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Life Course Origins of the Metabolic Syndrome in Middle-Aged Women and Men: The Role of Socioeconomic Status and Metabolic Risk Factors in Adolescence and Early Adulthood

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PURPOSE: To assess whether body mass index (BMI), blood pressure, and socioeconomic status in adolescence and early adulthood are independently related to the metabolic syndrome in adult women and men.
METHODS: We based our work on a Swedish prospective cohort study that recruited participants at 16 years of age (N = 1083 at age 16; 403 women and 429 men at age 43, 78% of those still alive [N = 1071]). Blood pressure (BP) and BMI were assessed when participants were 16 and 21 years of age. At age 43, the metabolic syndrome was defined according to the International Diabetes Federation guidelines. Socioeconomic status (SES) was operationalized by the participant (age 21 and 43) or parent's (age 16) occupational status. Information on smoking, snuff, alcohol, and inactivity was collected at age 43.
RESULTS: In women, SES at age 16 was independently related to the risk of metabolic syndrome. In women and men, BMI at age 16 was related to metabolic syndrome but was attenuated by BMI at age 21, which was significant in the final model; in women systolic BP displayed similar patterns.
CONCLUSIONS: Our data seem to suggest two independent life course pathways for metabolic syndrome: one metabolic pathway for both women and men operating through BMI (for women also systolic BP) in adolescence and early adulthood, and for women, an apparently independent pathway through adolescent socioeconomic disadvantage.
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KEY WORDS: Adolescence, Adult, Blood Pressure, Body Mass Index, Cohort Studies, Metabolic Syndrome X, Prospective Studies, Socioeconomic Status.

INTRODUCTION

The metabolic syndrome refers to the clustering of abdominal obesity, hypertension, disturbed glucose metabolism, and dyslipidemia, with adult prevalence estimates across the world varying between 7% and 43% in women and 8% and 24% in men (1). Childhood obesity has been suggested as a major etiological factor for the metabolic syndrome (2), supported by prospective studies demonstrating that metabolic risk factors in childhood and early adulthood, particularly body mass index (BMI) but also blood pressure and serum lipids, relate to the risk for the metabolic syndrome in adulthood (3, 4).

Not considered in these studies are the social inequalities in obesity (5) and metabolic syndrome (6, 7), particularly noticeable among women (7–11). Socioeconomic risks for the metabolic syndrome might also be partly determined by disadvantage earlier in life. This has been demonstrated in a prospective cohort study (12) in which parental class during childhood related to the metabolic syndrome in adulthood in women only, independently of adult class. Other investigators have reported inconsistent contributions of childhood socioeconomic status to adult metabolic syndrome risk (13–16) but have either comprised male-only samples (14), small samples with high attrition rate (13), and/or have used retrospective assessment of childhood socioeconomic conditions (14–16), an approach that notably might bias the studied association toward the null (17).

Thus, the metabolic syndrome seems to partly stem from hazardous metabolic profiles established during childhood but also possibly from early disadvantaged socioeconomic circumstances. The observation of childhood/adolescent origins of the metabolic syndrome implies that preventive efforts should be directed at young people (18). If any influences of socioeconomic conditions early in life are mediated by early metabolic risks, targeting these metabolic risks would be a sound preventive strategy. However, if early socioeconomic conditions do not operate through early metabolic risk factors, other strategies need to be used.

To our knowledge, no study has aimed at disentangling the contributions of adolescent socioeconomic and metabolic

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Selected Abbreviations and Acronyms

BMI = Body mass index
SES = socioeconomic status
HDL-C = high-density lipoprotein cholesterol
SBP = systolic blood pressure
DBP = diastolic blood pressure
OR = odds ratio
CI = confidence Interval

conditions to the subsequent risk of metabolic syndrome. This issue will be addressed by the present prospective cohort study, which aims (i) to assess whether socioeconomic status, BMI, and blood pressure in adolescence and early adulthood are related to the metabolic syndrome in mid-adulthood in women and men, and (ii) to investigate whether the contribution of the socioeconomic and metabolic predictors are independent from each other and from adult socioeconomic conditions and health behaviors.

METHODS

Study Population and Procedures

The participants were drawn from a 27-year prospective cohort study, the Northern Swedish Cohort, described in more detail elsewhere (19). The cohort comprised all adolescents who entered or should have entered the 9th (last) grade of compulsory school in the town of Luleå in 1981 (N = 1083; 506 girls and 577 boys), at the age of 16. The cohort was found to be representative of Sweden in various demographic comparisons (20). Follow-up surveys were conducted in 1983, 1986, 1995, and 2008, at participant age 18, 21, 30, and 43 years, respectively. At each survey, participants completed a comprehensive questionnaire about demographic and social circumstances, health-related symptoms, medication, health behaviors, and leisure activities. When participants were 16, 21, and 43 years of age, health examinations were performed (see Measures). All participants gave their informed consent, and ethical approval was obtained from the Regional Ethical Review Board in Umeå.

The present report is determined from data from the surveys administered when subjects were 16, 21, and 43years of age (N = 1003, 482 women and 521 men, 94% of those still alive at age 43; N = 1071) with the effective sample of main analyses comprising participants who had complete data available (N = 832, 403 women and 429 men, 78% 832/1071).

BMI and Blood Pressure at Age 16 and 21

When participants were 16 and 21 years of age, blood pressure was measured manually in the participant's right arm by trained medical personnel with a standard sphygmomanometer. Blood pressure was measured in the supine position after at least 10 minutes of resting. The mean of two measurements were used. In connection to the age 16 survey, all participants' weight (kg) and height (cm) were retrieved from school health records, as measured by the school nurses. At age 21, weight and height were self-reported by questionnaire. BMI (kg/m^2) was calculated for age 16 and 21 years.

Socioeconomic Status (SES) at Age 16, 21, and 43

Information on own (age 21 and 43) and parental (age 16) occupation was collected by questionnaire. Participants' own occupations at ages 21 and 43 were coded according to the socioeconomic classification system of Statistics Sweden (21) and were categorized according to a previously defined scheme (22). Manual workers were categorized as low SES (=1) whereas nonmanual employees and self-employed were categorized as high SES (=0). For participants who were not current working and for whom information on previous occupation was not available (only at age 21), e.g. for participants who were unemployed, studying, or performing military service, the highest educational attainment was used as a proxy (n = 206): university-preparatory high school or university studies indicated high SES (=0) whereas other types of high school education or lower was classified as low SES (=1). At age 16, parents' occupation was coded into three social groups. The classification of both parents in social group 3, corresponding to manual workers, was defined as low SES (=1), whereas at least one parent in social group 1 or 2 was defined as high SES (=0).

Metabolic Syndrome at Age 43

In 2008, a health examination after overnight fasting was performed by trained medical personnel at the participants' respective health care center. Blood pressure (mmHg) and waist circumference (cm) were measured according to the World Health Organization MONICA (Multinational MONItoring of trends and determinants in CArdiovascular disease) manual (23). Patients' blood pressures were measured with standard sphygmomanometer after resting with no change of position for at least 5 minutes, in a sitting position and with the right arm used. The mean of two consecutive measures was used. Waist circumference was measured with indoor clothing to the nearest 0.5 cm. Blood samples were collected and assessed for glucose, high-density lipoprotein cholesterol (HDL-C) and triglycerides according to the laboratory routines at the Department of Clinical Chemistry, Umeå University Hospital. Total precision (CV% at high/low concentration) was 1.5/1.2% for glucose, 2.8/2.8% for HDL-C, and 1.7/1.5% for triglycerides. External quality control is managed by EQUALIS (External QUality Assurance in Laboratory medicine In Sweden).

The definition of the International Diabetes Federation (24) was used to operationalize metabolic syndrome according to the following criteria: (i) waist circumference ⩾80 cm for women and ⩾94cm for men; and (ii) two or more of the following four criteria: (a) increased triglycerides (⩾1.7mmol/L) or specific treatment for that lipid abnormality; (b) reduced HDL-C (<1.29 mmol/L for women/<1.03 mmol/L for men) or specific treatment for that lipid abnormality, (c) increased blood pressure (systolic blood pressure [SBP] ⩾130 mmHg or diastolic blood pressure [DBP] ⩾85 mmHg) or treatment of hypertension, (d) increased fasting glucose (⩾5.6 mmol/L) or diagnosed type 2 diabetes. Sixty participants were treated for hypertension whereas no participants were treated specifically for either hypertriglyceridemia or low HDL-C. Current type 2 diabetes (n = 18) was based on self-reported presence of diabetes at age 43, excluding those who also reported diabetes at age 30 (n = 4; all of whom also reported diabetes at age 21) which were regarded as having type 1 diabetes.

Covariates at Age 43

Current health behaviors at age 43 were obtained by questionnaire self-reports of daily smoking (no/yes), daily snuff use (no/yes), and physical inactivity (seldom or never engaging in physical activity during the last 12 months/engaging each month or more often). All were coded as low risk = 0 and high risk = 1. Alcohol consumption was measured as daily consumption of pure alcohol derived from questions about typical frequency and quantity of beverage consumption. To take the beneficial effects of moderate drinking into account (25), alcohol consumption was coded into abstainers (0), low (1), and high (2). Because of the low estimated alcohol consumption, a low/high cut-off of 10 g/day for women and 15 g/day for men was used, yielding approximately 80% in the low drinking group. The main results were similar irrespective of coding scheme (e.g., dichotomization, using established cut-off limits, or more detailed categories).

Statistical Analysis

The total sample comprised 1003 participants (482 women and 521 men) with data available on at least one measure of interest. Because of drop-out on one or more measures (see Table 1) N varies somewhat between analyses and is lowest in analyses requiring the presence of all variables (N = 832, 403 women and 429 men), constituting the effective N for the main analyses. To examine potential selection bias, those with incomplete metabolic measures at age 43 were compared with those with complete measures. Women with incomplete data had greater systolic blood pressure at age 16 (M(s) = 122 [11.9] vs 117 [11.8], p = .005), whereas men with incomplete data had greater BMI at age 21 (M(s) = 23.7 [2.97] vs 22.9 [2.60], p = .036). High alcohol consumption was more prevalent in those with incomplete data, in both women (20% vs 8%, p = .008) and men (27% vs 11%, p < .001). There were no differences with respect to the other early metabolic risk factors, health behaviors or SES at either age (all p > .05).

All analyses were performed on women and men separately. The main analyses comprised a series of logistic regression analyses with metabolic syndrome on the independent variables, presented as odds ratios with 95% confidence interval. First, the crude odds ratios were calculated for each independent variable (Model 0). Second, a hierarchical logistic regression model with metabolic syndrome on exposures of interest was utilized to assess the independent associations. In Model 1, predictors at age 16 (SES, BMI, SBP, and DBP) were entered into the model, in Model 2 the corresponding predictors at age 21 (SES, BMI, SBP, and DBP) were added, and in Model 3 covariates at age 43 (SES and health behaviors) were added. To examine gender differences, predictors significant for either gender were followed by adding (centered) gender cross-product interaction terms.

RESULTS

The overall prevalence of the metabolic syndrome was estimated at 26.7%. Metabolic syndrome was substantially more prevalent in men (33.6%) than in women (19.1%), which may be related to disparities in triglycerides, blood pressure, and glucose levels above the cut-points (see Table 1). Men displayed greater BMI at age 21 and greater levels of systolic blood pressure at age 16 and 21, whereas women and men displayed similar BMI at age 16 as well as diastolic blood pressure at age 16 and 21.

BMI was highly stable between age 16 and 21 (r = .51 for women and r = .71 for men) whereas systolic (r = .33 for both women and men) and diastolic (r = .28 for women and r = .22 for men) blood pressure displayed weaker stability (see Table 2). Similarly, in men parental SES was rather strongly related to own socioeconomic attainment at age 21 (r = .32) and 43 (r = .33). In women, parental SES was less predictive of own SES at age 21 (r = .17) and 43 (r = .14), although comparable stability was observed for SES between age 21 and 43 in women (r = .31) and men (r = .42; see Table 2). Parental SES at age 16 was weakly related to concurrent BMI in both women (r = .10) and men (r = .09) but not to blood pressure, whereas SES at age 21 was not significantly related to either BMI or blood pressure (see Table 2).

In bivariate logistic regression analyses in women (Table 3, Model 0), low parental or low current SES was related to metabolic syndrome , as were BMI and SBP at both age 16 and 21. Mutual adjustment for predictors at age 16 (Model 1)

TABLE 1. Descriptive statistics of key variables and differences between women and men in the total sample (N = 1003)

	Women		Men		
Variable	Estimate*	n	Estimate*	n	*p* Value of difference
Metabolic syndrome	80 (19.1)	418	152 (33.6)	452	<.001†
Metabolic syndrome components					
High waist circumference	256 (60.8)	421	272 (59.3)	459	.640†
High triglycerides	49 (11.6)	423	168 (36.9)	465	<.001†
Low HDL cholesterol	125 (29.6)	422	136 (30.0)	453	.897†
High blood pressure	142 (31.7)	448	258 (53.4)	483	<.001†
High fasting glucose	43 (10.1)	425	115 (24.8)	464	<.001†
Body mass index (BMI, kg/m^2)					
BMI at age 16, M(s)	20.0 (2.67)	478	19.9 (2.77)	520	.272‡
BMI at age 21 , M(s)	21.3 (2.96)	476	23.0 (2.66)	518	<.001‡
Blood pressure (BP, mmHg)					
Systolic BP at age 16, M(s)	118 (11.9)	478	125 (13.6)	520	<.001‡
Systolic BP at age 21, M(s)	123 (12.3)	478	131 (12.7)	518	<.001‡
Diastolic BP at age 16, M(s)	68.7 (12.1)	478	68.5 (12.9)	520	.784‡
Diastolic BP at age 21, M(s)	72.5 (11.3)	478	72.8 (10.6)	518	.633‡
Socioeconomic status (SES)					
Low SES at age 16	174 (36.6)	476	204 (39.5)	517	.346†
Low SES at age 21,	273 (57.8)	472	352 (68.0)	518	.001†
Low SES at age 43	152 (31.5)	482	197 (38.0)	519	.033†
Health behaviors at age 43					
Daily smoking	113 (23.7)	477	90 (17.7)	508	.021†
Daily snuff use	62 (13.0)	478	156 (31.1)	502	<.001†
Alcohol consumption		477		510	.105†
Abstainers	38 (8.0)		32 (6.3)		
Low§	395 (82.8)		411 (80.6)		
High§	44 (9.2)		67 (13.1)		
Physical inactivity	79 (16.5)	480	127 (24.8)	513	.001†

*Estimates are n (%) of total number of participants (N) unless stated otherwise.
†χ^2 test.
‡*t* test.
§Low alcohol consumption = 0.1–9.9g/day for women and 0.1–14.9g/day for men; high alcohol consumption= ⩾10 g/day for women and ⩾15 g/day for men.

attenuated the contribution of SBP below significance. Both BMI and SBP at age 16 were further attenuated below significance by the addition of predictors at age 21 (Model 2), whereas the corresponding measures at age 21 remained significant after adding current health behaviors and SES (Model 3). Throughout the models, the substantial contribution of SES at age 16 remained largely unchanged.

Corresponding analyses in men (see Table 4) showed similar results with respect to BMI at age 16 and 21, but neither systolic blood pressure nor SES at age 16 reached significance. In the final model, only BMI at age 21 and physical inactivity contributed independently. These numerical gender discrepancies were confirmed for SES at age 16 (p interaction = .047 in model 1, p = .019 in model 2 and p = .003 in model 3), SBP at age 21 (p = .058 in model 2 and p = .044 in model 3), and physical inactivity (p = .041).

In complementary analyses the binary metabolic syndrome components (see Table 1) were regressed on BMI, SBP, and DBP at age 16 and 21 (data not shown). In brief, in both women and men high waist circumference (final Nagelkerke R^2=.15 in women and .17 in men) and high BP (R^2=.19 in women and .18 in men) were predicted by earlier BMI (p < .001) and SBP (p < .001-.005), respectively. BMI (age 16 in women; p = .035 and age 21 in men; p = .034) also contributed to adult high BP independently from SBP at age 16 and 21. The metabolic risk factors (particularly BMI at age 16) contributed to a lesser degree to adult triglycerides (R^2 = .02), HDL (R^2 = .04), and fasting glucose (R^2 = .04) in women, and to fasting glucose in men (R^2 = .05).

DISCUSSION

To the authors' knowledge, these results are the first reported from a prospective study simultaneously examining life course associations between metabolic risk factors and socioeconomic status in adolescence and early adulthood and the subsequent risk for metabolic syndrome in adulthood. The four main findings are that (i) parental socioeconomic status in adolescence was related to the risk of metabolic syndrome in women but not in men, (ii) BMI in adolescence was related to metabolic syndrome in both

TABLE 2. Zero-order correlations (Pearson's r) between exposures at age 16, 21, or 43 years for women (below diagonal, shaded) and men (above diagonal)

Variable (age)	BMI (16)	BMI (21)	SBP (16)	SBP (21)	DBP (16)	DBP (21)	Low SES (16)	Low SES (21)	Low SES (43)	Smoking (43)	Snuff (43)	Alcohol (43)	Inactivity (43)
BMI (16)	–	.72*	.19*	.03	−.01	.09†	.09†	.01	.09	.00	.00	−.01	.01
BMI (21)	.51*	–	.09	.08	.05	.01	.08	−.01	.04	−.04	.01	−.05	.01
SBP (16)	.16‡	.14‡	–	.33*	.19*	.09	.01	.07	.04	−.05	−.02	−.06	.00
SBP (21)	.08	.03	.33*	–	.12†	.25*	.12‡	.07	.08	.03	.09	.00	.02
DBP (16)	.09	.06	.27*	.09	–	.22*	.00	.02	.06	−.06	−.01	−.02	−.02
DBP (21)	.11†	.05	.14‡	.44*	.28*	–	.02	.05	.03	−.02	.00	−.03	.05
Low SES (16)	.10†	.05	.06	.05	−.02	.06	–	.32*	.33*	.03	.14‡	−.09	.13‡
Low SES (21)	.02	.07	.04	.01	−.02	−.05	.17*	–	.42*	.11†	.11†	−.07	.12†
Low SES (43)	.04	.06	.04	.03	.05	.06	.14‡	.31*	–	.10†	.08	−.12†	.18*
Smoking (43)	.01	.02	−.04	.00	−.06	−.10	−.07	.17*	.26*	–	.24*	.07	.24*
Snuff (43)	−.07	−.06	−.01	−.01	.04	−.05	.08	.04	−.01	.04	–	.04	.09
Alcohol (43)	−.08	−.05	.03	−.04	.00	−.05	−.02	−.04	−.05	.06	.07	–	.08
Inactivity (43)	−.03	−.01	−.04	.03	.09	.04	−.01	.04	.18*	.09	.03	−.01	–

BMI = body mass index; SBP/DBP = systolic/diastolic blood pressure; SES = socioeconomic status.
*$p < .001$.
†$p < .05$.
‡$p < .01$.

women and men, an association that appeared to be mediated by BMI in young adulthood, (iii) systolic blood pressure in adolescence was related to metabolic syndrome and appeared to be mediated by early adulthood systolic blood pressure in women but not in men, and (iv) that these life course predictors seemed to operate largely independent from each other and from adult health behaviors and SES.

At a general level, our results of an independent contribution of adolescent SES to adult health in women is in accordance with research on the diverse adult health effects of socioeconomic disadvantage in childhood (26–29). Although gender differences are seldom examined, associations tend to be stronger in women than in men (26, 30). One notable example particularly relevant for metabolic syndrome is research on adult obesity, which consistently has reported effects of childhood SES only for women (17). Specifically, our finding of a strong effect of adolescent SES on metabolic syndrome restricted to women is in accordance with the prospective study Langenberg et al. (12). This work, together with the present study, thus indicates that socioeconomic inequality in metabolic syndrome prevalence previously described in women (7–9) may partly stem from early disadvantage during childhood and adolescence. The failure of adult socioeconomic attainment in explaining the effect of adolescent SES in women is consistent with several reports on metabolic syndrome (15, 16) and obesity (31, 32). We extend these observations by demonstrating that the effect of adolescent SES on metabolic syndrome seems to be independent of several other suggested mediators: first and foremost body mass index and blood pressure in adolescence and young adulthood, a finding that contests the otherwise-plausible critical period model (33) of metabolic parameters mediating the effect of adolescent SES by tracking from young to adult age (34).

Because the effect of adolescent SES was independent from adolescent BMI, the association does not appear to be explained by the unfavorable socioeconomic trajectories caused by gendered discrimination of overweight women (19, 35). We notably also found no effect of adult health behaviors and that physical inactivity was significantly less strongly related to metabolic syndrome in women than in men, the reasons for which are unclear, although it is consistent with findings by Parker et al. (13). Others have found little influence of current health behaviors on metabolic syndrome in either gender (16), but most studies on the influence of childhood SES on metabolic syndrome (12, 36, 37) or obesity (17) have not adjusted for health behaviors.

Together, this lack of support for a range of putative mediators may suggest that childhood/adolescent SES operates over the life course through other pathways specifically in women. Potential mediators are unfavorable life trajectories of multiple adverse exposures (29, 38) particularly

TABLE 3. Hierarchical logistic regression models* in women (n = 403): metabolic syndrome at age 43 on socioeconomic and biological predictors in adolescence and young adulthood

	Model 0	Model 1	Model 2	Model 3
Independent variable	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)
Age 16				
Low SES	2.05 (1.25–3.36)	2.07 (1.23–3.48)	2.12 (1.24–3.64)	2.14 (1.22–3.75)
BMI (kg/m^2)	1.18 (1.08–1.29)	1.18 (1.07–1.29)	1.11 (<1.00–1.24)	1.11 (0.99–1.23)
SBP (5 mmHg)	1.15 (1.04–1.27)	1.10 (0.98–1.24)	1.05 (0.93–1.19)	1.05 (0.93–1.19)
DBP (5 mmHg)	1.07 (0.96–1.19)	1.03 (0.92–1.16)	1.03 (0.92–1.17)	1.04 (0.92–1.17)
Age 21				
Low SES	1.25 (0.75–2.07)		0.98 (0.56–1.69)	0.78 (0.43–1.41)
BMI (kg/m^2)	1.16 (1.06–1.27)		1.10 (1.01–1.21)	1.11 (1.01–1.21)
SBP (5 mmHg)	1.16 (1.05–1.28)		1.14 (1.02–1.28)	1.15 (1.02–1.29)
DBP (5 mmHg)	1.10 (<1.00–1.21)		0.99 (0.88–1.12)	0.98 (0.86–1.11)
Age 43				
Low SES	2.05 (1.24–3.39)			1.74 (0.93–3.23)
Smoking	1.25 (0.77–2.35)			1.44 (0.76–2.76)
Snuff	0.95 (0.46–1.98)			0.79 (0.33–1.86)
Alcohol (abstainers)	1.00			1.00
Alcohol (low)	0.86 (0.36–2.06)			0.99 (0.38–2.60)
Alcohol (high)	0.86 (0.25–2.90)			1.19 (0.30–4.65)
Inactivity	1.17 (0.61–2.23)			0.94 (0.44–1.99)

BMI = body mass index; CI = confidence interval; OR = odds ratio; SBP/DBP = systolic/diastolic blood pressure (per 5 mmHg); SES = socioeconomic status.
*Model 0 = crude ORs. Model 1= predictors at age 16 (SES, BMI, SBP, DBP), Model 2 = Model 1+ predictors at age 21 (SES, BMI, SBP, DBP), Model 3 = Model 2+ covariates at age 43 (SES, daily smoking, daily snuff use, alcohol consumption, physical inactivity).

common among disadvantaged women (39) or adverse psychosocial exposures in childhood (36). Adverse exposures might be further mediated by dysregulations of the cortisol regulation, which has been suggested as playing a pathophysiological role in the development of the metabolic syndrome (40) and which might be influenced by early disadvantage (22). Other unmeasured metabolic risk factors early in life, such as triglycerides and measures of glucose metabolism (3) are also possible mediators which cannot be ruled out by the present investigation. It is also possible that current lifestyle factor insufficiently captures influences on the long-term development of metabolic disease.

TABLE 4. Hierarchical logistic regression models* in men (n = 429): metabolic syndrome at age 43 on socioeconomic and biological predictors in adolescence and young adulthood

	Model 0	Model 1	Model 2	Model 3
Independent variables	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)
Age 16				
Low SES	1.08 (0.72–1.61)	0.93 (0.61–1.44)	0.79 (0.50–1.24)	0.68 (0.41–1.11)
BMI (kg/m^2)	1.20 (1.12–1.30)	1.20 (1.11–1.30)	1.07 (0.96–1.20)	1.07 (0.95–1.20)
SBP (5 mmHg)	1.06 (0.99–1.14)	1.03 (0.95–1.11)	1.04 (0.96–1.13)	1.05 (0.96–1.14)
DBP (5 mmHg)	0.92 (0.93–1.09)	0.99 (0.91–1.08)	1.00 (0.91–1.09)	1.01 (0.92–1.10)
Age 21				
Low SES	1.50 (0.97–2.30)		1.65 (1.02–2.66)	1.34 (0.79–2.29)
BMI (kg/m^2)	1.25 (1.15–1.35)		1.20 (1.07–1.35)	1.22 (1.08–1.38)
SBP (5 mmHg)	1.04 (0.96–1.12)		1.01 (0.92–1.11)	1.01 (0.91–1.11)
DBP (5 mmHg)	1.01 (0.92–1.11)		0.99 (0.89–1.09)	0.98 (0.88–1.09)
Age 43				
Low SES	1.79 (1.06–2.36)			1.36 (0.81–2.27)
Smoking	1.97 (1.20–3.25)			1.74 (0.97–3.14)
Snuff	1.15 (0.75–1.76)			0.96 (0.58–1.56)
Alcohol (abstainers)	1.00			1.00
Alcohol (low)	0.79 (0.34–1.84)			1.24 (0.46–3.38)
Alcohol (high)	2.00 (0.75–5.35)			2.83 (0.90–8.97)
Inactivity	2.75 (1.76–4.28)			2.34 (1.39–3.93)

BMI = body mass index; CI = confidence interval; OR = odds ratio; SBP/DBP = systolic/diastolic blood pressure (per 5 mmHg); SES = socioeconomic status.
*Model 0 = crude ORs. Model 1= predictors at age 16 (SES, BMI, SBP, DBP), Model 2 = Model 1+ predictors at age 21 (SES, BMI, SBP, DBP), Model 3 = Model 2+ covariates at age 43 (SES, daily smoking, daily snuff use, alcohol consumption, physical inactivity).

Of the adolescent/early adulthood metabolic risk factors, BMI in both genders and SBP in women contributed to metabolic syndrome prevalence. This finding is consistent with particularly body mass as important for the development of metabolic syndrome, as reported previously (3, 4, 18), in this study also showed to be independent from both life course SES and adult health behaviors. The contribution of SBP to metabolic syndrome specifically in women has to our knowledge not been reported previously. Metabolic syndrome parameters track moderately well from adolescence (41, 42) and our findings concerning BMI, and in women also SBP, indeed seemed to be mainly explained by tracking of these specific parameters, which has been documented for childhood (43), adulthood (44, 45), and from childhood to adulthood (46–49), although at least in women an effect of onset of puberty has been discussed (50, 51).

On the basis of retrospective data on men, it has been suggested that preventive efforts to reduce socioeconomic inequalities in metabolic syndrome should target health behaviors and obesity (14). Indeed, our results suggest that this statement might have some bearing in men. However, our data imply that interventions only directed at improving lifestyle and reducing obesity might be insufficient for the prevention of metabolic syndrome and related cardiovascular risks in women. Instead, it is of great importance to identify factors explaining the unfavorable metabolic prospects for socioeconomically disadvantaged adolescent girls, factors that might comprise targets for intervention.

Main strengths of the present study is the prospective design, which might be significant for valid inferences (17), and the high proportion of the original cohort participating assessed for metabolic syndrome according to standardized criteria. Limitations include difficulties in assessing SES at age 21, where education was used as a proxy because of a large part of participants studying or doing military service. The null result concerning SES at age 21 might partly stem from this issue. The cohort represents Swedish children who were 16 years of age in 1981, during a time were immigration into Sweden was limited; why we were unable to investigate the influence of ethnicity (9).

Moreover, BMI is not an optimal index of central obesity, which is of particular relevance for the metabolic syndrome. The use of self-reported data (BMI at age 21 and health behaviors at age 43) could possibly introduce bias. The correlation between self-reported and measured BMI at age 43 (data not used in the present manuscript) was high (Spearman's rho = .94 in women and .96 in men), but overreporting of physical inactivity could possibly explain the lack of contribution by this factor in women. Moreover, self-reported alcohol consumption was low in the cohort, possibly because of underreporting. Although the main results did not change with different alcohol coding schemes, the estimates concerning alcohol consumption should be interpreted with caution.

In summary, our results seem to support two pathways to metabolic syndrome operating over the life course, in parallel and independently of adult health behaviors: one metabolic pathway in both women and men with particularly early body mass, and for women also systolic blood pressure tracking into adulthood and thereby increasing the risk for metabolic syndrome. However, for women, early socioeconomic disadvantage seems to operate through other pathways independently of BMI and blood pressure in adolescence and young adulthood. These pathways have to be investigated in future research, an issue which we aim to pursue in the Northern Swedish Cohort.

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