample [53]. Interventions to change PA behavior are heterogeneous. Estimated ESs reported in the text are based on the random-effects model with the between-studies variance component σ_{θ}^2 estimated by weighted method of moments, unless otherwise designated [54]. When the between-study variance component is zero, the random- and fixed-effects results are identical.

We emphasized random-effects analyses because we anticipated considerable primary study heterogeneity. Statistical and clinical heterogeneity is an ongoing challenge in behavioral research where syntheses bring together studies that are diverse both clinically and methodologically [55,56]. Key contributors to heterogeneity are inclusion criteria differences, meaningful differences among interventions, dose variations, disease variations, study execution variations, length of follow up as well as study quality [55,57,58]. Meta-analysts have argued that heterogeneity should be viewed as the expectation, rather than the exception because it is so common in some types of quantitative syntheses [59]. Recent Cochrane reviews of behavioral interventions have accepted heterogeneity, used random-effects models to allow for and quantify heterogeneity, and conducted subgroup analyses to explore heterogeneity [56,60–62]. We planned four strategies to deal with heterogeneity. First, for the random-effects analyses, we reported not only the location parameter (mean ES) but also a variability parameter that represents variability of ESs about the location parameter to characterize the extent of heterogeneity [57]. Second, we conducted analyses using a random-effects model, which assumes true population ESs vary among the universe of study situations [57]. Third, we explored potential study-level moderators as an approach to understanding potential sources of heterogeneity [55,61]. Fourth, we interpreted our findings in light of existing heterogeneity. Testing for the presence of heterogeneity is less valuable than interpreting the extent to which heterogeneity affects meta-analysis conclusions [55].

The Common Language Effect Size was calculated to describe the probability that a random treatment subject would score better than a random control subject or, for pre-post comparisons, the probability that a random subject would score better at post-than pre-intervention. A Common Language Effect Size of 0.50 corresponds to an ES of $d = 0$. In addition, PA and blood pressure ESs from 2-group comparisons were translated to the original metric to enhance clinical interpretation of findings. Fitness, quality of life, and anthropometric outcomes could not be converted to an original metric due to extensive variations in measurement.

We conducted exploratory moderator analyses with 2-group comparisons to evaluate potential moderating variables of PA [63]. These preliminary analyses are intended as a hypothesis-generating contribution since there is an absence of previous evidence about intervention components and other attributes of study design to provide a firm basis for confirmatory hypothesis testing. Moderators for these exploratory analyses were selected based on availability in primary study reports. Our search focused on PA, and we conducted moderator analyses only with PA since other outcomes (e.g. blood pressure, quality of life) yielded far fewer comparisons. Continuous moderators were analyzed using a meta-analytic analogue of regression, and only if at least 6 studies reported data for the moderator; dichotomous moderators, a meta-analytic analogue of ANOVA and only if there were at least 3 studies at each moderator level [55,63]. Moderator analyses compare the amount of ES variability among levels of a study-level moderator with the amount of variability in observed ESs that would be expected by subject-level (and, for mixed-effects analyses, between-studies) sampling error alone. The effect of a continuous or dichotomous moderator is tested by the regression slope (β = unstandardized regression coefficient) or between-groups heterogeneity statistic (Q_B), respectively; heterogeneity beyond that due to the moderator is tested by the residual (Q_{residual}) or combined within-groups (Q_W) heterogeneity statistic, respectively. Although univariate moderator analyses were used instead of the more sophisticated multivariate mixed-effects approach preferred for non-independent ESs, the relatively few multiple-treatment pairs were not expected to unduly distort estimates and tests [64].

3. Results

Extensive search strategies resulted in 100 eligible samples from 79 research reports with a total of 11,877 subjects [6,7,8,10–13,42–51,65–126]. Most of the primary study reports were disseminated as journal articles ($s = 66$); the remainder were dissertations ($s = 11$) or presentation papers ($s = 2$) (s indicates number of reports, k indicates number of comparisons). A few reports appeared before 1990, and 36 appeared in 2000 or later. Most studies ($s = 53$) were funded. These reports enabled us to calculate an ES for 67 treatment-versus-control-group comparisons (9,564 subjects), 71 treatment group pre-post comparisons (4,204 subjects), and 31 control group pre-post comparisons (1,856 subjects). Data for 2 comparisons (2 treatment groups compared to a control group or 2 treatment group outcomes compared to baseline values) were available from 14 reports [12,33,46,47,50,68,82,89,90,99,101,109,112,126], a few reports ($s = 3$) contained 3 comparisons [65,67,110].

Funnel plots of ES by sampling variance showed some evidence of publication bias for PA outcomes among both treatment versus control group comparisons and among treatment group pre-post comparisons. The small numbers of comparisons for other variables rendered funnel plots difficult to assess. Nevertheless, anthropometric outcomes showed weak evidence of publication bias for treatment group pre-post comparisons and possibly for treatment versus control group comparisons. Plots are available from the senior author.

Primary study characteristics

Descriptive statistics for primary study characteristics are displayed in Table 3. Sample size varied extensively from 14 to 1,173 subjects, with a median of 67 subjects. The median value

for mean age was 59 years. Among the studies that reported gender distribution, typically about one-fourth of the subjects were women. Minority inclusion was infrequently reported: 16 reports indicated including African-Americans, 7 included Hispanics, and 2 included Native Americans. Only 1 paper reported on predominantly (50% or more) African-American subjects, and no reports focused on Hispanic or Native-American subjects. Attrition was typically modest, though a few studies experienced significant loss. Attrition was similar between treatment and control groups. Most studies ($k = 42$) measured PA behavior outcomes immediately after completing the intervention and 3 other papers reported PA outcomes within one month after interventions. Twenty-three studies measured PA at least 6 months post-intervention and only 12 studies reported PA one year or longer after interventions.

Tables 2 and 3 include intervention attributes. Typical attributes among studies with supervised exercise included between 2 and 3 weekly 60-minute sessions over 12 weeks. Fifteen studies included some form of mediated motivational or educational intervention delivery (e.g. telephone, mail, internet), and 7 used only mediated intervention delivery. Fitness testing ($s = 39$) and individualized exercise prescription ($s = 33$) were common intervention components. Modeling, either by research staff members or by people similar to subjects, occurred in 34 studies. Several studies asked subjects to monitor their PA behavior ($s = 24$) as a strategy to increase PA. Monitoring of PA behavior by research staff was also common ($s = 23$). Sixteen studies provided explicit feedback to subjects about their PA behavior. Several studies asked subjects to generate specific PA behavioral goals ($s = 17$) in an effort to change behavior. Twelve studies attempted to increase social support for PA. Twelve studies either taught problem-solving skills to subjects or included problem solving with the research staff. Twelve studies either provided rewards for subjects for increasing PA or taught subjects to reward themselves for PA. Ten studies specifically dealt with barriers to PA as a strategy to increase activity. Strategies infrequently reported include stimulus/cues to PA ($s = 5$), relapse prevention education ($s = 4$), decision making/balancing ($s = 4$), contracting ($s = 3$), cognitive modification ($s = 2$), self-management training ($s = 2$), shaping ($s = 1$), imagery ($s = 1$), and motivational interviewing ($s = 1$).

Overall effect of interventions

The overall effects of interventions on variables for 2-group comparisons are presented in Table 4 and for treatment group pre- versus post-test comparisons in Table 5. We will focus on random-effects results. The mean effect on PA in 2-group studies was .35. The treatment group pre- versus post-test ESs for PA were .49 ($\rho_{12} = .80$) and .57 ($\rho_{12} = .00$). The Common Language Effect Size for the 2-group PA comparisons was .60, indicating that 60% of the time a random treatment subject would have a better PA outcome than a random control subject. The Common Language Effect Size for treatment group pre- and post-intervention PA comparisons was .78 ($\rho_{12} = .80$ assumption reported for all Common Language Effect Sizes), meaning that 78% of the time a random treatment subject would score better on PA post-intervention than pre-intervention. To enhance interpretability, we transformed mean ESs for PA into kilocalories expended per week (kcal/wk) using results from appropriate reference groups averaged across available studies. For 2-group comparisons, the mean effect in terms of the combined treatment and control standard deviation (1054) implies a PA raw mean difference of $(.35 \times 1054 =) 368.9$ kcal/wk; relative to the mean of 1615 for control subjects, this further translates into a mean of $(1615 + 368.9 =) 1984$ kcal/wk for treatment subjects. These findings document that subjects with cardiovascular disease can increase PA behavior after interventions.

The fitness ES for 2-group studies was .17. The treatment group pre-post fitness ESs were .43 ($\rho_{12} = .80$) and .53 ($\rho_{12} = .00$). For fitness, the 2-group Common Language Effect Size was .55, and the treatment group pre- and post-intervention Common Language Effect Size was .75. For quality of life, the 2-group ES was .24 and the pre-pre-post ESs were .31 ($\rho_{12} = .80$).

and .40 ($p_{12} = .00$). The Common Language Effect Size for the 2-group quality of life comparisons was .57. The quality of life Common Language Effect Size for treatment group pre-post comparison was .69. The mean effect on subsequent cardiac events in 2-group studies was .23 with a Common Language Effect Size of .56. For 2-group comparisons on PA behavior, fitness, quality of life, and cardiac events, the mean ES was (statistically) significantly positive.

The 2-group comparison ESs for anthropometric measure and systolic and diastolic blood pressure were not significantly different from 0 (Table 4). We illustrated the small magnitudes of effects on blood pressure by converting the ES to the original metric. For 2-group comparisons, the mean effect in terms of the combined treatment and control standard deviation (16.3) is a systolic blood pressure raw mean difference of $(.02 \times 16.3 =) 0.3$; relative to the pooled mean of 131.6 for control subjects, this further translates into a systolic blood pressure mean of $(131.6 - .3 =) 131.3$ for treatment subjects. The mean diastolic blood pressure effect in terms of the combined treatment and control standard deviation (9.4) is a systolic blood pressure raw mean difference of $(.08 \times 9.4 =) 0.8$; relative to the mean of 78.5 for control subjects, this represents a diastolic blood pressure mean of $(78.5 - 0.8 =) 77.7$ for treatment subjects.

The treatment group pre-post anthropometric ESs were .18 ($p_{12} = .80$) and .16 ($p_{12} = .00$). For anthropometric measures, the treatment group pre-post comparison Common Language Effect Size was .61. The treatment group pre-post systolic blood pressure ESs were .09 ($p_{12} = .80$) and .10 ($p_{12} = .00$). The systolic Common Language Effect Size for treatment group pre-post comparison was .56. For diastolic blood pressure, the treatment group pre-post ESs were .15 ($p_{12} = .80$) and .21 ($p_{12} = .00$). The treatment group pre-post comparison Common Language Effect Size for diastolic blood pressure was .59. All of the treatment group pre- versus post-test comparisons were significantly different from 0 except for systolic blood pressure ($p_{12} = .00$). Many of the effects demonstrated significant ES heterogeneity according to the homogeneity test (Q in Table 4). With few studies for some outcomes, these estimates are imprecise and somewhat inflated, but they provide evidence that the true intervention effect varies among studies for these outcomes.

The mean effects of interventions for comparison/control group pre-post comparisons are found in Table 6. The mean ESs were not significantly different from 0 for PA behavior, anthropometric outcomes, systolic and diastolic blood pressure, or quality of life. Comparison groups experienced slight improvements in fitness with an ES of .12. The fitness Common Language Effect Size for control group pre- and post-intervention comparison was .58. Significant heterogeneity for many of the outcome variables suggests considerable variability among studies in the pre- versus post-intervention change for comparison groups. These findings document that overall, most comparison groups experience little benefit from participating in these studies.

Physical activity behavior moderator analysis

Tables 7 and 8 display results from the analyses of dichotomous and continuous moderator variables, respectively. Other potential moderators occurred infrequently. Neither funding nor publication year was significant moderator of ESs for PA. Gender distribution and sample mean age were unrelated to ESs for PA. Attrition proportion, which was typically modest, was unrelated to PA outcomes.

The amount of contact time between subjects and interventionists was related positively to PA outcomes ($\beta_1 = 0.205$ in terms of $\log_{10}$ minutes), though the number of weeks over which the intervention was delivered did not predict ES for PA. Studies with supervised exercise sessions reported larger PA mean ES (.46) after the supervised exercise had been completed than studies without supervised exercise (.31). Although exercise prescription was unrelated to PA

outcomes, studies using fitness testing had somewhat larger ESs for PA (.48) than studies without such testing (.33). Interventions that did not include goal setting reported larger ESs for PA (.40) than studies using goal setting (.21). Self-monitoring of PA behavior was unrelated to ESs for PA. Interventions that focused only on PA behavior reported larger ESs (.47) than interventions that attempted to change multiple behaviors (e.g. diet, smoking, medication adherence) (.32). Interventions delivered entirely by face-to-face encounter reported larger ESs (.41) than studies using any mediated delivery (e.g. telephone, mail) (.25). We found no PA differences between studies that recommended walking after the intervention when compared to those that did not recommend walking. Interventions that recommended more minutes per week of PA resulted in significantly larger ESs for PA ($\beta_1 = .004$).

4. Discussion

This research synthesis documented that treatment subjects performed more PA behavior, had better fitness results, experienced greater quality of life, and reduced cardiac events following interventions than did control subjects. In 2-group comparisons, anthropometric measures and systolic and diastolic pressure were not better among treatment subjects than control subjects. *Q* values documented substantial heterogeneity among primary studies. The considerable variation of intervention effects should stimulate further research to determine which characteristics of designs, interventions, and samples distinguish effective interventions from those that are less effective or even detrimental.

We included single-group pre-post comparisons to complement the information provided by 2-group comparisons. Although the two types of ESs yielded similar patterns of mean ESs across dependent variables, single-group treatment ES estimates tended to be slightly larger than their 2-group counterparts; ESs for anthropometric measures are a notable exception. Although control groups experienced little improvement over the course of research participation (only fitness improved significantly), findings from single-group pre-post comparisons should be interpreted with caution. We will limit further discussion of findings to 2-group main effects and moderators.

This is the first meta-analysis to examine PA outcomes among cardiac populations. The findings are important because they document that PA behavior change is possible. The ES of .35 is equivalent to a 368.9 kcal/wk better post-intervention PA mean among treatment subjects. For instance, if control subjects spent an averaged of 1,615 kcal/wk in energy, treatment subjects spent 1,984 kcal/wk. It is unclear whether this difference in PA outcomes is clinically meaningful because we do not yet know the doses required to achieve specific health benefits. This amount of PA increase does not meet current recommendations for healthy adults. The dose necessary to achieve health benefits among those with cardiovascular diseases is not well established. Evidence regarding incremental health changes as subjects move from entirely sedentary to modest activity to moderate PA is not available. For example, we do not know if people who are entirely sedentary and adopt a small amount of PA experience similar health gains as people who perform modest activity and move to moderate activity. The modest ES suggests we need interventions that increase PA more and among more subjects. Most studies reported PA outcomes immediately after interventions. Since it is long-term PA that bestows health benefits, this field needs more studies with extended follow-up to determine how well subjects persist in PA after interventions end [22].

Thompson et al. noted the urgent need to discover behavioral strategies and techniques that increase PA [22]. Our exploratory moderator analyses offer intriguing suggestive information about possible moderators of PA results. Interventions that included supervised exercise were more successful in attaining long-term behavior change. Although the availability of supervised exercise may be limited due to access or cost issues, it appears to be more successful

than other interventions components in changing PA [5]. Supervised exercise may provide participants with clear information about the form, intensity, and duration of exercise. Participants' consistent practice in both scheduling supervised PA and working through the supervised exercise sessions may teach them behavior patterns that foster continued PA. Exercise professionals who conduct supervised exercise may motivate in ways that are lacking in unsupervised PA at home. Studies using supervised exercise often included fitness testing, which was also associated with larger PA outcomes (albeit weakly). Fitness testing may give participants objective evidence about their physical health that they find motivating. It is also possible that fitness testing reassures some subjects that it is safe for them to perform PA.

The target behavior was important in the moderator analysis of PA. Interventions that focused solely on PA resulted in larger PA changes. This is consistent with findings from a meta-analysis of interventions to increase PA among older adults with diverse chronic illnesses [127]. Changing a single behavior can be a difficult endeavor, and cardiac populations may feel overwhelmed when asked to change PA, diet, medication, and possibly smoking behavior simultaneously. Although it may be ideal to change multiple cardiac risk behaviors at once, it may work better to apply interventions in sequence with the goal of changing one behavior at a time. The most successful interventions appear to be delivered face-to-face and with ample contact time to either groups or individuals. Recent efforts to design alternative delivery modes (e.g. interventions delivered by e-mail) should be carefully evaluated for their long-term effects.

Our moderator analysis documents the intriguing finding that interventions that recommended more minutes per week of exercise were more effective in promoting PA than those recommending less exercise. Similarly, Conn et al. reported that older adults told to perform moderate intensity exercise were more likely to increase PA than those told to perform low intensity PA [127]. It is possible that subjects more easily recognize rigorous recommendations -- more minutes per week or higher intensity exercise -- as a clear departure from their sedentary habits. The findings do not support the use of goal setting, self-monitoring, or recommending walking PA. The lack of support for goal setting and self-monitoring was somewhat surprising given the popularity of these strategies. Most intervention studies bundle behavior change interventions and rarely examine specific strategies, such as goal setting and self-monitoring. This meta-analysis is the first attempt to examine the effects of individual components. Unfortunately, we coded many PA behavior change intervention strategies but could not analyze them as moderators due to infrequent reporting. For some outcomes, the 2-group mean ES and variance component together suggest that certain studies in this body of research could plausibly yield true ESs as large as .50 (anthropometric measures), .60 (quality of life), or even .70 (PA). With better information on study-level features to model the ES heterogeneity, meta-analysts would be better able to identify types of studies that yield these impressive intervention effects. In other words, the heterogeneity of ESs suggests that better interventions may be present in the published literature, but without knowing more about moderator information it's difficult to know why the studies with higher ESs are getting such great results.

We found that gender and age distribution in samples were not related to PA, which suggests that interventions are equally effective for women and men of diverse ages [33]. The lack of studies targeting minority subjects prevented moderator analyses related to ethnicity. There is an urgent need for more intervention studies with these vulnerable populations. Functional status could be an important moderator of both PA behavior and health outcomes among studies designed to increase PA. We were unable to include New York Heart Association functional class as a variable because it was reported infrequently in primary studies. Future meta-analyses may be able to address important sample attributes, such as ethnicity and functional status, as potential moderators of ES as more studies report these attributes for the entire sample, focus on subjects with particular attributes, or report findings separately for sample subsets.

Regarding quality of life, the 2-group treatment versus comparison group ES was .24. Other research has noted modest improvements in quality of life among cardiac populations following PA or rehabilitation/education interventions [1,18,22]. These improvements may result from improved fitness and physical function or from the association between PA and reduced mild depression.

Fitness ESs were significantly better among treatment subjects as compared to control subjects, though the magnitude was small. Cardiorespiratory fitness may vary considerably among subjects undertaking identical exercise programs because of individual and genetic influences. The ideal PA to achieve fitness improvements is not yet known. Due to scant data, we could not synthesize the exercise characteristics, such as form and dose, that are essential to achieve favorable results. This is especially unfortunate because exercise variations may determine outcomes. A recent review was also unable to synthesize exercise dose effects across studies due to inadequate reporting in the literature [4]. Future work should examine the best exercise modes and doses for achieving optimal health outcomes. Although some primary studies reported co-morbid conditions, another potential influence on fitness outcomes, the reporting was not adequate to include them in the moderator analyses.

Subsequent cardiac events are important outcomes of any interventions with cardiac populations. Researchers have synthesized the effects of psycho-educational programs for cardiac populations and reported reduced cardiac events [32,128,129]. Dusseldorp et al. examined the influence of proximal changes on distal outcomes such as mortality and documented that infarction recurrence was 36% less in studies that changed proximal variables as compared to only 2% for studies that did not change proximal outcomes [32]. Although the magnitude of the statistically significant ES was modest in this meta-analysis and few studies reported these data, differences of even a few cardiac events are clinically very meaningful.

Previous meta-analyses have examined blood pressure outcomes and reported small decreases ranging from < 1 to 6 mmHg for systolic pressure and from 1 to 4 mmHg for diastolic pressure [5,19,25,26,28–30,130,131]. In this synthesis, reductions in systolic and diastolic blood pressure were small and not significantly different from 0. Our search may have yielded more diverse studies than some previous meta-analyses that used limited search strategies and inclusion criteria [1,5,19,25,26,131]. Research has established neither the body's mechanisms for changing blood pressure in response to PA nor the optimal characteristics of training programs for this purpose [19].

This synthesis used extensive and intensive search strategies to locate diverse studies. Even so, this does not ensure that their findings represent the effectiveness of interventions to increase PA among the general population of adults with cardiac diseases. Clark et al. suggested that the magnitude of effects may be somewhat smaller in research studies than what many people would achieve because both the treatment and control groups receive optimal medical care [1]. The effects of interventions to increase PA and deal with secondary preventive behaviors may be more beneficial among patients who are treated where care is less optimal [1]. It is possible that researchers purposefully recruited participants for these studies who were more sedentary than typical cardiac populations. But the opposite is also possible -- that researchers recruited healthier subjects with less risk due to human subjects concerns or patients' willingness to participate in interventions [1]. The differences between these subjects and the general population of adults with cardiac disease are unknown. The benefits of increasing PA are generally less than what pharmacological therapies can achieve for certain outcomes [22]. Other important outcomes, such as fitness, may only change with PA. The changes documented in these studies may be over and above the benefits patients accrue from medical management.

This meta-analysis was limited by the studies retrieved. We analyzed only studies that included PA results. Given the small number of studies, readers should cautiously interpret findings about fitness, quality of life, blood pressure, anthropometric measures, and subsequent cardiac events. Many important study features, such as treatment fidelity or subject attributes like smoking, were unreported and thus could not be analyzed [1,30,32]. We expected the mixed pattern of publication bias because meta-analyses of various populations often report such bias. Publication bias may result when investigators choose not to report studies unless they have statistically significant findings or when journals choose not to publish papers of this kind [35]. Investigators may believe that pilot projects do not need to be disseminated when interventions are unsuccessful. Studies that lack statistically significant findings may be more likely to be presented in published papers without adequate data for calculating ES, thus excluding them from meta-analyses. Only when both effective and ineffective interventions become widely known can researchers build efficiently on the work that came before them.

Overall, this meta-analysis documents improved PA outcomes following interventions. PA is especially important among cardiac populations because it may not only reduce problems associated with type 2 diabetes and obesity but also influence lipids, cardiovascular functional capacity, and the atherosclerotic process [22,31]. This field would benefit from further research to clarify which components of interventions are associated with better PA outcomes and to determine whether interventions are differentially effective for samples with particular characteristics.

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*Denotes studies included in meta-analysis

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Table 1
Inclusion Criteria for Primary Studies

1	Study reported between 1970 and mid 2005.
2	Research report in English.
3	Subjects were at least 18 years of age.
4	All subjects with diagnosed cardiovascular disease or results reported separately for this group.
5	Intervention designed to increase physical activity and/or exercise.
6	Minimum of 5 subjects who received the intervention with physical activity outcome data.
7	Pre-experimental, quasi-experimental, or experimental design.
8	Adequate data to calculate physical activity effect size (e.g., means and variability measures, <i>t</i> statistic, exact <i>p</i> value from <i>t</i> test) comparing treatment and control group or comparing treatment group baseline and outcome values.

Table 2
Intervention Characteristics Reported by at Least 10 of the 79 Studies

Characteristic	Description	Frequency
Supervised exercise	Provision of supervised exercise sessions at a specified location.	52
Fitness testing	Testing subjects' physical fitness when the testing was intended to change behavior. Not coded when fitness testing measured outcomes but information was not provided to subjects as a strategy to change behavior.	39
Exercise prescription	Individualized specific form, intensity, frequency and/or duration of exercise suggested to each subject. May include prescription for progression of exercise.	33
Self-monitoring	Subjects record physical activity behavior. Includes activity diaries.	24
Monitoring	Research staff monitors subjects' physical activity performance.	23
Social modeling	Opportunities to watch similar others exercise.	18
Goal setting	Subjects generate written or oral behavioral change goals.	17
Modeling	Opportunities to watch others exercise who may not be similar to subjects.	16
Feedback	Explicit information provided to subjects about their performance. Could include a comparison with a group norm or standard or with subjects' own past behavior.	16
Social support	Deliberate building of social relationships to foster increased physical activity (e.g. self-help groups, 'social support'). Includes team building but not team competition for rewards.	12
Problem-solving	Conducting problem-solving activities (name problems, generate potential solutions specific to exercise behavior, decide on a course of action, and evaluate effects of solution implemented) during sessions with the investigator or teaching problem solving to subjects.	12
Consequences	Specific planned tangible or intangible positive consequences of behavior change (e.g. lottery for prizes, praise, token economy, competition that has a reward, contracting for a reward, social reinforcement) when the research team or subject administers the reward.	12
Barriers management	Teaching subjects to overcome or manage barriers (obstacles, deterrents). Could include having subjects identify barriers and brainstorm strategies to overcome them or could be teaching methods to overcome researcher-identified barriers.	10

Table 3
Characteristics of Primary Studies Included in Cardiac Meta-Analyses

Characteristic	<i>k</i>	Min	<i>Q</i> ₁	<i>Mdn</i>	<i>Q</i> ₃	Max
Mean age (years)	76	35	56	59	64	78
Sample size per study	79	14	36	67	153	1173
Proportion of sample assigned to treatment group [*]	56	0.25	0.49	0.51	0.55	0.77
Proportion attrition from treatment group	62	0.00	0.05	0.12	0.22	0.39
Proportion attrition from comparison group [*]	47	0.00	0.05	0.13	0.22	0.59
Proportion female	65	0.00	0.14	0.25	0.31	1.00
Minutes of supervised exercise per session	33	10	50	60	60	75
Total number of supervised exercise sessions	35	6	26	36	48	202
Number of weeks intervention was delivered	69	1	8	12	26	208
Days after intervention outcome was measured	74	0	0	0	180	2109

Note. Includes all studies that contributed at least 1 independent-groups or pre-post effect size to primary analyses. Independent samples within studies aggregated by summing sample sizes before computing proportions and using weighted mean of other characteristics (weighted by sample size).

k = number of studies providing data on characteristic; *Q*₁ = first quartile, *Q*₃ = third quartile.

^{*} Computed only for studies that contributed 2-group comparisons.

Table 4
Fixed- and Random-Effects Estimates and Tests: Independent-Groups Comparisons at Distal Posttest

Dependent variable	k	Fixed-effects analysis				Random-effects analysis			
		<i>d</i>	<i>SE</i> (<i>d</i>)	95% CI for δ	<i>Q</i>	$\hat{\mu}_{\delta}$	<i>SE</i> ($\hat{\mu}_{\delta}$)	95% CI for μ_{δ}	$\hat{\sigma}_{\delta}^2$
Physical activity	62	0.31 [‡]	0.025	(0.26, 0.36)	116.65 [‡]	0.35 [‡]	0.039	(0.27, 0.42)	0.177
Fitness	18	0.15 [‡]	0.053	(0.05, 0.26)	32.06 [*]	0.17 [‡]	0.065	(0.04, 0.30)	0.117
Quality of life	16	0.20 [‡]	0.058	(0.09, 0.31)	25.38 [*]	0.24 [‡]	0.087	(0.07, 0.41)	0.184
Anthropometric	13	0.03	0.044	(-0.06, 0.11)	33.10 [‡]	0.07	0.083	(-0.10, 0.23)	0.219
Systolic BP	12	0.02	0.051	(-0.08, 0.12)	2.93	—	—	—	0.000
Diastolic BP	12	0.07	0.051	(-0.03, 0.17)	14.38	0.08	0.070	(-0.06, 0.22)	0.121
Cardiac events	6	0.23 [‡]	0.070	(0.09, 0.36)	4.68	—	—	—	0.000

Note. Fitness and systolic and diastolic blood pressure include 1 dependent multiple-treatment pair. PA and fitness include 1 dependent multiple-treatment triplet. Quality of life includes 3 dependent multiple-treatment pairs. PA includes 7 dependent multiple-treatment pairs.

k: number of comparisons; *d*: effect size; *Q*: heterogeneity statistic (*Q* is distributed approximately as chi-square with $df = k - 1$, where *k* is the number of (possibly dependent) observed effect sizes); $\hat{\mu}_{\delta}$: mean of true effect sizes; $\hat{\sigma}_{\delta}^2$: SD of true effect sizes.

* $p < .05$,

[‡] $p < .01$,

[#] $p < .001$ (for $H_0: \delta = 0$, $H_0: \mu_{\delta} = 0$, or $H_0: \sigma_{\delta}^2 = 0$).

Table 5
Fixed- and Random-Effects Estimates and Tests: Treatment Pretest versus Distal Posttest

Dependent variable	<i>k</i>	Fixed-effects analysis			Random-effects analysis				
		<i>d</i>	<i>SE(d)</i>	95% CI for δ	<i>Q</i>	$\hat{\mu}_{\delta}$	<i>SE</i> ($\hat{\mu}_{\delta}$)	95% CI for μ_{δ}	$\hat{\sigma}_{\delta}$
				$\rho_{12} = .80$					
Physical activity	51	0.33 [§]	0.012	(0.30, 0.35)	782.10 [§]	0.49 [§]	0.054	(0.39, 0.60)	0.355
Fitness	28	0.46 [§]	0.017	(0.43, 0.49)	197.69 [§]	0.43 [§]	0.051	(0.33, 0.53)	0.231
Anthropometric	27	0.16 [§]	0.013	(0.13, 0.18)	126.66 [§]	0.18 [§]	0.032	(0.12, 0.25)	0.136
Systolic BP	19	0.11 [§]	0.018	(0.08, 0.15)	102.37 [§]	0.09 [*]	0.049	(−0.00, 0.19)	0.177
Diastolic BP	19	0.18 [§]	0.018	(0.15, 0.22)	115.15 [§]	0.15 [‡]	0.051	(0.05, 0.25)	0.190
Quality of life	18	0.35 [§]	0.020	(0.31, 0.39)	101.54 [§]	0.31 [§]	0.055	(0.20, 0.42)	0.196
				$\rho_{12} = .00$					
Physical activity	56	0.45 [§]	0.026	(0.40, 0.50)	300.86 [§]	0.57 [§]	0.067	(0.44, 0.70)	0.419
Fitness	28	0.54 [§]	0.035	(0.47, 0.61)	33.49	0.53 [§]	0.044	(0.44, 0.61)	0.095
Anthropometric	27	0.16 [§]	0.029	(0.11, 0.22)	27.10	0.16 [§]	0.030	(0.10, 0.22)	0.031
Systolic BP	19	0.12 [‡]	0.039	(0.04, 0.19)	40.30 [‡]	0.10	0.064	(−0.03, 0.22)	0.186
Diastolic BP	19	0.21 [§]	0.039	(0.14, 0.29)	37.52 [‡]	0.21 [§]	0.062	(0.09, 0.33)	0.174
Quality of life	18	0.45 [§]	0.042	(0.36, 0.53)	24.92 [*]	0.40 [§]	0.059	(0.28, 0.51)	0.127

k: number of comparisons; *d*: effect size; *Q*: heterogeneity statistic (*Q* is distributed approximately as chi-square with $df = k - 1$, where *k* is the number of observed effect sizes); $\hat{\mu}_{\delta}$: mean of true effect sizes; $\hat{\sigma}_{\delta}$: SD of true effect sizes.

*
 $p < .10$,

[‡]
 $p < .05$,

[‡]
 $p < .01$,

[§]
 $p < .001$ (for $H_0: \delta = 0$, $H_0: \mu_{\delta} = 0$, or $H_0: \sigma_{\delta}^2 = 0$).

Table 6
Fixed- and Random-Effects Estimates and Tests: Comparison/Control Group Pretest versus Distal Posttest

Dependent variable	k	Fixed-effects analysis			Random-effects analysis				
		d	SE(d)	95% CI for δ	Q	μ̂ _δ	SE(μ̂ _δ)	95% CI for μ _δ	σ̂ _δ
				ρ ₁₂ = .80					
Physical activity	23	0.14 [‡]	0.020	(0.10, 0.17)	277.22 [‡]	0.06	0.075	(−0.09, 0.20)	0.332
Anthropometric	13	0.00	0.020	(−0.04, 0.04)	4.12	—	—	—	0.000
Fitness	11	0.12 [‡]	0.029	(0.06, 0.18)	31.93 [‡]	0.12 [‡]	0.059	(−0.00, 0.23)	0.150
Systolic BP	11	−0.10 [‡]	0.024	(−0.14, −0.05)	65.33 [‡]	−0.10	0.072	(−0.24, 0.04)	0.204
Diastolic BP	10	0.01	0.024	(−0.04, 0.05)	33.26 [‡]	0.05	0.054	(−0.06, 0.15)	0.134
Quality of life	7	0.14 [‡]	0.042	(0.06, 0.22)	20.96 [‡]	0.13	0.098	(−0.06, 0.32)	0.204
				ρ ₁₂ = .00					
Physical activity	23	0.16 [‡]	0.042	(0.07, 0.24)	68.21 [‡]	0.08	0.084	(−0.08, 0.25)	0.300
Anthropometric	13	0.00	0.044	(−0.08, 0.09)	0.82	—	—	—	0.000
Fitness	11	0.12 [*]	0.064	(−0.00, 0.25)	6.66	—	—	—	0.000
Systolic BP	11	−0.10 [*]	0.052	(−0.20, 0.00)	14.24	−0.12	0.073	(−0.26, 0.03)	0.123
Diastolic BP	11	−0.02	0.052	(−0.12, 0.09)	13.73	−0.02	0.070	(−0.16, 0.12)	0.114
Quality of life	7	0.15	0.092	(−0.03, 0.33)	4.53	—	—	—	0.000

k: number of comparisons; *d*: effect size; *Q*: heterogeneity statistic; $\hat{\mu}_{\delta}$: mean of true effect sizes; $\hat{\sigma}_{\delta}$: SD of true effect sizes;

ρ_{12} : a measure of association. Under homogeneity *Q* is distributed approximately as chi-square with *df* = *k* - 1, where *k* is the number of observed effect sizes.

* $p < .10$,

[‡] $p < .01$,

[‡] $p < .001$ (for $H_0: \delta = 0$, $H_0: \mu\delta = 0$, or $H_0: \sigma\delta^2 = 0$).

Table 7
Univariate Dichotomous Moderator Results: Independent-Groups Comparisons on Physical Activity at Distal Posttest

Moderator	Fixed-effects analysis						Mixed-effects analysis					
	k_0	k_1	$\hat{\sigma}_0$	$\hat{\sigma}_1$	SE_{diff}	Q_B	Q_W	$\hat{\mu}_{\delta_0}$	$\hat{\mu}_{\delta_1}$	SE_{diff}	Q_B	$\hat{\sigma}_{\delta}$
Funding	17	45	0.45 [§]	0.30 [§]	0.074	4.0 [†]	116.8 [§]	0.45 [§]	0.34 [§]	0.098	1.1	0.191
Ex. prescription	43	19	0.33 [§]	0.29 [§]	0.054	0.3	120.4 [§]	0.37 [§]	0.35 [§]	0.087	0.1	0.200
Fitness testing	42	20	0.30 [§]	0.38 [§]	0.059	1.8	118.9 [§]	0.33 [§]	0.48 [§]	0.091	2.7 [*]	0.197
Goal setting	51	11	0.34 [§]	0.22 [§]	0.060	3.9 [†]	116.9 [§]	0.40 [§]	0.21 [†]	0.105	3.3 [*]	0.196
Self-monitoring	48	14	0.31 [§]	0.36 [§]	0.064	0.5	120.2 [§]	0.35 [§]	0.41 [§]	0.096	0.3	0.198
Supervised ex.	37	25	0.27 [§]	0.42 [§]	0.055	7.6 [†]	113.2 [§]	0.31 [§]	0.46 [§]	0.080	3.9 [†]	0.187
Behavioral target	23	39	0.41 [§]	0.29 [§]	0.060	4.3 [†]	116.5 [§]	0.47 [§]	0.32 [§]	0.086	3.0 [*]	0.192
Int. social context	25	36	0.32 [§]	0.32 [§]	0.050	0.0	117.7 [§]	0.38 [§]	0.37 [§]	0.081	0.0	0.198
Mediated delivery	45	17	0.35 [§]	0.23 [§]	0.056	4.3 [†]	116.5 [§]	0.41 [§]	0.25 [§]	0.089	3.0 [*]	0.193
Rec. specific ex.	53	9	0.32 [§]	0.30 [§]	0.072	0.1	120.7 [§]	0.37 [§]	0.34 [§]	0.109	0.1	0.199

Moderator levels: funding (0 = no funding, 1 = funding); sex (1 = female, 0 = male); behavioral target (1 = multiple health behaviors, 0 = physical activity only); intervention social context (1 = group, 0 = individual); mediated delivery (1 = mail, telephone, mass media, or internet, 0 = face-to-face only); recommended specific exercise (1 = walking, 0 = other than walking); all others (1 = present, 0 = absent). k_j = number of (possibly dependent) ES estimates in group j ; heterogeneity statistics: Q_B = between groups (distributed as chi-square on $df = 1$ under H_0 : $\delta_0 = \delta_1$ or H_0 : $\mu\delta_0 = \mu\delta_1$), Q_W = combined within groups (distributed as chi-square on $df = k_0 + k_1 - 2$ under H_0 : $\sigma_{\delta_j}^2 = 0$); $\hat{\mu}_{\delta}$: mean of true effect sizes; $\hat{\sigma}_{\delta}$: SD of true effect sizes Moderator analysis conducted only if $k_0 \geq 3$ and $k_1 \geq 3$.

*
 $p < .10$,

†
 $p < .05$,

‡
 $p < .01$,

§
 $p < .001$ (for H_0 : $\delta_j = 0$, H_0 : $\delta_0 = \delta_1$, H_0 : $\sigma_{\delta_j}^2 = 0$, H_0 : $\mu\delta_j = 0$, or H_0 : $\mu\delta_0 = \mu\delta_1$).

Table 8
Univariate Continuous Moderator Results: Independent-Groups Comparisons on Physical Activity at Distal Posttest

Moderator	k	Fixed-effects analysis			Mixed-effects analysis				
		β_0	β_1	$SE(\beta_1)$	Q_{residual}	β_0	β_1	$SE(\beta_1)$	$\hat{\sigma}_\delta$
Publication year	62	0.412	−0.004	0.0039	119.9 [‡]	0.471	−0.004	0.0057	0.197
Mean age	61	0.134	0.003	0.0043	117.5 [‡]	0.093	0.005	0.0066	0.206
Proportion female	56	0.335	−0.032	0.1290	108.7 [‡]	0.375	−0.031	0.1871	0.200
Attrition proportion	48	0.358	−0.465	0.3140	85.5 [‡]	0.400	−0.431	0.4627	0.179
Contact time	26	−0.060	0.160 [‡]	0.0523	42.7 [*]	−0.137	0.205 [*]	0.0926	0.204
Weeks of int.	58	0.280	0.035	0.0331	117.9 [‡]	0.347	0.025	0.0600	0.208
Rec. min/wk	17	−0.087	0.004 [*]	0.0019	17.2	−0.042	0.004 [*]	0.0021	0.104

k = number of (possibly dependent) ES estimates. Q_{residual} = residual heterogeneity statistic, beyond that due to moderator (distributed as chi-square on $df = k - 2$ under H_0 : $\sigma_{\delta|x}^2 = 0$, where x is moderator value). Between-studies variance component σ_{δ}^2 estimated via weighted method of moments. Moderator analysis conducted only if $k \geq 6$.

k: number of comparisons; β_0 : regression intercept; β_1 : regression slope; Q : heterogeneity statistic; $\hat{\sigma}_\delta$: SD of true effect sizes.

* $p < .05$,

[‡] $p < .01$,

[‡] $p < .001$ (for H_0 : $\beta_1 = 0$ or H_0 : $\sigma_{\delta|x}^2 = 0$).