Published in final edited form as:
Am Heart J. 2012 January ; 163(1): 81–87.e1. doi:10.1016/j.ahj.2011.06.023.

Long-term effects of smoking and smoking cessation on exercise stress testing: Three-year outcomes from a randomized clinical trial

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Abstract

Background—The long-term effects of smoking and smoking cessation on markers of cardiovascular disease (CVD) prognosis obtained during treadmill stress testing (TST) are unknown. The purpose of this study was to evaluate the long-term effects of smoking cessation and continued smoking on TST parameters that predict CVD risk.

Methods—In a prospective, double-blind, randomized, placebo-controlled trial of 5 smoking cessation pharmacotherapies, symptom-limited TST was performed to determine peak METs, rate-pressure product (RPP), heart rate (HR) increase, HR reserve, and 60-second HR recovery, before and 3 years after the target smoking cessation date. Relationships between TST parameters and treatments among successful abstainers and continuing smokers were evaluated using multivariable analyses.

Results—At baseline, the 600 current smokers (61% women) had a mean age of 43.4 (SD 11.5) years and smoked 20.7 (8.4) cigarettes per day. Their exercise capacity was 8.7 (2.3) METs, HR reserve was 86.6 (9.6)%, HR increase was 81.1 (20.9) beats/min, and HR recovery was 22.3 (11.3) beats. Cigarettes per day and pack-years were independently and inversely associated with baseline peak METs ($P < .001$), RPP ($P < .01$, pack-years only), HR increase ($P < .05$), and HR reserve ($P < .01$). After 3 years, 168 (28%) had quit smoking. Abstainers had greater improvements than continuing smokers (all $P < .001$) in RPP (2,055 mm Hg beats/min), HR increase (5.9 beats/min), and HR reserve (3.7%), even after statistical adjustment (all $P < .001$).

Conclusions—Smokers with a higher smoking burden have lower exercise capacity, lower HR reserve, and a blunted exercise HR response. After 3 years, TST improvements suggestive of improved CVD prognosis were observed among successful abstainers.

Cigarette smoking is a powerful risk factor for cardiovascular disease (CVD) morbidity and mortality.[1,2] The mechanisms by which smoking increases CVD risk are not well understood.[3,4] Observational studies have demonstrated that exercise capacity is significantly impaired in smokers and that reduced smoke exposure may improve exercise parameters.[5,6] However, smoking cessation is associated with weight gain,[7-9] which affects exercise capacity,[10,11] and today's smokers are considerably heavier than those in past studies.[12-14] Therefore, the long-term effects of smoking cessation on exercise physiology are unclear in today's smokers. Several parameters measured during treadmill stress testing (TST) are predictive of future CVD events and mortality, including exercise capacity, rate-

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Clinical trial registration: Clinical Trials.gov no NCT00332644.

pressure product (RPP), peak heart rate (HR) increase, HR reserve, and 60-second HR recovery.[15-21] To our knowledge, the effects of smoking cessation and continued smoking on exercise parameters have not been investigated longitudinally in a contemporary cohort of smokers. The purpose of this study was to evaluate the long-term effects of smoking cessation and continued smoking on TST parameters that predict CVD risk.

Methods

Study participants and design

This study was approved by the Institutional Review Board at the University of Wisconsin School of Medicine and Public Health. All subjects provided written informed consent to participate in a 3-year longitudinal, randomized, double-blinded, placebo-controlled trial that evaluated the efficacy of 5 smoking cessation pharmacotherapies and the natural history of continued smoking and smoking cessation on CVD risk.[22] This report describes a prespecified analysis of data from the baseline and year 3 (final) study visits. Major inclusion criteria included being 18 years or older, smoking 10 or more cigarettes per day (cpd), and expired carbon monoxide (CO) higher than 9 ppm. Major exclusion criteria were blood pressure (BP) higher than 160/100 mm Hg, myocardial infarction within past 4 weeks, heavy alcohol use, use of contraindicated medications, and current pregnancy or breastfeeding.[22]

Study procedures

Participants were recruited from communities near and around Madison, WI, from January 2005 to June 2007. Baseline clinical visits included measurements of anthropometric data, fasting laboratory testing, completion of validated questionnaires and interviews, and TST. Smoking burden was defined as current cigarette smoking (cpd) and pack-years (cpd × years smoked). Recent smoke exposure was determined by exhaled CO levels. Smoking status was assessed by self-reported 7-day point-prevalence abstinence and and was confirmed by an expired CO level of less than 10 ppm. Three self-reported measures of environmental smoke exposure were evaluated at baseline: whether smoking was allowed inside the home, whether the subject lives with a partner/spouse who smokes, and whether smoking was allowed in the workplace.[23] Fasting blood samples were obtained by venipuncture and refrigerated. Plasma aliquots were isolated by centrifugation and frozen at −70°C. Physical activity was assessed by the International Physical Activity Questionnaire.[24]

Exercise testing

TST was conducted using a modified Balke protocol by an exercise physiologist, under physician supervision, using standards from the American Heart Association and American College of Sports Medicine.[25-27] Patients were asked to perform symptom-limited maximal exercise. At each stage of exercise, including peak exercise and 1 minute after exercise cessation, HR, BP, and estimated work load in metabolic equivalents (METs; 1 MET = 3.5 ml O_2 uptake/kg body weight/min) were determined.[15] After achieving the maximum work load, participants performed a minimum 3-minute cool-down at walking speed. All studies were interpreted for the presence of ischemia by a single physician, and peak METs, RPP (peak HR × peak systolic BP), HR increase, HR reserve (maximum HR/[220 – age in years], expressed as a percentage), and 60-second HR recovery (maximum HR – HR at 60 seconds) were calculated.

Data analysis

Analyses were performed with SPSS software (Version 17.0, SPSS, Inc., Chicago, IL). Continuous variables were described as means (SD); categorical variables were presented as

percentages. Because the cessation treatment condition was not significantly related to any TST variable or changes, it was not covaried. For the baseline analysis, Pearson and point-biserial correlations were used to identify univariate associations among TST parameters (peak exercise capacity, HR, RPP, HR increase, HR reserve, and HR recovery), smoking parameters (cpd, pack-years, CO), and participant characteristics. Separate multivariable analyses were performed to determine variables that were independently associated with each baseline exercise parameter, prior to the initiation of cessation therapy. All models included age, sex, resting HR, resting systolic and diastolic BP, diagnosis of diabetes mellitus (based on self-report or hemoglobin A_{1C} >6.5%), use of β-blockers, and use of any antihypertensive medication. Separate models were created for each smoking cessation parameter.

Next, we analyzed changes in TST parameters among participants who returned for their 3-year visit. *t* Test and χ^2 tests were used to evaluate differences in subject characteristics, TST parameters, and smoking parameters between those who returned and did not return for a 3-year visit and between abstainers and those who continued smoking. Multivariable regression analyses were used to determine variables that independently predicted changes in TST parameters. All models were adjusted for age, sex, resting HR, resting systolic and diastolic BP, use of β-blockers, body mass index, change in weight, quartile of moderate-vigorous leisure time activity, presence of a home smoking ban, and any other changes in variables that were correlated ($P < .10$) with changes in the TST parameter. HR reserve and its changes also were modeled as binary variables (≤12 vs >12 beats/min).

Results

Subject characteristics

Subject characteristics at baseline and the 3-year follow-up visit are in Table I. At baseline, the 600 current smokers (61% women) were 43.4 (11.5) years old and smoked 20.7 (8.3) cpd with a smoking burden of 26.7 (19.0) pack-years. BPs were normal (116.3 [14.3]/70.3 [10.3] mm Hg). Exercise capacity was 8.7 (2.3) METs with a peak RPP of 25,954.9 (5,431.4) mm Hg × beats/min, HR reserve of 86.6 (9.6)%, and 60-second HR recovery of 22.7 (11.3) beats. Only 27 (4.5%) subjects had ST segment changes suggestive of ischemia. Only 26 (4.3%) subjects were taking β-blockers (12 for hypertension); 4 (0.7%) were taking rate-lowering calcium-channel blockers (3 for hypertension), and 26 (4.3%) were taking antihypertensive medications (including individuals on β- or calcium-channel blockers for hypertension). Diabetes mellitus was present in 13 (2.2%) subjects. There were no differences between the treatment arms in the distributions of any demographic, anthropomorphic, smoking, TST, or laboratory parameters evaluated.

Baseline TST parameters and their relationships with smoking parameters

Baseline correlations between smoking and TST parameters are in Table II. Exercise capacity was correlated inversely with current smoking (cpd), smoking burden (pack-years), peak HR increase, and HR reserve (all $P < .001$). Exercise peak RPP and HR recovery were correlated inversely with smoking burden ($P < .001$ for both), but not with current smoking (cpd). No significant correlations were identified between CO and any TST parameter. In multivariable analyses (Table III), significant, independent associations between current smoking (cpd) and exercise capacity, peak HR increase, and HR reserve were identified, but not with exercise peak RPP or HR recovery. Significant, independent associations between smoking burden (pack-years) were identified for exercise capacity, peak RPP, peak HR increase, and HR reserve, but not with HR recovery.

Changes in TST parameters after 3 years

Smoking status was available on the 370 subjects (61.7%) who attended the 3-year follow-up. This percentage is almost exactly the same as the percentage that attended the 1-year visit (61.4%). This represents excellent long-term follow-up in a smoking cessation study.[8] Of these subjects, 168 (45.4% of returning subjects, 28.0% of original 600 subjects) successfully quit smoking ("abstainers"). Subjects who did not return for the 3-year visit were, on average, 1.9 years older ($P = .04$) and had slightly higher HR reserve (85% vs 87%, $P = .01$). Otherwise, subjects returning for the 3-year visit were similar to subjects who only attended the baseline visit in regard to age, sex, race, marital status, educational status, presence of diabetes mellitus, use of antihypertensive medications (including β-blockers), current smoking (cpd), smoking burden (pack-years), peak METs, peak RPP, peak HR increase, HR reserve, and HR recovery (see online Appendix Supplemental Table).

Compared with continuing smokers, abstainers had slightly lower CO levels at baseline ($P = .03$), but otherwise were similar in regard to all other parameters in Table I. After 3 years, abstainers had greater reductions in smoking parameters than continuing smokers (all $P < .001$) and greater increases in body mass index ($P < .001$) and waist circumference ($P = .01$).

In regard to TST parameters, abstainers had greater improvements than continuing smokers in peak RPP, peak HR increase, and HR reserve (all $P < .001$), differences that persisted after full adjustment (all $P < .001$, Table IV). No significant differences between abstainers and continuing smokers were observed for changes in peak METs ($P = .98$) and HR recovery ($P = .28$) after 3 years, whether the latter was coded as a continuous or categorical variable. No changes in TST parameters were related to sex, baseline smoking parameters, or treatment arm.

The effect of age on peak RPP and peak HR increase may reflect, in part, an initial values effect, since baseline levels were especially low in the older age groups. For both peak RPP and HR increase, there was less deterioration and eventual improvement by year 3 as a function of age, regardless of cessation status. However, among the smokers who were able to quit, RPP improved by year 3 in all but the first age quartile (<36 years) and improved the most among abstainers in the highest age quartile (>50 years). There also was a statistically significant weight change by abstinence interaction effect ($P = .03$). A larger HR increase at year 3 relative to baseline was observed among abstainers, but only when they gained less than 8.1 kg (Table IVB).

Discussion

Heavier smokers had lower TST measures of cardiore-spiratory fitness including peak exercise capacity, RPP ($P < .01$, pack-years only), peak HR increase ($P < .05$), and HR reserve ($P < .01$). To our knowledge, this is the first large study to serrially evaluate maximum exercise responses in a large cohort of modern smokers. Our finding of a dose effect of cigarette smoke on TST parameters that are predictive of CVD risk and mortality is important, as it helps identify a mechanism by which increased smoking burden increases CVD risk, namely, reduced exercise capacity and impaired physiological responses to exercise. These findings are consistent with previous reports from smaller studies that compared certain exercise responses (eg, peak HR, exercise capacity) among smokers with nonsmokers and reports that described the longitudinal effects of continued smoking, the effects of acute exposure to smoke, or varying levels of smoke exposure in young adults.[5,28-32] A recent report, however, suggested that younger smokers achieved a higher RPP during exercise than nonsmokers because of amplified BP changes.[6]

Second, we demonstrated that despite gaining weight, successful abstainers from smoking had greater improvements than continuing smokers in several TST parameters including RPP, peak HR increase, and HR reserve. These improvements were statistically significant even after adjusting for confounding variables, including baseline measures of smoking intensity, smoking burden, and CO levels. This is the first report, to our knowledge, of the longitudinal effects of smoking cessation on exercise parameters. Although exercise capacity and HR recovery did not improve significantly, the 3 parameters that did improve are important predictors of CVD and all-cause mortality.[15,17-21] Our longitudinal findings are consistent with some small cross-sectional studies of the short-term effects of smoking cessation on certain exercise parameters[30,33-35] and suggest that improved exercise physiology may be a mechanism for cessation-related reductions in CVD risk.

Importantly, the magnitude of improvement in TST parameters was not related to baseline smoking intensity or burden, suggesting that in the range of cigarette use reported in our subjects, TST parameters improve regardless of the degree of baseline smoking. That baseline smoking intensity and burden are not related to improvements in TST markers of CVD risk after cessation is consistent with our previous reports that improvements in HDL cholesterol[36] and endothelial function[8] in smokers who succesfully abstain are not related to baseline smoking intensity or burden either. This suggests a health benefit may be seen even amongst relatively light smokers. Also, older smokers who are able to quit may see the most improvement in TST parameters.

As expected, abstainers had larger increases in body mass index than individuals who continued to smoke cigarettes. Although increased weight is associated with reduced exercise capacity,[37] the benefical effects of smoking cessation on several TST parameters still were observed among abstainers. We did not formally evaluate mechanisms for the improved TST parameters we observed. Ischemic responses were very rare, so our findings are unlikely to be caused by fewer individuals with myocardial ischemia. Exercise responses are influenced by catecholamines and vagal tone; however, we did not see a change in HR recovery or resting HR. Exercise responses can be affected by leisure time activities, but they did not predict changes in TST parameters in our study. We previously demonstrated improved flow-mediated vasodilation after smoking cessation, so it is possible that improved endothelial function with improved cardiac and skeletal blood flow may mediate some of the improvements we observed.[8] Improved pulmonary function also may have contributed to our observations.

Limitations

Because this was a randomized clinical trial of smoking cessation interventions, there were no nonsmoking controls. Therefore, we cannot determine the extent to which the exercise parameters that improved among abstainers approached normal values. Because stress tests were obtained only at baseline and after 3 years, we could not evaluate the time course of improvement with quitting. It is common for subjects in smoking cessation studies who relapse to drop out or miss follow-up visits,[38-40] so we cannot exclude bias based on continued participation.

Although we observed longitudinal improvements in peak RPP, HR reserve, and HR increase with abstainence, we did not observe improvements in HR recovery or exercise capacity. Exercise capacity is a derived estimate of METs based on models related to VO_2 max stress testing results and achieved treadmill stress test workloads. It has significant variability and imprecision, so it may be more difficult to show longitudinal improvements in this TST parameter than those that rely on more direct measurements. Vagal reactivation is the main determinant of the HR decrease seen immediately after exercise.[15] It is possible that vagal tone does not improve quickly among individuals who abstain from smoking.

Indeed, we did not observe differences in resting HR among eventual abstainers and continuing smokers. It also is possible that continued exposure to second-hand smoke among ex-smokers blunted the expected physiological improvement in this parameter.

In our study, 38.3% of subjects did not return for their 1-year follow-up visit, which is consistent with the 30% to 43% 1-year drop-out rates reported in other recent clinical trials of smoking cessation pharmacotherapy.[38,39] Subjects who did not attend the 3-year visit were similar to subjects who only attended the baseline visit. Because second-hand smoke exposure was not quantified, the effects of smoking cessation on TST parameters may have been underestimated. Finally, the long-term effect of smoking cessation on CVD events was not evaluated, so the relationships between the changes in the TST parameters observed in this study and changes in long-term risk are not known.

Conclusions

Smokers with a higher smoking intensity and burden have lower exercise capacity and HR reserve, with a blunted HR response to exercise. After 3 years, physiological TST improvements suggestive of improved CVD prognosis were observed among successful abstainers.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

This research was supported by grant P50 DA019706 from NIDA to the University of Wisconsin-Center for Tobacco Research and Intervention and grant K05CA139871 from NCI to T.B.B. M.E.P. was supported by UL1RR025011 from the NCRR Clinical and Translational Science Award program. Medications were provided by GlaxoSmithKline. The authors are solely responsible for the design and conduct of this study, all analyses, drafting, and editing of the paper and its final contents.

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Table I

Subject characteristics at baseline and 3 years after the target quit date

		Relapsed*			Abstainers*			*P*	
	All subjects (baseline)	Baseline	Year 3	Δ Year 3 – baseline	Baseline	Year 3	Δ Year 3 – baseline	Baseline, relapsed vs abstainers	Δ Baseline – year 3
Number	600	213	213	–	155	155	–	–	–
Age (y)	43.4 (11.5)	43.8 (10.9)	46.8 (10.9)	–	44.6 (11.8)	47.6 (11.8)	–	.47	–
Sex (% female)	60.5	65.3	–	–	58.1	–	–	.16	–
Body mass index (kg/m^2)	28.3 (6.0)	28.3 (6.0)	28.9 (6.4)	0.65 (3.1)	28.9 (6.3)	31.1 (7.2)	2.2 (4.1)	.39	<.001
Waist circumference (cm)	94.7 (15.1)	94.3 (15.1)	95.9 (15.9)	1.5 (8.1)	96.8 (15.3)	100.8 (16.5)	3.6 (7.8)	.11	.01
Current smoking (cpd)	20.7 (8.4)	20.7 (8.3)	12.8 (8.2)	−8.0 (8.3)	20.0 (8.7)	0.0 (0.0)	−20.0 (8.7)	.41	<.001
Smoking burden (pack-years)	26.8 (19.1)	27.0 (18.2)	28.9 (18.8)	1.9 (1.2)	26.8 (20.6)	26.8 (20.6)	0.0 (0.0)	.92	<.001
CO (ppm)	27.0 (13.8)	28.5 (14.6)	18.7 (10.9)	−9.9 (14.3)	25.5 (13.2)	2.3 (1.4)	−23.2 (13.2)	.04	<.001
HR (beats/min)	71.9 (11.5)	72.3 (11.4)	74.5 (12.5)	2.1 (11.2)	71.9 (1.6)	74.7 (11.9)	2.8 (12.0)	.68	.57
Exercise capacity (maximum METs)	8.7 (2.3)	8.6 (2.2)	8.5 (2.3)	−0.1 (1.4)	8.8 (2.1)	8.6 (2.2)	−0.1 (1.6)	.58	.98
Exercise peak RPP (mm Hg × beats/min)	25,954.9 (5,431.4)	26,488.8 (5,381.5)	25,141.1 (5,015.4)	−1,289.6 (4,526.4)	25,998.9 (5,099.0)	26,823.4 (5,316)	791.5 (5,176.7)	.38	<.001
Exercise peak HR increase (beats/min)	81.1 (20.9)	81.7 (20.0)	74.1 (20.8)	−7.6 (14.8)	81.6 (20.2)	79.9 (20.6)	−1.7 (13.7)	.94	<.001
Exercise HR reserve (%)	86.6 (9.6)	87.4 (9.7)	84.3 (10.0)	−0.03 (.08)	87.3 (9.0)	88.0 (9.1)	0.01 (.08)	.97	<.001
HR recovery at 60 seconds (beats/min)	22.3 (11.3)	21.6 (10.6)	20.8 (9.2)	−2.3 (12.5)	22.3 (12.8)	22.3 (8.8)	1.1 (14.8)	.70	.28

All values are mean (SD).

CO indicates Carbon monoxide; *HR*, heart rate; *METs*, metabolic equivalents; *RPP*, rate-pressure product.

*Subjects with both baseline and year 3 TST measures only.

Table II

Baseline correlations between smoking and treadmill stress testing parameters

	Current smoking (cpd)		Smoking burden (pack-years)		Carbon monoxide (ppm)	
	r	***P***	***r***	***P***	***r***	***P***
Exercise capacity (maximum METS)	−0.22	<.001	−0.41	<.001	−0.06	.15
Exercise peak RPP (mm Hg × beats/min)	−0.08	.06	−0.28	<.001	−0.05	.24
Exercise peak HR increase (beats/min)	−0.23	<.001	−0.50	<.001	−0.08	.05
Exercise HR reserve (%)	−0.15	<.001	−0.25	<.001	−0.04	.37
Heart rate recovery at 60 seconds (beats/min)	−0.10	.10	−0.19	<.001	0.06	.35

Abbreviations as in Table I.

Table III

Best fitting models for associations of smoking parameters with baseline treadmill stress testing parameters

	Including current smoking (cpd)			Including smoking burden (pack-years)		
	Model	**Standardized β**	***P***	**Model**	**Standardized β**	***P***
Exercise capacity (maximum METs)	Adjusted $R^2 = 0.46$			Adjusted $R^2 = 0.45$		
	Exercise maximum HR (beats/min)	0.470	<.001	Exercise maximum HR (beats/min)	0.462	<.001
	Male sex	−0.280	<.001	Male sex	−0.282	<.001
	Resting HR (beats/min)	−0.275	<.001	Resting HR (beats/min)	−0.270	<.001
	Age (y)	−0.110	<.01	Smoking burden (pack-years)	−0.162	<.001
	Current smoking (cpd)	−0.115	<.001	Exercise maximum diastolic BP (mm Hg)	−0.081	.02
	Presence of diabetes mellitus	−0.109	<.001	Use of β-blockers	−0.001	.969
	Exercise maximum diastolic BP (mm Hg)	−0.082	.01			
	Resting systolic BP (mm Hg)	−0.074	.03			
	Use of β-blockers	0.025	.44			
Exercise peak RPP (mm Hg × beats/min)	Adjusted $R^2 = 0.29$			Adjusted $R^2 = 0.30$		
	Age (y)	−0.400	<.001	Age (y)	−0.333	<.001
	Resting systolic BP (mm Hg)	0.271	<.001	Resting systolic BP (mm Hg)	0.280	<.001
	Use of β-blockers	−0.162	<.001	Use of β-blockers	−0.161	<.001
	Resting HR (beats/min)	0.139	<.001	Resting HR (beats/min)	0.148	<.001
	Male sex	−0.118	<.01	Male sex	−0.124	<.001
	Current smoking (cpd)	−0.042	.25	Smoking burden (pack-years)	−0.122	<.01
Exercise peak HR increase (beats/min)	Adjusted $R^2 = 0.46$			Adjusted $R^2 = 0.40$		
	Age (y)	−0.513	<.001	Age (y)	−0.454	<.001
	Exercise maximum systolic BP (mm Hg)	0.301	<.001	Smoking burden (pack-years)	−0.172	<.001
	Resting systolic BP (mm Hg)	−0.209	<.001	Use of β-blockers	−0.153	<.001
	Current smoking (cpd)	−0.093	<.01			
	Use of β-blockers	−0.099	<.01			
	Presence of diabetes mellitus	−0.062	<.05			
Exercise HR reserve (%)	Adjusted $R^2 = 0.30$			Adjusted $R^2 = 0.31$		
	Exercise maximum systolic BP (mm Hg)	0.382	<.001	Exercise maximum systolic BP (mm Hg)	0.373	<.001
	Resting systolic BP (mm Hg)	−0.266	<.001	Resting HR (beats/min)	0.274	<.001

Am Heart J. Author manuscript; available in PMC 2013 January 01.

Including current smoking (cpd)			Including smoking burden (pack-years)		
Model	**Standardized β**	***P***	**Model**	**Standardized β**	***P***
Resting HR (beats/min)	0.266	<.001	Resting systolic BP (mm Hg)	−0.254	<.001
Use of β-blockers	−0.201	<.001	Use of β-blockers	−0.201	<.001
Current smoking (cpd)	−0.090	<.01	Smoking burden (pack-years)	−0.165	<.001
Male sex	0.082	.03	Male sex	0.072	.04
Age (y)	−0.072	.05	Age (y)	0.013	.79

Abbreviations as in Table I.

Models initially included age, sex, resting HR, resting systolic BP, resting diastolic BP, diagnosis of diabetes mellitus, use of β-blockers, and use of any antihypertensive medication as well as any other variable that was correlated significantly at baseline. Data are from the 368 subjects with baseline and 3-year stress tests.

Table IV

Best fitting models for changes in exercise peak rate-pressure product, peak heart rate increase, and heart rate reserve after 3 years, including abstinence status

A. Change in exercise peak RPP (year 3 – baseline)			
Adjusted R^2 = 0.14	**Standardized β**	***t***	***P***
Resting HR (beats/min)	−0.29	−5.89	<.001
Abstinence at year 3	0.20	4.03*	<.001
Age (y)	0.12	2.36	.02
B. Change in exercise peak HR increase (year 3 – baseline)			
Adjusted R^2 = 0.11	**Standardized β**	***t***	***P***
Abstinence at year 3	0.30	4.55	<.001
Change in weight from baseline (kg)	−0.10	−1.25	.21
Age (y)	0.11	2.20	.03
Interaction of change in weight and abstinence at year 3	−0.18	2.13	.03
C. Change in exercise HR reserve (year 3 – baseline)			
Adjusted R^2 = 0.17	**Standardized β**	***t***	***P***
Resting HR (beats/min)	−0.34	6.91	<.001
Abstinence at year 3	0.23	4.64	<.001

Models initially included abstinence status at year 3, age, sex, resting HR, resting systolic BP, use of β-blockers, body mass index, change in weight, quartile of moderate-vigorous leisure time activity, presence of a home smoking ban, as well as any other variable that was correlated significantly at baseline.

Only significant predictors are displayed above, except for Table 4B because it includes an interaction term.

Data are from the 368 subjects with baseline and 3-year stress tests.

Abbreviations as in Table I.