PEDIATRICS®
OFFICIAL JOURNAL OF THE AMERICAN ACADEMY OF PEDIATRICS

# Physical Fitness and Physical Activity at Age 13 Years as Predictors of Cardiovascular Disease Risk Factors at Ages 15, 25, 33, and 40 Years: Extended Follow-up of the Oslo Youth Study

Elisabeth Kvaavik, Knut-Inge Klepp, Grethe S. Tell, Haakon E. Meyer and G. David Batty

*Pediatrics* 2009;123;e80-e86
DOI: 10.1542/peds.2008-1118

ARTICLE

# Physical Fitness and Physical Activity at Age 13 Years as Predictors of Cardiovascular Disease Risk Factors at Ages 15, 25, 33, and 40 Years: Extended Follow-up of the Oslo Youth Study

**Elisabeth Kvaavik, PhD[a,b], Knut-Inge Klepp, MPH, PhD[a], Grethe S. Tell, MPH, PhD[c], Haakon E. Meyer, MD, PhD[d,e], G. David Batty, PhD[b]**

[a]Department of Nutrition and [e]Section for Preventive Medicine and Epidemiology, Faculty of Medicine, University of Oslo, Oslo, Norway; [b]MRC Social and Public Health Sciences Unit, University of Glasgow, Glasgow, Scotland; [c]Department of Public Health and Primary Health Care, University of Bergen, Bergen, Norway; [d]Norwegian Institute of Public Health, Oslo, Norway

The authors have indicated they have no financial relationships relevant to this article to disclose.

**What's Known on This Subject**

The few published studies suggest that higher childhood physical fitness is associated with lower levels of selected CVD risk factors in adulthood; however, interpretation of these studies is hampered by methodologic shortcomings, in particular a modest follow-up period.

**What This Study Adds**

In a study with extended follow-up, higher physical fitness in childhood but not physical activity seemed to confer protection against adult obesity and raised blood pressure.

## ABSTRACT

OBJECTIVE. Few studies have examined the association of childhood physical activity and physical fitness with cardiovascular disease risk factors in adulthood. Furthermore, interpretation of these findings is hampered by methodologic shortcomings. In a population-based cohort study, we explored the influence, if any, of childhood physical activity and physical fitness on later cardiovascular disease risk factors.

METHODS. Data were taken from the Oslo Youth Study, a prospective cohort study that began in 1979, when 1016 students (mean age: 13 years; range: 11–15 years) who were attending 6 schools were invited to participate in a health education intervention. Cardiovascular disease risk factor data were collected at baseline and again in 1981 (mean age: 15 years; range: 13–17 years), 1991 (mean age: 25; range: 23–27 years), 1999 (mean age: 33; range: 31–35 years), and 2006 (mean age: 40; range: 38–42 years).

RESULTS. At baseline, physical fitness was inversely related to BMI, triceps skinfold thickness, and blood pressure (systolic and diastolic; $N = 716$). These associations were also present in prospective analyses at ages 15 ($N = 472$), 25 ($N = 280$; except for systolic blood pressure), and 33 years ($N = 410$, only BMI measured)—albeit with progressively diminishing magnitude—but were lost at 40 years ($N = 294$). There were fewer relationships with cardiovascular disease risk factors when physical activity was the exposure of interest. Controlling for educational attainment of both the parent and the study member had little impact on these associations.

CONCLUSIONS. Although childhood physical fitness seems to reveal some inverse associations with obesity and blood pressure in early adulthood, these effects diminished markedly into middle age. *Pediatrics* 2009;123:e80–e86

www.pediatrics.org/cgi/doi/10.1542/peds.2008-1118

doi:10.1542/peds.2008-1118

Drs Tell and Klepp designed and conducted the baseline and early follow-up surveys for the Oslo Youth Study; Drs Kvaavik and Batty generated the idea for the manuscript, which was developed by the co-authors, and wrote the first draft around analyses conducted by Dr Kvaavik. The remaining authors commented on subsequent drafts of the manuscript.

**Key Words**
cohort, CVD, fitness, longitudinal, physical activity, risk factors

**Abbreviations**
BP— blood pressure
CVD— cardiovascular disease
LTPA—leisure time physical activity
$Vo_2$—maximum oxygen uptake
HDL— high-density lipoprotein
$HbA_{1c}$— glycosylated hemoglobin

Accepted for publication Sep 16, 2008

Address correspondence to Elisabeth Kvaavik, PhD, Department of Nutrition, Faculty of Medicine, University of Oslo, PO Box 1046 Blindern, N-0316 Oslo, Norway. E-mail: ekvaavik@medisin.uio.no



A SERIES OF cohort studies conducted in the past 6 decades identified a range of risk factors, measured in middle- and older-aged individuals, for cardiovascular disease (CVD),[1,2] a leading cause of death and morbidity in industrialized countries[3] and a prominent public health problem in low- to middle-income societies.[4,5] These well-documented predictors include obesity and overweight[6]; cigarette smoking[7]; raised blood glucose,[8,9] blood pressure (BP),[10] and serum cholesterol[11]; psychosocial factors[12]; and lower levels of physical activity or physical fitness[13–17]; however, it is evident that these risk factors, when measured solely in midlife, do not fully explain variations in CVD.[18–20] This observation has prompted work into emerging risk factors (eg, genetic markers, inflammatory factors, psychosocial indices), more detailed measurement of existing ones (eg, dietary characteristics), and the potential involvement of early life exposures.

Several of these risk factors when measured in early life seem to be predictive of CVD mortality and morbidity several decades later.[21] Thus, when measured between childhood and young adulthood, obesity and overweight,[22,23]

cigarette smoking,[24,25] high BP,[26] raised serum cholesterol,[24] and socioeconomic adversity[27,28] are associated with later elevated CVD risk. To date, however, there is a marked paucity of studies that have examined the links between preadult physical activity or physical fitness and later CVD mortality and morbidity. In the absence of these clinical end-point data, investigators have instead examined the links between physical activity and/or physical fitness in youth and risk factors for CVD in adulthood. In the 3 studies that hold data on physical fitness/physical activity in childhood or adolescence and CVD risk factors in adulthood,[29–31] there is a suggestion that higher physical fitness but not physical activity is associated with lower levels of CVD risk factors[32]; however, interpretation of findings is hampered by methodologic shortcomings that include a small sample size[29,31]; the absence of data on socioeconomic position, which, given its correlation with both CVD risk factors[33,34] and physical fitness/physical activity,[35,36] is a candidate confounding factor; and a modest follow-up period.[30] The last is particularly important: given the suggestion of progressively weaker tracking coefficients for CVD risk factors in children who were followed serially into adult life,[37] it is likely that the influence of physical fitness and physical activity on CVD risk factors will likewise diminish over time.

In the Oslo Youth Study, the cross-sectional associations among physical fitness, physical activity, and CVD risk factors measured when the study participants were ~13 years of age have been previously reported.[38] Subsequent selective measurement of CVD risk factors took place at ages 15, 25, 33 (self-reports of weight and height only), and 40 years. Analyses of these data therefore facilitate a more detailed examination of the influence of physical fitness and leisure time physical activity (LTPA) on CVD risk indices across the life course into middle age than has previously been possible.

## METHODS

The Oslo Youth Study began in 1979, when 1016 primary- and secondary-school students (mean age: 13 years; range: 11–15 years) who were attending 6 schools in socioeconomically disparate neighborhoods of Oslo were invited to participate in a health education intervention. Begun in the autumn semester of 1979 and described in detail elsewhere,[39–41] the objective of the intervention was to discourage smoking initiation and improve physical activity and dietary habits. As part of the schools-based intervention, students completed a self-administered questionnaire and underwent a health examination in 1979 (aged ~13 years) and 1981 (aged 15). In adulthood, resurveys took place in 1991 (aged 25), 1999 (questionnaire only; aged 33), and 2006 (aged 40). The study was initially approved by the Oslo City Health Authorities; subsequent surveys were approved by the Norwegian Data Inspectorate and the Regional committee for medical research ethics.

### Assessment of Physical Fitness and Physical Activity

In 1979, maximum oxygen uptake ($\dot{V}O_2$max; milliliters of $O_2$ per kilogram of body weight per minute) was indirectly assessed by trained staff who recorded pulse rate during a submaximal cycle–based exercise test. This approach to measuring $\dot{V}O_2$max is predicated on the assumption that from initiation of the workload and heartbeat frequency measured in steady state (usually after 4–6 minutes of exertion), $\dot{V}O_2$max can be estimated by extrapolating to the assumed maximum heart rate.[42] During the test, students pedaled at a steady rate of 50 pedal revolutions per minute with the workload adjusted by a mechanical breaking mechanism. Standard workloads were 50, 100, 150, and 175 W from an assumed end pulse of 140 to 160 beats per minute. Given that this protocol was originally devised for an adult population with an estimated maximum heart rate of 195 beats per minute, the predicted $\dot{V}O_2$max of a child population that has a higher maximum heart rate will be an underestimation. As previously described,[38] a correction factor of 1.18 was therefore used to convert $\dot{V}O_2$ max values herein.

Self-administered questionnaires that contained an inquiry regarding LTPA were administered in 1991, 1999, and 2006: "How often do you exercise for at least half an hour to the extent that you sweat and/or are short of breath?" (seldom [ie, less than following category]; 2–3 times per month; once a week; 2–3 times per week; 4–6 times per week; every day). The same inquiry was made in 1979 and 1981, with the exception that the response "4 to 6 times per week" was omitted. In addition to examining the relation of a single measurement of physical activity at age 13 with later CVD risk factors, we explored the influence of an accumulative score. To do so, we summed physical activity scores for the years 1979, 1991, and 1999 (possible range: 3–17); where responses from 1979 and/or 1999 were missing, values from 1981 (2) and 2006 (14) were substituted (corresponding substitutions for BMI were 3 and 27, respectively).

### Assessment of CVD Risk Factors

In 1979 and 1981, data collection took place in the schools and was conducted by 2 trained research workers. Subsequently, questionnaires were mailed to the study participants, and respondents were invited to attend a health examination at a study center in Oslo (in 1991, conducted by 2 trained nurses) or with their personal physician (in 2006). These examinations included measurement of height, weight, triceps skinfold thickness (1979, 1981, and 1991 only; mean of 2 measures are used in these analyses), waist and hip circumferences (1991 and 2006), and, during the school years, pubertal development using the Tanner scale[43] (based on external genitalia for boys and breasts for girls[44]). BMI was computed on the basis of the usual formula (weight/[height]$^2$ kg/m$^2$).

Resting BP was ascertained using a random-0 sphygmomanometer in 1979, 1981 (for both of these visits, 2 measurements were taken by the same research worker by using a sphygmomanometer), and 1991 (3 readings taken by 2 trained nurses by using a Dinamap device). In 2006, 2 measurements were taken by the participant's general practitioner (type of instrument not recorded).

The mean of these values for each year was used herein. Venipuncture was made for measurements of serum total and high-density lipoprotein (HDL) cholesterol, triglycerides, and glycosylated hemoglobin ($HbA_{1c}$; 2006 only). Whether the study member had fasted was recorded for all years. Fasting and nonfasting values for triglyceride, cholesterol, and $HbA_{1c}$ were pooled because the analyses restricted to either group revealed essentially the same results. Both fasting and nonfasting values were used for total and HDL cholesterol and $HbA_{1c}$ analyses. The analyses of $HbA_{1c}$ in 2006 facilitated an assessment of the presence of the metabolic syndrome in a subgroup ($n$ = 118) on the basis of the following criteria among fasting participants: triglyceride level ≥1.70 mmol/L, $HbA_{1c}$ level >5.45%, systolic BP level ≥130 mm Hg or diastolic BP level ≥85 mm Hg, HDL cholesterol level <1.30 mmol/L for women and <1.04 mmol/L for men, and waist circumference ≥80 cm for women and ≥94 cm for men.

Educational attainment of the parent (range: elementary school (7 years) to college/university) was ascertained from a parental questionnaire administered in 1979/1981 and was used as our indicator of early-life socioeconomic position. When data on both mothers and fathers were available, those with the highest level of attained education were used in the analyses. The same inquiries were made of the study participants when they were aged 25 to 40 years.

### Statistical Analyses

Linear regression analysis was used to summarize the relation of physical fitness and LTPA in 1979 with subsequent measurement of BMI, waist circumference, total serum cholesterol, HDL cholesterol, triglycerides, systolic and diastolic BP, and $HbA_{1c}$. In preliminary analyses, there was no evidence of differential associations in men and women, so data were pooled and adjusted for gender. Given the narrow age range of the study participants, no adjustment was made for chronological age. Control for pubertal development, original study intervention group, and smoking status had no impact on the physical fitness/physical activity–CVD risk factor associations, so these adjustments are not shown here. We adjusted for socioeconomic position on the basis of parents' and study participants' educational attainment, the latter recorded at each survey. Furthermore, in the analyses involving LTPA as the exposure of interest, we controlled for physical fitness with the aim of examining whether the impact of activity on CVD risk factors was mediated via fitness. SPSS 14.0 (SPSS, Inc, Chicago, IL) was used in all analyses.

Of the 1016 students who were invited to take part in the study in 1979, 827 (81.4%) participated. Complete data on all variables included herein were available for 716 study participants (mean age: 13.0 years; range: 11.0–15.0), 472 in 1981 (mean age: 15.0 years; range: 13.0–17.0), 280 in 1991 (mean age: 25.1 year; range: 23.0–27.0), 410 in 1999 (mean age: 33.0 years; range: 31.0–35.0), and 294 (self-reported weight and height) and 199 (measured variables) in 2006 (mean age: 40.0 years; range: 38.0–42.0). Study participants who were pregnant or reported experiencing anorexia/bulimia nervosa were excluded from analyses in 1999 ($n$ = 16) and 2006 ($n$ = 3).

TABLE 1 **Baseline Characteristics (1979) According to Participation in a Later Follow-up Survey (1991): The Oslo Youth Study**

| Baseline Characteristics (1979) | Participant in 1991 ($N$ = 254) | Nonparticipant in 1991 ($N$ = 462) | $P$ for Difference |
|---|---|---|---|
| Female gender, % | 54.0 | 44.0 | .007 |
| Parental education, % high school graduate | 51.0 | 39.0 | .001 |
| Physical fitness, mL/kg per min | 49.4 | 49.8 | .716 |
| BMI, kg/m$^2$ | 18.4 | 18.5 | .703 |
| Triceps skinfold thickness, mm | 11.6 | 11.8 | .519 |
| Total cholesterol, mmol/L | 4.7 | 4.7 | .960 |
| HDL cholesterol, mmol/L | 1.3 | 1.3 | .666 |
| Triglyceride, mmol/L | 0.6 | 0.7 | .110 |
| Tanner stage, scale 1–5 | 2.6 | 2.4 | .076 |
| Systolic BP, mm Hg | 110.0 | 108.0 | .096 |
| Diastolic BP, mm Hg | 58.0 | 58.0 | .703 |
| LTPA, % twice weekly | 57.0 | 58.0 | .831 |

The presence of these missing data raises obvious concerns regarding selection bias; that is, the characteristics of people who participated in the study differed from those who did not. Table 1 presents a comparison of baseline (1979) data according to whether original study members took part in the 1991 survey (tables for the same comparisons according to later participation are available on request). The only significant differences were that responders in 1991 were more likely to be female and have parents with higher educational attainment ($P < .05$ for both). In 1999, the only difference between responders and nonresponders was seen for systolic BP at baseline (110 mm Hg vs 108 mm Hg; $P$ = .022). In 2006, the disparities were similar to those seen in 1991 with the addition that participants were marginally more physically mature in 1979 than nonparticipants (2.6 vs 2.4; $P$ = .037).

## RESULTS

Table 2 presents the associations between childhood physical fitness and later CVD risk factors. For the purpose of comparison, we also tabulate results from a previous report of the cross-sectional analyses when the study participants were aged 13 years.[38] The inverse relation between physical fitness and BMI present at age 13 was also apparent at 15, 25, and 33 years of age; however, these gradients progressively diminished in magnitude with increasing follow-up time such that the association was lost at 40 years of age. Although there was also evidence of an inverse relation between fitness and triceps skinfold thickness as measured at age 15, there was no apparent link with our third measure of adiposity, waist-to-hip ratio as measured at ages 25 and 40 years. At baseline, high fitness was associated with lower total cholesterol and triglycerides and higher HDL cholesterol. These relationships were lost at follow-up, except for total cholesterol, which became somewhat stronger and significant at 25 years of age. A negative

TABLE 2 **Physical Fitness at Age 13 Years in Relation to CVD Risk Factors in Later Life (Standardized Regression Coefficients): The Oslo Youth Study**

| Parameter | Age, y | *n* | Gender-Adjusted | *P* | SEP-Adjusted | *P* |
|---|---|---|---|---|---|---|
| BMI | 13 | 716 | −0.38 | <.001 | −0.36 | <.001 |
| | 15 | 472 | −0.27 | <.001 | −0.24 | <.001 |
| | 25 | 280 | −0.20 | .001 | −0.20 | .001 |
| | 33 | 410 | −0.17 | <.001 | −0.17 | <.001 |
| | 40 | 294 | −0.09 | .118 | −0.10 | .088 |
| Triceps skinfold thickness | 13 | 716 | −0.40 | <.001 | −0.40 | <.001 |
| | 15 | 472 | −0.20 | <.001 | −0.19 | <.001 |
| | 25 | 280 | −0.17 | .003 | −0.17 | .002 |
| Waist-to-hip ratio | 25 | 280 | −0.02 | .736 | −0.02 | .638 |
| | 40 | 199 | −0.03 | .628 | −0.04 | .506 |
| Total cholesterol | 13 | 716 | −0.09 | .030 | −0.10 | .010 |
| | 15 | 472 | −0.002 | .961 | −0.01 | .828 |
| | 25 | 280 | −0.11 | .059 | −0.14 | .016 |
| | 40 | 199 | −0.11 | .107 | −0.14 | .053 |
| HDL cholesterol | 13 | 716 | 0.10 | .014 | 0.09 | .025 |
| | 15 | 472 | 0.10 | .045 | 0.08 | .108 |
| | 25 | 280 | −0.06 | .290 | −0.04 | .470 |
| | 40 | 199 | −0.09 | .179 | −0.10 | .211 |
| Triglycerides | 13 | 716 | −0.16 | <.001 | −0.16 | <.001 |
| | 15 | 472 | −0.09 | .067 | −0.09 | .074 |
| | 25 | 280 | −0.04 | .474 | −0.06 | .266 |
| | 40 | 199 | 0.10 | .138 | 0.09 | .186 |
| Systolic BP | 13 | 716 | −0.19 | <.001 | −0.17 | <.001 |
| | 15 | 472 | −0.15 | .001 | −0.14 | .002 |
| | 25 | 280 | −0.09 | .081 | −0.09 | .078 |
| | 40 | 199 | 0.04 | .551 | 0.05 | .501 |
| Diastolic BP | 13 | 716 | −0.20 | <.001 | −0.20 | <.001 |
| | 15 | 472 | −0.14 | .002 | −0.13 | .005 |
| | 25 | 280 | −0.15 | .013 | −0.14 | .020 |
| | 40 | 199 | −0.02 | .734 | −0.01 | .876 |
| $HbA_{1c}$ | 40 | 199 | 0.00 | .999 | 0.005 | .943 |

SEP indicates socioeconomic position and comprises parental education when the study members were 13/15 years of age and participants' own education at 25 to 40 years of age.

fitness–BP (both components) gradient was seen in cross-sectional analyses at age 13 and also at ages 15 and 25 (diastolic BP only), although the effect was much attenuated. Fitness at age 13 was not related to $HbA_{1c}$ at age 40. Controlling for education—both parental (baseline) and participant's own (follow-up)—had essentially no impact on the association between physical fitness and CVD risk factors.

In Table 3, the associations between physical activity at age 13 and later CVD risk factors are depicted. There was no evidence of an association with any of the risk factor outcomes, the only exception being an inverse relation with triglyceride in cross-sectional analyses at baseline; however, this relationship was attenuated after control for markers of socioeconomic position and was not present at follow-up. When the relation of accumulated physical activity score with CVD risk factors at age 40 was evaluated (Table 4), there was a suggestion that participants who were more active across the life course had a lower BMI; however, this relation was no longer significant after statistical adjustment for fitness and socioeconomic status.

We also examined the associations of physical fitness and physical activity with later measurements of BMI, waist circumference, and waist-to-hip ratio by using standard categorizations advanced by the World Health Organization[45] and International Diabetes Federation.[46] The results were essentially the same as reported already for the continuous data. Both fitness and activity were unrelated to the metabolic syndrome (17 cases in 118 participants) in any of our analyses. Finally, we also ran the previously described analyses having restricted the analytical sample only to study members who participated in all waves of the study ($n = 104$), and the results were essentially unchanged (all results available on request).

## DISCUSSION

The aim of this study was to examine the relation of LTPA and physical fitness at 13 years of age with future CVD risk factors up to 27 years later. At baseline, physical fitness was inversely related to adiposity (BMI and triceps skinfold thickness) and BP (systolic and diastolic). These associations were present in prospective analyses at ages 15, 25, and 33 years—albeit with progressively diminishing magnitude—but were lost at 40 years. There were few, if any, associations when physical activity was the exposure of interest, perhaps because this inquiry was somewhat basic. That physical fitness demonstrates stronger predictive power than activity is consistent with the few other cohort studies in this area.[29–31]

Evidence from 2 spheres of physical activity epidemiology, when taken together, gave us reason to anticipate that both physical activity and physical fitness would reveal associations with later measurement of other CVD risk factors. First, CVD risk factors "track" between childhood and adulthood such that higher risk children tend to become higher risk adults.[47–49] Second, cross-sectional studies of children indicate that higher levels of physical fitness and physical activity have a favorable impact on CVD risk factors.[50] That the fitness/activity–CVD risk factor associations that are apparent in cross-sectional studies of children are lost in prospective studies may be partially ascribed to reverse causality. Thus, although it is biologically plausible that increased fitness and activity may be related to lower levels of CVD risk factors in cross-sectional studies, it is equally possible that selected risk factors, such as obesity, may make physical activity uncomfortable, thereby lowering fitness, and this may explain the seeming inverse relation.[14] Although it would of course have been valuable to examine the tracking of cardiorespiratory fitness across the life course, fitness was measured only twice in this study, and these 2 time points were very close together (1979 and 1981); that is, too short a period to investigate tracking of fitness in any meaningful way.

The strengths of this study include the measurement of both physical activity and physical fitness, repeat assessment of risk factors and covariate data across the life course, and its population-based sampling. It is not of course without its shortcomings. First, given that this is a longitudinal study, there was marked attrition at each attempt at following up the study members; however,

TABLE 3 **LTPA at Age 13 Years in Relation to CVD Risk Factors in Later Life (Standardized Regression Coefficients): The Oslo Youth Study**

| Parameter | Age, y | *n* | Gender-Adjusted | *P* | Fitness-Adjusted | *P* | SEP-Adjusted | *P* |
|---|---|---|---|---|---|---|---|---|
| BMI | 13 | 706 | −0.02 | .584 | 0.03 | .393 | 0.03 | .347 |
| | 15 | 463 | 0.04 | .439 | 0.06 | .180 | 0.07 | .105 |
| | 25 | 276 | 0.02 | .778 | 0.04 | .476 | 0.05 | .417 |
| | 33 | 403 | −0.05 | .341 | −0.02 | .669 | −0.01 | .801 |
| | 40 | 289 | −0.09 | .107 | −0.07 | .207 | −0.07 | .231 |
| Triceps skinfold thickness | 13 | 706 | −0.06 | .106 | −0.003 | .921 | 0.001 | .987 |
| | 15 | 463 | 0.01 | .797 | 0.03 | .716 | 0.03 | .455 |
| | 25 | 276 | −0.03 | .560 | −0.004 | .939 | −0.003 | .959 |
| Waist-to-hip ratio | 25 | 276 | 0.06 | .160 | 0.07 | .124 | 0.07 | .095 |
| | 40 | 195 | −0.07 | .237 | −0.06 | .269 | −0.06 | .288 |
| Total cholesterol | 13 | 706 | −0.04 | .272 | −0.03 | .459 | −0.03 | .481 |
| | 15 | 463 | −0.02 | .720 | −0.02 | .594 | −0.02 | .600 |
| | 25 | 276 | 0.04 | .540 | 0.07 | .275 | 0.08 | .197 |
| | 40 | 195 | −0.03 | .677 | −0.02 | .741 | −0.01 | .857 |
| HDL cholesterol | 13 | 706 | 0.07 | .079 | 0.05 | .185 | 0.05 | .184 |
| | 15 | 463 | −0.02 | .683 | −0.03 | .510 | −0.03 | .525 |
| | 25 | 276 | 0.04 | .413 | 0.05 | .373 | 0.04 | .484 |
| | 40 | 195 | 0.06 | .407 | 0.06 | .365 | 0.06 | .384 |
| Triglyceride | 13 | 706 | −0.08 | .034 | −0.05 | .148 | −0.05 | .160 |
| | 15 | 463 | −0.02 | .621 | −0.02 | .633 | −0.03 | .575 |
| | 25 | 276 | 0.09 | .189 | 0.09 | .122 | 0.10 | .081 |
| | 40 | 195 | 0.003 | .964 | −0.003 | .963 | 0.01 | .926 |
| Systolic BP | 13 | 706 | −0.02 | .542 | 0.002 | .953 | 0.003 | .937 |
| | 15 | 463 | −0.06 | .233 | −0.04 | .386 | −0.03 | .488 |
| | 25 | 276 | −0.03 | .533 | −0.02 | .656 | −0.02 | .676 |
| | 40 | 195 | −0.05 | .494 | −0.05 | .463 | −0.04 | .525 |
| Diastolic BP | 13 | 706 | −0.06 | .110 | −0.04 | .326 | −0.03 | .366 |
| | 15 | 463 | 0.04 | .425 | 0.05 | .259 | 0.05 | .240 |
| | 25 | 276 | −0.08 | .158 | −0.07 | .236 | −0.07 | .220 |
| | 40 | 195 | −0.05 | .446 | −0.05 | .466 | −0.05 | .494 |
| $HbA_{1c}$ | 40 | 195 | 0.01 | .938 | 0.01 | .939 | 0.01 | .948 |

SEP indicates socioeconomic position and comprises parental education when the study members were 13/15 years of age and participants' own education at 25 to 40 years of age.

analyses of the characteristics of people who were lost to follow-up versus those who were retained revealed few differences. Differences that were present—for gender and parents' educational level—although not unimportant, are consistent with reports from large British birth cohort studies.[51,52] It is notable, however, that even with this attrition, the Oslo study is larger than others with a comparably long duration of CVD risk factor surveillance, such as the Amsterdam Growth and Health Longitudinal Study.[29] Second, given the range of CVD risk factors measured on a number of occasions, we necessarily conducted a large number of statistical tests. It is therefore plausible that some of the few positive results have arisen by chance.

TABLE 4 **Accumulated LTPA in Relation to CVD Risk Factors at Age 40 Years (Standardized Regression Coefficients): The Oslo Youth Study**

| Parameter | *n* | Gender-Adjusted | *P* | Fitness-Adjusted | *P* | SEP-Adjusted | *P* |
|---|---|---|---|---|---|---|---|
| BMI | 248[a] | −0.12 | .044 | −0.10 | .100 | −0.09 | .150 |
| Waist-to-hip ratio | 164[b] | −0.08 | .215 | −0.07 | .260 | −0.05 | .379 |
| Total cholesterol | 164 | −0.11 | .172 | −0.10 | .174 | −0.07 | .332 |
| HDL cholesterol | 164 | −0.001 | .993 | −0.004 | .953 | −0.02 | .832 |
| Triglyceride | 164 | 0.04 | .579 | 0.05 | .538 | 0.10 | .376 |
| Systolic BP | 164 | −0.01 | .869 | −0.01 | .888 | 0.01 | .849 |
| Diastolic BP | 164 | −0.01 | .942 | 0.001 | .987 | 0.01 | .901 |
| $HbA_{1c}$ | 164 | −0.01 | .942 | −0.004 | .962 | −0.01 | .883 |

SEP indicates socioeconomic position and comprises parental education when the study members were 13/15 years of age and participants' own education at 25 to 40 years of age.

[a] Analyses are based on a subsample of participants who had data on LTPA for 1979 or 1981, for 1991, and for 1999 or 2006 and also self-reported weight/height in 2006, parental education in 1979/1981, own education in 2006, and fitness data for 1979.

[b] Analyses are based on a subsample of participants who had data on LTPA for 1979 or 1981, for 1991, and for 1999 or 2006 and also health examination data in 2006, parental education in 1979/1981, own education in 2006, and fitness data for 1979.

## CONCLUSIONS

On the basis of the results of this study, there is a suggestion that higher physical fitness may confer protection against later obesity and raised BP, although these effects diminished over time. Additional work to examine the long-term stability of fitness from childhood to adult life and its association with adult CVD risk factors is warranted.


## ACKNOWLEDGMENTS

The Oslo Youth Study was initially supported by the Norwegian Cancer Society. Subsequent support includes grants from the Norwegian Research Council, the EXTRA funds from the Norwegian Foundation for Health and Rehabilitation, and from the Norwegian Health Association. Dr Kvaavik is currently funded by a grant from the Norwegian Research Council; Dr Batty is a UK Wellcome Trust Fellow (WBS U.1300.00.006.00012.01).


## REFERENCES


1. Yusuf S, Reddy S, Ounpuu S, Anand S. Global burden of cardiovascular diseases: part I—general considerations, the epidemiologic transition, risk factors, and impact of urbanization. *Circulation.* 2001;104(22):2746–2753
2. Labarthe DR. *Epidemiology and Prevention of Cardiovascular Diseases*. Gaithersburg, MD: Aspen Publishers, Inc, 1998
3. *Mortality Statistics: Cause, 1995*. London, England: The Stationery Office; 1997
4. Murray CJ, Lopez AD. *The Global Burden of Disease*. Boston, MA: WHO, Harvard School of Public Health, World Bank, 1996
5. *The World Health Report: Making a Difference*. Geneva, Switzerland: World Health Organization; 1999
6. Batty GD, Shipley MJ, Jarrett RJ, Breeze E, Marmot MG, Davey Smith G. Obesity and overweight in relation to disease-specific mortality in men with and without existing coronary heart disease in London: the original Whitehall study. *Heart.* 2006; 92(7):886–892
7. Parish S, Collins R, Peto R, *et al*. Cigarette smoking, tar yields, and non-fatal myocardial infarction: 14,000 cases and 32,000 controls in the United Kingdom. The International Studies of Infarct Survival (ISIS) Collaborators. *BMJ.* 1995;311(7003): 471–477
8. Fuller JH, Shipley MJ, Rose G, Jarrett RJ, Keen H. Coronary-heart-disease risk and impaired glucose tolerance: the Whitehall study. *Lancet.* 1980;1(8183):1373–1376
9. Batty GD, Kivimäki M, Davey Smith G, Marmot M, Shipley MJ. Post-challenge blood glucose concentration and stroke mortality rates in non-diabetic men in London: 38-year follow-up of the original Whitehall prospective cohort study. *Diabetologia.* 2008;51(7):1123–1126
10. Lewington S, Clarke R, Qizilbash N, Peto R, Collins R. Age-specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies. *Lancet.* 2002;360(9349):1903–1913
11. Lewington S, Whitlock G, Clarke R, *et al*. Blood cholesterol and vascular mortality by age, sex, and blood pressure: a meta-analysis of individual data from 61 prospective studies with 55,000 vascular deaths. *Lancet.* 2007;370(9602):1829–1839
12. Smith GD, Ben Shlomo Y, Beswick A, Yarnell J, Lightman S, Elwood P. Cortisol, testosterone, and coronary heart disease: prospective evidence from the Caerphilly study. *Circulation.* 2005;112(3):332–340
13. Batty GD, Lee IM. Physical activity and coronary heart disease. *BMJ.* 2004;328(7448):1089–1090
14. Batty GD. Physical activity and coronary heart disease in older adults: a systematic review of epidemiological studies. *Eur J Public Health.* 2002;12(3):171–176
15. Batty GD, Lee IM. Physical activity for preventing strokes. *BMJ.* 2002;325(7360):350–351
16. Lee CD, Folsom AR, Blair SN. Physical activity and stroke risk: a meta-analysis. *Stroke.* 2003;34(10):2475–2481
17. Blair SN. Physical activity, fitness, and coronary heart disease. In: Bouchard C, Shephard R, Stephens T, eds. *Physical Activity, Fitness, and Health*. Champaign, IL: Human Kinetics; 1994: 579–590
18. Marmot MG, Shipley MJ, Rose G. Inequalities in death: specific explanations of a general pattern? *Lancet.* 1984;1(8384): 1003–1006
19. Yarnell JW. The PRIME study: classical risk factors do not explain the severalfold differences in risk of coronary heart disease between France and Northern Ireland. Prospective Epidemiological Study of Myocardial Infarction. *QJM.* 1998; 91(10):667–676
20. Batty GD, Der G, MacIntyre S, Deary IJ. Does IQ explain socioeconomic inequalities in health? Evidence from a population based cohort study in the west of Scotland. *BMJ.* 2006; 332(7541):580–584
21. McCarron P, Davey Smith G. Physiological measurements in children and young people, and risk of coronary heart disease in adults. In: Gates A, ed. *A Lifecourse Approach to Coronary Heart Disease Prevention Scientific and Policy Review*. London, England: The Stationery Office; 2003:49–78
22. Baker JL, Olsen LW, Sorensen TI. Childhood body-mass index and the risk of coronary heart disease in adulthood. *N Engl J Med.* 2007;357(23):2329–2337
23. Lawlor DA, Martin RM, Gunnell D, *et al*. Association of body mass index measured in childhood, adolescence, and young adulthood with risk of ischemic heart disease and stroke: findings from 3 historical cohort studies. *Am J Clin Nutr.* 2006; 83(4):767–773
24. Dyer AR, Stamler J, Shekelle RB. Serum cholesterol and mortality from coronary heart disease in young, middle-aged, and older men and women from three Chicago epidemiologic studies. *Ann Epidemiol.* 1992;2(1–2):51–57
25. McCarron P, Davey Smith G, Okasha M, McEwen J. Smoking in adolescence and young adulthood and mortality in later life: prospective observational study. *J Epidemiol Community Health.* 2001;55(5):334–335
26. McCarron P, Davey Smith G, Okasha M, McEwen J. Blood pressure in young adulthood and mortality from cardiovascular disease. *Lancet.* 2000;355(9213):1430–1431
27. Galobardes B, Lynch JW, Davey Smith G. Childhood socioeconomic circumstances and cause-specific mortality in adulthood: systematic review and interpretation. *Epidemiol Rev.* 2004;26:7–21
28. Galobardes B, Smith GD, Lynch JW. Systematic review of the influence of childhood socioeconomic circumstances on risk for cardiovascular disease in adulthood. *Ann Epidemiol.* 2006; 16(2):91–104
29. Twisk JW, Kemper HC, Van Mechelen W. The relationship between physical fitness and physical activity during adolescence and cardiovascular disease risk factors at adult age: the Amsterdam Growth and Health Longitudinal Study. *Int J Sports Med.* 2002;23(suppl 1):S8–S14
30. Boreham C, Twisk J, Neville C, Savage M, Murray L, Gallagher A. Associations between physical fitness and activity patterns during adolescence and cardiovascular risk factors in young adulthood: the Northern Ireland Young Hearts Project. *Int J Sports Med.* 2002;23(suppl 1):S22–S26
31. Lefevre J, Philippaerts R, Delvaux K, *et al*. Relation between cardiovascular risk factors at adult age, and physical activity

during youth and adulthood: the Leuven Longitudinal Study on Lifestyle, Fitness and Health. *Int J Sports Med.* 2002;23(suppl 1):S32–S38
32. Twisk JW, Kemper HC, Van Mechelen W. Prediction of cardiovascular disease risk factors later in life by physical activity and physical fitness in youth: general comments and conclusions. *Int J Sports Med.* 2002;23(suppl 1):S44–S49
33. Kivimäki M, Lawlor DA, Smith GD, *et al*. Early socioeconomic position and blood pressure in childhood and adulthood: the Cardiovascular Risk in Young Finns Study. *Hypertension.* 2006; 47(1):39–44
34. Blane D, Hart CL, Davey Smith G, Gillis CR, Hole DJ, Hawthorne VM. Association of cardiovascular disease risk factors with socioeconomic position during childhood and during adulthood. *BMJ.* 1996;313(7070):1434–1438
35. Van Lenthe FJ, Boreham CA, Twisk JW, Strain JJ, Savage JM, Smith GD. Socio-economic position and coronary heart disease risk factors in youth: findings from the Young Hearts Project in Northern Ireland. *Eur J Public Health.* 2001;11(1):43–50
36. Freitas D, Maia J, Beunen G, *et al*. Socio-economic status, growth, physical activity and fitness: the Madeira Growth Study. *Ann Hum Biol.* 2007;34(1):107–122
37. Beckett LA, Rosner B, Roche AF, Guo S. Serial changes in blood pressure from adolescence into adulthood. *Am J Epidemiol.* 1992;135(10):1166–1177
38. Tell GS. Vellar OD. Physical fitness, physical activity, and cardiovascular disease risk factors in adolescents: the Oslo Youth Study. *Prev Med.* 1988;17(1):12–24
39. Tell GS, Vellar OD. Noncommunicable disease risk factor intervention in Norwegian adolescents: the Oslo Youth Study. In: Hetzel B, Berebson GS, eds. *Cardiovascular Risk Factors in Childhood: Epidemiology and Prevention*. Amsterdam, Netherlands: Elsevier Science Publishers BV; 1987:203–217
40. Tell GS, Klepp KI, Vellar OD, McAlister A. Preventing the onset of cigarette smoking in Norwegian adolescents: the Oslo youth study. *Prev Med.* 1984;13(3):256–275
41. Klepp KI, Tell GS, Vellar OD. Ten-year follow-up of the Oslo Youth Study Smoking Prevention Program. *Prev Med.* 1993; 22(4):453–462
42. Astrand PO, Rodahl K. *Textbook of Work Physiology*. New York, NY: McGraw-Hill; 1986
43. Tanner JM. *Growth at Adolescence*. Oxford, England: Blackwell Scientific Publications; 1962
44. Tell GS. Cardiovascular disease risk factors related to sexual maturation: the Oslo Youth Study. *J Chronic Dis.* 1985;38(8): 633–642
45. World Health Organization. *Physical Status: The Use and Interpretation of Anthropometry—Report of a WHO Expert Committee. Who Technical Report Series*. Geneva, Switzerland: WHO; 1995
46. *The IDF Consensus Worldwide Definition of the Metabolic Syndrome*. Brussels, Belgium: International Diabetes Federation; 2006
47. Kelder SH, Osganian SK, Feldman HA, *et al*. Tracking of physical and physiological risk variables among ethnic subgroups from third to eighth grade: the Child and Adolescent Trial for Cardiovascular Health cohort study. *Prev Med.* 2002;34(3): 324–333
48. Andersen LB. Tracking of risk factors for coronary heart disease from adolescence to young adulthood with special emphasis on physical activity and fitness. *Dan Med Bull.* 1996;43(5): 407–418
49. Webber LS, Srinivasan SR, Wattigney WA, Berenson GS. Tracking of serum lipids and lipoproteins from childhood to adulthood: the Bogalusa Heart Study. *Am J Epidemiol.* 1991; 133(9):884–899
50. Ortega FB, Ruiz JR, Castillo MJ, Sjöström M. Physical fitness in childhood and adolescence: a powerful marker of health. *Int J Obes (Lond).* 2008;32(1):1–11
51. Atherton K, Fuller E, Shepherd P, Strachan DP, Power C. Loss and representativeness in a biomedical survey at age 45 years: 1958 British birth cohort. *J Epidemiol Community Health.* 2008; 62(3):216–223
52. Wadsworth ME, Butterworth SL, Hardy RJ, *et al*. The life course prospective design: an example of benefits and problems associated with study longevity. *Soc Sci Med.* 2003;57(11): 2193–2205

# Physical Fitness and Physical Activity at Age 13 Years as Predictors of Cardiovascular Disease Risk Factors at Ages 15, 25, 33, and 40 Years: Extended Follow-up of the Oslo Youth Study

Elisabeth Kvaavik, Knut-Inge Klepp, Grethe S. Tell, Haakon E. Meyer and G. David Batty

*Pediatrics* 2009;123;e80-e86
DOI: 10.1542/peds.2008-1118