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Kinesiophobia and exercise self-efficacy as mediators of functional gain after cardiac rehabilitation in older patients with coronary heart disease

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Article

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Abstract

Kinesiophobia is common in older patients with coronary heart disease and is widely assumed to limit functional recovery, yet whether reducing it explains the functional benefit of cardiac rehabilitation has not been examined in trial data. In this secondary analysis of a single-centre randomised trial, 139 patients aged 65 years or older with coronary heart disease and baseline kinesiophobia above the entry threshold received a staged, Health Belief Model-based rehabilitation programme or usual care, and were assessed at admission, discharge, and one and three months after discharge. Change in exercise self-efficacy and in kinesiophobia served as mediators of the Barthel Index at three months, adjusted for baseline. The programme increased self-efficacy and reduced kinesiophobia, but only self-efficacy produced a direction-consistent indirect effect on function (1.36 points, 95% confidence interval 0.26 to 2.77; 15.4% of the total effect), with a borderline mediator–outcome path (P = 0.052). Kinesiophobia produced an inverse indirect effect that disappeared once the mediator preceded the outcome. Most of the functional benefit was therefore not transmitted through either psychological change, although a small self-efficacy-mediated component could not be excluded. Reduction of kinesiophobia should not be assumed to translate into earlier functional recovery.

Introduction

Kinesiophobia—an excessive and irrational fear that physical activity will precipitate a cardiac event—is highly prevalent in patients with coronary heart disease (CHD). A meta-analysis of 15 studies from six countries estimated a pooled prevalence of 61.0% (95% CI 49.4 to 72.6) among patients with heart disease[1]; in Chinese patients with CHD, a meta-analysis of 27 studies reported a prevalence of 64% (95% CI 54 to 73)[2]. In a prospective cohort of 149 patients, of whom 82 were referred for cardiac rehabilitation, each one-point increase on the 13–52 version of the Tampa Scale for Kinesiophobia (TSK) was associated with 8% lower odds of initiating the programme (OR 0.92, 95% CI 0.85 to 0.99)[3]. Such findings have led to fear of movement being treated as a plausible, potentially modifiable barrier, and have encouraged interventions designed to reduce it, on the implicit assumption that removing the barrier will restore physical activity and function.

That assumption rests on weaker ground than its clinical appeal suggests. The association between kinesiophobia and physical activity is small: a meta-analysis of 63 studies including 12,278 participants reported r = − 0.19 (95% CI − 0.26 to − 0.13), with a statistically significant negative association in the cardiac subgroup[4]. Exercise-based cardiac rehabilitation is itself effective—a Cochrane review of 85 trials found reduced myocardial infarction (RR 0.72, 95% CI 0.55 to 0.93) and all-cause hospitalisation (RR 0.58, 95% CI 0.43 to 0.77) at 6–12 months[5]—yet participation remains low: in a 14-country survey only 19.6% of enrolled patients with CHD attended at least half of the programme, which corresponds to 57.1% of those offered one[6], and among older patients uptake and participation decline further with age and comorbidity[7]. Closing the gap between an effective therapy and its underuse requires knowing which patient-level factors actually drive the functional benefit, rather than assuming that the most visible psychological barrier is the operative mechanism.

The Health Belief Model (HBM) offers a structured way to target such factors. An HBM-based education programme improved health beliefs and lifestyle in 120 patients with CHD[8], and in a trial of older adults with type 2 diabetes an HBM-based programme increased self-efficacy for diabetes self-management alongside glycaemic control[9]. Among the constructs of the model, self-efficacy is the one most consistently linked to exercise behaviour: in a secondary analysis of a rehabilitation trial among patients with psychological distress, baseline exercise self-efficacy predicted physical activity three months later[10], and in a cohort of 801 patients self-efficacy contributed to exercise adherence at six months[11]. Cross-sectional studies have combined these observations into a mediation model in which self-efficacy links kinesiophobia to physical activity, with a mediated proportion of 35.6% in 509 Chinese patients with CHD[12] and a comparable pattern after percutaneous coronary intervention[13].

Whether this pathway operates prospectively, and in older patients in particular, is unclear. In a parallel-mediation study of 376 patients after coronary artery bypass grafting, kinesiophobia and self-efficacy both mediated the association between fatigue and physical activity, but the pattern differed by age: in the 61–75-year stratum only self-efficacy carried the indirect effect, whereas kinesiophobia did so only in the 45–60-year stratum[14]. Functional outcomes have rarely been examined. The only study we identified that combined the Barthel Index with the TSK-SV Heart, in 244 patients with chronic heart failure, modelled activities of daily living (ADL) and kinesiophobia as parallel mediators of the association between cardiac function and health status rather than as a causal chain, and was cross-sectional[15]. It therefore remains unknown whether the functional gain produced by a psychologically oriented rehabilitation programme is attributable to the reduction of kinesiophobia, to the enhancement of self-efficacy, or to neither.

We analysed data from a randomised controlled trial of a staged, HBM-based cardiac rehabilitation programme in older patients with CHD[16]. The trial reported that the programme reduced kinesiophobia and improved ADL and exercise self-efficacy; in its conclusion the authors recommended that structural equation modelling be used to validate the mediating pathway “fear of exercise—self-efficacy—ADL”. We tested the proposed sequence in trial data, using path analysis rather than the structural equation model the authors suggested, and examined three hypotheses: that change in exercise self-efficacy mediates the effect of the programme on 3-month ADL (H1); that change in kinesiophobia does so (H2); and that the two act in series, with reduction of kinesiophobia preceding the gain in self-efficacy (H3). Because mediation analyses in trials are frequently underpowered and inconsistently reported[17], the models and a set of sensitivity analyses were fixed in a written analysis plan, and the precision of each estimate is reported.

Results

Sample and outcome profile

The analysed sample comprised 139 patients (69 intervention, 70 control); mean age was 76.6 (SD 7.0) years and 83 (59.7%) were men. Sixty-nine patients were in NYHA class I, 41 in class II and 29 in class III; 92 (66.2%) had at least one other chronic condition. Baseline scores did not differ significantly between groups in the parent report (all $P > 0.05$); observed values were 76.07 ± 18.71 versus 74.42 ± 22.50 for the Barthel Index, 43.16 ± 4.41 versus 43.72 ± 4.30 for the TSK-SV Heart and 40.50 ± 17.01 versus 41.77 ±

16.92 for the SEE (Table 1). As reported previously, the programme produced significant time, group and interaction effects for all three outcomes[16].

Table 1
Observed values of the three outcomes by group and time point.

Measure	Group	Admission (T0)	Discharge (T1)	1 month (T2)	3 months (T3)	Change T0→T3
Barthel Index (0–100)	Control (n = 70)	76.07 ± 18.71	81.21 ± 17.14	84.71 ± 16.17	86.71 ± 16.39	10.64 ± 16.48
Barthel Index (0–100)	Intervention (n = 69)	74.42 ± 22.50	88.26 ± 14.45	93.19 ± 9.35	95.07 ± 8.29	20.65 ± 20.05
TSK-SV Heart (17–68)	Control (n = 70)	43.16 ± 4.41	42.87 ± 3.88	42.24 ± 2.94	42.16 ± 3.33	−1.00 ± 6.15
TSK-SV Heart (17–68)	Intervention (n = 69)	43.72 ± 4.30	42.22 ± 3.63	40.26 ± 3.53	39.57 ± 4.61	−4.16 ± 6.52
SEE (0–90)	Control (n = 70)	40.50 ± 17.01	40.47 ± 18.04	43.07 ± 21.56	43.81 ± 23.05	3.31 ± 15.15
SEE (0–90)	Intervention (n = 69)	41.77 ± 16.92	48.06 ± 17.76	51.22 ± 18.29	56.91 ± 18.26	15.14 ± 17.55

Values are mean ± SD. Values at all four time points are reproduced from the parent trial report[16] for orientation; between-group tests are not repeated. Higher Barthel and SEE scores indicate better function and greater confidence; higher TSK-SV Heart scores indicate greater kinesiophobia. SEE, Self-Efficacy for Exercise scale; TSK-SV Heart, Tampa Scale for Kinesiophobia Heart.

The timing of the changes is relevant to the mediation models (Table 1). The gain in ADL was concentrated before discharge (unadjusted mean change 13.8 points in the intervention group versus 5.1 in the control group), whereas the two groups changed to a similar degree between discharge and three months (6.8 versus 5.5). Exercise self-efficacy rose early, with an in-hospital gain of 6.3 points in the intervention group compared with 0.0 in the control group (40.50 to 40.47), and rose further in both groups thereafter. Kinesiophobia, by contrast, was essentially unchanged at discharge (− 1.5 versus − 0.3) and declined only during follow-up. At three months, 66 of 139 patients (47.5%) had a Barthel Index of 100.

Within individuals, the changes in the two domains were only weakly related: the correlation between change in ADL and change in SEE was 0.11 during hospitalisation and 0.23 from admission to three months, and the corresponding correlations with change in kinesiophobia were 0.22 and 0.32.

Change in exercise self-efficacy as a mediator

In Model A, the programme increased SEE from T0 to T3 by 11.75 points (95% CI 6.29 to 17.21; P < 0.001). Each point of increase in SEE was associated with a 0.12-point higher Barthel Index at three months after

adjustment for baseline ADL, but this mediator–outcome path was borderline (95% CI 0.00 to 0.23; P = 0.052). The total effect of allocation on 3-month ADL was 8.86 points (5.06 to 12.67; P < 0.001) and the direct effect 7.50 points (3.49 to 11.50; P < 0.001), leaving an indirect effect of 1.36 points (0.26 to 2.77), or 15.4% of the total effect (Table 2).

Table 2
Mediation models for the effect of allocation on the Barthel Index at three months (adjusted for baseline).

Model	a path (95% CI)	b path (95% CI)	Total effect c (95% CI)	Direct effect c' (95% CI)	Indirect effect ab (95% CI)	Proportion mediated	E-value (b path)
A:change in SEE	11.75 (6.29 to 17.21)	0.12 (0.00 to 0.23)	8.86 (5.06 to 12.67)	7.50 (3.49 to 11.50)	1.36 (0.26 to 2.77)	15.4%	1.54
B: change in kinesiophobia	−3.27 (− 5.33 to − 1.20)	0.75 (0.47 to 1.03)	8.86 (5.06 to 12.67)	11.31 (7.71 to 14.92)	−2.45 (− 4.41 to − 0.86)	Not interpretable	2.14
C: parallel (both mediators)	11.75 (SEE); −3.27 (TSK)	0.05 (SEE); 0.72 (TSK)	8.86 (5.06 to 12.67)	10.58 (6.67 to 14.50)	0.62 (SEE); −2.35 (TSK); total − 1.73 (− 3.95 to 0.21)	Not interpreted	—
D:serial (kinesiophobia → self-efficacy → ADL)	0.61 (0.22 to 0.99)	0.05 (− 0.06 to 0.16)	8.86 (5.06 to 12.67)	—	−0.11 (− 0.37 to 0.08)	—	—

Confidence intervals for indirect effects are percentile bootstrap intervals (5,000 resamples). In Model B the indirect effect is opposite in sign to the hypothesised pathway and the direct effect exceeds the total effect, an inconsistent-mediation pattern; the proportion mediated is therefore not a meaningful quantity. The a path is the effect of allocation on change in the mediator, adjusted for baseline Barthel Index; the b path is the effect of change in the mediator on the outcome, adjusted for allocation and baseline Barthel Index. The E-value applies to the mediator–outcome path and was computed as described in Methods; the values for the confidence limit nearest the null were 1.04 (Model A) and 1.77 (Model B). In Model C both mediators are entered simultaneously, so the a and b paths are reported for each mediator, the total indirect effect is their sum, and the difference between the two indirect effects was 2.97 points (1.05 to 5.03). In Model D the a path is the first link of the serial chain, expressed in SEE points per point of change in TSK-SV Heart, and the b path is the second link (change in SEE to the Barthel Index); all values in Models C and D are those reported in the Results.

Change in kinesiophobia as a mediator

In Model B, the programme reduced kinesiophobia by 3.27 points (95% CI − 5.33 to − 1.20; P = 0.002). The mediator–outcome path was positive and statistically significant (b = 0.75, 0.47 to 1.03; P < 0.001), indicating that a greater reduction in kinesiophobia was associated with a lower—not higher—Barthel Index

at three months once allocation and baseline ADL were taken into account. The resulting indirect effect was − 2.45 (− 4.41 to − 0.86), opposite in sign to the hypothesised pathway, and the direct effect (11.31, 7.71 to 14.92) exceeded the total effect (8.86). This is an inconsistent-mediation pattern; the estimate was therefore not interpreted as evidence that kinesiophobia mediates the functional benefit, and the proportion mediated is not a meaningful quantity. A specification in which the mediator precedes the outcome is reported in the Results.

Parallel and serial models

When both mediators were entered simultaneously (Model C; Table 2), the path from change in SEE to 3-month ADL fell to 0.05 (− 0.06 to 0.16; P = 0.346) and its indirect effect to 0.62 (− 0.49 to 1.80), whereas the path from change in kinesiophobia remained 0.72 (0.43 to 1.01; P < 0.001) with an indirect effect of − 2.35 (− 4.23 to − 0.82). The total indirect effect was − 1.73 (− 3.95 to 0.21) and the direct effect 10.58 points (6.67 to 14.50); the difference between the two indirect effects was 2.97 points (1.05 to 5.03).

The serial pathway specified in H3 was not supported, and its first link ran in the direction opposite to the hypothesis: a greater reduction in kinesiophobia was associated with a lower, not a higher, subsequent self-efficacy (0.61 points of SEE per point of change in TSK, 95% CI 0.22 to 0.99; P = 0.007). The second-stage path was 0.05 (− 0.06 to 0.16; P = 0.346) and the serial indirect effect was − 0.11 (95% CI − 0.37 to 0.08).

Sensitivity analyses

The self-efficacy indirect effect was not robust (Table 3). It persisted when individual slopes replaced change scores (0.61, 0.04 to 1.26; mediator–outcome path P = 0.060) but disappeared after adjustment for age, sex, NYHA class and comorbidity (0.98, − 0.16 to 2.49), after exclusion of patients with a Barthel Index of 100 at three months (n = 73; −0.21, − 4.09 to 3.34) and after exclusion of those with a baseline score ≥ 95 (1.91, − 0.82 to 4.99). The second of these subsets was defined by the outcome, and the mediator–outcome path within it was null (b = − 0.01, P = 0.917); that analysis therefore does not test whether a ceiling effect weakens the mediator–outcome association. Re-scaling the SEE to mean item scores divides the a path and the indirect effect by nine (a = 1.31; ab = 0.15) and leaves all P values unchanged.

Table 3
Sensitivity analyses of the indirect effects.

Analysis	n	SEE: a path	SEE: b path (P)	SEE: ab (95% CI)	Kinesiophobia: a path	Kinesiophobia: b path (P)	Kinesiophobia: ab (95% CI)
Primary	139	11.75	0.12 (0.052)	1.36 (0.26 to 2.77)	−3.27	0.75 (< 0.001)	−2.45 (− 4.41 to − 0.86)
S1: linear slopes	139	3.61	0.17 (0.060)	0.61 (0.04 to 1.26)	−1.08	1.11 (< 0.001)	−1.20 (− 2.16 to − 0.41)
S2a: Barthel < 100 at T3	73	18.83	−0.01 (0.917)	−0.21 (− 4.09 to 3.34)	−0.52	0.63 (0.007)	−0.32 (− 2.11 to 1.74)
S2b: baseline Barthel < 95	100	17.88	0.11 (0.235)	1.91 (− 0.82 to 4.99)	−2.31	0.87 (< 0.001)	−2.01 (− 4.66 to 0.22)
S3a: in-hospital phase (T0–T1)	139	6.32	0.05 (0.719)	0.32 (− 1.11 to 1.79)	—	—	—
S3b: post-discharge phase (T1–T3)	139	5.51	0.15 (0.025)	0.81 (0.01 to 1.98)	—	—	—
S4: adjusted for age, sex, NYHA class, comorbidity	139	11.71	0.08 (0.178)	0.98 (− 0.16 to 2.49)	−3.12	0.71 (< 0.001)	−2.23 (− 3.96 to − 0.75)

In S1 the a paths are expressed in points per month. Phase-specific models (S3) were estimated for the primary mediator only, because the total effect of allocation on post-discharge change in ADL was null. S2a selected on the three-month outcome and is therefore uninformative about ceiling effects. S5 (re-scaling of the SEE to mean item scores) divides the SEE a path and indirect effect by nine and leaves all P values unchanged, and is not tabulated separately. NYHA, New York Heart Association; SEE, Self-Efficacy for Exercise scale.

The phase-specific analyses pointed to the timing of change as the likely explanation. During hospitalisation, when most of the functional gain occurred, the programme raised SEE by 6.32 points (3.08 to 9.56), but change in SEE did not predict change in ADL over the same interval (b = 0.05, − 0.22 to 0.33; P

= 0.719), and the indirect effect was 0.32 (− 1.11 to 1.79). After discharge the mediator–outcome path was nominally significant (b = 0.15, 0.02 to 0.28; P = 0.025) and the indirect effect 0.81 (0.01 to 1.98), but the total effect of allocation on post-discharge change in ADL was null (1.31, − 2.07 to 4.69; P = 0.448), so no mediated effect can be inferred.

Precision

The 95% CIs for the two primary indirect effects were 2.5 and 3.6 Barthel points wide (Table 2)—the same order of magnitude as the minimal clinically important difference of 3.1–3.6 points[20]—and ranged from 1.2 to 7.4 points across the sensitivity analyses. The smallest indirect effect detectable with 80% power was 2.15 points, 1.6 times the 1.36 points observed; the corresponding value for the kinesiophobia model was 2.58 points. A post hoc power calculation based on the observed effect merely restates the imprecision of the estimate, so we report the detectable effect rather than the achieved power as the informative quantity.

Specification of the mediator and the timing of measurement

Table 4 sets the three specifications side by side. Adjusting the mediator for its own baseline, the specification that matches the model used for the outcome, left the a path for self-efficacy essentially unchanged (12.06) but raised the mediator–outcome coefficient from 0.12 to 0.15 (P = 0.010) and the indirect effect from 1.36 to 1.87 points (95% CI 0.69 to 3.34), or 21.1% of the total effect; the indirect effect remained 1.85 (0.53 to 3.47) after adjustment for age, sex, NYHA class and comorbidity. The indirect effect is therefore sensitive to how the mediator is specified: it is smaller and less stable when the mediator is analysed as a change score without adjustment for its own baseline.

Table 4
Change-score, baseline-adjusted and lagged specifications of the two mediator models.

Specification	Mediator	a path	b path (P)	Indirect effect ab (95% CI)	Proportion mediated	ab adjusted for covariates (95% CI)
Change score T0→T3 (primary)	Self-efficacy	11.75	0.12 (0.052)	1.36 (0.26 to 2.77)	15.4%	0.98 (− 0.16 to 2.49)
Change score T0→T3 (primary)	Kinesiophobia	−3.27	0.75 (< 0.001)	−2.45 (− 4.41 to − 0.86)	NA	−2.23 (− 3.96 to − 0.75)
T3 adjusted for T0	Self-efficacy	12.06	0.15 (0.010)	1.87 (0.69 to 3.34)	21.1%	1.85 (0.53 to 3.47)
T3 adjusted for T0	Kinesiophobia	−2.60	0.92 (< 0.001)	−2.38 (− 4.40 to − 0.78)	NA	−2.23 (− 4.19 to − 0.74)
T2 adjusted for T0 (lagged)	Self-efficacy	7.12	0.13 (0.049)	0.96 (0.14 to 2.11)	10.9%	0.87 (− 0.09 to 2.06)
T2 adjusted for T0 (lagged)	Kinesiophobia	−2.00	0.30 (0.308)	−0.60 (− 1.72 to 0.51)	NA	−0.68 (− 1.78 to 0.46)

Values are path coefficients and indirect effects. The change-score rows reproduce Table 2 and the covariate-adjusted column of Table 3 (analysis S4). In the two other specifications the mediator at the stated wave replaces the change score and is adjusted for its own baseline value; in the lagged specification the mediator at T2 precedes the outcome at T3. Confidence intervals for indirect effects are percentile bootstrap intervals (5,000 resamples). Effects are expressed per point of the mediator at the stated wave; in the change-score rows, per point of change. The proportion mediated is not reported for kinesiophobia because the indirect effect is opposite in sign to the total effect. T0, admission; T2, one month after discharge; T3, three months after discharge.NA: Proportion mediated was not calculated/reported for kinesiophobia because the indirect effect is opposite in sign to the total effect (inconsistent mediation).

Enforcing the temporal order changed the picture for both mediators. With the mediator measured at T2, the programme still raised self-efficacy by 7.12 points (P = 0.004), but the mediator–outcome coefficient fell to 0.13 (P = 0.049) and the indirect effect to 0.96 points (0.14 to 2.11), 10.9% of the total effect; after covariate adjustment the interval included zero (0.87, − 0.09 to 2.06; b path P = 0.128). For kinesiophobia the inverse coefficient did not survive this specification: the programme reduced fear by 2.00 points at T2, but the change in fear no longer predicted the Barthel Index (b = 0.30, P = 0.308), and the indirect effect was − 0.60 (− 1.72 to 0.51).

Robustness to unmeasured mediator–outcome confounding

The two mediator–outcome associations differed in their robustness to unmeasured confounding (Table 2). For change in self-efficacy, the mediator–outcome coefficient of 0.12 in the change-score model corresponded to a standardised difference of 0.15, an approximate risk ratio of 1.14 and an E-value of 1.54:

an unmeasured confounder associated with both change in self-efficacy and the three-month Barthel Index by a risk ratio of 1.54 each, beyond the measured covariates, would be sufficient to explain the association away. The E-value for the confidence limit nearest the null was 1.04, so an association of almost any strength would carry that limit across zero. For change in kinesiophobia the coefficient of 0.75 corresponded to a standardised difference of 0.37, an approximate risk ratio of 1.39 and an E-value of 2.14 (1.77 for the confidence limit). The inverse indirect effect in Model B is therefore unlikely to arise from weak confounding of the mediator–outcome path; to explain it away a confounder would have to be at least as strong as this, or the explanation would have to lie elsewhere—in the timing of measurement, in reverse causation, or in the specification of the model (see Discussion).

Discussion

In this secondary analysis of a randomised trial, the programme improved function and self-efficacy and reduced kinesiophobia[16], yet neither of the two psychological changes accounted for most of the functional benefit. Self-efficacy produced a direction-consistent indirect effect that was small, imprecisely estimated and not robust to sensitivity analyses. Kinesiophobia produced an indirect effect of the opposite sign, with a direct effect larger than the total effect—an inconsistent-mediation pattern that cannot be read as a mediating mechanism, and that was close to zero in the specification in which the mediator preceded the outcome (see Results). The serial pathway proposed at the end of the original report, in which reduction of fear precedes the gain in self-efficacy and thereby improves ADL, was not supported: its first link ran in the opposite direction and its second link was absent.

The cross-sectional literature provided a coherent rationale for expecting mediation. In 509 Chinese patients with CHD, self-efficacy mediated 35.6% of the association between kinesiophobia and physical-activity-related self-management[12], and a similar pattern was reported after percutaneous coronary intervention[13]. Our data do not reproduce that structure when the variables are measured repeatedly and anchored to a randomised contrast. Part of the discrepancy is likely to be methodological: cross-sectional models estimate the covariation of states, whereas the present analysis estimates whether a change in one variable transmits the effect of an intervention to a later change in another, a considerably stricter requirement.

The study that most closely anticipates ours is a mediation analysis of 376 patients after coronary artery bypass grafting[14]. There, the pathway differed by age: in the 61–75-year stratum only self-efficacy carried the indirect effect, whereas kinesiophobia carried it only in the 45–60-year stratum. Our cohort has a mean age of 76.6 years and therefore lies beyond the oldest stratum examined, so the comparison is an extrapolation. In our primary model only self-efficacy produced a direction-consistent indirect effect, whereas the kinesiophobia estimate was inverse and did not survive the lagged specification. Read alongside that study, our findings raise the possibility that confidence, rather than fear, is the more relevant psychological target in this age group, although our estimates are too imprecise to establish a difference between the two paths.

Three observations from our data are pertinent. First, the timing does not fit a mediating chain. Most of the functional gain had accrued by discharge, at which point kinesiophobia had barely moved; the reduction in fear appeared during the following weeks, after the functional benefit had already been realised. A variable that changes after the outcome cannot transmit the intervention's effect to it. Second, during the phase in which the functional gain occurred, change in self-efficacy was unrelated to change in ADL at the individual level (b = 0.05), and the within-person correlations between functional and psychological change were weak throughout (r = 0.11 to 0.32). Third, the association between kinesiophobia and physical activity in the wider literature is small (r = − 0.19)[4], which limits how much functional benefit could plausibly be transmitted through that pathway even if it were fully engaged.

The inverse sign of the kinesiophobia indirect effect deserves comment, because it invites the wrong interpretation. It does not indicate that reducing fear harms function. It arises because the mediator–outcome association, estimated within allocation group and conditional on baseline function, was positive while the intervention lowered the mediator, so that the direct effect exceeded the total effect. Associations of this kind can occur when the mediator and the outcome are measured over the same interval, when the outcome is constrained by a ceiling, when an unmeasured variable influences both, or when the mediator is a change score and patients with more extreme baseline values have more room to move. The specification analyses reported in the Results narrow this down. When the mediator was measured one month before the outcome, the mediator–outcome coefficient fell from 0.75 to 0.30 (P = 0.308) and the indirect effect crossed zero (Table 4), whereas adjusting the mediator for its own baseline did not remove the inverse sign. Simultaneity of measurement, rather than suppression, is therefore the most parsimonious reading, and we treat the same-interval estimate as a consequence of the design rather than as a finding.

The programme coupled belief-focused education with supervised exercise, individualised Borg-guided prescriptions, functional tasks such as sitting-to-standing exercises, and supervised sessions in a simulated home environment with a family member before discharge[16]. These behavioural and physical components are plausible sources of the functional gain, and they operated during the admission, exactly when the gain occurred. Our design cannot apportion the effect between them and the belief-focused components, because the intervention was delivered as a bundle. The practical implication, however, is that the functional benefit did not depend on the psychological changes being complete: patients who were still fearful at discharge nonetheless regained function. That observation is itself useful for clinicians who are reluctant to mobilise patients until fear has resolved.

Several features of the analysis constrain interpretation. First, the Barthel Index was at the maximum of its scale in 47.5% of patients at three months, and a restricted outcome compresses variance, which may attenuate mediator–outcome covariance. The analysis that excluded patients at the maximum cannot settle this, because the subset was defined by the outcome and the mediator–outcome coefficient within it was null (b = − 0.01); how much the ceiling attenuated the self-efficacy path therefore remains unresolved, and these data cannot separate a ceiling effect from a genuinely weak association. Second, the trial was powered for its total-effect comparisons, and the smallest indirect effect detectable in this sample was 2.15 Barthel points, against the 1.36 points observed; this is a limitation on precision, not evidence that

mediation is absent. Third, the mediator and the outcome were measured over the same intervals, which weakens causal ordering even when the exposure is randomised; the lagged specification reported in the Results addresses this directly and yielded a smaller indirect effect for self-efficacy and a null one for kinesiophobia.

A further consideration concerns the robustness of the two mediator–outcome associations. The E-value for the self-efficacy path was 1.54, and that for the confidence limit nearest the null was 1.04, so a confounder only slightly stronger than the measured covariates would be sufficient to explain the association away; for the kinesiophobia path the corresponding values were 2.14 and 1.77. The E-value quantifies rather than excludes confounding of the mediator–outcome path. The contrast is consistent with the rest of the results: the self-efficacy component is the more fragile of the two, whereas the inverse kinesiophobia estimate does not come from an association that any confounder could remove, which is why we treat its sign as the problem rather than its precision.

We chose an ANCOVA-style outcome specification because it is unbiased under randomisation and more efficient than analysing change scores[21, 22]. Regression to the mean affects both arms equally and therefore does not bias the between-group contrasts on which the total and direct effects rest. It does not, however, exclude the possibility that part of the within-arm mediator–outcome association reflects the greater scope for change among patients with more extreme baseline values; we list this among the possible explanations for the inverse sign (see Discussion).

For practice, the findings argue against making the relief of kinesiophobia a precondition for functional rehabilitation, and against evaluating such programmes solely by their effect on fear. Functional training can proceed while fear is still resolving. For research, they suggest that the mechanism question should be posed with finer temporal resolution than four time points allow, and with measures of the behavioural dose actually received—exercise minutes, adherence and supervision—rather than of beliefs alone. Self-efficacy may still matter where the outcome is maintenance of activity rather than recovery of function, since it predicts adherence at six months but less clearly at twelve[11].

This was a single-centre secondary analysis of 139 patients followed for three months, and it was not designed or powered for mediation. The Barthel Index limits discrimination among patients who are independent, and no objective measure of functional capacity (6-minute walk distance or peak oxygen uptake) was available. Exercise adherence was assessed in the intervention group only, and only as a three-level rating at three months, so we could not examine a relation between exercise dose and functional gain. The mediator–outcome association is observational even though the exposure is randomised; the E-value analysis quantifies the confounding that would be required to explain each mediator–outcome association away, but it cannot exclude confounding of that strength. Finally, the Chinese SEE used here was preliminarily validated in community-dwelling older adults[19] and has also shown predictive validity in middle-aged outpatients with CHD[24]; its performance in hospitalised older patients with CHD is inferred rather than established.

In older patients with coronary heart disease, the parent trial showed that a staged, HBM-based cardiac rehabilitation programme improved function and self-efficacy and reduced kinesiophobia. In this secondary

analysis, most of that functional benefit was not explained by either psychological change, although a small self-efficacy-mediated component could not be excluded. Reduction of kinesiophobia in particular did not transmit the intervention's effect to function: its inverse association in the same-interval model disappeared once the mediator preceded the outcome, and the pathway proposed previously was not supported. Programmes of this kind should be evaluated by their direct effects on function, and mechanistic work should examine the timing of change and the behavioural dose delivered rather than assuming that removing fear restores activity.

Methods

Design and setting

This is a secondary mediation analysis of a single-centre, single-blind randomised controlled trial conducted in the Department of Nursing, Suining Central Hospital, Sichuan, China, between November 2024 and October 2025[16]. The parent trial enrolled 140 patients aged ≥ 65 years with CHD and a TSK-SV Heart score > 37; one patient in the intervention group was lost to follow-up, leaving 139 patients (69 intervention, 70 control) in the analysed set. Allocation was generated with the Medsci randomisation tool (odd/even numbering) and concealed in sequentially numbered, sealed, opaque envelopes; outcome assessment and scale scoring were performed by a data manager not involved in intervention delivery, and participants and interventionists were not blinded. The trial was approved by the Ethics Committee of Suining Central Hospital (KYLLKS20240193) and registered with the China Clinical Trials Registry (ChiCTR2500097008). All participants gave written informed consent. All methods were performed in accordance with the relevant guidelines and regulations, including the Declaration of Helsinki. The present analysis used only the previously collected, de-identified dataset; no additional contact with participants was undertaken.

The models reported here, the rule used to classify inconsistent mediation and the list of sensitivity analyses were fixed in a written analysis plan before the manuscript was prepared, but after exploratory inspection of the dataset. The analysis is therefore not a blinded pre-specification and should be regarded as exploratory with respect to the hypotheses tested.

Intervention and outcomes

The intervention has been described in detail elsewhere[16]. In brief, participants in the intervention group received usual care plus a structured, four-phase programme organised around the constructs of the HBM: assessment of perceived susceptibility and severity on the first day of admission; a group session and supervised low-intensity exercise on days 2–3; an individualised, Borg-scale-guided prescription with quantified short-term goals from day 4 to discharge, comprising corridor walking and bedside sitting-to-standing exercises; one or two supervised sessions in a simulated home environment, accompanied by a nurse or family member, before discharge; and structured telephone and social-media follow-up for three months after discharge. The control group received usual care, comprising admission education, monitoring, medication management and a standardised walking prescription at discharge.

Outcomes were assessed at admission (T0), discharge (T1), and one (T2) and three (T3) months after discharge. Kinesiophobia was measured with the Chinese version of the TSK-SV Heart (17 items, four dimensions, total 17–68; higher scores indicate greater fear), which has acceptable reliability and validity in Chinese patients with CHD (Cronbach's α 0.859, test–retest reliability 0.79)[18]. Exercise self-efficacy was measured with the Chinese version of the Self-Efficacy for Exercise (SEE) scale, comprising nine items each scored 0–10[19]. In the dataset analysed here, and in the tables of the parent report, the SEE is recorded as the sum of the nine items (range 0–90); the methods of the parent report describe a 0–10 item mean[16], so the two reports differ in the stated metric but not in the values analysed; the a paths and indirect effects, expressed per point of the sum score, can be converted to the mean-item scale by dividing by nine, whereas the mediator–outcome coefficients convert in the opposite direction (0.12 × 9 = 1.08). P values and the proportion mediated are unaffected. ADL was measured with the Barthel Index (0–100; higher scores indicate greater independence), for which a minimal clinically important change of 3.1–3.6 points and a smallest detectable change of 3.0 points have been reported in geriatric rehabilitation[20].

Statistical analysis

The primary results of the trial—time, group and interaction effects, and between-group comparisons at follow-up—have been reported and are not repeated here[16]. This report uses the same dataset but addresses a different question and reports none of the trial's primary comparisons.

All models used ordinary least squares path analysis. The exposure was randomised allocation (0 = control, 1 = intervention). The primary mediator was change in SEE from T0 to T3 and the secondary mediator was change in TSK-SV Heart from T0 to T3; each mediator was regressed on allocation and baseline Barthel Index, so the a paths (11.75 and − 3.27) differ marginally from the crude between-group differences in change (11.83 and − 3.16). The outcome was the Barthel Index at T3 with the baseline value as a covariate, an ANCOVA-style specification that is unbiased under randomisation and more efficient than the analysis of change scores[21, 22].

Four models were estimated, in the order fixed in the analysis plan (Fig. 1). Model A estimated the indirect effect through change in SEE; Model B through change in kinesiophobia; Model C entered both mediators simultaneously (parallel model), including the contrast between their indirect effects; and Model D estimated the serial pathway kinesiophobia → self-efficacy → ADL (H3). Indirect effects were quantified as the product of path coefficients, with 95% confidence intervals from 5,000 percentile bootstrap resamples (fixed seed). We report the a path, the b path (mediator to outcome, adjusted for the exposure and baseline ADL), the total effect (c), the direct effect (c'), the indirect effect (ab) and the proportion of the total effect mediated. Exposure–mediator interaction was not modelled, so the estimates are controlled direct and indirect effects under the assumption of no interaction. An indirect effect whose sign was opposite to the hypothesised direction, or for which c' exceeded c, was classified in advance as inconsistent mediation and was not interpreted as evidence of a mediating mechanism; the proportion mediated was not interpreted where the total effect was null.

Seven sensitivity analyses were planned. Five were executed: (S1) individual linear slopes across the four time points in place of change scores; (S2) exclusion of patients at the ceiling of the Barthel Index at T3,

and separately of those with a baseline score ≥ 95; (S3) separate estimation for the in-hospital (T0–T1) and post-discharge (T1–T3) phases, for the primary mediator only; (S4) additional adjustment for age, sex, NYHA class and the presence of other chronic conditions; and (S5) re-scaling of the SEE to mean item scores. The two that could not be executed were an analysis including the participant lost to follow-up, for whom no values existed at T3, and the Monte Carlo power simulation described in the analysis plan; power is instead reported from the Sobel standard error. Sequential ignorability was assumed throughout: the mediator–outcome association is observational. Because that assumption cannot be verified, the robustness of the mediator–outcome association to unmeasured confounding was quantified, as described in the final paragraph of this section.

All 139 patients in the analysed set had complete mediator and outcome data at all four time points. The one participant lost to follow-up (intervention group) was excluded before analysis and no imputation was performed, so a complete-case analysis was the only option.

Analyses were performed in Python 3.13 (NumPy 2.2, SciPy 1.14) and all estimates were cross-checked with an independent implementation of the same models. Two-sided P values < 0.05 were considered statistically significant; estimates are reported with 95% confidence intervals, and borderline results are described as such. Reporting follows the AGReMA short form for mediation analyses conducted as a secondary aim[23].

Two further specifications were estimated for each mediator, after the primary models and outside the original analysis plan. In the first, the mediator at T3 replaced the change score and was adjusted for its own baseline value, the counterpart for the mediator of the ANCOVA specification used for the outcome; the two models differ only by the inclusion of the baseline mediator. In the second, the mediator measured one month after discharge (T2) predicted the Barthel Index at T3, with the baseline mediator and the baseline Barthel Index as covariates, so that the mediator preceded the outcome. Both were estimated with and without the covariates used in S4, and confidence intervals came from 5,000 percentile bootstrap resamples. These specifications are reported as exploratory checks of model specification and temporal ordering rather than as pre-specified contrasts.

Unmeasured mediator–outcome confounding was quantified with the E-value[25]. Because allocation was randomised, the association exposed to confounding is the mediator–outcome path. The E-value is the minimum strength of association, on the risk-ratio scale, that an unmeasured confounder would need with both the mediator and the outcome, beyond the measured covariates, to explain that association away; a larger value indicates a more robust association. The mediator–outcome coefficient was expressed as a standardised mean difference, $d = b \times SD(\Delta M)/SD(Y)$, where $SD(\Delta M)$ is the pooled standard deviation of the change score and $SD(Y)$ the pooled standard deviation of the Barthel Index at three months; d was converted to an approximate risk ratio as $RR \approx \exp(0.91 \times d)$, and the E-value computed as $RR + \sqrt{[RR \times (RR - 1)]}$, the same calculation being applied to the confidence limit nearest the null. For a continuous outcome the conversion is approximate and assumes a binary unmeasured confounder; the E-value therefore supplements rather than replaces the assumption of sequential ignorability.

Declarations

Data availability

The de-identified dataset generated and analysed during the current study, together with the analysis code, are available from the corresponding author on reasonable request, subject to approval by the institutional ethics committee.

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Author contributions

Nan Xie conceptualized the study, designed the methodology and supervised the project. Xiaoqin Zheng performed the statistical analysis and data curation and drafted the original manuscript. Ya Tan contributed to data acquisition and clinical resources and critically revised the manuscript. All authors read and approved the final manuscript.

Additional information

Competing interests

The authors declare no competing interests.

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Figures

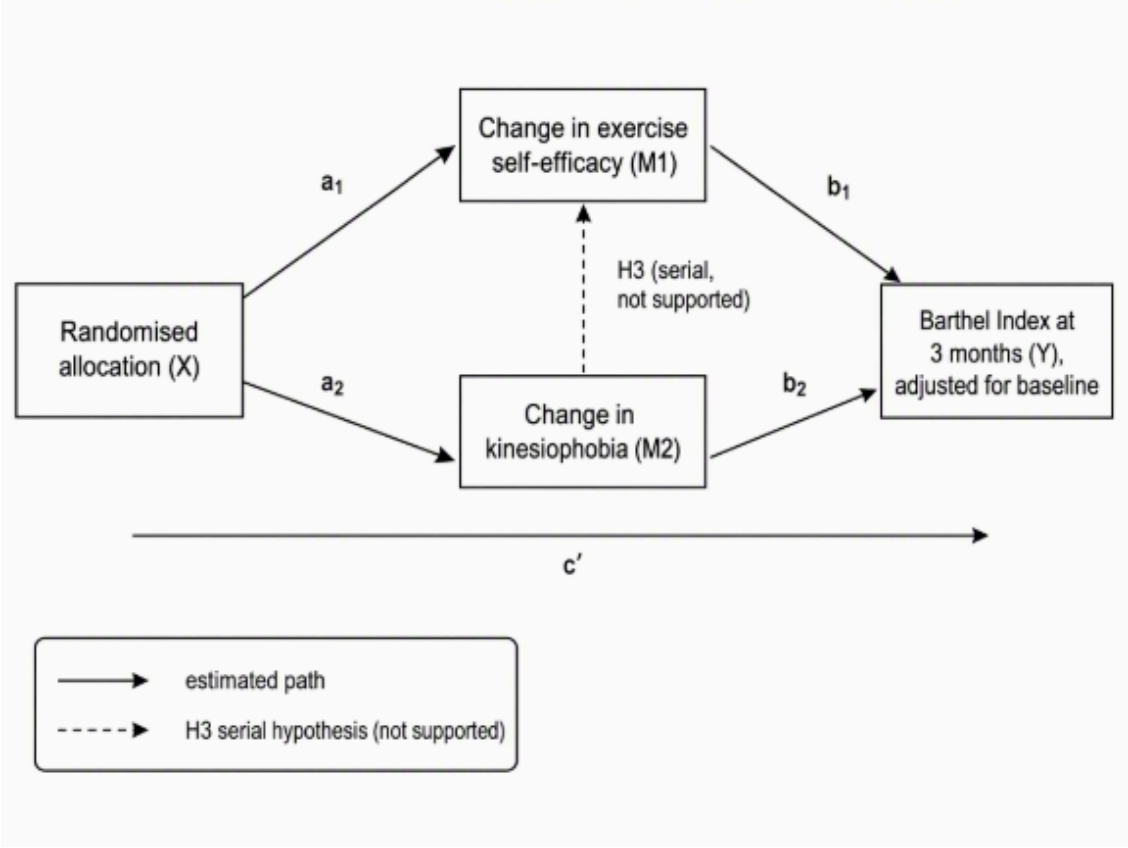


Figure 1

Conceptual mediation model tested. Randomised allocation (X) was modelled as affecting the Barthel Index at three months (Y), adjusted for the baseline Barthel Index, directly (c') and indirectly through change in exercise self-efficacy (M1) and change in kinesiophobia (M2); the two mediators were entered simultaneously in the parallel model. The dashed arrow denotes the serial hypothesis H3 (change in kinesiophobia → change in exercise self-efficacy), which was specified a priori and was not supported. a1 and a2 are the effects of allocation on M1 and M2; b1 and b2 are the effects of M1 and M2 on the outcome. Estimates differ between models and are given in Table 2 for the single-mediator, parallel and serial models and in Table 4 for the alternative specifications. The diagram is schematic and not drawn to scale.