Decline in Physical Fitness From Childhood to Adulthood Associated With Increased Obesity and Insulin Resistance in Adults

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OBJECTIVE — To examine how fitness in both childhood and adulthood is associated with adult obesity and insulin resistance.

RESEARCH DESIGN AND METHODS — A prospective cohort study set in Australia in 2004–2006 followed up a cohort of 647 adults who had participated in the Australian Schools Health and Fitness Survey in 1985 and who had undergone anthropometry and cardiorespiratory fitness assessment during the survey. Outcome measures were insulin resistance and obesity, defined as a homeostasis model assessment index above the 75th sex-specific percentile and BMI ≥30 kg/m^2, respectively.

RESULTS — Lower levels of child cardiorespiratory fitness were associated with increased odds of adult obesity (adjusted odds ratio [OR] per unit decrease 3.0 [95% CI 1.6–5.6]) and insulin resistance (1.7 [1.1–2.6]). A decline in fitness level between childhood and adulthood was associated with increased obesity (4.5 [2.6–7.7]) and insulin resistance (2.1 [1.5–2.9]) per unit decline.

CONCLUSIONS — A decline in fitness from childhood to adulthood, and by inference a decline in physical activity, is associated with obesity and insulin resistance in adulthood. Programs aimed at maintaining high childhood physical activity levels into adulthood may have potential for reducing the burden of obesity and type 2 diabetes in adults.

Diabetes Care 32:683–687, 2009

The increasing burden of adult obesity and type 2 diabetes has made the search for prevention more urgent. Trials indicate that the prevalence of obesity and incidence of type 2 diabetes can be reduced by physical activity interventions in adulthood even over 3 years (1).

The role of physical activity in childhood is unclear. With little cohort and no trial data, the decline in physical activity from childhood to adulthood has been linked to adult adiposity, but the associations have been variable (2–6). The inability to find a clear association may be due to measurement error in these studies, which compared questionnaire data over time.

Change in cardiorespiratory fitness in an individual is strongly correlated with a change in habitual energy expenditure and physical activity undertaken during leisure time (7). Data on change in cardiorespiratory fitness from one time point to another might be a better marker of change in physical activity (8). Change in fitness between the ages 17 and 25 years has been more strongly associated with adult measures of adiposity than with change in physical activity by questionnaire (2). No study has reported on the association of change in cardiorespiratory fitness between childhood and adulthood with adult insulin resistance, a type 2 diabetes precursor.

We examined the association of change in cardiorespiratory fitness from childhood to adulthood with obesity and insulin resistance in adulthood.

RESEARCH DESIGN AND METHODS

— The Childhood Determinants of Adult Health study is a prospective cohort study (9,10). Baseline data were collected in 1985 on a representative sample of 8,498 children aged 7–15 years as part of the Australian Schools Health and Fitness Survey. Sampling procedures and methods of data collection are presented elsewhere (11). Children who were aged 9, 12, or 15 years underwent additional measurements, with 2,595 completing a submaximal cardiorespiratory fitness test and an estimate of lean body mass. The follow-up study was performed from 2004 to 2006 across 34 clinics. In total, 60.8% (n = 5,170) provided follow-up data, with 19.5% (n = 1,658) unable to be traced, 9.6% (n = 817) not responding to contact, 9.0% (n = 767) refusing to participate, and 1.0% (n = 86) deceased. Of the original sample with child fitness, 61.3% (n = 1,590) participated in the follow-up, and 647 of these also had an adult measure of cardiorespiratory fitness and lean body mass at a clinic. The childhood characteristics of those with child fitness who also had adult fitness measured were very similar to those who did not, with the exception of socioeconomic status (SES) (i.e., mean age 11.8 vs. 10.8 years, male 51.6 vs. 50.6%, mean BMI 18.5 vs. 18.2 kg/m^2, and mean fitness 2.71 vs. 2.75 W/kg, lower SES 22.8% vs. higher SES 31.3% in the highest quartile; $P < 0.01$). Further, the child-to-adulthood trajectory for BMI increase for these 647 was very similar to

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Received 4 September 2008 and accepted 15 December 2008.
Published ahead of print at http://care.diabetesjournals.org on 23 December 2008. DOI: 10.2337/dc08-1638.

that of all of the participants in the follow-up (mean increase in BMI 6.4 vs. 6.9 kg/m^2; $P = 0.62$, respectively). The studies were ethics board approved and had attained written informed consent.

Outcome variables

Obesity. BMI was calculated as the ratio of weight in kilograms to squared height in meters (kg/m^2). Obesity in adulthood was defined as a BMI ≥30 kg/m^2.

Insulin resistance. Fasting plasma glucose levels were measured by the Olympus AU5400 automated analyser (Olympus, Middlesex, U.K.). Two methods of insulin determination were used during the follow-up study. Fasting plasma insulin was measured by a microparticle enzyme immunoassay kit (AxSYM; Abbot Laboratories, Abbot Park, IL) and by electrochemiluminescence immunoassay (Elecsys Modular Analytics E170; Roche Diagnostics, Mannheim, Switzerland) with interassay standardization.

Insulin sensitivity was estimated by the homeostasis model assessment (HOMA) index, defined as the product of fasting insulin in μU/ml and fasting glucose in mmol//l divided by 22.5 (12). Insulin resistance was defined as HOMA index at or above the 75th sex-specific percentile (13).

Predictor variable: cardiorespiratory fitness. Cardiorespiratory fitness was estimated at baseline and follow-up as physical working capacity at a heart rate of 170 bpm on a bicycle ergometer (Monark Exercise AB, Vansbro, Sweden) pedaled at a rate of 60 rpm (14). Baseline and follow-up cardiorespiratory fitness was expressed in relative terms as watts per kilogram of lean body mass. Relative cardiorespiratory fitness is preferred because the absolute workload achieved is a function of muscle mass (15).

Lean body mass in childhood and adulthood was calculated from body density and percent body fat equations that used baseline and follow-up measures of skinfold thickness. In 1985, triceps, biceps, subscapular, and suprailiac skinfolds were measured at locations determined by reference to anatomical landmarks on the right side of the body (16) using Holtain Calipers (Holtain, Crymych, U.K.) to the nearest 0.1 mm. Skinfold measures >40 mm at follow-up were truncated, and skinfold values were imputed from BMI and waist circumference. Body density was estimated from the log of the sum of four skinfolds using age-specific regression equations (16–18). Calculations of body fat were made (19), and lean body mass was estimated by subtracting fat mass from total body mass.

Table 1—*Summary of study variables in childhood and adulthood*

	Male		Female	
	n	Statistic	*n*	Statistic
Childhood (1985)				
Age (years)	334	11.9 ± 2.4	313	11.8 ± 2.4
SES	324		305	
Highest quartile		31.2		31.5
3rd quartile		25.9		26.9
2nd quartile		33.3		35.7
Lowest quartile		9.6		5.9
Relative cardiorespiratory fitness (W/kg)	334	3.02 ± 0.62	313	2.48 ± 0.65
Lean body mass (kg)	334	36.4 ± 11.3	313	31.7 ± 7.3
BMI (kg/m^2)	334	18.4 ± 2.7	313	18.5 ± 2.8
Smoking prevalence	328	10.4	308	12.0
Adulthood (2004–2006)				
Age (years)	334	31.9 ± 2.5	313	31.7 ± 2.6
Relative cardiorespiratory fitness (W/kg)	334	3.07 ± 0.59	313	2.97 ± 0.69
Lean body mass (kg)	334	64.4 ± 7.6	313	45.0 ± 6.7
Glucose (mmol/l)	329	5.19 ± 0.42	306	4.83 ± 0.41
Insulin (μU/ml)	323	7.36 ± 5.27	301	6.66 ± 3.92
HOMA index	323	1.73 ± 1.35	301	1.46 ± 0.93
BMI (kg/m^2)	334	26.4 ± 3.9	313	25.0 ± 5.5
Smoking prevalence	319	21.6	306	16.7
Alcohol consumption (g/week)	284	47 (17–100)	275	28 (11–83)
Physical activity (steps/day)	276	9,171 ± 3,802	258	9,121 ± 3,083
Obese	334	15.3	313	14.4
Insulin resistance	323	24.8	301	24.9

Data are means ± SD, %, or median (interquartile range) unless otherwise indicated.

Covariates

Socioeconomic status. The residential postcode of each child was used to derive area-level SES based on the Australian Bureau of Statistics index of relative socioeconomic disadvantage derived from the 1981 population census (20).

Alcohol consumption. Alcohol consumption at follow-up was calculated from an alcohol questionnaire using a frequency grid (21).

Smoking. Cigarette smoking at baseline and follow-up was measured by self-administered questionnaire. Those smoking daily were classified as regular smokers.

Dietary habits. Information about food habits was collected at follow-up. Scores from three questions linked to fat intake (milk type, meat fat, and use of spreads) formed a variable for fat intake.

Physical activity. In the adult survey only, pedometers (Yamax Digiwalker SW-200; Yamax USA, San Antonio, TX) were worn during waking hours for seven consecutive days to measure ambulatory

Table 2—*Daily step counts and relative fitness in adulthood according to changes in fitness from childhood to adulthood*

	Physical activity		Fitness	
	n	Steps/day	*n*	W/kg
Persistent unfit	82	8,948 ± 2,593	89	2.33 ± 0.29
Decreasing fitness	167	8,461 ± 3,093	163	2.65 ± 0.38
Persistent moderate fitness	63	9,300 ± 2,921	66	2.97 ± 0.18
Increasing fitness	148	9,908 ± 4,407	164	3.45 ± 0.46
Persistent high fitness	74	9,262 ± 3,164	77	3.81 ± 0.51
P		0.007		<0.001

Data are means ± SD unless otherwise indicated.

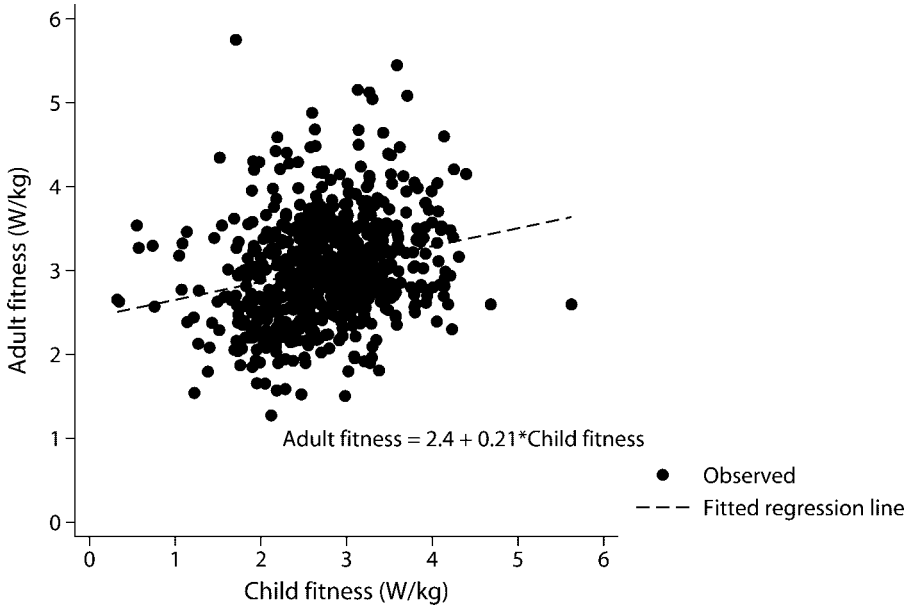

Figure 1—*Scatter plot of adult fitness level versus child fitness level.*

activity (22). For participants with four or more measurement days, the average number of steps per day was calculated. Physical activity was also measured by questionnaire in both childhood and adulthood. The associations for both childhood physical activity and change in physical activity were not statistically significant except for change in physical activity and obesity ($P = 0.05$).

Statistical analyses

The characteristics of subjects included in this analysis were compared with other subjects using Student's *t* test for continuous variables and the χ^2 test for categorical variables. A life course analysis (23) was taken to relate cardiorespiratory fitness to the outcomes of BMI and HOMA, obesity status, and insulin resistance. Linear and logistic regression was used for the continuous or binary outcomes, respectively. The coefficient (or OR) for child fitness quantifies the total effect of poor child fitness (i.e., mediated via adult fitness and unmediated) (24). The coefficient for change in fitness quantifies the additional detriment that results from decreasing fitness between childhood and adulthood. Adjustment for confounders is listed in table footnotes.

Also, a composite fitness variable was created. Subjects who were in the lowest fitness category both in childhood and adulthood were classified as "persistent low fitness" (lowest tertile in both childhood and adulthood), and those who dropped by one or two categories between childhood and adulthood were classified as "decreasing fitness." The other categories were "persistent moderate fitness" (middle tertile at both life stages), "increasing fitness" (an increase of one or two categories), and "persistent high fitness" (highest tertile at both life stages). The relationship between this profile and the number of steps at adulthood and fitness level at adulthood are examined with ANOVA. Tests for interaction did not show differential effects between male and female subjects.

RESULTS — Table 1 shows that in childhood but not adulthood, male subjects had higher mean values for fitness and lean body mass than female subjects. Mean fitness increased for females but not male subjects from childhood to adulthood, and BMI increased substantially for both.

Table 2 indicates that there is a clear association between increasing composite child-adult fitness and mean step count ($P = 0.007$). Pedometer step count was associated with adult fitness ($P < 0.001$).

Figure 1 shows that for each unit increase in child fitness, adult fitness increased by 0.21 (95% CI 0.14–0.28) units. Table 3 shows that subjects who were unfit as children had increased odds of obesity and insulin resistance. Similar findings were evident for serum insulin level (data not shown).

Fit children were more likely to be fit

Table 3—*Effect of change in fitness from childhood to adulthood on obesity and insulin resistance in adulthood for all subjects*

		Adjusted for sex and baseline age only		Fully adjusted*	
Outcome	Predictor	Statistic	*P*	Statistic	*P*
Obesity	Poorer child fitness	3.0† (1.8–5.1)	<0.001	3.0† (1.6–5.6)	<0.001
	Decreasing fitness	3.9‡ (2.5–6.1)	<0.001	4.5‡ (2.6–7.7)	<0.001
Insulin resistance	Poorer child fitness	1.9† (1.3–2.9)	0.001	1.7† (1.1–2.6)	0.01
	Decreasing fitness	2.2‡ (1.5–3.0)	<0.001	2.1‡ (1.5–2.9)	<0.001
Adult BMI (kg/m^2)	Poorer child fitness	1.30§ (0.56–2.04)	<0.001	0.96§ (0.34–1.58)	0.002
	Decreasing fitness	1.86‖ (1.27–2.44)	<0.001	1.72‖ (1.22–2.21)	<0.001
Adult HOMA index	Poorer child fitness	0.26§ (0.08–0.45)	0.007	0.18§ (−0.0003 to 0.36)	0.05
	Decreasing fitness	0.38‖ (0.24–0.52)	<0.001	0.33‖ (0.19–0.48)	<0.001

Data are OR (95% CI) or regression coefficient (95% CI) unless otherwise indicated. Obesity, adult BMI ≥ 30 kg/m^2. Insulin resistance, adult HOMA index above the 75th sex-specific percentile. Sample sizes were 601 for the analyses of obesity status and BMI and 581 for the analyses of the HOMA outcomes. *Adjusted for sex, age, SES at baseline, and education level at follow-up. Analyses of obesity status and BMI additionally adjusted for BMI at baseline; analyses of HOMA outcomes additionally adjusted for waist circumference at baseline. †OR for a 1-unit decrease in childhood fitness. ‡OR for a 1-unit decrease in fitness change. §Coefficient, i.e., the mean increase in the outcome for a 1-unit decrease in childhood fitness. ‖Coefficient, i.e., the mean increase in the outcome for a 1-unit decrease in fitness change.

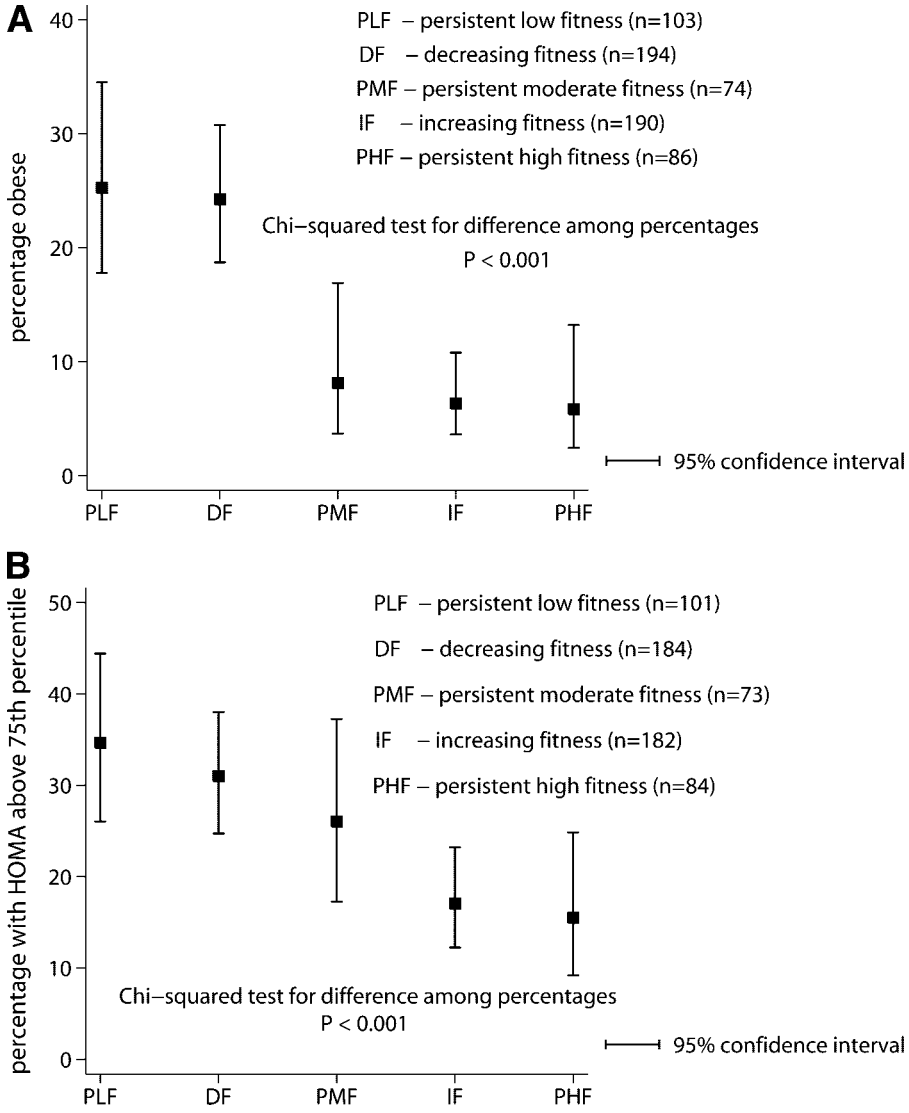

Figure 2—*Percentage of obese subjects and those with HOMA above the 75th percentile for each of the five fitness-change categories: persistent low fitness (PLF), decreasing fitness (DF), persistent moderate fitness (PMF), increasing fitness (IF), and persistent high fitness (PHF). A: The numbers of subjects in the categories were 103 (PLF), 194 (DF), 74 (MMF), 190 (IF), and 86 (PHF). B: The numbers of subjects in the categories were 101 (PLF), 184 (DF), 73 (MMF), 182 (IF), and 84 (PHF).*

adults (Fig. 1), and so fitness in childhood has an important indirect effect, mediated by adult fitness. The results also suggested that there were clear disadvantages for subjects who decreased their fitness levels between childhood and adulthood, with this predictor showing even stronger associations with adult outcomes than did degree of child fitness. The estimated coefficients and ORs were similar to those from analyses in which further adjustments for smoking status and alcohol and fat intake in adulthood were made (data not shown).

Figure 2 shows that the proportion of subjects with obesity and the proportion of subjects with insulin resistance were higher in the decreasing-fitness and persistent-low-fitness groups than in the persistent-high-fitness and increasing-fitness groups. Taken together, these findings suggest that confounding by initial child fitness status or BMI does not explain the association of decreasing fitness with higher adult obesity and higher insulin resistance. The principal factor associated with low risk of obesity and insulin resistance in early adulthood is a relatively high level of adulthood fitness. To assess the robustness of these estimates to loss to follow-up, population-weighted analyses of the following variables measured at baseline were implemented: sex, age, socioeconomic status, smoking status, BMI, and relative fitness. The weight for each subject was the inverse of the probability of providing follow-up data given their status on the above factors. The results for the weighted analyses were similar to the nonweighted analyses, suggesting that there is no loss–to–follow-up bias due to lack of representativeness on the variables used to calculate the weights.

We restricted the analysis to nonobese children only and again found that lower child fitness (adjusted OR 2.9 [95% CI 1.8–4.8]; $P < 0.001$) and decreasing fitness (3.9 [2.5–6.1]; $P < 0.001$) were associated with adult obesity, showing that the findings did not reflect reverse causality.

The association between fitness decrease and insulin resistance persisted after adjustment for both child BMI and waist circumference. Even after accounting for adult obesity, poor child fitness and fitness decrease were associated with a higher risk of insulin resistance, with adjusted ORs of 1.5 (95% CI 1.0–2.3; $P = 0.06$) and 1.6 (1.1–2.2; $P = 0.011$), respectively.

CONCLUSIONS — The findings indicate that a decline in fitness, and by inference a decline in physical activity from childhood to adulthood, is a stronger predictor of adult obesity and insulin resistance than low levels of fitness in childhood. This is supported by data from one study that focused on change in physical activity from childhood to early adulthood (3).

These findings indicate that programs that encourage physical activity earlier in life are also likely to be important. First, a fit child is more likely to be a fit adult partaking in more physical activity in adulthood (3). Child fitness is an important determinant of adult fitness at the population level. Further, after accounting for change in fitness over time, children with low fitness were more likely to have unfavorable obesity and insulin resistance outcomes. Importantly, the findings here indicate that programs that help fit individuals to maintain fitness and unfit children to improve fitness will be important.

Change in fitness is likely to be a good proxy for change in physical activity, as supported by the finding that physical activity measured by pedometer in adulthood was higher in the persistent-high-fitness or increasing-fitness groups. Alcohol consumption, smoking, and fat intake in adulthood were excluded as potential confounders of the observed asso-

ciations. Bias due to subject selection is also unlikely to explain the results. The childhood characteristics of those with child fitness who also had adult fitness measured were very similar to those who did not, with the exception of socioeconomic status. Lower-SES individuals in this cohort were more likely to become obese over time. The loss of these individuals from the decreasing-fitness group would have the effect of biasing the association toward the null; thus, the findings are likely conservative. The longitudinal fitness sample was similar to recent Australian surveys with regard to the proportion of subjects undertaking sufficient physical activity (male 42.4 vs. 44.1%; female 42.8 vs. 49%) (24) and the proportion of overweight or obese (male 55.8 vs. 57.7%; female 29.0 vs. 35.4%) (25). Furthermore, the results from the weighted analysis indicated that the findings were robust and that loss to follow-up was unlikely to have influenced the findings.

The effect of longitudinal fitness decrease on insulin resistance persisted after adjustment for child BMI, adult BMI, or adult obesity status. This indicates that the effect of a decline in fitness on insulin resistance is not solely due to an associated lower adult BMI. Possible direct effects via changes in muscle metabolism, for example, must be considered.

In conclusion, these data strongly suggest that a decline in cardiorespiratory fitness from childhood to adulthood, and by inference a decline in physical activity, is associated with a higher prevalence of obesity and insulin resistance in adulthood. Programs aimed at maintaining childhood physical activity levels into adulthood have potential for reducing the burden of obesity and type 2 diabetes in adults.

Acknowledgments— The study was supported by the National Health and Medical Research Council's Population Health Capacity Building Grant 436914 and by assistance from the Heart Foundation, the Tasmanian Community Fund, and Veolia Environmental Services.

Financial support was also provided by the Sanitarium Health Food Company, ASICS Oceania, and Target Australia. No other potential conflicts of interest relevant to this article were reported.

We gratefully acknowledge the contributions of the Childhood Determinants of Adult Health Study's project manager, Marita Dalton, all other project staff and volunteers, and the study participants. Dr. Leigh Blizzard provided valuable statistical advice. Louise Thwaites assisted in the manuscript's preparation.

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