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## The relationship between physical fitness and clustered risk, and tracking of clustered risk from adolescence to young adulthood: eight years follow-up in the Danish Youth and Sport Study

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### Abstract

**Introduction:** Cardiovascular disease (CVD) is usually caused by high levels of many risk factors simultaneously over many years. Therefore, it is of great interest to study if subjects stay within rank order over time in both the biological risk factors and the behaviour that influences these risk factors. Many studies have described stability (tracking) in single risk factors, especially in children where hard endpoints are lacking, but few have analysed tracking in clustered risk.

**Methods:** Two examinations were conducted 8 years apart. The first time, 133 males and 172 females were 16–19 years of age. Eight years later, 98 males and 137 females participated. They were each time ranked into quartiles by sex in four CVD risk factors all related to the metabolic syndrome. Risk factors were the ratio between total cholesterol and HDL, triglyceride, systolic BP and body fat. The upper quartile was defined as being at risk, and if a subject had two or more risk factors, he/she was defined as a case (15–20 % of the subjects). Odds ratios (OR) for being a case was calculated between quartiles of fitness in both cross-sectional studies. The stability of combined risk was calculated as the OR between cases and non-cases at the first examination to be a case at the second examination.

**Results:** ORs for having two or more risk factors between quartiles of fitness were 3.1, 3.8 and 4.9 for quartiles two to four, respectively. At the second examination, OR were 0.7, 3.5 and 4.9, respectively. The probability for "a case" at the first examination to be "a case" at the second was 6.0.

**Conclusions:** The relationship between an exposure like physical fitness and CVD risk factors is much stronger when clustering of risk factors are analysed compared to the relationship to single risk factors. The stability over time in multiple risk factors analysed together is strong. This relationship should be seen in the light of moderate or weak tracking of single risk factors, and is strong evidence for early intervention in children where risk factors cluster.

## Introduction

Hard endpoints of atherosclerotic CVD are not prevalent in young people, but as atherosclerosis develops gradually over the years, it may be effective to prevent high levels of CVD risk factors already present in children. The rationale behind this thinking is that risk factors track, i.e. subjects keep rank order within a risk factor. Most CVD risk factors show moderate tracking [1,2]. A tracking coefficient is a measure of variability between two time points. Variation in a risk factor value is caused by assessment error, short time fluctuations, and more permanent changes in mean risk factor levels. Only the latter is interesting in the prediction of atherosclerotic development.

Many of the risk factors show great variability caused by assessment problems and day-to-day variation, i.e. blood pressure and cholesterol show great short-term fluctuations and physical activity is usually self reported with great assessment problems [3,4]. A more correct tracking coefficient can be obtained by minimising some of the short-term variation, which can be removed by repeated measurements, i.e. two to three blood samples over a week both at baseline and at the follow-up. However, this is rarely done in epidemiological studies. Also, assessment error can often be diminished, but it may be expensive in large longitudinal studies. Therefore, it may be more convenient to analyse the stability of clustered risk.

Many of the risk factors are related to a common causal factor, the metabolic syndrome [5]. In an insulin-insensitive person, the risk factors related to the metabolic syndrome will be elevated simultaneously, and this may, to some extent, overrule the error variation. We have shown earlier in cross-sectional studies that physical fitness is weakly related to each single CVD risk factor [6], but probably due to a strong effect of training on insulin sensitivity, a strong relationship is found between fitness and clustered risk [7]. The aims of the present study were to assess the relationship between physical fitness and clustered risk in two cross-sectional studies and to calculate tracking of clustered risk in an eight-year longitudinal study from adolescence to young adulthood.

## Methods

In 1983, 1298 subjects participated in a school-based study. Thirty six schools, 18 vocational and 18 high schools, were selected to be representative of the whole country, and all pupils in the selected classes were invited to participate. No subjects refused to participate. Among these, every fourth pupil on the class lists were invited to participate in the present study. Only 2.3% refused to participate. In the first cross-sectional study in 1983, 133 boys and 172 girls, at that time 16–19 years of age, took part. In 1991, 98 men (73.7%) and 137 women (79.7%)

from the original sample, now 23–27 years of age, participated in a second examination.

Variables include demographic and health habit variables, blood pressure, blood lipids, fatness and physical fitness. Body height was measured to the nearest cm, and body weight to within 100 g. Skinfolts were measured with a Harpenden skinfold calliper above the patella on both knee's, and 1/3 of the distance from umbilicus to spina iliaca anterior superior on both sides of the abdomen. The sums of these four skinfolts were used to calculate body fat% using the following equation:

Body fat (kg) =  $0,184 \times SF_{abd} + 0,145 \times SF_{knee} - 0,66$ , where  $SF_{abd}$  is the sum of right and left abdominal skinfolts and  $SF_{knee}$  is the sum of the skinfolts above the left and right knee [8]. Arterial BP was measured with a mercury sphygmomanometer after 20 min of supine rest followed by another 5 min in the sitting position. A fasting venous blood sample was drawn from a superficial vein in the arm. The plasma was enzymatically analyzed for total cholesterol, HDL-cholesterol (HDL-C) and triglycerides (Gilford 3500 Auto Analyzer).

Self-reported leisure physical activity was obtained as type and quantity of organized sport activities in clubs and at the schools, of unorganized sports activities, and as other physical activities including physical labor, walking, cycling, and dance. A physical activity index was constructed from the reported intensity and duration of the activities. Maximal oxygen uptake was directly measured during a maximal progressive cycle test. In the study from 1984, expired air was collected in Douglas bags and analysed with an Electrochemistry (S-3a) analyser, and in the study from 1991, in an open MedGraphics system. Detailed description of all methods has been published previously [9,10].

## Statistics

All data were analysed using SPSS (version 11). Participants in each cross-sectional examination were ranked into quartiles by sex in four CVD risk factors all related to the metabolic syndrome. Risk factors were the ratio between total cholesterol and HDL, triglyceride, systolic BP and body fat. The upper quartile was defined as being at risk. Clustering of risk was defined in those subjects having two or more risk factors (~15% of the subjects). Logistic regression was used to calculate the odds ratio (OR) between quartiles of fitness for being a case at the first examination, and the same analysis was performed at the second examination. Logistic regression was also used to calculate tracking of clustered risk factors. The OR of being a "case" at the follow up for those who were a "case" at the first examination compared to those who were a "non-case" at baseline.

**Table 1: Pearson correlation coefficients between values in single CVD risk factors assessed in 1983 and 1991.**

	Men			Women		
	n	r	P<	n	r	P<
Body fat (%)	80	0.72	**	97	0.46	**
BMI (kg m <sup>-2</sup> )	86	0.80	**	114	0.77	**
VO <sub>2max</sub> (l min <sup>-1</sup> )	78	0.51	**	90	0.72	**
VO <sub>2max</sub> (ml min <sup>-1</sup> kg <sup>-1</sup> )	78	0.35	**	90	0.48	**
Systolic BP (mm)	80	0.49	**	97	0.54	**
Diastolic BP (mm)	80	0.44	**	97	0.38	**
Cholesterol (mmol l <sup>-1</sup> )	75	0.39	**	88	0.51	**
HDL-C (mmol l <sup>-1</sup> )	75	0.29	*	89	0.49	**
Triglyceride (mmol l <sup>-1</sup> )	59	0.32	*	81	0.33	*
Physical activity (h w <sup>-1</sup> )	88	0.31	**	114	0.20	NS

\* P < 0.01; \*\* P < 0.001; NS non-significant

## Results

The cross-sectional relationship between physical fitness and single risk factors was weak in 1983 and in 1991, and the relationship between physical activity and risk factors was not significant. In both genders, fitness related to cholesterol, HDL-C/C, triglyceride and waist/hip girth in 1991. Relationships were strongest in men with r<sup>2</sup>-values between 0.10 and 0.18. In women, r<sup>2</sup>-values of 0.06 to 0.10 were found in 1991. In both genders, the closest relation to fitness was found in cholesterol. Physical activity did not relate to any of the risk factors in either sex. A similar picture was found in 1983.

Tracking coefficients in single risk factors were between 0.20 and 0.72 (table 1). Physical activity and triglyceride were in the low end and variables related to anthropometry, such as BMI and absolute VO<sub>2max</sub>, in the high end.

Self-reported physical activity was not associated with clustered risk in the first or second study. At the first examination, ORs between quartiles of fitness for having 2 or more risk factors were 3.1 (95% CI: 1.0–9.3), 3.8 (95% CI: 1.3–11.2) and 4.9 (95% CI: 1.7–14.2) for quartiles two to four, respectively. At the first examination 19.3% were defined as cases. At the second examination, ORs were 0.7 (95% CI: 0.1–4.3), 3.5 (95% CI: 0.9–14.5), and 4.9 (95% CI: 1.2–19.7), respectively, and the number of subjects defined as cases were 15.2%. Further, the fitness level at the first examination was used as a predictor for being a "case" at the second examination, both with and without adjustment for fitness level at the second examination. No significant relationships were found.

The probability for "a case" at the first examination to be "a case" at the second was 6.0 (95% CI: 2.1–16.9) compared to non-cases. The number of risk factors an individ-

ual had at baseline was related to the number at the second examination with an r-value of 0.42 (P < 0.001).

## Discussion

The main conclusions of the present study were: 1) Even if there were weak relationships between CVD risk factors in each cross-sectional study, there was a strong relationship between clustered risk and physical fitness, and 2) Single CVD risk factors only tracked modestly, and 3) Subjects with clustered risk factors were at great risk of having clustered risk eight years later.

Many studies have analysed tracking in single risk factors [11-13]. Most of these studies have found moderate and comparable tracking coefficients in the specific CVD risk factors. The size of the coefficients reflects both true variation in the true mean, error variation due to measurement error and day-to-day fluctuations. It is possible to diminish the error variation, but it is usually very expensive in epidemiological studies. Many studies have solved these problems with fitness variables, and studies using direct measurement of VO<sub>2max</sub> have only little error variation in this variable [2,14]. Other variables are assessed with substantial error variation, both in biological CVD risk factors, and especially in physical activity measures. Assessment of physical activity could be improved by using objective measurement, i.e. accelerometer. Some of the error variation would then be eliminated, but we cannot say if that would result in a significant relationship between physical activity and clustered risk.

We therefore decided to analyse clustered risk. Subjects who were insulin-insensitive were supposed to reflect this in high levels of many risk factors simultaneously, and clustered risk might therefore show greater stability. Further, CVD usually results from high levels of many risk factors for many years, not high levels of a single risk fac-

tor. This makes it plausible to analyse for clustered risk and tracking of clustered risk. Significant changes have been found in CHD risk factors in school-based interventions. Detection of subjects at risk is, therefore, important [15].

Aerobic training is known to increase insulin sensitivity [16], and we found a strong relationship between physical fitness and clustered risk in both cross-sectional studies. Also, clustered risk tracked strongly from adolescence to young adulthood. Subjects at risk may be screened more effectively by analysing clustered risk instead of looking at single risk factors. A suggestion for screening is to use a surrogate measure for insulin sensitivity, such as fasting insulin or an oral glucose tolerance test.

We did not find that adolescent fitness level predicted adult clustered risk. A reason for this lack of association could be the relatively limited number of cases.

## Conclusions

In conclusion, tracking of CVD risk and the relationship between physical fitness and CVD risk factors become stronger when clustered risk is analysed.

## Competing interests

None declared.

## Authors' contributions

LBA collected all data, carried out the analyses, and made the first draft. HH discussed the idea, and participated in data analysis. VG assisted in drafting the paper and developing the analysis. SEH assisted with the statistical analysis and discussion of the concept. KF assisted in drafting the paper and developing the analysis. All authors read and approved the final manuscript.

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