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Fitness, fatness, and coronary heart disease risk in adolescents: the Northern Ireland Young Hearts Project

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ABSTRACT

BOREHAM, C., J. TWISK, L. MURRAY, M. SAVAGE, J. J. STRAIN, and G. CRAN. Fitness, fatness, and coronary heart disease risk in adolescents: the Northern Ireland Young Hearts Project. Med. Sci. Sports Exerc., Vol. 33, No. 2, pp. 270–274, 2001. Purpose: The purpose of this study was to examine the independence and relative strengths of association between coronary heart disease (CHD) risk status and both body fatness and cardiorespiratory (C-R) fitness in 12- and 15-yr-old adolescents. Methods: The study cohort consisted of 1015 schoolchildren aged 12 and 15 yr (251 12-yr-old boys, 258 12-yr-old girls, 252 15-yr-old boys, and 254 15-yr-old girls), representing a 2% random sample of each population group. For each child, height, weight, sexual maturity (pubertal status), skin-fold thicknesses (4 sites), blood pressure (random zero sphygmanometer), nonfasting serum total, and high density lipoprotein (HDL)-cholesterol and C-R fitness (20-m shuttle run; 20-MST) were determined under standardized conditions. Socioeconomic status and habitual physical activity were also determined from questionnaire information. Multiple regression analyses were carried out to examine relationships between five CHD risk factors, and fitness and fatness and to examine the relative strengths of fitness and fatness on CHD risk status, correcting for potential confounding variables. Results: Our main findings were: 1) Relationships between fatness and CHD risk factors are invariably stronger than between fitness and the same risk factors. For example, partially adjusted standardized regression coefficients for 12-yr-old boys revealed significant relationships between all five CHD risk factors and fatness, compared with three of five for fitness. The corresponding figures for 12-yr-old girls were three of five (fatness) and one of five (fitness). Broadly similar results were apparent for 15-yr-olds. 2) Although relationships between fitness and CHD risk factors do not survive further adjustment for fatness, the relationships between fatness and CHD risk are more robust and are unaffected by further adjustment for fitness. Conclusion: Our results indicate that the observed relationships between C-R fitness and CHD risk status in adolescents are mediated by fatness, whereas the observed relationships with fatness are independent of fitness. Primary prevention of CHD during childhood should therefore concentrate upon preventing or reversing undue weight gain. Key Words: OBESITY, AEROBIC FITNESS, CARDIOVASCULAR HEALTH, YOUNG PEOPLE

In adult populations, strong inverse associations have been demonstrated between aerobic fitness and both coronary heart disease (CHD) risk factor status (16,37,45) and CHD mortality (13,34). These relationships hold for men and for women, and are independent of other potential confounding risk factors, including body fatness. In children and adolescents, however, these relationships appear to differ. Although higher levels of fitness are consistently associated with more favorable CHD risk factor profiles in children, several studies have indicated that such relationships may be mediated by body fatness rather than aerobic fitness per se (5,17,20,33,39). Because CHD is a lifelong process (4), such generational differences in the etiology of risk, if confirmed, would have major implications for risk reduction strategies employed at different stages of the life course. The purpose of the present study was to further examine the relationships between CHD risk status and both fatness and fitness in 12- and 15-yr-old adolescents, and to establish the relative independence and strengths of association so that appropriate CHD preventive strategies can be formulated for this age group.

MATERIALS AND METHODS

The Young Hearts Project is a longitudinal study of CHD risk factors in a sample of young people from Northern Ireland. The original baseline survey, which provides the basis for the current investigation, was completed in 1990 (9).

Subjects. The study population consisted of 1015 schoolchildren (251 12-yr-old boys, 258 12-yr-old girls, 252 15-yr-old boys, and 254 15-yr-old girls) selected from 16 schools in a random, stratified sample representative of each of the five regional school boards, as well as the two sectors of postprimary education in Northern Ireland. The target sample size of 250 per age/gender group was based on the variability of pilot study results and represented a 2% random sample of each population group. A more detailed account of sampling methodology and response characteristics is given elsewhere (9). The overall response rate was 0.195-9131/01/3302-0270/$3.00/0

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with no discernible pattern for nonparticipation that would influence the validity of the random nature of the sample. Ethical approval was obtained from the Medical Research Ethical Committee of The Queen’s University of Belfast, and written consent was obtained from the parent or guardian and all participating children.

Testing procedures. For each child the test protocol included a medical examination during which height, weight, and sexual maturity by pubertal status (38) were determined. Skin-fold thicknesses were obtained from four sites (biceps, triceps, subscapular, and suprailiac) and summed for the estimation of body fatness (44). Blood pressure was measured twice from the right arm, using a Hawksley random-zero sphygmomanometer, with subjects sitting quietly beforehand for at least 5 min. Systolic blood pressure (SBP) was recorded as the mean of the two values for Korotkoff phase I, whereas the diastolic blood pressure (DBP) was based on the mean of two values for phase V (15-yr-olds) or phase IV (12-yr-olds). Nonfasting blood samples were drawn from the antecubital vein under local anesthesia and separated into serum/plasma within 4 h. Total cholesterol concentration was estimated by an enzymatic technique (CHOD-PAP, Boehringer Mannheim, Germany) and HDL cholesterol concentration by phosphotungstic magnesium reagents. All assays were performed in a laboratory conforming to World Health Organization standards. During the period of the study, intra-assay coefficients of variation (C-vs) were <3.0% for total and HDL cholesterol measurements, and inter assay C-vs were <5.0%. Socioeconomic status was determined from occupational information provided by the parent or guardian of each child and was classified according to the Office of Population Census and Surveys (28). A physical activity score was computed for each subject based on confidential questionnaire information on everyday physical activities (for example, method of transportation to and from school, activities during breaks in the school day, sports participation after school, and so on). Children were “cued” with a time-based framework using the structure of a typical school day. This method was felt to be more appropriate than cueing by activities, which is the more traditional method (3). Answers were coded according to frequency, intensity, and duration, appropriately weighted, and the activity score (from 1 to 100) computed (30). Although specific psychometric data are not available for this questionnaire, it has demonstrated both concurrent validity (25) and predictive validity in relation to physical fitness (10). Cardiorespiratory fitness (C-R fitness) was determined by the 20-m endurance shuttle run. This test requires subjects to run back and forth between two lines set 20 m apart. Running pace is determined by audio signals, emitted from a prerecorded cassette tape, the initial velocity being 8.5 km·h⁻¹, and increasing by 0.5 km·h⁻¹ every minute. A constant level of encouragement is given to participants throughout the test. The test is terminated when the child fails to reach the end lines concurrent with the audio signals on two consecutive occasions. Scoring is by “laps” completed, and the test has been validated for use with children (11,43). Throughout the duration of the project, all tests were carried out in the same order by the same members of the testing team.

Statistical analyses. Data were coded, checked, and entered onto an ICL 3980 mainframe computer for subsequent analysis using SPSSX. To assess the relationship between fitness and fatness, Pearson product moment correlation coefficients were calculated between these two parameters for all four age and gender groups. To examine the relationship between CHD risk status and fitness and fatness and to examine the relative importance of fitness and fatness, several multiple regression analyses were carried out. First, a “crude” analysis was performed in which fitness and fatness were related to CHD risk factors separately, correcting for social class and maturation. Second, multiple linear regression analyses were carried out in which both fitness and fatness were analyzed together in relation to CHD risk factors, again correcting for social class and maturation; the second analysis will be referred to as “adjusted” analysis. Both crude and adjusted analysis were performed for all four age and gender groups.

RESULTS

Table 1 shows the descriptive data for the main variables measured at age 12 and 15 yr, and the Pearson’s product moment correlations between aerobic fitness and fatness for all four age/sex groups. The correlation coefficients indicate moderate to strong relationships for all groups.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Boys 12 yr (N = 251)</th>
<th>Boys 15 yr (N = 252)</th>
<th>Girls 12 yr (N = 258)</th>
<th>Girls 15 yr (N = 254)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Height (cm)</td>
<td>149.7 (7.9)</td>
<td>168.9 (7.2)</td>
<td>151.0 (7.5)</td>
<td>160.1 (5.9)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>42.6 (9.4)</td>
<td>58.6 (9.4)</td>
<td>44.0 (9.0)</td>
<td>56.6 (8.6)</td>
</tr>
<tr>
<td>Sum of four skinfolds (cm)</td>
<td>37.9 (20.6)</td>
<td>34.1 (16.4)</td>
<td>43.6 (15.8)</td>
<td>52.1 (16.8)</td>
</tr>
<tr>
<td>20-m shuttle run (no. of laps)</td>
<td>58.7 (18.8)</td>
<td>79.2 (21.3)</td>
<td>44.3 (13.2)</td>
<td>45.4 (13.5)</td>
</tr>
<tr>
<td>Systolic BP (mm Hg)</td>
<td>111.0 (11.6)</td>
<td>123.2 (12.4)</td>
<td>111.6 (12.2)</td>
<td>118.2 (11.8)</td>
</tr>
<tr>
<td>Diastolic BP (mm Hg)</td>
<td>80.0 (9.5)</td>
<td>73.4 (9.4)</td>
<td>70.3 (8.1)</td>
<td>74.2 (8.8)</td>
</tr>
<tr>
<td>Total cholesterol (mmol L⁻¹)</td>
<td>4.6 (0.8)</td>
<td>4.2 (0.7)</td>
<td>4.7 (0.8)</td>
<td>4.6 (0.8)</td>
</tr>
<tr>
<td>HDL (mmol L⁻¹)</td>
<td>1.4 (0.3)</td>
<td>1.3 (0.3)</td>
<td>1.4 (0.3)</td>
<td>1.4 (0.3)</td>
</tr>
<tr>
<td>TC/HDL</td>
<td>3.5 (1.0)</td>
<td>3.5 (1.0)</td>
<td>3.6 (0.9)</td>
<td>3.5 (0.8)</td>
</tr>
<tr>
<td>Correlation between fitness and fatness</td>
<td>-0.62</td>
<td>-0.46</td>
<td>-0.50</td>
<td>-0.41</td>
</tr>
</tbody>
</table>

**TABLE 1.** Mean and standard deviation (s) of the variables of interest.
Tables 2 and 3 show the standardized regression coefficients for relationships between aerobic fitness, fatness, and CHD risk factors for boys and girls, respectively. The tables are arranged so that regression coefficients can be compared between crude analyses (whereby statistical correction is made for social class and maturation) and adjusted analyses, which includes additional adjustment for fitness (if the independent variable is fatness) or fatness (if the independent variable is fitness). Such an arrangement allows a judgement on the relative importance of fitness and fatness, regarding their relationship with CHD risk factors.

It is clear from both Tables 2 and 3 that, first, relationships (crude) between fatness and CHD risk factors are invariably stronger than between fitness and the same risk factors. Second, although the relationships between fitness and CHD risk factors do not, in the main, survive further adjustment for fatness, the relationships between fatness and CHD risk factors are more robust and are unaffected by further adjustment for fitness. Thus, the relationship observed for fitness in the crude analyses can be explained by the association with fatness, whereas the observed relationships with fatness are independent of fitness. Finally, in general, the above relationships are stronger for male rather than female subjects, and for younger rather than older adolescents.

DISCUSSION

In keeping with previous studies in children, the present investigation revealed moderate to strong correlations between fitness and fatness (17,20,39) and between fitness and CHD risk factors (5,33,39). However, the latter were strongly confounded by body fatness, whereas the more robust associations found between fatness and CHD risk factors proved to be independent of fitness and other potential confounders. Although such results may be partly influenced by the more objective nature of the fatness test compared with the C-R fitness test employed in the present study, the overall findings confirm that children’s health status may be influenced more by body fatness than by aerobic fitness. This is in contrast to the reported findings from adult studies, which have, on the whole, shown that fitness and changes in fitness are strong predictors of CHD risk and mortality, which are largely independent of body fatness (7,8,13,16,34,37,45).

At least part of the explanation for this discrepancy may lie in the different end points used by children’s and adult studies. Although the latter have recourse to CHD mortality as a dependent variable, studies on children are restricted to levels of precursor risk factors for atherosclerosis. Thus, different independent relationships may exist between fitness and CHD mortality in adults, and fatness and CHD risk status in children. It is known, for example, that higher levels of physical activity and fitness in adults may protect against acute cardiac events by a variety of mechanisms including a reduced myocardial vulnerability to ventricular arrhythmias, reduced platelet stickiness and aggregability, and increased fibrinolysis (21,24). Acute coronary events are extremely rare in children, however, and so this protective mechanism of physical activity and fitness is not manifest (or is of little relevance).

Rather, factors affecting precursors of atherosclerosis are of paramount importance, because atherosclerosis is now established as a lifelong degenerative process (4) beginning in childhood. In this regard, our study, in keeping with others (5,17,18,20,33,39) indicates that high fatness rather than low fitness is related independently to poorer CHD risk factors in children. It is known, for example, that higher levels of physical activity and fitness in adults may protect against acute cardiac events by a variety of mechanisms including a reduced myocardial vulnerability to ventricular arrhythmias, reduced platelet stickiness and aggregability, and increased fibrinolysis (21,24). Acute coronary events are extremely rare in children, however, and so this protective mechanism of physical activity and fitness is not manifest (or is of little relevance).

Rather, factors affecting precursors of atherosclerosis are of paramount importance, because atherosclerosis is now established as a lifelong degenerative process (4) beginning in childhood. In this regard, our study, in keeping with others (5,17,18,20,33,39) indicates that high fatness rather than low fitness is related independently to poorer CHD risk factors status in children.

Childhood obesity has been strongly implicated in the clustering of risk factors (2,6,18,36,41) and their tracking into young adulthood (2,14,42). Pathological evidence has also linked antemortem levels of fatness in children with the extent of atherosclerosis in major arteries at autopsy (4,23),

**TABLE 2. Standardized regression coefficients regarding the relationship between fitness, fatness and CHD risk factors for boys.**

<table>
<thead>
<tr>
<th></th>
<th>Boys (12 yr)</th>
<th></th>
<th></th>
<th>Boys (15 yr)</th>
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<tbody>
<tr>
<td></td>
<td>Fitness</td>
<td>Fatness</td>
<td>Fitness</td>
<td>Fatness</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Crude</td>
<td>Adjusted</td>
<td>Crude</td>
<td>Adjusted</td>
<td></td>
</tr>
<tr>
<td>Systolic BP</td>
<td>-0.16*</td>
<td>0.01</td>
<td>0.29**</td>
<td>0.27**</td>
<td></td>
</tr>
<tr>
<td>Diastolic BP</td>
<td>-0.11</td>
<td>0.11</td>
<td>0.30**</td>
<td>0.35**</td>
<td></td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>-0.28**</td>
<td>-0.11</td>
<td>0.36**</td>
<td>0.27**</td>
<td></td>
</tr>
<tr>
<td>HDL cholesterol</td>
<td>0.06</td>
<td>-0.05</td>
<td>-0.13*</td>
<td>-0.17*</td>
<td></td>
</tr>
<tr>
<td>TC:HDL</td>
<td>-0.22**</td>
<td>-0.04</td>
<td>0.31**</td>
<td>0.29**</td>
<td></td>
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</table>

Crude, correcting for social class and maturation; Adjusted, also correcting for either fitness (20-m shuttle run score) or fatness (sum of four skinfolds).

* P < 0.05; ** P < 0.01.

**TABLE 3. Standardized regression coefficients regarding the relationship between fitness, fatness, and CHD risk factors for girls.**

<table>
<thead>
<tr>
<th></th>
<th>Girls (12 yr)</th>
<th></th>
<th></th>
<th>Girls (15 yr)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Fitness</td>
<td>Fatness</td>
<td>Fitness</td>
<td>Fatness</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Crude</td>
<td>Adjusted</td>
<td>Crude</td>
<td>Adjusted</td>
<td></td>
</tr>
<tr>
<td>Systolic BP</td>
<td>0.01</td>
<td>0.11</td>
<td>0.12*</td>
<td>0.18*</td>
<td></td>
</tr>
<tr>
<td>Diastolic BP</td>
<td>-0.10</td>
<td>0.02</td>
<td>0.22**</td>
<td>0.23**</td>
<td></td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>0.04</td>
<td>0.12</td>
<td>0.11</td>
<td>0.17*</td>
<td></td>
</tr>
<tr>
<td>HDL cholesterol</td>
<td>0.16*</td>
<td>0.08</td>
<td>-0.22**</td>
<td>-0.18*</td>
<td></td>
</tr>
<tr>
<td>TC:HDL</td>
<td>-0.11</td>
<td>0.03</td>
<td>0.28**</td>
<td>0.27**</td>
<td></td>
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</table>

Crude, correcting for social class and maturation; Adjusted, also correcting for either fitness (20-m shuttle run score) or fatness (sum of four skinfolds).

* P < 0.05; ** P < 0.01.
whereas a longer-term study (27) has shown that overweight in adolescence was a more powerful predictor of adult morbidity and mortality than overweight in adulthood. It would also appear that relationships between fatness and CHD risk status are somewhat stronger than those previously reported between physical activity and CHD status for this population (10).

Such evidence suggests that childhood obesity may well be the single most important modifiable risk factor in the pathogenesis of CHD during childhood. What are the implications of such findings for public health and primary prevention strategies? It is clear that an emphasis on improving physical fitness levels in middle-aged men and women as a strategy for the prevention of CHD is justified based on the available evidence from adult studies (8,13,34). Evidence for the benefits of weight loss for cardiovascular health in adults is more equivocal. Indeed, advocating weight loss in middle-aged individuals may even increase the risk of CHD (15). However, for children there appears to be a prima facia case for the primary focus on prevention of CHD to be on the avoidance or reversal of undue weight gain. Prevention is of particular importance, because it is estimated that 80% of obese adolescents become obese adults (33). More worryingly, despite the acknowledged adverse health outcomes associated with childhood obesity and despite increasing obesity among the pediatric populations of developed countries (40), it is estimated that only 20% of obese adolescents receive treatment for their condition. The cornerstones of such preventive measures on a population basis are enhanced physical activity levels (10,26,29) and adoption of a healthier diet (32). Indeed, an earlier analysis of this population (10) indicated that for adolescent females at least, there is an inverse relationship between physical activity levels and percentage body fat. As physical activity is a behavior, whereas physical fitness is an attribute (31), behavioral intervention at a young age is required. Specific measures relating to a reduction of television viewing time (1,12), increased exercise (19), and healthier school meals (22) may prove particularly effective.

In conclusion, the results of the present-day study confirm previous reports that fatness is strongly and independently associated with CHD risk status in adolescents and that aerobic fitness is not. Primary prevention of CHD during childhood should therefore concentrate upon preventing or reversing undue weight gain.

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