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# A Meta-Analysis of Psychoeducational Programs for Coronary Heart Disease Patients

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In a meta-analysis of 37 studies, the effects of psychoeducational (health education and stress management) programs for coronary heart disease patients were examined. The results suggest that these programs yielded a 34% reduction in cardiac mortality; a 29% reduction in recurrence of myocardial infarction (MI); and significant ($p < .025$) positive effects on blood pressure, cholesterol, body weight, smoking behavior, physical exercise, and eating habits. No effects of psychoeducational programs were found in regard to coronary bypass surgery, anxiety, or depression. The results also suggest that cardiac rehabilitation programs that were successful on proximal targets (systolic blood pressure, smoking behavior, physical exercise, emotional distress) were more effective on distal targets (cardiac mortality and MI recurrences) than programs without success on proximal targets.

*Key words:* cardiac rehabilitation, stress management, health education, coronary heart disease, meta-analysis

About 30 years ago, it was widely believed that physical training should be the cornerstone of cardiac rehabilitation programs, which were designed to improve physical fitness and return the patient to work. Over the last few decades, the objectives of cardiac rehabilitation have gradually expanded to include facilitating the patient's return to his or her usual way of life before the cardiac event not only in a professional sense but also in a much wider physical, personal, and social sense (Mulcahy, 1990). In addition, there is growing awareness that secondary prevention should be added to the traditional goals. In this context, *secondary prevention* refers to the reduction of cardiac mortality and morbidity, through pharmacological therapy, surgery, and risk factor modification. This implies that the patient should be encouraged to return to his or her former way of life, with the exception of tobacco smoking, physical inactivity, alcohol abuse, and other behaviors conducive to hyperlipidaemia, hypertension, diabetes, and excessive weight.

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A number of qualitative and quantitative reviews have assessed the effects of various types of cardiac rehabilitation programs. In the meta-analyses of Oldridge, Guyatt, Fischer, and Rimm (1988) and O'Connor et al. (1989), exercise-based rehabilitation programs were proved to reduce all-cause mortality, cardiovascular mortality, and the fatal reinfarction rate. According to the authors of these meta-analyses, however, the fact that most trials involved formal or informal nonexercise components precluded the possibility of reaching definitive conclusions about "exercise only" trials.

Whereas Oldridge et al. (1988) and O'Connor et al. (1989) focused mainly on exercise-based rehabilitation, in a meta-analysis of 18 controlled studies Nunes, Frank, and Kornfeld (1987) showed that a reduction in Type A behavior was a consequence of treatments involving various combinations of education, behavior modification, cognitive therapy, psychodynamic therapy, relaxation, imaging, and emotional support. The morbidity and mortality findings have to be interpreted cautiously, however, because these data were based on only 2 studies. The results suggest that a combination of treatment techniques was most effective in reducing Type A behavior and cardiac recurrences.

In addition, Ketterer (1993) compared risk reduction associated with behavioral therapies (psychoeducational interventions) with risk reduction associated with medical therapy offered to ischemic heart disease patients. With the possible exception of aspirin use in patients with unstable angina, the relative risk reduction of nonfatal myocardial infarctions (MIs) and cardiac deaths observed so far for behavioral therapies are superior to those for any form of medical therapy. Medical therapies varied from administration of beta blockers, antiplatelets, and calcium blockers to aspirin, intravenous heparin, and even coronary artery bypass grafting (CABG). Ketterer's review thus documents promising outcomes for behavioral therapies as compared with medical therapy.

Psychosocial or psychoeducational interventions may affect rehabilitation outcomes in two ways. First, they can facilitate psychosocial recovery, including the patient's return to everyday activities. Second, they can play an important role in secondary prevention, by encouraging compliance with medical advice and behavior change related to risk factor modification. Most interventions of the first type can be characterized as stress management (SM) programs, and interventions of the second type focus on behavior modification and health education (HE).

In two meta-analyses, a rough distinction was made between HE and SM programs. Mullen, Mains, and Velez's (1992) meta-analysis of 28 controlled trials emphasized the effects of education programs in cardiac rehabilitation, whereas W. Linden, Stossel, and Maurice's (1996) meta-analysis of 23 controlled trials included all interventions that appeared psychological or of a counseling nature while excluding studies of educational interventions. However, both of these meta-analyses included programs that combined HE and SM. The sample of studies used in the two meta-analyses showed an overlap of three trials. Favorable effects were observed by Mullen et al. on mortality, systolic blood pressure, exercise, and diet, but not on recurrence of MI and smoking. Mullen et al. also investigated the effects of several moderating variables. Program quality (i.e., a total score on five educational principles: individualization, feedback, facilitation, relevance, and reinforcement) showed a significant positive association with study effect size. Length of intervention, length of measurement period, intervention emphasis, and channel of intervention were not significantly associated with study effect size. W. Linden et al. found an overall reduction in mortality and nonfatal MI recurrence at short-term follow-up (2 years or less) and at long-term follow-up (2–8 years). The review also showed positive effects on systolic blood pressure and cholesterol, although only four trials reported results on these measures. The effects of moderating variables were not investigated by W. Linden et al.

These two meta-analyses leave room for theoretical and methodological improvements on several important points. First, the model on which most of the research reviewed is based assumes that emotional distress and risk factors and related behaviors contribute to cardiac morbidity and mortality. The rationale for SM and HE programs is that by reducing stress, risk factors, or both, recurrences of and death from coronary heart disease (CHD) also will be reduced. Effects of both SM and HE programs on proximal targets (e.g., reduced Type A behavior, reduced emotional distress, smoking cessation) as well as on distal outcomes need, therefore, to be evaluated, taking into account the changes in proximal targets.[1] Existing meta-analyses have failed to do so. Second, the two meta-analyses fail to provide an operational definition of the types of interventions under investigation, resulting in an agglomeration of different program types. In our view, distinguishing among types of psychoeducational programs is vital to a better understanding of why some programs are more effective than others. Third, if a study reported two trials using the same control group, both trials were used to estimate the population effect size: This procedure violates the assumption of independence of study effect sizes (Matt & Cook, 1994). Fourth, posttest effect sizes were not adjusted for pretest differences. When quasi-experiments are included, as in Mullen et al.'s (1992) review, adjustment for pretest differences is recommended (Heinsman & Shadish, 1996). Although W. Linden et al. (1996) stated that their review included only randomized trials, their sample did include one quasi-experiment (i.e., Munro, Creamer, Haggerty, & Cooper, 1988). In our view, in both randomized and nonrandomized trials population effect size estimates will be more accurate when pretest differences are taken into account. As Ketterer (1993) stated, correcting for base rates is "the only meaningful way to compare efficacy of therapies" (p. 480). Fifth, comparison of the results of the reviews is difficult, because different effect size statistics were used.

In the present meta-analysis we tried to overcome these limitations by (a) taking proximal targets into account in analyses of distal outcomes; (b) distinguishing among SM programs, HE programs, and programs combining both components; (c) selecting only one trial in studies reporting multiple trials; (d) adjusting posttest effect sizes by pretest differences; and (e) expressing population effect sizes in different ways. Furthermore, this review complements the existing reviews in that it examines a larger sample of studies ($k = 37$), including more recent ones, and explores the moderating influence of study features, which the existing reviews did not examine.

The first objective of this meta-analysis was to assess the effects of psychoeducational programs for CHD patients on cardiac outcomes and physical health outcomes in a quantitative way. We focused on two types of psychoeducational programs: SM and HE. The second objective was to test the hypothesis that success on proximal targets contributes to a reduction of cardiac mortality and cardiac recurrences. The third objective was to explore the moderating effects of other key study features. We examined the following cardiac outcome variables: cardiac mortality, recurrence of MI, CABG, and incidence of angina pectoris, and we examined the following proximal targets: risk factors (blood pressure, cholesterol, weight, smoking) and related behaviors (physical exercise, eating habits) and emotional distress (anxiety, depression).

## Method

### Selection of Studies

*Literature search.* A PsychLIT and MedLINE computer search was performed to locate relevant studies published between 1974 and 1998. Three types of key words were used. The first type concerned patient characteristics: *MI, heart surgery, coronary bypass, percutaneous transluminal coronary angioplasty* (*PTCA*), *CHD, heart disorder,* and *cardiovascular disorder.* The second type

[1] We would like to acknowledge the recommendation of Peter Barnett, who raised this point.

of key word concerned the outcome variables: *mortality, morbidity, blood pressure, cholesterol, overweight, weight, obesity, smoking, physical exercise, nutrition, food, anxiety, depression, anger, emotional distress, well-being,* and *quality of life.* The third type of key word concerned the type of program: *cardiac rehabilitation, psychological or psychoeducational intervention, education, stress management, training, therapy, counseling,* and *relaxation.* The reference lists from empirical and review studies found in the computer search were used to locate additional studies.

*Inclusion criteria.* We limited the meta-analysis to studies with patients who had experienced a cardiac event within the 6 months prior to the start of the treatment. A cardiac event was defined as MI, CABG, PTCA, or some combination of these.

Because the primary focus of this meta-analysis was effects on cardiac outcomes and physical health outcomes, studies reporting effects on emotional distress were included only if effects on risk factors, related behaviors, morbidity, or cardiac mortality also were reported.

Additional criteria for inclusion involved study design. Only relatively high-quality studies were included in the meta-analysis, that is, studies that included a control condition or comparison condition (i.e., randomized experiments or quasi-experiments, see Cook & Campbell, 1979). Quasi-experiments were included only when samples were stratified or matched pairwise, or when a certain time period was used as an assignment rule for patients from the same hospital. For risk factors, related behaviors, and psychological variables, a pretest measurement was required. Studies reporting that the difference between treatment and control group was "not significant," without giving exact results, were included.

*Problems of multiplicity.* We made decisions about four types of problems of multiplicity. The first type concerned multiple articles reporting the same study. When this occurred, the one key article reporting the most data was selected. The other articles were used to locate supplementary data (about follow-up measurements or other outcome measures). The second type concerned multiple experimental conditions reported within a single study, implying multiple comparisons between them (this occurred in nine studies). To avoid dependency of study effect sizes (Matt & Cook, 1994), one comparison was selected from each of these nine studies. One selection rule was used for all studies: Given random assignment, the chosen comparison was the one between the experimental condition with the most extensive psychosocial treatment versus a standard care condition. The remaining types of multiplicity concerned multiple measurement points and multiple measures, which are discussed below, following the *Effect Size Computation* section.

## Coding

Two independent raters (one methodologist and one health psychologist) coded study features and effect size data. In cases of divergence between the coding of the two raters, a third rater (a second health psychologist) chose the most plausible coding. The studies were categorized by type of evaluation, which was a direct consequence of the classification of the type of treatment condition and control–comparison condition (e.g., if the treatment condition received HE and physical training, and the comparison condition received only physical training, then the type of evaluation was HE). *Health education* was defined as instructional activities organized in a systematic way, involving personal contacts between a health professional and coronary heart patients (and partners) to facilitate positive changes in risk factors for CHD and related unhealthy behaviors. *Stress management* was defined as psychotherapeutic interventions or relaxation training or supportive interventions (i.e., the opportunity or facility to express emotions or talk about problems). Both SM and HE had to include at least one face-to-face session. *Physical training* was defined as actual exercise training (ET). Information provision, which was not organized in a systematic way (e.g., personal communication with the cardiologist, free provision of leaflets for all patients), was considered *standard care.* This resulted in five types of evaluation: SM only; HE only; HE and ET; HE and SM; and HE, SM, and ET. Additionally, the following study features were coded: year of publication (of the key article), use of random assignment, use of matching, the country where the research took place, type of patient (MI, CABG, PTCA, or combinations), mean age of patients, gender of patients (percentage female), exclusion criteria in selection of patients (e.g., only severe patients, only smokers), setting of the program (inpatient, outpatient, or both), the measurement point of the pretest (number of weeks after the coronary event), length of the program (in weeks and in sessions), profession of program providers, participation of partners, target of the treatment (individual patient, patient group, or both), and five indicators of program quality (i.e., those used by Mullen et al. [1992], see beginning of article).

Moreover, data were coded for the computation of effect sizes: the measurement point of each posttest (number of weeks after start of the program); the operationalization of each outcome variable (description and unit of measurement); and, for each outcome variable and each measurement (also, if applicable, for the pretest), the exact results (observed cell frequencies for categorical data, and observed means, standard deviations, and sample sizes for continuous data). If exact results were not reported, other available statistics were coded (e.g., $p$ values). If an article reported only that an effect was nonsignificant, this was coded as well.

Additionally, the quality of the measurement and the quality of the data were coded for each outcome variable separately. Finally, the proximal outcomes were coded on whether they were a targeted outcome of the intervention. The distal outcomes, cardiac mortality and cardiac recurrences, were coded on whether the study had succeeded in achieving the proximal intervention target(s). When a study did not formulate the proximal targets explicitly, the following procedure was applied. For studies categorized as evaluating HE, risk factors and related behaviors were considered proximal intervention targets. For studies categorized as evaluating SM, measures of emotional distress (i.e., anxiety, depression) were considered proximal intervention targets. Success on proximal targets was defined as significant favorable results on all proximal targets measured in the study, at one or more measurement points. When both significant and nonsignificant results were achieved on these targets, a study was considered as having had partial success. When no significant or significant unfavorable results were achieved, a study was considered as having failed.

## Effect Size Computation

For continuous data, we computed Cohen's $d$ from the observed means and standard deviations of the treatment and control conditions (Rosenthal, 1994, p. 237). For categorical data, we calculated odds ratios and phi coefficients (Fleiss, 1994, pp. 249–251, Formulas 17-18 and 17-11). If any cell frequency was zero, we added 0.5 to all cell frequencies. To compare and to combine effect sizes on the basis of both continuous and categorical data, we converted the above-mentioned effect sizes into correlation measures ($r$). Cohen's $d$ was converted directly into $r$ (Rosenthal, 1994, p. 239, Formula 16-24). Odds ratios were first converted into a chi-square measure (Fingleton, 1984, p. 6, second formula) and then into $r$ (Rosenthal, 1994, p. 237, Formula 16-17). The resulting $r$ values were almost equal to the phi coefficients in the case of high cell frequencies but were lower, and thus more conservative, for low cell frequencies. Therefore, we used the converted odds ratios, not the phi coefficients in further analyses (for an extensive overview of problems with the phi coefficient, see Fleiss, 1994).

If exact results were missing and only a significance level was reported, we looked up the corresponding $z$ value in a table of standard normal deviates and then converted this $z$ value into $r$ (Rosenthal, 1994, p. 237, Formula 16-17). When a study reported only a nonsignificant effect, we assumed a one-tailed $p$ level of .50 for continuous data, resulting in an effect size ($r$) of zero. In the case of categorical data, equal cell frequencies were assumed, resulting in an odds ratio of 1.

We made decisions concerning the two remaining types of multiplicity: multiple measurement points and multiple measures for one outcome variable. With respect to the first type, we classified the measurement points used in the posttests into three measurement periods: less than 1 year after start of the program (short-term period), 1–2 years (medium-term period), or longer than 2 years (long-term period); within each of these periods only one effect size per study was computed (if available). If a study reported several posttests within a measurement period, the last posttest within that period was chosen. In addition to these three periods, we defined a total measurement period as the period ending at a study's final posttest measurement. With respect to the second type of multiplicity, if some studies reported several measures for the same outcome variable, the measure used in most studies was selected to increase the reliability of the effects over studies (see Results section).

### Statistical Analysis

To estimate a population effect size for each outcome variable, we multiplied the study effect size estimates ($r$s) within one measurement period by the corresponding sample sizes and then combined them using Fisher's $r$-to-$z$ transformation (Rosenthal, 1994; Shadish & Haddock, 1994). To facilitate comparison of our results (see beginning of article), we also expressed the population effect size as a standardized mean difference (Cohen's $d$), by converting the weighted average effect size (WAES) $r$ (Rosenthal, 1994, p. 239, Formula 16-24).

For the dependent variables with a pretest measurement, we estimated the population effect size with the adjusted posttest effect sizes. To obtain adjusted effect sizes, we performed a weighted linear regression analysis with the pretest difference as predictor (expressed as an effect size, see Hedges & Olkin [1985] and Heinsman & Shadish [1996]) and the posttest effect size as dependent variable. Before performing the regression analyses, we transformed the study effect sizes using Fisher's $r$-to-$z$ transformation.

To enable comparison of our results for mortality and morbidity with those in the review of W. Linden et al. (1996), we combined the odds ratios using the Mantel–Haenszel summary estimate of the odds ratio (Shadish & Haddock, 1994, p. 271, Formula 18-14).

Because the evaluated psychoeducational programs for CHD patients can be seen as a sample of all psychoeducational programs implemented for CHD patients, a random-effects model was assumed. In this model the variation of observed effect sizes partly mirrors the true variation of the population effect size; therefore, the observed variance can be considered to have two components: true population variance and sampling error. We used the software program META 5.3 (Schwarzer, 1989a) to estimate the WAES $r$ and its variance by means of the Hunter–Schmidt method (Schwarzer, 1989b). To reduce the chance of Type I errors due to multiple testing, we used the false discovery rate controlling procedure (Benjamini & Hochberg, 1995, p. 293, Formula 1) within each outcome measure. The correction was computed with $p^* < .025$, where $p^*$ indicates the controlled one-tailed $p$ value. The number of tested hypotheses ($m$) varied for each outcome measure (e.g., for cardiac mortality, $m = 6$; for recurrence of MI, $m = 10$).

Effect sizes were considered to be homogeneous if the following two conditions were satisfied: The percentage of observed variance accounted for by sampling error was at least 75%, and the chi-square test of homogeneity was not significant (see Schwarzer, 1989b). In cases of heterogeneity, moderators (i.e., the study features described in the *Coding* section) were sought that might account for the systematic variation in the effect sizes. Moderators were not sought if the number of studies was too small (five or fewer) to make a meaningful examination of differences between subgroups of studies defined by the moderator. In the first place, we investigated the effect of the moderator "success on proximal targets" for the studies reporting cardiac mortality, cardiac recurrences, or both. Because there were no a priori hypotheses about the effects of other moderators, we subsequently performed disjoint cluster analysis (using META 5.3) to identify possible clusters of similar effect sizes. Additionally, we examined study features as potential bases for differences in effect sizes. For each level of a moderator, we computed an estimate of the population effect size separately. A study feature was considered to be a moderator if its categories (or combinations of categories) identified distinct homogeneous sets of study effect sizes.

## Results

### Description of Study Features

Thirty-seven studies, reported in 52 publications, were included in the meta-analysis. The earliest study was published in 1974, and the most recent study was published in 1997 (see Table 1 for a complete overview of studies and features). Most studies evaluated the effects of an HE-and-SM program. In 4 of these studies the treatment group also followed an ET program, but this program was offered to the comparison group as well. In 3 of the studies evaluating the effect of SM, the treatment group also followed an HE or ET program, but this program was offered to the comparison group as well. The Cohen's kappa for this classification of the 37 studies was .85, indicating good agreement of the raters (Landis & Koch, 1977).

Nine studies used a quasi-experimental design. Two of these studies used matching by pairs (Perk, Hedbäck, & Engvall, 1990; Stransky, Kälin, Schwarzenbach, & Schär, 1986), four studies used matching by stratified sampling (Dracup et al., 1984; Hedbäck & Perk, 1987; Munro et al., 1988; Salonen, Hämynen, & Heinonen, 1985), and the remaining three studies used a certain time period as a rule for assigning patients to conditions.

One study included only "severe" patients: Stern, Gorman, and Kaslow (1983) selected patients with low exercise capacity and high anxiety, depression, or both. One study included only nonsmokers (Friedman et al., 1986), and two studies included only smokers (Salonen et al., 1985; Taylor, Houston-Miller, Killen, & DeBusk, 1990).

On average, the pretest measurement occurred 4 weeks after the cardiac event. The programs varied substantially in

Table 1
*Study Features Categorized by Type of Evaluation*

| Study | $N^a$ | Co | Ra | Typ | Ag | Fe | Cou | Set | Pr | Dur | Ses | Mu | Psy | Tar | Par |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Health education, stress management, and exercise training | | | | | | | | | | | | | | | |
| Bengtsson (1983) | 126 | SC | yes | MI | 56 | 15 | Eur | o ≥ 6 | 4 | 13 | 26 | yes | no | in + g | yes |
| DeBusk et al. (1994) | 585 | SC | yes | MI | 57 | 21 | Am | i + o | 1 | 52 | 18 | no | no | in | no |
| Engblom, Rönnemaa, et al. (1992) | 228 | SC | yes | CA | 54 | 12 | Eur | i + o | 0 | 26 | 38 | yes | yes | in + g | no |
| Erdman & Duivenvoorden (1983) | 64 | SC | yes | MI | 51 | 0 | Eur | o ≥ 6 | 26 | 26 | 52 | yes | yes | g | no |
| Fridlund et al. (1991) | 116 | SC | yes | MI | 56 | 13 | Eur | i + o | 2 | 26 | 33 | yes | no | in + g | yes |
| Kallio et al. (1979) | 375 | SC | yes | MI | 54 | 20 | Eur | o < 6 | 2 | 14 | | yes | yes | in | no |
| B. Linden (1995) | 34 | SC | yes | MI | | | Eur | i + o | 1 | 8 | 4 | no | no | in | yes |
| Oldenburg et al. (1989) | 40 | SC | yes | MI | 59 | 24 | Aus | i + o | 2 | 10 | 27 | yes | yes | in + g | no |
| Salonen et al. (1985) | 96 | SC | no | MI | | 0 | Eur | i + o | 2 | 52 | 24 | yes | yes | in + g | yes |
| Health education and stress management | | | | | | | | | | | | | | | |
| Dracup et al. (1984) | 36 | ET | no | Mix | 57 | 10 | Am | o ≥ 6 | | 10 | 10 | no | no | g | yes |
| Frasure-Smith & Prince (1985) | 461 | SC | yes | MI | 58 | 0 | Can | o < 6 | 2 | 52 | 18 | no | no | in | no |
| Frasure-Smith et al. (1997) | 1,376 | SC | yes | MI | 59 | 34 | Can | o < 6 | 2 | 52 | 18 | no | no | in | no |
| Horlick et al. (1984) | 116 | SC | yes | MI | 53 | 9 | Can | o < 6 | 2 | 6 | 6 | yes | yes | g | yes |
| Mayou (1983) | 85 | SC | yes | MI | 51 | 0 | Eur | o < 6 | 0 | 4 | 4 | no | yes | in | yes |
| Oldenburg et al. (1985) | 29 | SC | yes | MI | 56 | 11 | Aus | i | 1 | 2 | 10 | yes | yes | in | no |
| Pozen et al. (1977) | 38 | SC | yes | MI | 58 | 21 | Am | i + o | 1 | 26 | 35 | no | no | in + g | yes |
| Rahe et al. (1975) | 57 | SC | yes | MI | 50 | 7 | Am | o < 6 | 1 | 12 | 6 | yes | yes | g | yes |
| Sivarajan et al. (1983) | 125 | ET | yes | MI | 56 | 13 | Am | o < 6 | 1 | 8 | 8 | no | no | g | yes |
| Stern et al. (1983) | 64 | SC | yes | MI | 54 | 14 | Am | o ≥ 6 | 26 | 12 | 12 | yes | yes | g | no |
| Taylor et al. (1990) | 130 | SC | yes | MI | 53 | 14 | Am | i + o | 1 | 26 | 9 | no | no | in | no |
| Theorell (1982) | 177 | SC | no | MI | | | Eur | o < 6 | 6 | 1 | 2 | yes | no | g | yes |
| van Elderen, Maes, & Van den Broek (1994) | 48 | ET | yes | MI | 57 | 18 | Eur | i + o | 1 | 8 | 10 | yes | yes | in + g | yes |
| van Elderen, Maes, Seegers, et al. (1994) | 204 | ET | yes | Mix | 55 | 17 | Eur | o < 6 | 4 | 8 | 8 | yes | yes | g | yes |
| Health education and exercise training | | | | | | | | | | | | | | | |
| Hedbäck & Perk (1987) | 305 | SC | no | MI | 57 | 15 | Eur | o < 6 | 1 | 38 | 30 | yes | no | in + g | yes |
| Perk et al. (1990) | 147 | SC | no | CA | 57 | 20 | Eur | o ≥ 6 | 0 | 14 | 26 | yes | no | in + g | no |
| Young et al. (1982) | 201 | SC | no | MI | 51 | 14 | Am | i | 1 | 2 | 14 | yes | no | in | yes |
| Health education | | | | | | | | | | | | | | | |
| Karvetti (1981) | 143 | SC | yes | MI | 46 | 0 | Eur | o < 6 | 1 | 52 | 9 | no | no | in + g | no |
| Marshall et al. (1986) | 80 | SC | no | CA | 59 | 30 | Am | i | 0 | 2 | | no | no | in | yes |
| Stransky et al. (1986) | 50 | SC | no | MI | | 0 | Eur | o < 6 | 4 | 104 | 12 | yes | no | g | yes |
| Stress management | | | | | | | | | | | | | | | |
| Fielding (1979) | 45 | SC | yes | MI | | 0 | Eur | o < 6 | | 6 | 6 | | | g | no |
| Friedman et al. (1986) | 771 | HE | yes | MI | 53 | 10 | Am | o ≥ 6 | 26 | 234 | 62 | yes | yes | g | yes |
| Gilliss et al. (1993) | 143 | SC | yes | CA | 59 | 20 | Am | i + o | 0 | 9 | 7 | no | no | in + g | yes |
| Ibrahim et al. (1974) | 79 | SC | yes | MI | 54 | 18 | Am | o < 6 | 3 | 50 | 50 | no | yes | g | no |
| Jones & West (1996) | 2,158 | SC | yes | MI | | | Eur | o < 6 | 4 | 7 | 7 | yes | yes | g | yes |
| Mitsibounas et al. (1992) | 43 | SC | yes | MI | 53 | 12 | Eur | o < 6 | 2 | 52 | 26 | yes | yes | g | no |
| Munro et al. (1988) | 57 | ET | no | MI | 52 | 0 | Am | o ≥ 6 | 13 | 12 | 12 | no | no | g | yes |
| Van Dixhoorn et al. (1989) | 156 | ET | yes | MI | 55 | 6 | Eur | o < 6 | 4 | 6 | 6 | yes | yes | in | no |

*Note.* Co = nature of the comparison condition; Ra = random assignment to conditions; Typ = type of patient in the sample; Ag = mean age; Fe = percentage of female patients in the study sample; Cou = country; Set = setting of the program; Pr = pretest (number of weeks after the cardiac event [rounded]); Dur = duration of program in weeks; Ses = total number of sessions; Mu = multidisciplinary team; Psy = involvement of a psychologist, psychiatrist, or psychotherapist; Tar = target of the program; Par = participation of partners in treatment; SC = standard care; MI = myocardial infarction; Eur = European; o = outpatient; ≥6 = 6 or more weeks after discharge; in + g = both individual and group; Am = American; i = inpatient; CA = coronary artery bypass grafting; g = group; in = individual; <6 = within 6 weeks of discharge; Aus = Australian; ET = exercise training; Mix = mixed types; Can = Canadian; HE = health education.
[a]Actual *N*s varied between different outcomes; largest is reported.

duration and number of sessions. Five programs were shorter than 6 weeks; 10 programs lasted longer than half a year. The average duration was 28 weeks, and the average number of sessions was 18. In 22 studies, a multidisciplinary team was involved in providing the program; in 15 of these a psychologist, psychotherapist, or psychiatrist took part. In the remaining 15 unidisciplinary studies the program was provided by a nurse (10 studies); by a psychologist, psychotherapist, or psychiatrist (2 studies); by a dietitian (Karvetti, 1981); or by a researcher (Munro et al., 1988). In 1 study the treatment provider was not reported.

### *Population Effect Sizes and Moderators*

An overview of the studies included in the population effect size estimates for each outcome variable and each measurement period is displayed in Table 2.[2] Table 3 shows the results for cardiac mortality, recurrence of MI, CABG, and incidence of angina pectoris. We report results separately for each of these outcome measures. The total measurement period for the assessment of cardiac mortality ranged from a half year to 10 years. The estimate of the population effect size (i.e., the WAES *r*) was significant for the long term and for the part/success cluster. For the long term, the odds of surviving were 1.52 times higher for the treatment group than for the control group, equivalent to a 34% reduction in cardiac mortality. The distribution of the study effect sizes for the total measurement period did not suggest a homogeneous set of studies. Success on proximal targets was found to be a moderator: Studies without success on proximal targets formed a homogeneous set (failure cluster), and studies with success or with partial success on proximal targets formed a homogeneous set (part/success cluster). For the part/success cluster, the odds of surviving were 1.44 times higher for the treatment group than for the control group, indicating a 31% reduction in cardiac mortality. The studies in this cluster had success on systolic blood pressure, cholesterol, body weight, smoking behavior, physical exercise, emotional distress, or some combination of these.

The total measurement period of MI recurrence ranged from 1 year to 10 years. The WAES *r* was significant at all measurement periods, except for the short term. For the total-term, medium-term, and long-term periods the odds ratios reflect respectively a 20%, 26%, and 29% reduction in recurrence of MI. The effect sizes for these measurement periods were not homogeneous, however; success on proximal targets was again found to be a moderator. Studies without success or with partial success and with a sample size higher than 100 formed a homogeneous set (part/fail cluster), as did those with success on proximal targets and a sample size higher than 100 (success cluster). The population effect sizes were significant only for the success cluster. The odds ratios for this cluster reflect, respectively, a 36%, 42%, and 41% reduction in recurrence of MI. The studies of this cluster had success in regard to systolic blood pressure, smoking behavior, physical exercise, and emotional distress. Studies with a sample size of fewer than 100 were not taken into account, because the sets of effect sizes were very heterogeneous (either very high, e.g., an odds ratio of 18.77, or very low, e.g., an odds ratio of 0.38).

The total measurement period of CABG ranged from 1 year to 10 years, and that of incidence of angina pectoris ranged from 6 weeks to 3 years. All sets of study effect sizes were homogeneous, but the estimated population effect size was significant only for the short term for angina pectoris. The odds ratio of 1.22 reflects an 18% reduction in incidence of angina.

Table 4 shows the population effect size estimates for the risk factors and the psychological variables. A long-term measurement period is not displayed in this table, because only three studies reported long-term effects on one or more risk factors or psychological variables. All effect sizes (except for smoking behavior) were adjusted for pretest differences. The population effect size estimates for the risk factors are in general higher than those for mortality and cardiac recurrences.

Mean systolic blood pressure was assessed in seven studies, and one study (Hedbäck & Perk, 1987) assessed the percentage of patients with hypertension. We estimated an effect size *r* for this study, based on the odds ratio (see *Effect Size Computation* section), and included this estimate. The three sets of adjusted study effect sizes were homogeneous. For the total period and the medium term, the WAES *r* was significant, indicating that the relevant interventions were associated with decreased systolic blood pressure of the rehabilitated patients, especially between 1 and 2 years after the start of the program.

Total serum cholesterol (in mmol/l, or mg/dl) was assessed in seven studies. For the total measurement period, the set of study effect sizes for this variable was equal to that for the medium term. At all measurement terms the WAES *r* was significant, indicating that the relevant studies showed decreased cholesterol levels in the rehabilitated patients. However, the sets of study effect sizes were not homogeneous. No moderators were found.

Weight was assessed in eight studies. Mean weight (in kg or lbs) was assessed in five studies. One study reported mean body mass index, and one study reported a body fat measure (Dracup et al., 1984). One study reported only change scores (in kg) for patients who were overweight (Sivarajan et al., 1983). The WAES *r* was significant for all measurement periods.

Smoking behavior was assessed in 21 studies. The number of patients who had quit smoking or continued smoking by the posttest was used as a measure of smoking behavior. Because this measure was dependent on the number of smokers at pretest, adjustment for pretest differences was not necessary. The number of smokers at pretest was considered the sample size of the study. When a study did not report this number, we used the total sample size. The WAES *r* was significant for the total measurement period and for the medium term. However, at each measurement term the population effect size estimates suggested a heterogeneous set of studies. For the total measurement period no moderator was found. For the medium term, studies reporting the exact number (or percentage) of pretest smokers who had quit smoking by the posttest showed a significant WAES *r* (the exact cluster). All of these studies had been published since 1985. The observed odds ratio of 2.71 indicated that the patients who had followed a psychoeducational program were about three times more likely to quit smoking than patients who had not. This corresponds to a 63% reduction in smoking. Studies reporting inexact data had a nonsignificant WAES *r* and had all been published before 1985 (the inexact cluster).

Physical exercise was assessed in eight studies by self-report. The effects are not reported in Table 4, because the quality of the measurement was low, and no single indicator could be determined. In four studies *exercise behavior* was defined as the number of patients engaging in regular

[2] A copy of an appendix containing the effect size data is available from Elise Dusseldorp.

Table 2
*Categorization of Studies by Outcome Variable, Measurement Period, and Cluster*

| Measurement period | Cluster | Studies |
|---|---|---|
| | | Cardiac mortality |
| Short term | | Horlick et al. (1984), Rahe et al. (1975), Stern et al. (1983) |
| Medium term | | DeBusk et al. (1994), Frasure-Smith et al. (1997), Frasure-Smith & Prince (1989), Friedman et al. (1986), Hedbäck, Perk, & Perski (1985), Rahe et al. (1975), Stern et al. (1983), Van Dixhoorn (1997) |
| Long term | | Frasure-Smith & Prince (1989), Friedman et al. (1986), Hedbäck, Perk, & Wodlin (1993), Kallio et al. (1979), Rahe, Ward, & Hayes (1979), Van Dixhoorn (1997) |
| | | Recurrence of myocardial infarction |
| Short term | | Rahe et al. (1975), Stern et al. (1983), Young et al. (1982) |
| Medium term | Part/fail | Bengtsson (1983), DeBusk et al. (1994), Frasure-Smith et al. (1997), Fridlund et al. (1991), Jones & West (1996), Theorell (1982), Van Dixhoorn (1997), Young et al. (1982) |
| Medium term | Success | Frasure-Smith & Prince (1989), Friedman et al. (1986), Hedbäck & Perk (1987), Perk et al. (1990) |
| Long term | Part/fail | Kallio et al. (1979), Van Dixhoorn (1997) |
| Long term | Success | Frasure-Smith & Prince (1989), Friedman et al. (1986), Hedbäck, Perk, & Wodlin (1993) |
| | | Coronary artery bypass grafting |
| Short term | | Rahe et al. (1975), Stern et al. (1983) |
| Medium term | | DeBusk et al. (1994), Frasure-Smith et al. (1997), Frasure-Smith & Prince (1989), Fridlund et al. (1991), Oldenburg, Perkins, & Andrews (1985), Rahe et al. (1975), Stern et al. (1983), Van Dixhoorn (1997) |
| Long term | | Frasure-Smith & Prince (1989), Fridlund et al. (1991), Hedbäck, Perk, & Wodlin (1993), Rahe, Ward, & Hayes (1979), Van Dixhoorn (1997) |
| | | Angina pectoris |
| Short term | | Fridlund et al. (1991), Jones & West (1996), Marshall et al. (1986), Mayou (1983), Oldenburg, Allan, & Fastier (1989), Stern et al. (1983), Van Dixhoorn, Duivenvoorden, Staal, & Pool (1989), Young et al. (1982) |
| Medium term | | Bengtsson (1983), Fridlund et al. (1991), Mayou (1983), Oldenburg, Allan, & Fastier (1989), Oldenburg, Perkins, & Andrews (1985), Perk et al. (1990), Stern et al. (1983), Young et al. (1982) |
| Long term | | Fridlund et al. (1991), Kallio et al. (1979), Van Dixhoorn, Duivenvoorden, Staal, Pool, & Verhage (1987) |
| | | Systolic blood pressure |
| Short term | | Dracup et al. (1984), Engblom, Rönnemaa, et al. (1992), Mitsibounas et al. (1982), Munro et al. (1988), Van Dixhoorn, Duivenvoorden, Staal, & Pool (1989) |
| Medium term | | Engblom, Rönnemaa, et al. (1992), Hedbäck & Perk (1987), Kallio et al. (1979), Mitsibounas et al. (1982), Stransky et al. (1986) |
| | | Total cholesterol |
| Short term | | DeBusk et al. (1994), Engblom, Rönnemaa, et al. (1992), Mitsibounas et al. (1982), Oldenburg, Allan, & Fastier (1989) |
| Medium term | | DeBusk et al. (1994), Engblom et al. (1996), Kallio et al. (1979), Karvetti (1981), Mitsibounas et al. (1982), Oldenburg, Allan, & Fastier (1989), Stransky et al. (1986) |
| | | Weight |
| Short term | | Dracup et al. (1984), Mitsibounas et al. (1982), Oldenburg, Allan, & Fastier (1989), Sivarajan et al. (1983), Young et al. (1982) |
| Medium term | | Engblom, Rönnemaa, et al. (1992), Kallio et al. (1979), Karvetti (1981), Mitsibounas et al. (1982), Oldenburg, Allan, & Fastier (1989), Young et al. (1982) |
| | | Smoking |
| Short term | | DeBusk et al. (1994), Engblom, Rönnemaa, et al. (1992), Erdman & Duivenvoorden (1983), Fridlund et al. (1991), Horlick et al. (1984), Jones & West (1996), B. Linden (1995), Mayou et al. (1981), Mitsibounas et al. (1982), Pozen et al. (1977), Salonen et al. (1985), Sivarajan et al. (1983), Taylor, Houston-Miller, Killen, & DeBusk (1990), van Elderen, Maes, Seegers, et al. (1994), van Elderen, Maes, & Van den Broek (1994), Young et al. (1982) |
| Medium term | Inexact | Bengtsson (1983), Ibrahim et al. (1974), Mayou et al. (1981), Theorell (1982), Young et al. (1982) |
| Medium term | Exact | DeBusk et al. (1994), Fridlund et al. (1991), Engblom, Rönnemaa, et al. (1992), Hedbäck & Perk (1987), Mitsibounas et al. (1982), Oldenburg, Allan, & Fastier (1989), Salonen et al. (1985), Taylor, Houston-Miller, Killen, & DeBusk (1990), van Elderen, Maes, Seegers, et al. (1994), van Elderen, Maes, & Van den Broek (1994) |

Table 2
*(continued)*

| Measurement period | Cluster | Studies |
|---|---|---|
| | | Physical training |
| Short term | | Dracup et al. (1984), Engblom, Rönnemaa, et al. (1992), Erdman & Duivenvoorden (1983), Fridlund et al. (1991), Gilliss et al. (1993), Jones & West (1996), van Elderen, Maes, Seegers, et al. (1994), van Elderen, Maes, & Van den Broek (1994) |
| Medium term | | Engblom, Rönnemaa, et al. (1992), Fridlund et al. (1991), van Elderen, Maes, Seegers, et al. (1994), van Elderen, Maes, & Van den Broek (1994) |
| | | Healthy eating habits |
| Short term | | DeBusk et al. (1994), Marshall et al. (1986), Sivarajan et al. (1983), van Elderen, Maes, Seegers, et al. (1994), van Elderen, Maes, & Van den Broek (1994) |
| Medium term | | DeBusk et al. (1994), Karvetti (1981), van Elderen, Maes, Seegers, et al. (1994), van Elderen, Maes, & Van den Broek (1994) |
| | | Anxiety |
| Short term | | Erdman & Duivenvoorden (1983), Fielding (1979), Horlick et al. (1984), Jones & West (1996), B. Linden (1995), Oldenburg, Allan, & Fastier (1989), Taylor, Houston-Miller, Smith, & DeBusk (1997), van Elderen, Maes, Seegers et al. (1994), van Elderen, Maes, & Van den Broek (1994) |
| Medium term | | Fielding (1979), Frasure-Smith et al. (1997), Oldenburg, Allan, & Fastier (1989), Taylor, Houston-Miller, Smith, & DeBusk (1997), van Elderen, Maes, Seegers, et al. (1994), van Elderen, Maes, & Van den Broek (1994) |
| | | Depression |
| Short term | | Engblom, Hämäläinen, et al. (1992), Fielding (1979), Fridlund et al. (1991), Gilliss et al. (1993), Horlick et al. (1984), Jones & West (1996), B. Linden (1995), Oldenburg, Allan, & Fastier (1989), Stern et al. (1983), Taylor, Houston-Miller, Smith, & DeBusk (1997), van Elderen, Maes, Seegers, et al. (1994), van Elderen, Maes, & Van den Broek (1994) |
| Medium term | | Fielding (1979), Frasure-Smith et al. (1997), Fridlund et al. (1991), Oldenburg, Allan, & Fastier (1989), Stern et al. (1983), Taylor, Houston-Miller, Smith, & DeBusk (1997), van Elderen, Maes, Seegers, et al. (1994), van Elderen, Maes, & Van den Broek (1994) |

*Note.* Short term: shorter than 1 year; Medium term: from 1 through 2 years; Long term: longer than 2 years.

exercise; in two studies as the mean number of hours spent exercising; and in two studies as the number of patients who engaged in walking, bicycling, climbing, or weightlifting (in this case only the results for walking were used). The WAES $r$ was significant for all three measurement terms (.071 for the short term, .200 for the medium term, and .074 for the total term), but all three sets of studies were heterogeneous. Moderator variables were not found.

Healthy eating habits were measured in six studies by self-report. The effects are not reported in Table 4, because the quality of the measurement was low, and no common indicator could be determined. The WAES $r$ was significant for all three measurement terms (.140 for the short term, .135 for the medium term, and .096 for the total term), but all three sets of study effect sizes were heterogeneous. Moderator variables were not found.

Anxiety was measured in 10 studies; all but 1 of these used a validated questionnaire to measure anxiety. Depression was measured in 13 studies; all but 2 of these used a validated questionnaire to measure depression. Several studies used a cutoff score to distinguish between high and low levels of anxiety or depression, whereas other studies used mean scores to measure effects. Although this distinction between categorical and continuous data could be made, there was no discrepancy in the results for either anxiety or depression. All sets of study effect sizes were homogeneous. The WAES $r$ was not significant for any of the measurement terms, indicating that psychoeducational interventions do not generally succeed in reducing anxiety or depression.

Three study features were found to act as moderators: success on proximal targets, quality of the data, and year of publication. The features of random assignment, several program characteristics (type of evaluation, setting and length of the program, profession of the program provider, individual or group treatment, participation of partners), and patient characteristics (mean age, type of cardiac event, percentage of women) were not found to be moderators of treatment success.

## Discussion

The principal effects of HE and SM programs for CHD patients identified in this meta-analysis include a 34% reduction in cardiac mortality and a 29% reduction in recurrence of MI at 2–10 years' follow-up and significant ($p < .025$) positive effects on the risk factors and related behaviors at 6 weeks–2 years' follow-up. No principal effects were found on CABG or on anxiety or depression.

Moreover, a moderator effect was found for success on proximal targets. At 1–10 years' follow-up, the reduction in recurrence of MI in the intervention studies with success on proximal targets was 36% versus 2% in studies without success or with only partial success on proximal targets. The reduction in cardiac mortality in studies with success or

Table 3
*Population Effect Size Estimates for Cardiac Mortality, Recurrence of Myocardial Infarction (MI), Coronary Artery Bypass Grafting (CABG), and Incidence of Angina Pectoris*

| Measurement period | Cluster | *k* | *N* | Population effect sizes | | | | Hom. |
|---|---|---|---|---|---|---|---|---|
| | | | | WAES *r* | 95% CI | *d* | OR | |
| **Cardiac mortality** | | | | | | | | |
| Total | | 10 | 4,266 | .023 | −.008–.053 | .05 | 1.30 | no |
| Total | Failure | 4 | 1,705 | −.020 | −.068–.028 | −.04 | 0.88 | yes |
| Total | Part/success | 6 | 2,561 | .051 | .012–.089* | .10 | 1.44 | yes |
| Short term | | 3 | 237 | .017 | −.112–.146 | .03 | 1.20 | yes |
| Medium term | | 8 | 3,767 | −.003 | −.035–.029 | .00 | 1.06 | yes |
| Long term | | 6 | 1,999 | .070 | .027–.112* | .14 | 1.52 | yes |
| **Recurrence of MI** | | | | | | | | |
| Total | | 16 | 7,084 | .032 | .008–.055* | .06 | 1.25 | no |
| Total | Part/fail | 8 | 5,150 | .007 | −.020–.035 | .01 | 1.02 | yes |
| Total | Success | 4 | 1,684 | .091 | .043–.138* | .18 | 1.56 | yes |
| Short term | | 3 | 318 | −.027 | −.138–.084 | −.05 | 0.84 | yes |
| Medium term | | 15 | 6,739 | .036 | .012–.060* | .07 | 1.35 | no |
| Medium term | Part/fail | 8 | 4,889 | .016 | −.012–.044 | .03 | 1.17 | yes |
| Medium term | Success | 4 | 1,684 | .081 | .033–.129* | .16 | 1.71 | yes |
| Long term | | 7 | 2,209 | .065 | .023–.106* | .13 | 1.41 | no |
| Long term | Part/fail | 2 | 531 | −.060 | −.145–.025 | −.12 | 0.71 | yes |
| Long term | Success | 3 | 1,537 | .100 | .050–.149* | .20 | 1.69 | yes |
| **CABG** | | | | | | | | |
| Total | | 9 | 3,117 | .003 | −.032–.038 | .01 | 1.02 | yes |
| Short term | | 2 | 121 | −.086 | −.262–.096 | −.17 | 1.75 | yes |
| Medium term | | 8 | 2,842 | .006 | −.031–.043 | .01 | 1.02 | yes |
| Long term | | 5 | 1,063 | .037 | −.023–.097 | .07 | 1.29 | yes |
| **Angina pectoris** | | | | | | | | |
| Total | | 12 | 3,450 | .036 | .002–.069 | .07 | 1.16 | yes |
| Short term | | 8 | 2,878 | .048 | .011–.084* | .10 | 1.22 | yes |
| Medium term | | 8 | 792 | .070 | −.000–.140 | .14 | 1.36 | yes |
| Long term | | 3 | 549 | .024 | −.061–.107 | .05 | 1.08 | yes |

*Note.* *k* = number of studies; WAES *r* = weighted average effect size *r*; CI = confidence interval; *d* = mean standardized difference (Cohen's *d*); OR = Mantel–Haenszel common odds ratio; Hom. = homogeneity of the set of study effect sizes; short term = less than 1 year after start of program; medium term = from 1 through 2 years; long term = longer than 2 years.
*One-tailed overall $p < .025$, using a discoverywise correction within each outcome measure.

partial success was 31% versus an increase of 14% for studies with no success. These findings suggest that our hypothesis can be confirmed, because they show that success on risk factors, related behaviors, or emotional distress contributes to reduction in cardiac event recurrences and mortality.

The results for mortality and morbidity are partly consistent with those reported in two previous meta-analyses (W. Linden et al., 1996; Mullen et al., 1992). Regarding mortality, W. Linden et al. (1996) found both short-term and long-term effects (odds ratios of 1.76 and 1.39). Mullen et al. (1992) found a significant Cohen's *d* of .24 for a homogeneous set of studies over the total follow-up period. We found a significant effect only for the long term. This difference may be attributable to the fact that W. Linden et al. and Mullen et al. included some studies that did not distinguish between cardiac mortality and mortality from other sources. Our results on recurrence of MI are consistent with the results of W. Linden et al., who found, in addition to a short-term effect, a significant effect for measurement periods of more than 2 years (odds ratio = 1.64, number of studies [*k*] = 3).

Regarding smoking behavior, our results differ from those of Mullen et al.'s (1992) meta-analysis, in which no effect was found for a homogeneous set of studies (*d* = .07, *k* = 9). W. Linden et al. (1996) did not evaluate this variable. In line with the positive effects reported by W. Linden et al. on blood pressure and cholesterol level, and by Mullen et al. on blood pressure, exercise, and diet, our meta-analysis showed favorable effects on blood pressure, cholesterol level, physical exercise, and eating habits. These results are not entirely comparable, however, because Linden et al. and Mullen et al. did not make adjustments for pretest differences.

Contrary to the positive findings of W. Linden et al. (1996), the present meta-analysis showed no favorable effects on anxiety and depression. The inclusion of two recent trials (Frasure-Smith et al., 1997; Jones & West, 1996) in this review may have been responsible for this

Table 4
*Population Effect Size Estimates for Risk Factors and Psychological Variables*

| Measurement period | Cluster | $k$ | $N$ | Population effect sizes: WAES $r$ | Population effect sizes: 95% CI | Population effect sizes: $d$ | Population effect sizes: OR | Hom. |
|---|---|---|---|---|---|---|---|---|
| | | | | Systolic blood pressure[a] | | | | |
| Total | | 8 | 1,063 | .121 | .061–.180* | .24 | | yes |
| Short term | | 5 | 471 | .077 | −.014–.168 | .16 | | yes |
| Medium term | | 5 | 831 | .106 | .038–.173* | .21 | | yes |
| | | | | Total serum cholesterol[a] | | | | |
| Total | | 7 | 1,206 | .249 | .100–.211* | .53 | | no |
| Short term | | 4 | 812 | .299 | .234–.360* | .65 | | no |
| Medium term | | 7 | 1,206 | .249 | .100–.211* | .53 | | no |
| | | | | Weight[a] | | | | |
| Total | | 8 | 1,049 | .088 | .027–.149* | .18 | | yes |
| Short term | | 5 | 393 | .158 | .059–.255* | .32 | | yes |
| Medium term | | 6 | 936 | .085 | .020–.148* | .17 | | yes |
| | | | | Smoking behavior | | | | |
| Total | | 21 | 3,940 | .064 | .033–.095* | .13 | 1.27 | no |
| Short term | | 16 | 3,441 | .033 | .000–.067 | .07 | 1.14 | no |
| Medium term | | 15 | 1,573 | .154 | .104–.202* | .31 | 1.83 | no |
| Medium term | Inexact | 5 | 625 | .029 | −.050–.108 | .06 | 1.12 | yes |
| Medium term | Exact | 10 | 948 | .233 | .171–.294* | .49 | 2.71 | yes |
| | | | | Anxiety[a] | | | | |
| Total | | 10 | 3,960 | .009 | −.023–.040 | .02 | | yes |
| Short term | | 9 | 2,796 | −.015 | −.052–.022 | −.03 | | yes |
| Medium term | | 6 | 1,588 | .045 | −.005–.094 | .09 | | yes |
| | | | | Depression[a] | | | | |
| Total | | 13 | 4,258 | .031 | .001–.061 | .06 | | yes |
| Short term | | 12 | 3,097 | .020 | −.016–.055 | .04 | | yes |
| Medium term | | 8 | 1,734 | .049 | .002–.096 | .10 | | yes |

*Note.* $k$ = number of studies; WAES $r$ = weighted average effect size $r$; CI = confidence interval; $d$ = mean standardized difference (Cohen's $d$); OR = Mantel–Haenszel common odds ratio; Hom. = homogeneity of the set of study effect sizes; short term = less than 1 year after start of the program; medium term = from 1 through 2 years.
[a]Effect sizes are adjusted for pretest difference.
*One-tailed overall $p < .025$, using a discoverywise correction within each outcome measure.

inconsistency in results. Because both of these studies showed null findings on emotional distress and included very large numbers of patients, they had a considerable negative impact on our findings. Nevertheless, the absence of effects in these two studies requires further consideration. Lewin, Thompson, Johnston, and Mayou (1997) attributed the null findings of Frasure-Smith et al. (1997) to the fact that the program was delivered at home by nurses untrained in psychological skills. In Jones and West's (1996) study, however, the stress management program was offered by clinical psychologists. In response to the null findings of Jones and West, Irving (1997) raised the question of why psychological interventions are ineffective. We disagree with Irving's conclusion that the influence of psychological factors in precipitation of and recovery from MI may have been overemphasized in earlier studies. There is accumulating evidence of the large impact of cardiac events on psychological functioning and of the roles of anxiety and depression in both the onset and progression of CHD (Anda et al., 1993; Appels, 1997; Shapiro, 1996). About 15%–30% of cardiac patients are severely distressed after their cardiac event (Follick et al., 1988; Frasure-Smith, Lesperence, & Talajic, 1993; Gonzalez et al., 1996; Langeluddecke, Fulcher, Baird, Hughes, & Tennant, 1989; Schleifer et al., 1989); however, the majority of patients cope with their cardiac event in a functional way. These patients do not require intensive or extended stress management programs; such programs are, however, needed by the minority of patients who do not cope in a functional way with their cardiac event. Therefore, a second explanation may lie in the presence of floor effects. For example, in Frasure-Smith's (1991) study, the treatment program had an impact only among patients who reported high levels of stress symptoms in the hospital. Finally, W. Linden (personal communication, August 1998) mentioned a few other potential reasons for the paucity of effects on emotional distress. There may be sex-specific and age-specific differences in outcomes (e.g., Frasure-Smith et al., 1997) or negative outcomes that are related to being sick, old, alone, or poor and which cannot be considered as maladaptive and targeted in a Beck-type approach to cognitive restructuring. In addition to these arguments, we discuss another explanation concerning the lack of moderator effects for type of evaluation and target of the treatment.

Our results suggest that several study features, including patient characteristics and program characteristics, as moderators did not influence the strength of the effects. The basis for concluding that these findings reflect a true absence of moderation by these factors is limited because (a) some moderators in our study showed very little variation (e.g., mean age of the patients, type of cardiac event; see Table 1), (b) some outcome variables (e.g., CABG) were reported in too few studies, and (c) a restrictive definition of a moderator was used. The lack of finding a moderator effect for type of evaluation and target of the treatment needs further consideration.

In general, the studies included in this meta-analysis did not shed sufficient light on effective mechanisms or components of cardiac rehabilitation programs. In most studies, programs are described only vaguely, without explicit reference to a theoretical model or to empirical findings supportive of specific causal relationships between a given strategy or intervention and positive effects on outcome or intermediate indicators of success. The absence of clear descriptions of targets of the program and of program components can be an additional explanation for the lack of effects on anxiety and depression. Program components explicitly focusing on the reduction of anxiety and depression are rarely elaborated on in the studies. For example, some SM programs were described only in general terms as counseling for stress or anxiety, or as group discussion of ideas, thoughts, and feelings about the heart attack and its effects. Furthermore, the absence of clear descriptions of program components could have impeded differential effectiveness of health education versus stress management or of psychoeducational programs with versus without physical training. Also, sometimes HE programs were vaguely described, and therefore the boundary between HE and information supply (categorized as standard care) was sometimes not clear cut. For example, some education programs were merely described as counseling about risk factors. The absence of a homogeneous set of larger effect sizes for programs including physical training speaks against the conclusions advanced by Oldridge et al. (1988) and O'Connor et al. (1989). However, these authors have themselves argued that definite conclusions are not possible because most of the trials included formal and informal components, making it difficult to distinguish between, on the one hand, comprehensive programs involving physical training, health education, and stress management and, on the other hand, programs with fewer components.

Although the present meta-analysis provides a strong basis for the conclusion that psychoeducational programs show promising effects, several methodological limitations should be noted. The results for the risk factors and related behaviors are less accurate than those for the other outcome variables. In general, the quality of measurement of these two types of outcome measures was low. Only four studies reported using a standardized procedure to measure blood pressure or cholesterol (DeBusk et al., 1994; Dracup et al., 1984; Engblom, Rönnemaa, et al., 1992; Munro et al., 1988). Smoking behavior was measured by self-reports in all but two studies (DeBusk et al., 1994; Taylor et al., 1990). Physical exercise and eating habits were operationalized in a variety of different ways; moreover, some studies reported only change scores for these outcome measures, which led to less accurate estimated effect sizes. In addition, the present meta-analysis might have overestimated true population effect sizes, because only published studies were included. We tried to attenuate overestimation by using a conservative method of estimation (i.e., using zero as an estimate for nonsignificant results, not using phi coefficients, and adjusting for pretest differences), by considering only the last posttest within a given measurement period, and by using a random-effects model when estimating aggregated effect sizes. Finally, it should be noted that the summary odds ratio is not entirely proportional to the WAES $r$ and Cohen's $d$. This is probably due to the fact that the odds ratio, contrary to other measures of effect size, is not affected by unequal sample sizes (Haddock, Rindskopf, & Shadish, 1998).

In this meta-analysis we did not consider the results for randomized trials separately for several reasons. First, in research on cardiac rehabilitation full randomization is sometimes disadvantageous. The probability that patients from different conditions may interact with each other has to be reduced for both methodological and ethical considerations: methodologically, to avoid diffusion of treatment effects, and ethically, to avoid feelings of discrimination. Randomization based on time periods can therefore be an appropriate alternative design. Second, the result of the moderator analyses indicated no effect for the variable representing random or nonrandom assignment, suggesting that the results for randomized trials separately did not differ remarkably from the present results. Third, the distinction between randomized experiments or quasi-experiments is not always clear cut. Sometimes a randomized experiment became a quasi-experiment because of differential attrition in the various conditions (Cook, Campbell, & Peracchio, 1990). For example, in Frasure-Smith and Prince's (1985) study, about twice as many treatment patients as control patients chose not to take part in the study, most probably because of differences in respondent burden associated with the two conditions (see Frasure-Smith & Prince, 1989, p. 489). Finally, in some studies, despite random assignment of patients, pretest differences were apparent. We therefore adjusted posttest effect sizes for pretest differences in both randomized experiments and quasi-experiments to estimate overall effects more precisely.

An important practical implication of this meta-analysis is that the development of psychoeducational programs in cardiac rehabilitation should be stimulated. Risk factor modification and reduction of emotional distress should be targeted in CHD patients to decrease their chances of a fatal or nonfatal recurrence of MI. The development of psychoeducational programs, however, has to be based on theory-driven research focusing on the relationship between specific components of interventions and changes in proximal and distal targets related directly to the needs of the individual patient.

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References marked with an asterisk indicate studies included in the meta-analysis. References marked with two asterisks were also used for effect size data and study features.


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