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The higher the better? Interval training intensity in coronary heart disease

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ABSTRACT

Objectives: Peak oxygen uptake (VO_{2peak}) increases more after high intensity interval training compared to isocaloric moderate exercise in patients with coronary heart disease (CHD). We assessed the impact of exercise intensity during high intensity intervals on the increase in VO_{2peak}.
Design/Methods: We included 112 patients with coronary heart disease who had participated in randomized trials of interval training consisting of four times four minutes intervals at 85–95% of heart rate maximum (HR_{max}) for 12 weeks. Exercise intensity was calculated for each patient using HR during the two last minutes of each interval, expressed as percentage of HR_{max}. We used a univariate general linear model with VO_{2peak} increase as the dependent variable and percentage of HR_{max}, age, number of exercise sessions, and baseline VO_{2peak} as covariates. Exercise intensity was also divided into three categories; <88%, 88–92%, and >92% of HR_{max}, and these categories were used as a fixed factor in the model.
Results: VO_{2peak} increased by 3.9 (SD 3.1) mL kg^{-1} min^{-1}, equal to 11.9% after 23.4 exercise sessions. Percentage of HR_{max} had a significant effect on increase in VO_{2peak}, both as a continuous ($p = 0.019$) and categorical variable ($p = 0.020$). The estimated marginal means and 95% confidence intervals of the increase in VO_{2peak} for the three intensity categories were 3.1 (2.0, 4.2), 3.6 (2.8, 4.4), and 5.2 (4.1, 6.3) for the <88%, the 88–92%, and the >92% category, respectively.
Conclusions: Even *within* the high intensity training zone, exercise intensity was an important determinant for improving VO_{2peak} in patients with coronary heart disease.

1. Introduction

Aerobic fitness is recognized as a robust indicator of cardiovascular health and a well-established predictor of total and cardiovascular mortality in subjects with and without coronary heart disease (CHD).[1,2] Direct measurement of maximum or peak oxygen uptake (VO_{2max}/VO_{2peak}) is considered to be the gold standard for aerobic fitness, and changes in aerobic fitness are related to changes in mortality risk.[3] The increase in VO_{2peak} after a period of exercise training depends upon the intensity, frequency, and duration of each exercise session, as well as the length of the training program, and the initial fitness level of the subject. Out of these factors, exercise intensity has been argued to be the key factor in producing improvements in VO_{2peak}, as increasing intensity up to 100% of VO_{2peak} produces the greatest improvements across all frequencies, durations, program lengths, and initial fitness levels of the subjects.[4] The increase in VO_{2peak} depends on the time spent near VO_{2max} during each training session. In other words, high intensity training protocols should be designed to allow the maintenance of a high percentage of VO_{2max} for a long duration in order to improve maximal aerobic power in healthy individuals and in CHD patients.[5]

In line with this, studies comparing equal volumes, and thus energy expenditure, of moderate continuous training and higher intensity aerobic interval training, have found significantly larger increases in VO_{2peak} after high intensity aerobic interval training (AIT) in both healthy subjects[6] and in CHD patients.[7–9] The training model used in some of these studies[6–8], consisted of four times four minutes of exercise at an intensity of 85–95% of individual heart rate maximum (HR_{max}). This model has been broadly used in cardiac rehabilitation in Norway, and the risk associated with such exercise training has recently been documented to be low.[10] However, when working with patients undergoing cardiac rehabilitation, it is of value to know *how high* the exercise intensity should

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Table 1
Subject characteristics at baseline, in each intensity category. Mean (standard deviation) if not otherwise indicated.

	Intensity category		
	<88% $n=29$	88–92% $n=55$	>92% $n=28$
Age, years	59.6 ± 9.6	56.5 ± 9.1	58.2 ± 8.5
Male/female – no. patients	24/5	52/3	21/7
Body mass index, kg/m^2	28.1 ± 3.9	27.7 ± 3.5	27.7 ± 3.8
Prior myocardial infarction – no. patients (%)	24 (83%)	41 (75%)	19 (%)
Prior percutaneous coronary intervention – no. patients (%)	13 (45%)	13 (24%)	5 (18%)
Prior coronary bypass surgery – no. patients	1 (3%)	2 (13%)	1 (4%)
Diabetes mellitus type 2 – no. patients	3 (10%)	4 (7%)	–
Hypertension – no. patients (%)	8 (28%)	6 (11%)	4 (14%)
Smoking – no. patients (%)			
Current	4 (14%)	5 (9%)	1 (4%)
Former	18 (62%)	25 (46%)	9 (32%)
Never	7 (24%)	25 (46%)	18 (63%)
Initial VO_{2peak} in mL kg^{-1} min^{-1}	30.9 ± 5.5	34.0 ± 6.5	27.7 ± 3.8
β-Receptor antagonists – no. patients (%)	23 (80%)	41 (75%)	23 (82%)
Number of exercise sessions	23.9 ± 3.8	23.4 ± 3.5	22.8 ± 2.1

be to induce the greatest improvements in VO_{2peak}[11]. The purpose of the present study was, therefore, to investigate the impact of the precise exercise intensity during AIT on the increase in VO_{2peak} in patients with CHD. Due to the superiority of AIT compared to moderate intensity training in improving VO_{2peak}, we hypothesized that higher relative intensity during intervals would elicit a greater increase in VO_{2peak}.

2. Methods

We pooled data from patients with CHD who participated in one out of four (one study is yet unpublished) randomized controlled trials (RCTs) on AIT conducted within our research group.[8,12,13] Patients were both men and women above 18 years with established CHD, either presented as acute coronary syndrome or angina pectoris. In one of the studies, the patients ($n=8$) were all stable CHD patients with the event occurring >6 months prior to inclusion[8]. In two of the studies the patients ($n=64$ and $n=29$) were included 4–16 weeks after an acute coronary event)[12,13]. In the last study, the patients ($n=11$) were included 1–2 weeks after a percutaneous coronary intervention either due to stable angina pectoris or acute coronary syndrome (Madssen et al., unpublished). The patients in our study can be considered to be quite fit, but are representative for all patients in the studies used.[8,12,13] (and Madssen et al, unpublished). All patients were on optimal medical treatment and/or underwent revascularization before entering the RCT and can hence be regarded as having stable CHD. None of the patients had chronic heart failure or disabilities limiting physical activity. Criteria for inclusion and exclusion in the studies we pooled the data from are outlined in supplementary material (Table 2). Patients were divided into three groups, based on their exercise intensity as described below, and subject characteristics according to exercise intensity category are presented in Table 1. The three categories were comparable in all other variables than exercise intensity.

Supplementary material related to this article can be found, in the online version, at http://dx.doi.org/10.1016/j.jsams.2013.07.007.

All patients signed an informed written consent form before entering the RCTs that the present data were derived from. All of the included RCTs were approved by the Regional Committee for Medical and Health Research (REC, Norway), as were the present analyses. The approval number for the study was 2012/1246. The study conformed to the principles outlined in the declaration of Helsinki.

VO_{2peak} was measured on a treadmill and respiratory gas was analyzed (Oxycon Pro, Jaeger, Germany/Metamax II, Cortex, Germany). Twelve-lead electrocardiograms were monitored continuously during all testing. We used an individualized ramp protocol adjusted to last 8–12 minutes after warm-up.[14] Most patients walked the whole test and we then increased the load by raising the incline of the treadmill every minute or every second minute. For patients who were capable of running, we held the inclination constant and increased the speed 0.5–1 km/h based on the subject's response. Reasons for terminating the test were exhaustion or standard clinical criteria.[15] The patients were taking their prescribed medications at all tests as recommended by the American College of Sports Medicine.[16]

According to the AIT model used (Fig. 1), patients warmed up for about ten minutes at a moderate intensity (60–70% of HR_{max}). Then, patients were then instructed to increase the intensity to a level corresponding to 85–95% of individual HR_{max} for four minutes and repeated this four times with moderate intensity breaks (60–70% of HR_{max}) of three minutes in between and at the end. All patients were encouraged to exercise with a HR in the 85–95% of HR_{max} zone and the actual exercise intensity (HR) was self-selected. During intervals, most patients used 1.5–2 min to reach their predefined target HR. If they had not reached at least 85% of HR_{max} after 2 min, the patients were encouraged to increase the work load. Each patient's HR was recorded at 2 min and at the end (4 min) of each interval. We then calculated the relative intensity for each patient using the HR at these time points and expressed intensity as a percentage of the individual HR_{max} (Fig. 1). All patients wore HR monitors at all training sessions (Polar, Oulu, Finland). Only patients with complete training intensity registrations were included in the present study. All patients exercised for 12 weeks. In one study [13] both treadmill, group exercise training and outdoor uphill walking were used, while in the other three studies the exercise model was treadmill walking or running.[8,12]

We used a univariate general linear model with increase in VO_{2peak} (in mL kg^{-1} min^{-1}) as the dependent variable, and percentage of HR_{max}, age, number of exercise sessions, and baseline VO_{2peak} as covariates. We also categorized exercise HR into three categories, <88%, 88–92%, and >92% of HR_{max} and used these categories as a fixed factor in the model. P-values below 0.05 were considered significant. All statistics were conducted using IBM SPSS Statistics 20.

3. Results

We experienced no adverse events during exercise or in the first hour after exercise in any of the RCTs that the current data analysis was derived from. Overall, VO_{2peak} increased by 3.9 (SD 3.1) mL kg^{-1} min^{-1} (paired sample t-test $p<0.001$), equal to 11.9