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# Adolescent predictors of life course trajectories of body fatness

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Nienke J Wijnstok, Jos WR Twisk, Erik H Serne, Etto C Eringa, Yvo M. Smulders.

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## Abstract

*Introduction:* It is unknown which adolescent risk factors predict an unfavourable developmental pattern of body fat over a prolonged period of time.

*Methods:* Latent class growth analyses was used to examine the presence of distinct 30-year developmental patterns of body fat using total fat (S4SF) and fat distribution (SFratio) measured by skinfold-thickness, separate for boys and girls. Logistic regression analyses were used to determine adolescent lifestyle predictors for the relatively unfavourable developmental patterns. Also, predictive effects of biological parameters (i.e. blood pressure, lipid profile) in adolescence were examined.

*Results:* For SFratio, high caloric intake and (OR=0.35; p=0.01) and physical activity (OR= 0.20; p<0.01) are protective, while alcohol use is a predictor of a relatively unfavourable developmental pattern (OR=1.79; p=0.05). For S4SF, smoking appears protective (OR=0.44; p=0.10) for a relatively unfavourable developmental pattern. Adolescent blood pressure and lipid profile had no added value over lifestyle factors in predicting life courses of total fat or fat distribution.

*Conclusion:* In adolescents, low physical activity and use of alcohol are predictors of a relatively unfavourable 30-year developmental pattern of central fat distribution. Total fatness is not clearly associated with adolescent life style factors, apart from a weak apparent protective effect of smoking.

**Key words:** adolescence, life-course, lifestyle, obesity, predictors

## Introduction

The epidemic of overweight and obesity is a worldwide problem that is still growing. Overweight and obesity, in particular with a more central distribution pattern, are risk factors for early morbidity and mortality from a wide range of diseases including cardiovascular disease (CVD), chronic obstructive pulmonary disease (COPD), muscle and joint disorders and some types of cancer<sup>194–196</sup>.

An important aspect of overweight and obesity is its pattern of development during the course of life. Identification of critical periods for future weight gain may be relevant for prevention and intervention. Previous studies suggest adolescence as one of these critical periods<sup>197–201</sup>. Adolescence is regarded by many as the episode in life during which lasting changes in anthropometry occur, and there is indeed evidence to support this<sup>197–201</sup>. Studies addressing adolescence, however, are often limited by selected populations and/or limited follow-up into adulthood<sup>198,199,201–205</sup>.

Apart from solid longitudinal data on developmental patterns in body fat, there is also a disturbing lack of insight into adolescent predictors of such patterns. Such predictors may be causal or relate to subtle characteristics of the adult obese phenotype. Several parameters are suggested to predict high body fat and a central pattern of body fat in later life<sup>206</sup>. These parameters include lifestyle parameters such as high caloric intake, low physical activity, low physical fitness, smoking and alcohol use or biological parameters such as blood pressure and lipid profile<sup>196,197,200,204,205,207–210</sup>. Again, however, limited availability of complete longitudinal data represents a problem in adequately pinpointing adolescent predictors for unfavourable developmental patterns of body fat throughout life<sup>211</sup>.

The aim of the current study was to identify adolescent predictors of long-term developmental patterns in body fat. We analyzed the predictive value of lifestyle for relatively unfavourable developmental patterns of body fat in apparently healthy boys and girls. Furthermore, we assessed whether biological factors during adolescence, lipid levels and blood pressure, predict unfavourable developmental patterns of body fat independent from life-style factors.

## Methods

### Study Population

The studied population is part of the Amsterdam Growth and Health Longitudinal Study (AGHLS), which started in 1976. The main aim of the study was to describe normal growth and health, but extended to etiological questions from adolescence to adulthood. Measurements were performed at 10 time points during the past 30 years, from 13 to 42 years of age. More specific information on the AGHLS can be found elsewhere<sup>26</sup>.

In the study, adolescents from 2 schools were examined. In the first 4 years, a specific study design was followed to test the requirement for -and effects of- repeated measures: in one school, adolescents were studied annually, and in the other school, a random sample of 25% of included adolescents was tested each year. For those with repeated measures of potential body fat trajectory predictors in adolescence, the mean value of the first 4 years was used. The medical ethics committee of the VU University Medical Center (Amsterdam, Netherlands) approved the study, and all participants (provided by subjects' parents during 13-16 years of age) gave written informed consent.

### **Anthropometrics**

For defining the developmental patterns of body fat, both the total sum of four skinfolds (S4SF) and a skinfold thickness ratio (SFratio) were used. Skinfold thickness was measured at the biceps, triceps, subscapular and supra-iliac site, to the nearest 0.1 mm with a skinfold calliper (Holtain London, UK) according to guidelines from the International Biological Program<sup>23</sup>. SFratio was defined as (subscapula + supra-iliac) / (biceps + triceps).

### **Lifestyle predictors in the adolescent period**

Lifestyle related variables included caloric intake, alcohol use, smoking, physical activity and physical fitness. Habitual food intake was measured by a cross-check dietary history face-to-face interview<sup>48</sup>. From this interview, food characteristics were calculated with the Dutch food and nutrition table. Total energy intake (in kCal) and the total amount of consumed alcohol (expressed in yes/no) were assessed. Smoking (expressed as yes/no) was determined by a separate questionnaire. Physical activity information was obtained using a structured interview covering the 3 months before the interview, and was expressed in METs/week<sup>209</sup>. The interview covered subjects of both the duration and intensity of daily physical activities during work, school, sports, transportation, and leisure time. Physical fitness was measured using maximum oxygen uptake (VO<sub>2</sub>-max) during a running test on a treadmill and was expressed in ml · min<sup>-1</sup> · kg<sup>-2/3</sup><sup>209</sup>.

### **Biological parameters in the adolescent period**

Biological parameters were systolic blood pressure (SBP), diastolic blood pressure (DBP), total cholesterol, and HDL cholesterol. Both SBP and DBP were measured manually. A standard pressure cuff was placed around the left upper arm. With a sphygmomanometer both SBP and DBP were measured twice, the lowest values were recorded. Mean arterial pressure (MAP) was calculated as (2\*DBP + SBP)/3. Non-fasting venous blood samples were taken from the antecubital vein. Total and HDL cholesterol levels were determined using standard in-home assays. Cholesterol ratio was calculated as total cholesterol/

HDL cholesterol. Finally, retrospective information on birth weight was obtained in the year 2000.

### **Statistical analyses**

Descriptive information is presented as mean (SD), median (IQR), or percentage (%) as appropriate.

The statistical approach to the data consisted of 3 steps: 1] identification of developmental patterns 2] predicting such developmental patterns with lifestyle variables, and 3] assessing if biological variables improved this prediction.

Growth patterns were defined using Latent Class Growth Analyses. This part of the analyses is described in detail elsewhere<sup>212</sup>. Briefly, latent class growth analysis is a method defining distinct classes over time based on variance at each of the time points. Each of the defined groups is based on within-group homogeneity and between-group heterogeneity. These analyses were performed separately for boys and girls.

In the second step, logistic regression analyses were used to detect lifestyle-related adolescent predictors of unfavourable growth into adulthood. These analyses were performed for both S4SF and SFratio, using a backward procedure which excluded lifestyle predictors until only parameters with  $p \leq 0.10$  remained.

In the final step, the potential improvement of the prediction model by adding biological risk factors was analysed.

LCGA were performed in Mplus version 5.21 (Mplus, Los Angeles, USA) and logistic regression analyses in PASW Statistics version 18.0 (PASW Statistics Inc., Chicago, USA).

## **Results**

### **Identification of developmental patterns**

In [Figure 6](#), the developmental patterns of S4SF and SFratio over time are presented. Descriptive information on lifestyle and biological parameters for these groups is presented in [table 8](#). As shown in the adult characteristics, these subjects are relatively healthy.

### **Crude analyses of the predictors**

[Table 9](#) shows the crude logistic regression analyses for each of the lifestyle and biological parameters. Separate analyses for boys and girls yielded similar results, and the gender groups were thus combined to increase statistical power. Since none of the continuous relations met linearity assumptions, crude analyses are presented in quartiles with the lowest quartile as reference group. In the logistic regression analyses, the favourable developmental patterns are used as reference group.

**Table 8.** Descriptive information of the Study population (AGHLS).

	<b>Sum of 4 skinfolds (S4SF)</b>		<b>Skinfolds ratio (SFratio)</b>	
	favourable (N=192)	Unfavourable (N=67)	favourable (N=161)	Unfavourable (N=98)
<b>Lifestyle variables</b>				
Gender (% boys)	46%	42%	47%	41%
Smoking (%yes)	15.6%	7.5%	13.7%	13.3%
Alcohol use (%yes)	37.5%	38.8%	36.3%	39.8%
Caloric intake (Kcal / day)	1122 (409)	1049 (449)	1181 (427)	937 (376)
VO2max (ml · min <sup>-1</sup> ·kg <sup>-2/3</sup> )	10.1 (1.23)	10.1 (1.5)	10.2(1.3)	9.8 (1.3)
Physical activity (mean of reported MET's per day)	17421 (3200)	17357 (3432)	18159 (3365)	16170 (2653)
<b>Biological risk factors</b>				
Systolic blood pressure (mmHg)	124 (8)	126 (10)	126 (8)	122 (9)
Total cholesterol (mmol/L)	4.39 (0.75)	4.44 (0.76)	4.35 (0.73)	4.50 (0.76)
HDL cholesterol (mmol/L)	1.39 (0.27)	1.38 (0.28)	1.37 (0.26)	1.42 (0.29)
Birthweight (grams)	3383 (714)	3467 (634)	3428 (700)	3371 (683)
<b>Adult characteristics</b>				
BMI (kg/m <sup>2</sup> )	23.7 (2.7)	24.7 (3.3)	24.5 (3.9)	24.3 (3.7)
Systolic blood pressure (mmHg)	114 (16)	116 (14)	116 (14)	115 (15)
HDL cholesterol	1.68 (0.41)	1.86 (0.45)	1.73 (0.42)	1.72 (0.44)

Data is presented as mean (SD) or frequencies when appropriate.

### **Lifestyle predictors of developmental patterns**

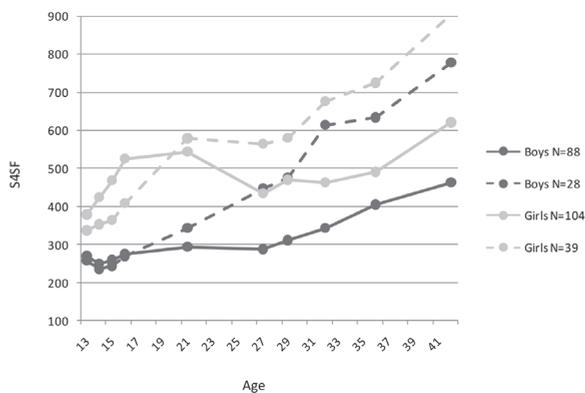
For both S4SF (table 10a) and SFratio (table 10b), the logistic regression models are presented. For the total fat parameter S4SF, only smoking came close to predicting a favourable developmental pattern, although the OR of 0.44 did not reach conventional limits of statistical significance ( $p=0.10$ ). All other lifestyle variables failed to predict the unfavourable developmental pattern even close to statistical significance. For an unfavourable developmental pattern of the central fat parameter SFratio, the final model includes caloric intake (favourable), physical activity (favourable) and alcohol use (unfavourable). Figure 7 illustrates the risk of an unfavourable developmental pattern of SFratio across categories of adolescent alcohol use and physical activity, adjusted for caloric intake.

### **Biological risk factors do not improve prediction**

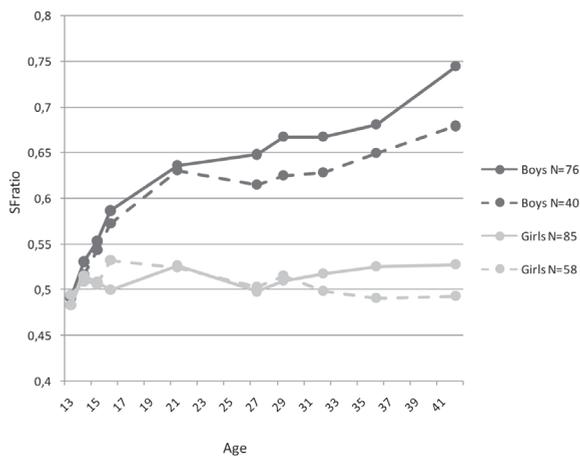
Adding blood pressure and cholesterol to the final model (table 10b) did not improve models for both S4SF and SFratio. Analyses with alternatives to the currently presented variables in the model, such as mean arterial pressure instead of systolic blood pressure and cholesterol-ratio instead of HDL-cholesterol, showed essentially identical results (data not shown).

**Figure 6.** Developmental patterns of the a. S4Sf and b. SFratio<sup>212</sup>

**A.** Sum of four skinfold thickness (in mm)



**B.** Skinfolths thickness ratio (subscapula and suprailiac/ biceps and triceps)



The dark-grey lines represent the boys and the light-grey lines represent the girls. Solid lines represent the normal developmental patterns and the dashed lines have an unfavourable developmental pattern.

**Table 9.** Crude univariate logistic regression analyses with S4SF and SFratio.

Predictor variable		S4SF		SFratio	
		OR	P-value	OR	P-value
Smoking (yes vs no)		0.44 (0.16– 1.17)	0.10	0.97 (0.46– 2.02)	0.93
Alcohol use (yes vs no)		1.06 (0.60 – 1.87)	0.85	1.14 (0.68 – 1.92)	0.61
Caloric intake	Quartile 2	0.70	0.35	0.81	0.53
	Quartile 3	0.58	0.17	0.44	0.02*
	Quartile 4	0.58	0.17	0.25	< 0.01*
Cardiopulmonary fitness	Quartile 2	0.79	0.56	1.05	0.90
	Quartile 3	0.66	0.31	0.81	0.55
	Quartile 4	1.20	0.63	0.50	0.06*
Physical activity	Quartile 2	0.75	0.48	0.78	0.48
	Quartile 3	0.88	0.74	0.51	0.06*
	Quartile 4	1.06	0.90	0.17	< 0.01*
Systolic blood pressure	Quartile 2	0.38	0.03*	0.85	0.67
	Quartile 3	0.78	0.51	0.58	0.12
	Quartile 4	1.07	0.86	0.37	0.02*
HDL Cholesterol	Quartile 2	0.92	0.84	0.60	0.19
	Quartile 3	1.17	0.68	1.27	0.49
	Quartile 4	0.78	0.52	1.12	0.75
Birth weight	Quartile 2	1.05	0.91	1.05	0.88
	Quartile 3	1.87	0.10	0.92	0.82
	Quartile 4	1.81	0.13	0.99	0.97

**Table 10.** Final models for prediction of normal versus unfavourable developmental pattern of S4SF and SFratio.

**Model A: S4SF**

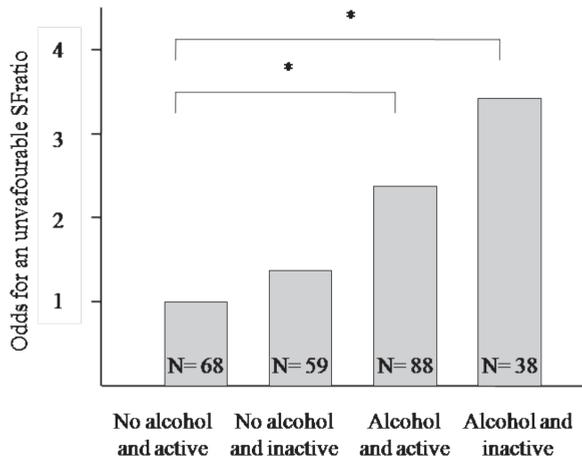
Predictor variable	OR	Pvalue
Smoking (yes vs no)	0.44	0.10

**Model B: SFratio**

Predictor variable	OR	Pvalue
Alcohol	<b>1.79</b>	<b>0.05</b>
Caloric intake	Group 2	0.89
	Group 3	<b>0.48</b>
	Group 4	<b>0.35</b>
Activity questionnaire	Group 2	0.83
	Group 3	0.60
	Group 4	<b>0.20</b>

**Bold figures** are characterised by a p-value of <0.10

**Figure 7.** Odds ratio for an unfavourable developmental pattern of central fatness across categories of adolescent alcohol use and physical activity.



Between group differences are marked with an \* when  $p < 0.01$ .

Analysis is corrected for caloric intake.

## Discussion

The salient finding of our study is that an unfavourable developmental pattern with respect to central fat distribution (SFratio) is predicted by alcohol use, physical activity, and caloric intake. Remarkably, however, high caloric intake appeared favourable, rather than unfavourable. An unfavourable developmental pattern of total fat (S4SF) is not clearly predicted from a collection of adolescent lifestyle factors commonly suspected to predispose to future overweight and obesity. Only smoking was weakly associated with having a lower odds of an unfavourable developmental pattern. Finally, blood pressure and lipid profile, both markers of the adult obese phenotype, have no predictive value for long-term developmental patterns of body fatness.

Although this study is not the first to explore possible predictors of life courses of body fat, it is, to the best of our knowledge, the first study to present adolescent predictors of 30-year developmental patterns of body fat. Latent class growth modelling is a relatively new technique that is used to identify these developmental patterns from longitudinal data<sup>213</sup>. This method identifies groups solely on variance, in contrast to a classical approach using pre-defined cut-off points of body fat<sup>213,214</sup>. Such developmental patterns over a specified period do not necessarily identify the same individuals who would classify as 'favourable' or 'unfavourable' in a cross-sectional analysis at the end of a follow-up period.

The currently used life course developmental patterns of body fat were computed using both S4SF and SFratio. Measures of waist circumference were not included in all of the measurement rounds, and therefore could not be used to compute developmental patterns. For BMI, measures were performed at each of the measure round, but distinct BMI trajectories could not be identified. This may have to do with the fact that BMI depends on length changes, which poses particular problems to longitudinal data obtained during periods with wide variations in length growth<sup>215</sup>. Also, studies have indicated that in non-obese subjects, skinfolds are a better estimate of actual body fat than BMI, which is also dependent on lean, muscle and bone mass<sup>216-218</sup>. The current population based study in largely non-obese subjects therefore uses skinfold measurements to identify life course developmental patterns of body fat and body fat distribution.

In our study, the only life-style factor that was close to predicting a favourable developmental pattern for total body fat was smoking ( $p=0.10$ ). It is needless to say that smoking should never be encouraged, certainly not in adolescents, but this study is not the first to suggest an inverse relation between smoking and body fat<sup>218,219</sup>. This association may be explained by nicotine induced effects, such as on appetite regulation and metabolic rate<sup>219</sup>.

The lack of predictive effects of other lifestyle factors with respect to developmental patterns of total body fat is noteworthy. Comparative data from similar studies are not available, and our study may be underpowered to reveal such effects, particularly if they are relatively subtle. The take-away message however does seem to be that there is no strong or straightforward association between commonly implicated lifestyle factors in adolescence, and unfavourable life course developmental patterns of total body fat.

Alcohol use and physical inactivity were identified as predictors of an unfavourable developmental pattern of central body fat. Alcohol use is well-known as a strong correlate of central body fat in both adolescents as well as young adults<sup>220,221</sup>, which is equally true for physical activity<sup>210</sup>. We have now shown that, in adolescents, these associations project into the future of body fat distribution.

As for caloric intake, our data suggest a favourable effect of caloric intake in the highest quartile, whereas the opposite might have been expected. This may be explained by residual confounding, for example by physical activity, which was conceivably not fully captured by the physical activity questionnaire. In addition, average adolescent caloric intake in the current sample was 1500 Kcal, whereas average caloric intake nowadays is 1800 kcal for boys and 2200 Kcal for girls<sup>222</sup>. Hence, the highest quartile of current adolescent caloric intake is much higher than that of our baseline sample, and intake congruent with the current upper quartile may well predispose to future (central) body fatness.

Theoretically, classical biological variables have the advantage of being more stable in tracking into adulthood<sup>41</sup>. The literature is, however, not unequivocal in that blood pressure or lipid levels predict (central) body fat, and our study suggests that they do not appear to enhance the prediction of developmental patterns of (central) body fat over and above lifestyle factors.

In conclusion, in healthy adolescents, predictors of 30-year developmental patterns are different for measures of total fat compared to measures of central fat distribution. Relatively high caloric intake does not appear to be a key adverse factor. Physical activity and use of alcohol, however, are. More life course research, particularly in populations at higher risk, is needed.

### **Conflicts of interest Statement**

The study is financially supported by The Netherlands Heart Foundation grant number (2007- B178). No other conflict of interest was declared.

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