Longitudinal change in coronary heart disease risk factors in older runners

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Abstract

Background: it is currently not clear how coronary heart disease (CHD) risk factors change over time in chronic exercisers. Therefore, the purpose of this study is to describe the longitudinal change in CHD risk factors in chronically endurance-trained men and women, and to determine the exercise and nutritional factors associated with those respective changes.

Methods and results: ninety-one middle-aged runners (56 male, 35 female) were tested on two occasions approximately 10 years apart (aged 50.8 ± 8.0 versus 60.0 ± 7.9 years at respective visits). Body composition, VO2max, blood pressure (BP) and blood chemistries were measured, and the subjects’ self-reported training and nutritional history. Data were analysed by factorial analysis of variance (ANOVA) and multivariate step-wise regression. Among the entire sample, training volume decreased (61.1 ± 28.2 versus 44.7 ± 24.6 km/week, P < 0.05) but nutritional variables did not change. Body fat (16.9 ± 5.3% for men versus 21.1 ± 5.3% for women, P < 0.05), blood lipids, blood glucose and systolic and diastolic BP all changed negatively over the study duration. These changes occurred similarly in both genders and irrespective of menstrual and hormone replacement status among the women. Lastly, the changes in CHD risk factors were not predicted by change in exercise or nutritional patterns.

Conclusions: despite the maintenance of significant volumes of exercise and the absence of changes in diet, most CHD risk factors demonstrated unfavourable changes over 10 years in chronic men and women runners. However, the absolute values for most CHD risk factors remained better than those reported for sedentary peers of comparable age.

Keywords: endurance exercise, blood lipids, blood pressure; elderly

Introduction

The study of older athletes had focussed on describing primary age-related changes in physical fitness and sports performance [1–6], with less attention focussed on health-related changes such as those that reflect coronary heart disease (CHD). While exercise clearly protects against CHD and premature mortality [7, 8], risk is not absent in chronic exercisers, and there is a need to understand the factors that may change over time and contribute to that risk.

The few studies that have described longitudinal changes in CHD risk factors in older athletes [6, 9, 10], reflecting small, men-only samples, have demonstrated little or no change in various CHD risk factors over 7–33 years. It is
plausible that CHD risk factors may not change over time in chronic exercisers, as health-related variables are responsive to relatively low volumes and intensities of physical activity [11, 12]. However, cross-sectional research has clearly demonstrated a dose–response relationship between CHD risk factors, exercise volume [13, 14] and exercise intensity [13] in several thousand men and women. Therefore, declines in training volume and intensity, possibly a requisite with advancing age [16], may lead to negative changes in CHD risk factors. Moreover, several studies have suggested that changes in CHD risk factors may reflect primary ageing [17, 18]. It is also possible that CHD risk factors may change differently over time in men and women runners considering the influence of menopause [19].

Therefore, the purpose of this study is to describe the longitudinal change in CHD risk factors in chronically endurance-trained men and women, and to determine the exercise and nutritional factors associated with those respective changes. We hypothesised that CHD risk factors would change negatively over time in these chronic runners, and that these CHD risk factor changes would be related to changes in training, fitness and nutrition.

**Methods**

**Subjects**

Ninety-one runners (56 male, 35 female) were selected from 232 older athletes (147 male, 85 female) participating in a longitudinal study previously described in Wiswell et al. (2001) [20]. The subjects were tested biannually for various parameters of physical fitness and health. The subjects’ first and last visits were selected for comparison in this study. Subjects from the parent study were excluded if they had been tested on only one occasion or had less than five years between their first and last test, had a known cardiovascular disease, including hypertension, at the first visit, or were on medication at either visit that would influence any of the CHD risk factors. All subjects gave written informed consent according to guidelines established by the Institutional Review Board of the University of Southern California.

**Anthropometric measures**

Subjects reported to the laboratory following a 12-h overnight fast. On arrival in the laboratory, their height was measured with a stadiometer, and weight determined on a calibrated Homs beam-scale. Residual lung volume was assessed using the nitrogen dilution technique [21], and body composition determined by hydrodensitometry [20].

**Pre-exercise examination**

Resting ECG was recorded on a Hewlett Packard 1500 Electrocardiograph (Palo Alto, CA). Blood pressure (BP) was measured in a supine position using an auscultatory technique. The study physician examined subjects, and an in-dwelling angiocatheter was inserted into a forearm vein. Resting blood samples were drawn into pre-chilled ethylenediaminetetraacetic acid (EDTA) Vacutainer tubes. The samples were immediately centrifuged, pipetted into storage vials and stored at −80°C until analysis.

**Maximal aerobic capacity**

\( \text{VO}_{2\text{max}} \) was determined using a continuous, incremental protocol on a motorised treadmill. The initial speed and grade were 2.5 mph and 0%, respectively, with increases of 0.5 mph and 2%, respectively, every 2 min of exercise. The test was terminated at the point of subjective exhaustion. The volume of expired air (VE), volume of oxygen consumption (VO\(_2\)) and volume of carbon dioxide production (VCO\(_2\)) were determined on an Ametek metabolic system (Pittsburgh, PA).

**Blood chemistry**

Fasting glucose, triglyceride, total cholesterol and high-density lipoprotein (HDL) cholesterol concentrations were determined from the resting blood sample. Samples were analysed using an Ortho-Clinical Diagnostics Ectachem DT60 (Johnson & Johnson, Rochester, NY). Coefficients of variation for the various assays at 5 mm were 1% for glucose, 1.5% for triglycerides and 2.5% for the total cholesterol and HDL cholesterol assays.

**Cardiovascular risk**

Estimated risk of CHD was determined using the Framingham Risk equation [22]. This method uses age, HDL cholesterol, total cholesterol, systolic BP, smoking history, diabetes and ECG-related left ventricular hypertrophy (LVH) to predict CHD risk. The risk estimates are reported according to the 10-year probabilities per 100 of developing CHD. The 10-year risk estimate was then compared with the ‘average’ 10-year risk for each subject’s age and gender group [22].

**Performance**

The subjects’ endurance training and performance data were self-reported via a questionnaire. The parameters included years of training, distance run per week, days run per week, personal best for 5 km, 10 km and marathon distances and best performance within the preceding year for the same distances. The subjects also recorded cross training in swimming, cycling and/or resistance exercise, as well as any periods of inactivity due to injury/illness since their previous test. The subject responses were confirmed by oral interview on the day of testing.

**Dietary history**

The subjects were asked to keep a 3-day dietary history prior to testing. Subject responses were confirmed by oral interview on the day of testing. These diet histories were analysed using the Food Processor Plus program (ESHA v. 2, Salem Oregon) for total caloric consumption in kilocalories.
and nutrient intake in grams, including fat, protein (Pro), carbohydrate (CHO), saturated fat and cholesterol.

**Menstrual history**

Female subjects completed a menstrual history questionnaire, including age at menarche, current menstrual history, age at menopause (if relevant) and hormone replacement therapy (HRT) use. Self-reported values were confirmed by oral interview.

**Statistics**

Data were analysed using the Statistical Package for Social Sciences (SPSS) software v. 11.0. Factorial analysis of variance (ANOVA) was used to determine significant main and interaction effects within a 2 (Time) × 2 (Gender) design. Predicted CHD risk was compared to population risk estimates using independent sample t-tests. Differences among women groups based upon hormonal status were determined by one-way ANOVA. Multivariate step-wise regression was used to investigate variables that predicted change in CHD risk factors. Data are reported as mean ± SD, and significance was pre-determined at P<0.05.

**Results**

As no interactions were noted, we report differences between two time points and between genders. The mean age at baseline was 50.8 ± 8.0 years and at post-testing 60.0 ± 7.9 years. Table 1 shows the subject characteristics for the entire cohort. Body mass index (BMI) and body fat changed significantly over time (P<0.05, Table 1), whereas lean body mass (LBM) did not. By gender, men had greater body mass (71.4 ± 7.2 versus 55.4 ± 5.3 kg), height (177.3 ± 6.1 versus 164.1 ± 6.1 cm), LBM (61.0 ± 5.5 versus 43.7 ± 4.0 kg), and lower body fat (14.5 ± 4.1 versus 20.8 ± 4.7%) compared to women (P<0.05). Exercise training and nutritional variables for the entire sample are presented in Table 2. Training volume, training frequency and fitness parameters all declined significantly (P<0.05), whereas none of the nutritional variables changed. By gender, men had greater oxygen consumption (4.0 ± 0.7 versus 2.6 ± 0.4 l/ min, P<0.05) and fitness (56.2 ± 9.3 versus 47.3 ± 7.2 ml/kg/min, P<0.05), while training volume and frequency did not differ. Men also had a greater total caloric (2277 ± 698 versus 1921 ± 564 kcal, P<0.05) and CHO intake (328 ± 122 versus 264 ± 88 g, P<0.05) compared to women. No other dietary variables differed by gender.

Table 3 demonstrates the CHD risk factor variables for the entire cohort. Total cholesterol, triglycerides, glucose, systolic BP and diastolic BP increased, and HDL cholesterol decreased (P<0.05). Men had significantly lower HDL cholesterol (67 ± 17 versus 82 ± 17 mg/dl, P<0.05), and significantly higher blood glucose (96 ± 12 versus 88 ± 9 mg/dl, P<0.05), systolic BP (128 ± 11 versus 115 ± 10 mmHg, P<0.05), and diastolic BP (78 ± 8 versus 72 ± 8 mmHg, P<0.05) than women. Among men, there were two smokers, two diabetics and six with LVH. No women had diabetes or LVH, nor were there any smokers. Since LVH in chronic runners is considered a physiological and not a pathological adaptation [23], we did not include those with diagnosed LVH at baseline as having evidence of CHD.

Among the women subjects, 11 (42.6 ± 2.3 years at visit 1) were pre-menopausal at both visits and 24 (51.3 ± 7.0 years at visit 1) were post-menopausal at one or both visits. Of the latter, 19 women (52.0 ± 7.6 years) were on HRT at the post visit and 5 (50.8 ± 6.5 years) were not. While differences existed between the pre- and post-menopausal women for several variables (age, weight, LBM, total cholesterol, predicted CHD risk) there were no differences between the post-menopausal groups based on HRT status for any of the reported CHD risk factor variables or CHD risk estimate. Therefore, age appears to be a greater influence than menstrual status in this cohort. The 10-year Framingham Risk of CHD increased (P<0.001) in the entire cohort from 4.2 ± 3.8% to 9.2 ± 7.2%. Men had significantly greater 10-year predicted CHD risk than women (5.7 ± 4.0% versus 1.7 ± 1.4%, P<0.05). However, the estimated 10-year risk of CHD was significantly lower than the average 10-year risk for age- and gender-matched population norms developed by the original authors [22]. In brief, the authors developed prediction equations for CHD endpoints, which were based on measurements of several known risk factors using 5573 original and offspring subjects from the Framingham Heart Study. For comparison, the calculated population

| Table 2. Exercise training and nutritional variables at baseline and post-testing (mean ± SD) |
|------------------------------------------|------------------------------------------|------------------|
| Training volume (km/week)               | 61.1 ± 28.2                              | 44.7 ± 24.6      |
| Training frequency (days/week)          | 5.8 ± 1.0                                | 4.9 ± 1.5        |
| VO_{max} (l/min)                        | 3.5 ± 0.9                                | 3.0 ± 0.8        |
| VO_{max} (ml/kg/min)                    | 52.9 ± 9.6                               | 45.0 ± 9.0       |
| Total calories (kcal)                   | 2144 ± 667                               | 2172 ± 555       |
| Carbohydrates (g)                       | 304 ± 116                                | 289 ± 91         |
| Fat (g)                                 | 63 ± 27                                  | 69 ± 33          |
| Protein (g)                             | 82 ± 30                                  | 84 ± 24          |

*Main effect of time, baseline to post comparison for the entire sample.
risk factor variables. Variables did not predict any of the noted changes in CHD revealed that training, nutritional and body composition for these analyses, gender data were combined and age the change in CHD risk factors as the dependent variable. (LBM, % fat) variables were entered as predictors, with calories, CHO, Pro and Fat intake) and body composition (fitness, km/week and days/week) and nutritional (total triglycerides, total cholesterol and adiposity were inversely related to running volume [13, 14]. Therefore, we assumed that the 27% reduction in training volume in these subjects related to running volume [13, 14]. Therefore, we assumed that the 27% reduction in training volume in these subjects would lead to negative changes in CHD risk factors. While the latter did occur, we did not see any association between the respective changes in training volume and CHD risk factors, making the influence of reduced exercise training on the changes in BP and blood chemistries unclear. Furthermore, menopause and the replacement of oestrogen did not appear to influence the change in CHD risk factors in the women runners in this study. This is similar to the reports by Williams and Dreyer et al. [18], suggesting improved lipid profiles in post-menopausal chronic runners regardless of oestrogen replacement status.

The interaction of exercise and nutrition has been shown to have a strong influence on CHD risk factors compared to exercise alone [24]. Therefore, changes in nutritional patterns could confound the interpretation of how exercise influences blood chemistries and body composition with age. In fact, Mengelkoch et al. suggest that favourable changes in dietary habits may be an explanation for the improved total cholesterol profile noted in their athletes [10]. In this current study, dietary habits of the athletes compare favourably with established healthy eating patterns [25] and did not change over the course of the study. Moreover, there were no associations between change in dietary habits and change in CHD risk factors. Therefore, as with exercise training, it is unclear what impact nutritional changes have had on the results from this study.

It could be argued that the increases in CHD risk factors in these older runners reflect primary age-related changes not influenced by lifestyle patterns such as exercise and nutrition [17]. This would be consistent with reported patterns for age-related loss of fitness and performance variables [1–6]. However, exercise clearly establishes healthier profiles that ultimately increase functional capacity at most ages and reduces the lifetime risk of disease and disability. These chronic exercisers demonstrated better HDL cholesterol, triglyceride and BP profiles than reported in the literature for sedentary adults of similar age [8, 26]. This is consistent with previous reports comparing chronic exercisers to sedentary adults [27, 28].

Predicted CHD risk increased over time in this group but remained lower than sedentary normative values. Indeed, at baseline (≈ 50 years of age) and post-testing (≈ 60 years of age), the predicted CHD risk of this group was approximately half that of age- and gender-matched population norms. We anticipated this outcome, since exercise is well known to protect against CHD and premature mortality [7, 8]. The reduced risk reflects the high fitness level of this cohort (VO\textsubscript{2max} ≈ 45 ml/kg/min at 60 years of age) compared to non-athletes of similar age (VO\textsubscript{2max} ≈ 30 ml/kg/min), as well as favourable CHD risk factor profiles. Of interest, at pre-testing it was predicted that 4.5% of the athletes would

Table 3. Coronary heart disease risk factor variables at baseline and post-testing (mean ± SD)

<table>
<thead>
<tr>
<th></th>
<th>Baseline (N = 91)</th>
<th>Post (N = 91)</th>
<th>P-value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol (mmol/l)</td>
<td>5.04 ± 0.93 (195 ± 36)</td>
<td>5.56 ± 0.93 (215 ± 36)</td>
<td>0.001</td>
</tr>
<tr>
<td>HDL cholesterol (mmol/l)</td>
<td>1.89 ± 0.49 (73 ± 19)</td>
<td>1.66 ± 0.49 (64 ± 19)</td>
<td>0.001</td>
</tr>
<tr>
<td>Triglycerides (mmol/l)</td>
<td>0.84 ± 0.41 (74 ± 36)</td>
<td>0.99 ± 0.60 (88 ± 53)</td>
<td>0.025</td>
</tr>
<tr>
<td>Glucose (mg/dl)</td>
<td>5.11 ± 0.67 (92 ± 12)</td>
<td>5.44 ± 0.78 (98 ± 14)</td>
<td>0.02</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>123 ± 13</td>
<td>131 ± 18</td>
<td>0.001</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>76 ± 8</td>
<td>81 ± 10</td>
<td>0.001</td>
</tr>
</tbody>
</table>

*a Main effect of time, baseline to post comparison for the entire sample.
CHD risk factors in older runners

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Low serum carotenoids and development of severe walking disability among older women living in the community: the Women’s Health and Aging Study I

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Abstract

Objective: to determine whether low serum carotenoid levels, an indicator of low intake of fruits and vegetables, are associated with the progression of disability in older women.

Design: longitudinal analysis in a population-based cohort.

Setting: moderately–severely disabled women, ≥65 years, living in the community in Baltimore, Maryland (the Women’s Health and Aging Study I).

Participants: 554 women without severe walking disability (inability to walk or walking speed <0.4 m/s) at baseline.

Main outcome measure: incidence of severe walking disability assessed every 6 months over 3 years.

Results: 155 women (27.9%) developed severe walking disability during follow-up. Rates of development of severe walking disability per 100 person-years among women in the lowest and in the three upper quartiles of total carotenoids were, respectively, 13.8 versus 10.9 (P = 0.0017). Adjusting for confounders, women in the lowest quartile of total carotenoids were more likely to develop severe walking disability (hazards ratio 1.57, 95% confidence interval 1.24–2.00, P = 0.0002) compared with women in the three upper quartiles.

Conclusion: low serum carotenoid levels, an indicator of low intake of fruits and vegetables, are independent predictors of the progression towards severe walking disability among older women living in the community.

Keywords: ageing, carotenoids, disability, risk factors, women, elderly

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