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Socio-economic status and cardiovascular risk factors in rural and urban areas of Vellore, Tamilnadu, South India

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Background We examined associations between socio-economic status (SES) indicators and cardiovascular disease (CVD) risk factors among urban and rural South Indians.

Methods Data from a population-based birth cohort of 2218 men and women aged 26–32 years from Vellore, Tamilnadu were used. SES indicators included a household possessions score, attained education and paternal education. CVD risk factors included obesity, hypertension, impaired glucose tolerance or diabetes, plasma total cholesterol to high density lipoprotein (HDL) ratio and triglyceride levels and consumption of tobacco and alcohol. Multiple logistic regression analysis was used to assess associations between SES indicators and risk factors.

Results Most risk factors were positively associated with possessions score in urban and rural men and women, except for tobacco use, which was negatively associated. Trends were similar with the participants' own education and paternal education, though weaker and less consistent. In a concurrent analysis of all the three SES indicators, adjusted for gender and urban/rural residence, independent associations were observed only for the possessions score. Compared with those in the lowest fifth of the score, participants in the highest fifth had a higher risk of abdominal obesity [odds ratio (OR) = 6.4, 95% CI 3.4–11.6], high total cholesterol to HDL ratio (OR = 2.4, 95% CI 1.6–3.5) and glucose intolerance (OR = 2.8, 95% CI 1.9–4.1). Their tobacco use (OR = 0.4, 95% CI 0.2–0.6) was lower. Except for hypertension and glucose intolerance, risk factors were higher in urban than rural participants independently of SES.

Conclusion In this young cohort of rural and urban south Indians, higher SES was associated with a more adverse CVD risk factor profile but lower tobacco use.

Keywords Socio-economic indicators, CVD risk factors, India, birth cohort studies

Introduction

In recent years, cardiovascular disease (CVD) has emerged as a leading cause of death in developing countries.[1] It is important to identify and target people who are at risk, given that a third of all deaths are expected to be due to CVD by 2020. Studies have shown socio-economic patterning in the prevalence of risk factors for CVD, including obesity,[2,3] smoking[4] and lipid profile.[5] In developed countries, the association between socio-economic status (SES) and CVD risk factors is negative, with a higher prevalence of CVD risk factors among people of lower SES.[6,7] However, the evidence from developing countries, including India, has been inconsistent.[4,8–11] In addition, there is scant information on differences in socio-economic patterning of CVD risk factors between urban and rural areas of India.[12]

Our primary goal was to assess the prevalence of CVD risk factors, and their associations with SES, in urban and rural Indian settings. Most previous studies have used a single indicator of SES such as education, income or wealth index. However, these different indicators may have differing effects.[13] Education reflects degree of knowledge and skill, along with the ability to attract material wealth. On the other hand, income reflects current economic or materialistic welfare. Wealth index, which is based on asset ownership, could be considered an indicator of long-term economic status, as household assets are unlikely to change in response to short-term economic shocks. Since SES indicators are interrelated to a certain extent, the effects of each of these indicators could be masked by the others. For instance, education could have a direct effect on CVD risk factors or reflect the effect of income or wealth. Further, differences in CVD risk factors between SES groups could arise in early life, and studying paternal education as a measure of childhood SES would provide an opportunity to compare the relative effects of current and childhood SES on CVD risk factors. Thus, examining the independent effect of these indicators could provide better understanding regarding the underlying mechanisms and help identify specific target groups in CVD prevention programmes.

We have therefore used multiple indicators of SES (a score based on household possessions, educational status and paternal education) to assess the independent effect of each of these indicators on CVD risk factors among South Indian adults.

Methods

Participants and settings

Original study

We used data from a cohort of infants born during 1969–73 in Vellore district, in Tamilnadu state. Twenty-four wards in Vellore town representing different socio-economic strata, and 25 of 42 villages from nearby rural settings were randomly selected. The rural sample was chosen from a geographically defined region 15 miles from the town. The main occupation in this area is agriculture, followed by beedi (local cigarette) manufacturing. The urban sample was selected from Vellore town (population 906 745; 2001 census) which is the district headquarters. A large section of the population is involved in trade, commerce and government service.

In the original study, a total of 14 147 pregnancies were identified, which resulted in 10 691 singleton live births. Of these, 47% moved outside the study area as many mothers traditionally moved to their parents' home for delivery and were not available for further examination. The remainder ($n = 5753$) were measured (birthweight, length and head circumference) within 120 h of delivery by trained personnel. These measurements were repeated during infancy (1–3 months), childhood (6–8 years) and adolescence (10–15 years). Further details about the cohort are described elsewhere.[14]

Follow-up study

In 1998–2002, we retraced members of the original cohort (now aged 26–32 years) to study the relationship between early childhood growth and adult cardiovascular risk factors.[15] All subjects who were singleton births and whose parental and birth measurements were available ($n = 4052$) were eligible for the follow-up study. Of the latter, 2572 were traced by health workers and 2218 (55%) agreed to participate in the study, 997 from urban areas (men: 544; women: 453) and 1221 from rural areas (men: 617; women: 604). Information on SES indicators, anthropometry and CVD risk factors were obtained during this period (1998–2002).

Variables

SES indicators

We used the individual's education level, paternal education and a score based on household possessions as indicators of SES. Income data are often poorly reported in developing settings, and it was thought that a possessions score would be a more reliable way of assessing SES. Education variables were recorded as one of four categories from 'no schooling' (category 1, 0 years), primary and middle school (category 2, 1–8 years), high school and higher secondary (category 3, 9–12 years) and >12 years of schooling (category 4, >12 years). Paternal education was used as a measure of childhood SES.

For the possessions score, participants were asked if the household owned each of the following items/amenities: electricity, fan, bicycle, radio, motorized two-wheeled vehicle, gas stove, television, cable television, electric mixer, electric grinder, electric air cooler, washing machine, car, air conditioner, computer, television antenna and telephone. One approach to such data involves summing the number

of possessions, but this has the disadvantage of assigning equal weight to each item, regardless of its value or utility. We therefore created a composite score using weights obtained from principal component analysis (PCA)[16] and grouped the first principal component by quintiles.

Anthropometry and biochemical analyses

Participants attended the clinic after an overnight fast. Urban participants came to the main hospital in Vellore town and rural subjects visited the Rural Unit for Health and Social Affairs (RUHSA). Physical measurements included weight, height, waist and hip circumferences and blood pressure. These were made according to standard protocols by one of two physicians who were trained, and their methods standardized, before the start of the study. Blood pressure was measured using an OMRON 711 automated device,[17] with the appropriate cuff size for the measured mid-upper-arm circumference, and after the subject had been seated at rest for at least 5 min. Two readings were made, re-applying the cuff for each, and the average of two readings was used for the definition of hypertension. Plasma glucose and lipid levels were measured fasting (12 h overnight) and glucose was measured 30, 60 and 120 min following a 75-g oral glucose load. Blood samples from the rural clinic were transported on ice to the main hospital laboratory and centrifuged within 3 h of collection. Plasma glucose was measured by a glucose oxidase/peroxidase method, and serum lipids using commercial enzymatic kits (Roche Diagnostics, Germany), on a Hitachi 911 autoanalyser (USA). Roche Precinorm and Precipath controls were used for quality assessment of these parameters. The biochemistry laboratory also belongs to WHO and BioRad schemes for external quality assurance.

Definitions of CVD risk factors

Body mass index (BMI) was calculated as weight (kg) divided by height (m) squared. BMI was categorized as underweight (<18.5), normal (18.5–22.9), overweight (23.0–24.9) and obese (>25).[18] Abdominal obesity was defined as a waist circumference >90 cm for men and >85 cm for women.[19] Hypertension was defined as a systolic blood pressure $\geqslant$140 mmHg and/or diastolic blood pressure $\geqslant$90 mmHg.[20] Using self-report, participants who had been diagnosed by a doctor as having hypertension and used anti-hypertensive drugs ($n=1$) were classified as hypertensive. A high total cholesterol to high density lipoprotein (HDL) ratio was defined as $\geqslant$4.5 and a high triglyceride concentration as $\geqslant$1.69 mmol/l.[21] Diabetes was defined as a fasting blood glucose $\geqslant$7 mmol/l or a 120-min value of $\geqslant$11.1 mmol/l. Impaired glucose tolerance (IGT) was defined as fasting blood glucose <7 mmol/l and a 120-min value of $\geqslant$7.8 mmol/l but <11.1 mmol/l, and impaired fasting glucose (IFG) as a fasting blood glucose of $\geqslant$6.1 mmol/l and <7 mmol/l.[22] Participants who had been diagnosed by a doctor as having diabetes and used medication for diabetes ($n=8$) were classified as diabetic.

Current alcohol consumption was assessed by questioning the participants about their frequency and volume of intake of spirits, beer and wine, and these were converted into units of alcohol per week (1 unit = 574 ml beer or 125 ml wine or 23 ml spirits). They were categorized as 0 = none (0 units), 1 = mild ($\leqslant$7 units), 2 = moderate (8–21 units) and 3 = heavy ($\geqslant$21 units) and dichotomized into current consumers or non-consumers of alcohol. Tobacco consumption was recorded as whether the subjects smoked (cigarettes, bidis, cigars or hookah), chewed (raw tobacco or with pan) or inhaled (snuff). Subjects were categorized simply as current tobacco users or as nonusers (ex-users + never).

Statistical analysis

All descriptive analyses of the risk factors were performed separately for men and women and for urban and rural participants. Analyses were carried out using the risk factors as both continuous and dichotomous variables; the patterns were similar for both and we present results only for the latter. Kendall's τ rank correlation coefficients were obtained to assess the relationship between the three indicators of SES. We examined the prevalence rates (and 95% CI) of risk factors across the categories of the socio-economic indicators. *P*-values for trends in CVD risk factors were obtained by treating the SES indicators as continuous variables in logistic regression analyses. We used multiple logistic regression analyses to estimate independent effects of the different socio-economic indicators on risk factors, including gender, place of residence, possessions score, adult educational status and paternal educational status in the models as covariates. Interaction tests were used to examine differences in associations between SES and risk factors according to urban–rural residence; none were found. All analyses were performed using STATA 10.0 (StataCorp, College Station, TX, USA).

Results

Study population

Data from a total of 2218 participants were available for the analysis, 997 urban participants (544 men and 453 women) and 1221 rural participants (617 men and 604 women). Their age ranged from 26 to 32 years with a mean of 28.3 years (SD = 1.1).

Urban–rural differences

Table 1 shows the mean and SD (for continuous measures) and the prevalence (dichotomous variables) of CVD risk factors stratified by gender and place of residence. Urban men had the highest

Table 1 Prevalence, mean and standard deviation of CVD risk factors stratified by urban–rural status and gender

	Urban ($n=997$)		Rural ($n=1221$)		
Mean levels of CVD risk factors	Men ($n=544$) Mean (SD)	Women ($n=453$) Mean (SD)	Men ($n=617$) Mean (SD)	Women ($n=604$) Mean (SD)	***P*-value[b]**
Age (years)	28.5 (1.1)	28.0 (1.2)	27.9 (1.0)	28.1 (1.3)	<0.001
BMI (kg/m^2)	21.5 (3.6)	22.3 (4.3)	19.9 (3.2)	19.7 (3.7)	<0.001
Waist circumference (cm)[a]	79.6 (71.5–88.6)	75.1 (67.3–81.6)	74.8 (69.1–82.9)	68.0 (62.8–74.5)	<0.001
Total cholesterol to HDL ratio	4.5 (1.3)	3.9 (1.0)	4.1 (1.2)	3.6 (0.9)	<0.001
Triglycerides (mmol/l)[a]	1.1 (0.8–1.7)	0.8 (0.6–1.1)	0.9 (0.7–1.3)	0.8 (0.6–1.0)	<0.001
SBP (mmHg)	114.2 (10.9)	101.1 (10.2)	110.6 (10.9)	101.6 (10.9)	<0.001
DBP (mmHg)	74.7 (9.2)	72.9 (8.2)	71.0 (9.0)	72.2 (8.6)	<0.001
FBG (mmol/l)[a]	5.5 (5.2–5.8)	5.4 (5.1–5.8)	5.4 (5.1–5.7)	5.3 (4.9–5.6)	<0.001
Glucose 120 min (mmol/l)[a]	6.3 (5.2–7.4)	6.6 (5.7–7.7)	6.0 (5.2–7.1)	6.4 (5.5–7.4)	<0.001
Prevalence of CVD risk factors (%)					
Underweight (<18.5 kg/m^2)	22.1	21.8	39.2	43.8	<0.001
Overweight (23.0–24.9 kg/m^2)	16.6	17.4	8.8	9.1	<0.001
Obesity I (25.0–29.9 kg/m^2)	14.7	19.8	7.8	7.1	<0.001
Obesity II (>30.0 kg/m^2)	1.8	5.6	0.49	1.5	<0.001
Abdominal obesity (men >90 cm; women >85 cm)	18.9	14.1	10.4	5.8	<0.001
High total cholesterol to HDL ratio ($\geqslant$4.5)	47.4	23.5	31.8	13.1	<0.001
High triglyceride ($\geqslant$1.69 mmol/l)	25.0	8.6	17.1	4.3	<0.001
Hypertension (SBP $\geqslant$ 140 mm Hg or DBP $\geqslant$ 90 mm Hg)	4.9	1.3	2.1	1.5	0.032
Diabetes (FBG $\geqslant$7.0 mmol/l or 120-min value $\geqslant$11.1 mmol/l)	4.6	2.6	1.5	2.6	0.016
IGT (FBG <7.0 mmol/l and 120-min value $\geqslant$7.8 mmol/l, but <11.0 mmol/l)	17.2	22.5	13.7	15.5	0.0004
IFG (FBG $\geqslant$6.1 mmol/l and 120-min value <7.0 mmol/l)	6.3	7.5	5.7	4.6	0.104
Tobacco use	46.5	1.6	40.1	3.2	0.11
Alcohol use	60.3	–	49.5	–	<0.001

FBG: fasting blood glucose; SBP: systolic blood pressure; DBP: diastolic blood pressure.
[a]Summarized as median and inter-quartile range.
[b]Age- and sex-adjusted *P*-values were obtained for the comparison of CVD risk factors between rural and urban populations.

prevalence of abdominal obesity, high total cholesterol to HDL ratio and triglyceride levels, hypertension, diabetes and tobacco and alcohol use. The prevalence of obesity, overweight and IGT was highest among urban women. Rural women had the highest prevalence of underweight. Similar percentages of men in the urban and rural populations used tobacco, whereas urban men were more likely to consume alcohol. Very few women were tobacco users, and none consumed alcohol.

Relationship between SES indicators

Household possessions score was positively correlated with education status [Kendall's $\tau=0.44$, $P<0.001$] and paternal education status [Kendall's $\tau=0.42$, $P<0.001$], as were individual and paternal education status [Kendall's $\tau=0.36$, $P<0.001$]. As shown in Table 2, rural residents were less likely than urban participants to be in the highest fifth of the possessions score, or in the highest education category. Paternal education levels showed large urban–rural differences; 42.2% of the rural adults had fathers who received no formal education, compared with 22.7% of urban adults. However, the adults' own education levels did not vary greatly; 7.5% of urban adults had no formal education compared with 11.5% of rural adults.

Table 2 Distribution of SES indicators by urban–rural status and gender

SES Indicators	Urban ($n=997$)		Rural ($n=1221$)		*P* value[a]
	Men ($n=544$)	Women ($n=453$)	Men ($n=617$)	Women ($n=604$)	
Household possessions score (%)					
1 (Lowest)	7.2	7.2	28.0	32.9	
2	15.9	17.0	25.5	19.9	
3	21.3	22.9	19.6	21.4	
4	21.5	21.4	16.7	16.6	
5 (Highest)	33.5	31.5	10.2	9.4	<0.001
Education (%)					
No education (0 years)	3.7	11.9	5.7	17.4	
Primary/middle school (1–8 years)	35.3	40.2	34.5	49.2	
High/secondary school (9–12 years)	44.1	34.9	49.9	31.1	
>12 years of schooling	16.9	13.0	9.9	2.3	<0.001
Paternal education (%)					
No education (0 years)	22.5	22.9	44.5	40.2	
Primary/middle school (1–8 years)	55.6	53.9	46.0	49.2	
High/secondary school (9–12 years)	19.9	20.1	8.9	8.6	
>12 years os schooling	2.2	3.1	0.5	1.9	<0.001

[a]Age- and sex-adjusted *P*-values were obtained for the comparison of SES indicators between rural and urban populations.

BMI and waist circumference

Tables 3–5 show the prevalence of risk factors according to the SES indicators, stratified by gender and place of residence. In general, there were increasing trends in the prevalence of obesity, overweight and abdominal obesity with the possessions score, in both sexes and in both urban and rural areas (Table 3). The prevalence of obesity was highest among urban women, and that of abdominal obesity highest among urban men, from the highest fifth of the possessions score. In contrast, the prevalence of underweight decreased with increasing possessions score and was highest among rural women in the lowest fifth of the possessions score. Although there were similar trends in the obesity measures with other SES indicators (Tables 4 and 5), these were weaker and less consistent, especially among rural participants.

Lipid profile, blood pressure and glucose tolerance

The prevalence of a high total cholesterol to HDL ratio, high triglycerides, hypertension and glucose intolerance (either IGT, IFG or diabetes) tended to increase with higher possessions score in both genders and in both urban and rural participants (Table 3). These trends were stronger in urban than in rural participants, and in men than women, with the exception of glucose intolerance. In contrast, associations of risk factors with educational status (Table 4) were present only for high total cholesterol to HDL ratio in urban men, and those with paternal education (Table 5) were present only for high total cholesterol to HDL ratio in rural men, hypertriglyceridaemia in urban women, and glucose intolerance in rural women.

Tobacco and alcohol use

These analyses were carried out for men only. The prevalence of tobacco use was highest among urban men in the lowest fifth of the possessions score and fell with increasing score (Table 3), with increasing educational status in urban and rural men (Table 4), and with increasing paternal educational status in rural men (Table 5). Alcohol use was unrelated to any of the SES indicators among urban men, but fell with increasing possessions score and educational status among rural men.

Independent effects of SES indicators on risk factors

Table 6 shows the ORs for risk factors according to fifths of the SES variables, adjusted for each other along with gender and place of residence. Higher household possessions score was associated with increased odds of obesity, overweight, abdominal obesity, high total cholesterol to HDL ratio, high triglycerides and glucose intolerance. Household possessions score was inversely associated with underweight and tobacco use. There were no independent effects of the individual's education level on CVD risk factors in this analysis, except that higher educational status

Table 3 Prevalence (%) (95% CI) of CVD risk factors according to quintiles of household possessions score, stratified by urban–rural status and gender

	Household possessions score (fifths)					
CVD risk factors	1 (Lowest)	2	3	4	5 (Highest)	*P*-value for trend[a]
Obesity						
UM	2.4 (0.06–12.5)	8.0 (3.3–16.1)	12.9 (7.4–20.4)	20.5 (13.6–28.9)	23.6 (17.6–30.4)	<0.001
UW	9.1 (2–24)	7.8 (3–16.1)	24.2 (16.3–33.7)	25.0 (16.7–34.8)	40.0 (31.8–48.6)	<0.001
RM	4.6 (2–8.9)	5.7 (2.6–10.7)	5.8 (2.3–11.6)	12.6 (6.9–20.6)	22.2 (12.7–34.5)	0.001
RW	5.0 (2.4–9.0)	5.9 (2.4–11.7)	6.9 (3.2–12.8)	15.0 (8.6–23.5)	19.6 (10.2–32.4)	0.01
Overweight						
UM	7.1 (1.5–19.4)	13.9 (7.4–23.1)	12.9 (7.4–20.4)	18.8 (12.1–27.1)	20.9 (15.2–27.5)	0.005
UW	9.1 (1.9–24.3)	15.5 (8.3–25.6)	18.4 (11.4–27.2)	16.6 (9.8,25.6)	20.0 (13.7–27.5)	0.09
RM	5.7 (2.8–10.3)	6.4 (3.1–11.4)	6.6 (2.9–12.7)	15.6 (9.1,23.9)	15.8 (7.9,27.3)	0.02
RW	6.0 (3.1–10.2)	9.2 (4.7–15.9)	9.3 (4.9–15.7)	10.0 (4.9–17.6)	17.8 (8.9–30.4)	0.21
Underweight						
UM	30.9 (17.6–47.0)	30.2 (20.7–41.1)	26.7 (18.9–35.7)	17.9 (11.5–26.1)	15.9 (10.9–22.1)	0.018
UW	42.4 (25.4–60.7)	37.7 (26.8–49.4)	17.5 (10.6–26.2)	25.0 (16.7–34.8)	9.3 (5.1–15.4)	0.006
RM	45.7 (38.1–53.4)	48.1 (40.0–56.2)	43.3 (34.3–52.6)	26.2 (18.0–35.8)	12.7 (5.6,23.4)	<0.001
RW	55.7 (48.1–62.3)	43.6 (34.6–53.1)	50.3 (41.5–59.3)	30.0 (21.2–39.9)	12.5 (5.1–24.1)	<0.001
Abdominal obesity						
UM	4.8 (0.5–16.1)	9.1 (4.0–17.3)	12.9 (7.4–20.4)	21.3 (14.3–29.9)	29.1 (22.6–36.3)	<0.001
UW	0.0 (0.0)	3.8 (0.8–10.9)	11.5 (6.1–19.2)	15.4 (8.9–24.2)	23.9 (17.2–31.8)	<0.001
RM	4.6 (2.0–8.9)	7.0 (3.5–12.1)	9.9 (5.2–16.7)	14.5 (8.3–22.9)	28.6 (17.9–41.3)	<0.001
RW	3.5 (1.4–7.1)	1.6 (0.2–5.8)	5.4 (2.2–10.9)	9.0 (4.1–16.3)	17.8 (8.9–30.3)	<0.001
High total cholesterol to HDL ratio						
UM	30.9 (17.6–47.0)	41.8 (31.3–52.9)	43.9 (34.5–53.4)	46.4 (37.0–56.1)	56.9 (49.3–64.2)	0.001
UW	6.0 (0.7–20.2)	15.5 (8.3–25.6)	28.8 (20.3–38.5)	20.0 (12.5–29.4)	30.0 (23.0–38.8)	0.004
RM	23.8 (17.6–30.9)	29.4 (22.4–37.3)	31.1 (22.9–40.2)	40.5 (30.9–50.8)	46.0 (33.3–59.0)	<0.001
RW	12.5 (8.2–17.9)	12.5 (7.2–19.7)	9.3 (4.9–15.8)	17.0 (10.2–25.9)	17.8 (8.9–30.3)	0.28
High triglycerides						
UM	16.7 (6.9–31.3)	17.2 (9.9–26.8)	22.4 (15.1–31.1)	27.3 (19.5–36.3)	30.7 (24.1–38.0)	0.005
UW	3.0 (0.07–15.7)	7.7 (2.9–16.1)	6.7 (2.7–13.4)	10.3 (5.1–18.2)	10.5 (6.1–16.9)	0.14
RM	16.1 (11.0–22.5)	15.9 (10.6–22.6)	12.5 (7.1–19.8)	23.3 (15.5–32.6)	20.6 (11.4–32.6)	0.21
RW	4.5 (2.1–8.4)	3.3 (0.9–8.3)	2.3 (0.5–6.6)	6.0 (2.3–12.7)	7.1 (2.0–17.3)	0.44
Hypertension						
UM	2.3 (0.0–12.5)	1.1 (0.002–6.2)	2.5 (0.5–7.3)	6.0 (2.4–11.9)	8.2 (4.6–13.2)	0.007
UW	–	5.1 (1.4–12.7)	1.0 (0.02–5.2)	1.0 (0.02–5.6)	–	0.06
RM	1.1 (0.1–4.1)	1.3 (0.1–4.5)	1.6 (0.2–5.8)	3.8 (1.0–9.6)	4.7 (0.09–13.2)	0.04
RW	2.5 (0.8–5.7)	–	–	4.0 (1.0–9.1)	–	0.72
Diabetes/ IGT/IFG						
UM	26.2 (12.3–40.0)	16.1 (8.0–23.9)	18.9 (11.8–26.2)	25.6 (17.7–33.7)	31.9 (25.0–38.9)	0.01
UW	12.1 (0.0–23.9)	22.1 (12.6–31.6)	34.6 (25.3–43.9)	28.9 (19.6–38.0)	30.3 (22.3–37.9)	0.08
RM	10.9 (6.3–15.7)	17.2 (11.2–23.1)	23.1 (15.6–30.8)	20.4 (12.4–28.4)	31.8 (19.9–43.5)	<0.001
RW	16.1 (10.9–21.2)	15.8 (9.2–22.4)	23.3 (15.8–30.6)	27.0 (18.1–35.8)	32.1 (19.5–44.7)	0.001
Tobacco use						
UM	71.4 (55.4–84.2)	50.5 (39.6–61.4)	46.5 (37.2–56.0)	51.2 (41.8–60.2)	35.7 (28.7–43.1)	<0.001
RM	49.7 (42.0–57.3)	45.2 (37.3–53.3)	34.7 (26.2–43.9)	32.0 (23.1–41.9)	25.3 (15.2–37.9)	<0.001
Alcohol use						
UM	61.9 (45.6–76.4)	55.1 (44.1–65.8)	63.7 (54.3–72.5)	65.8 (56.5–74.3)	56.5 (49.0–63.9)	0.79
RM	53.7 (46.0–61.3)	53.5 (45.4–61.4)	46.3 (37.2–55.6)	45.6 (35.7–55.7)	41.2 (29.0–54.3)	0.03

UM: urban men; UW: urban women; RM: rural men; RW: rural women.
[a]*P*-values for trend were obtained from simple logistic regression analysis.

Table 4 Prevalence (%) (95% CI) of CVD risk factors according to level of education, stratified by urban–rural status and gender

	Educational status of the adult				
CVD risk factors	No education (0 years)	Primary/Middle school (1–8 years)	High/Secondary school (9–12 years)	>12 years of schooling	*P*-value for trend[a]
Obesity					
UM	10.0 (1.2–31.7)	13.0 (8.6–18.7)	15.4 (11.0–20.7)	28.2 (19.3–38.7)	0.03
UW	16.7 (7.9–29.3)	21.5 (15.7–28.2)	28.2 (21.3–35.9)	37.9 (25.5–51.7)	0.01
RM	8.5 (1.8–23.0)	7.0 (4.0–11.3)	9.8 (6.7–13.7)	4.9 (1.0–13.7)	0.83
RW	4.7 (1.5–10.7)	7.4 (4.7–11.0)	12.7 (8.3–18.4)	7.1 (0.1–33.8)	0.17
Overweight					
UM	25.0 (8.7–49.0)	13.0 (8.6–18.7)	15.8 (11.4–21.1)	23.9 (15.6–33.9)	0.43
UW	9.2 (3.0–20.3)	16.0 (11.0–22.1)	21.7 (15.5–29.0)	17.2 (8.5–29.4)	0.05
RM	8.5 (1.8–23.0)	7.0 (4.0–11.3)	10.1 (6.9–14.1)	8.2 (2.7–18.1)	0.81
RW	5.7 (2.1–12.0)	9.4 (6.4–13.3)	10.1 (6.2–15.3)	14.2 (1.8,42.9)	0.49
Underweight					
UM	35.0 (15.3–59.2)	27.7 (21.5–34.6)	22.1 (17.0–27.9)	7.6 (3.1–15.1)	0.004
UW	27.8 (16.4–41.7)	27.0 (20.8–34. 2)	18.5 (12.8–25.6)	8.6 (2.8–18.9)	0.26
RM	45.7 (28.8–63.3)	40.8 (34.1–47.7)	38.2 (32.7–43.9)	34.4 (22.7–47.6)	0.26
RW	58.1 (48.1–67.7)	43.9 (38.2–49.8)	36.1 (29.3–43.4)	35.7 (12.8–64.9)	0.006
Abdominal obesity					
UM	10.0 (1.2,31.7)	13.0 (8.6–18.6)	18.3 V(13.6–23.8)	34.7 (25.1–45.4)	<0.001
UW	5.5 (1.1–15.3)	12.1 (7.7–17.8)	15.8 (10.5–22.4)	23.7 (13.6–36.5)	0.005
RM	8.5 (1.8–23.0)	8.9 (5.4–13.5)	12.6 (9.1–16.9)	4.9 (1.0–13.7)	0.80
RW	1.9 (0.2,6.7)	5.0 (2.8–8.2)	9.6 (5.8–14.8)	–	0.03
High total cholesterol to HDL ratio					
UM	27.8 (9.6–53.4)	44.4 (37.2–51.8)	47.1 (40.6–53.6)	58.8 (48.1–69.2)	0.008
UW	24.1 (13.4–37.6)	22.2 (16.4–29.0)	22.9 (16.7–30.3)	28.9 (17.8–42.1)	0.64
RM	22.8 (10.4–40.1)	28.8 (22.8–35.4)	34.8 (29.5–40.4)	32.2 (20.6–45.6)	0.12
RW	11.4 (6.0–19.1)	12.8 (9.2–17.1)	15.4 (10.5–21.3)	–	0.63
High triglycerides					
UM	30.0 (11.9–54.2)	23.9 (18.1–30.6)	22.1 (17.0–27.8)	33.7 (24.1–44.3)	0.43
UW	7.4 (2.0–17.9)	9.8 (5.9–15.1)	7.5 (3.9–12.8)	8.4 (2.8–18.7)	0.94
RM	20.0 (8.4–36.9)	19.8 (14.6–25.8)	15.2 (11.4–19.7)	14.7 (6.9–26.1)	0.21
RW	3.8 (1.0–9.4)	4.3 (2.3–7.4)	4.8 (2.2–8.9)	–	0.89
Hypertension					
UM	10.0 (1.2–31.7)	2.6 (0.8–5.9)	4.1 (2.1–7.5)	10.8 (5.3–19.0)	0.12
UW	3.7 (0.4–12.7)	1.0 (0.1–3.9)	1.2 (0.1–4.4)	–	0.13
RM	–	–	2.5 (1.1–5.0)	4.9 (1.0–13.7)	0.04
RW	1.9 (0.2–6.7)	0.6 (0.08–2.4)	2.6 (0.8–6.0)	–	0.77
Diabetes/ IGT/IFG					
UM	15.0 (0.0–32.1)	23.9 (17.8–30.0)	22.5 (17.1–27.8)	34.7 (24.8–44.8)	0.06
UW	31.5 (18.7–44.2)	27.5 (20.9–34.0)	26.5 (19.6–33.5)	32.2 (19.9–44.4)	0.88
RM	25.7 (10.4–40.9)	19.7 (14.3–25.1)	16.9 (12.6–21.1)	19.7 (9.0–29.9)	0.29
RW	19.1 (11.4–26.7)	19.2 (14.6–23.7)	22.3 (16.3–28.4)	50.0 (20.0–79.9)	0.16
Tobacco use					
UM	45.0 (23.1–68.4)	59.3 (52.1–66.4)	41.6 (35.3–48.1)	32.6 (23.1–43.1)	<0.001
RM	57.1 (39.3–73.6)	47.8 (41.1–54.8)	38.9 (33.4–44.6)	9.8 (3.6–20.1)	<0.001
Alcohol use					
UM	35.0 (15.3–59.2)	62.5 (55.2–69.3)	64.1 (57.7–70.2)	51.1 (40.4–61.7)	0.81
RM	62.9 (46.0–79.6)	56.3 (49.6–63.0)	47.4 (41.8–53.0)	29.5 (17.8–41.2)	<0.001

UM: urban men; UW: urban women; RM: rural men; RW: rural women.
[a]*P*-values for trend were obtained from simple logistic regression analysis.

Table 5 Prevalence (%) (95% CI) of CVD risk factors according to paternal education status, stratified by urban–rural status and gender

	Paternal educational status				
CVD risk factors	No education (0-years)	Primary/Middle school (1–8 years)	High/Secondary school (9–12 years)	>12 years of schooling	*P*-value for trend[a]
Obesity					
UM	14.7 (8.3–21.1)	13.6 (9.7–17.5)	24.1 (15.8–32.3)	41.6 (8.9–74.3)	0.06
UW	20.2 (12.3–28.0)	23.6 (18.2–29.0)	34.1 (24.1–43.9)	38.5 (7.9–69.0)	0.29
RM	6.6 (3.6–9.5)	8.8 (5.5–12.1)	12.7 (3.6–21.8)	–	0.07
RW	6.1 (3.1–9.2)	8.4 (5.2–11.6)	11.5 (2.5–20.5)	50 (16.8–83.1)	0.02
Overweight					
UM	17.2 (10.4–24.0)	17.6 (13.2–21.9)	12.9 (6.5–19.4)	16.6 (0.0–41.3)	0.96
UW	13.4 (6.7–20.1)	18.2 (13.3–23.1)	17.5 (9.6–25.6)	30.8 (1.7–59.8)	0.51
RM	7.3 (4.1–10.4)	8.8 (5.5–12.1)	16.4 (6.2–26.4)	–	0.10
RW	6.1 (3.1–9.2)	11.4 (7.8–15.1)	11.5 (2.6–20.5)	–	0.17
Underweight					
UM	20.4 (13.2–27.8)	24.2 (19.4–29.1)	17.6 (10.3–24.9)	25.0 (0.0–53.8)	0.66
UW	36.5 (27.1–45.9)	19.9 (14.8–24.9)	12.1 (5.2–18.9)	7.6 (0.0–24.5)	<0.001
RM	39.8 (33.9–45.6)	40.2 (34.5–46.0)	32.7 (19.9–45.5)		0.83
RW	48.9 (42.6–55.3)	43.2 (37.6–48.9)	26.9 (14.4–39.4)	25.0 (0.0–53.7)	0.13
Abdominal obesity					
UM	16.3 (9.7–23.0)	17.8 (13.5–22.2)	21.2 (13.4–29.1)	50.0 (16.9–83.1)	0.09
UW	10.5 (4.6–16.6)	13.9 (9.5–18.3)	17.5 (9.6–25.6)	21.4 (0.0–46.0)	0.12
RM	8.0 (4.7–11.2)	11.2 (7.6–14.9)	16.4 (6.2–26.4)	–	0.04
RW	2.4 (0.1–4.4)	6.0 (3.3–8.7)	11.5 (2.5–20.6)	41.7 (8.9–74.4)	<0.001
High total cholesterol to HDL ratio					
UM	45.4 (36.5–54.5)	45.4 (39.8–51.1)	56.1 (46.5–65.7)	41.7 (8.9–79.4)	0.30
UW	19.6 (11.8–27.5)	24.2 (18.9–29.8)	27.4 (18.1–36.8)	14.3 (0.0–35.2)	0.36
RM	27.8 (22.4–33.1)	32.9 (27.3–38.4)	44.4 (30.8–58.1)	–	0.02
RW	12.7 (8.5–16.9)	12.7 (8.9–16.7)	17.6 (6.9–28.5)	8.3 (0.0–26.7)	0.76
High triglycerides					
UM	23.8 (16.1–31.4)	24.5 (19.6–29.3)	27.8 (19.1–36.4)	25.0 (0.0–53.8)	0.58
UW	13.4 (6.7–20.1)	8.1 (4.7–11.6)	3.2 (0.0–7.0)	14.2 (0.0–35.3)	0.04
RM	15.2 (10.9–19.6)	17.9 (13.4–22.4)	22.2 (10.8–33.7)	–	0.27
RW	3.2 (1.0–5.5)	4.0 (1.7–6.3)	9.6 (1.3–17.9)	8.3 (0.0–26.7)	0.13
Hypertension					
UM	3.3 (0.0–6.4)	4.9 (2.5–7.4)	6.4 (1.8–11.1)	8.3 (0.0–26.7)	0.23
UW	1.9 (0.0–4.6)	1.2 (0.0–2.6)	–	7.1 (0.0–22.5)	0.76
RM	1.0 (0.0–2.3)	2.5 (1.0–4.2)	5.4 (0.0–11.6)	–	0.08
RW	1.6 (0.0–3.3)	1 (0.0–2.0)	1.9 (0.0–5.7)	8.3 (0.0–26.7)	0.74
Diabetes/IGT/IFG					
UM	26.2 (18.3–34.1)	24.5 (19.6–29.3)	24.1 (15.8–32.2)	25.0 (0.0–53.7)	0.69
UW	29.8 (20.8–38.7)	26.6 (21.1–32.2)	34.1 (24.1–43.9)	7.1 (0.0–22.5)	0.71
RM	18.2 (13.5–22.8)	18.7 (14.1–23.2)	21.8 (10.6–33.1)		0.77
RW	16.5 (11.8–21.1)	22.6 (17.8–27.3)	23.1 (11.2–34.9)	58.3 (25.6–91.0)	0.006
Tobacco use					
UM	51.6 (42.7–60.6)	45 (39.3–50.6)	43.5 (34.0–53.0)	58.3 (25.6–91.0)	0.29
RM	45.8 (39.8–51.7)	36.7 (30.9–42.2)	30.9 (18.3–43.5)	–	0.008
Alcohol use					
UM	63.1 (54.4–71.8)	59.6 (54.0–65.1)	58.3 (48.9–67.8)	66.7 (35.4–97.9)	0.54
RM	53.1 (47.2–59.0)	47.8 (42.0–53.8)	41.8 (28.3–55.3)	–	0.08

UM: urban men; UW: urban women; RM: rural men; RW: rural women.
[a]*P*-values for trend were obtained from simple logistic regression analysis.

Table 6 Multiple logistic regression analysis of all three SES indicators adjusted for each other, gender and urban–rural status

SES Indicators	Obesity OR 95%CI	Overweight OR 95%CI	Underweight OR 95%CI	Abdominal obesity OR 95%CI	High total cholesterol to HDL ratio OR 95%CI	High triglyceride OR 95%CI	Hypertension OR 95%CI	Diabetes /IGT/IFG OR 95%CI	Tobacco use OR 95%CI	Alcohol use OR 95%CI
Gender (female)	1.6 (1.2–2.1)	1.3 (1.0–1.7)	1.2 (0.9–1.5)	0.6 (0.5–0.8)	0.3 (0.2–0.4)	0.2 (0.1–0.3)	0.4 (0.2–0.8)	1.1 (0.9–1.4)	–	–
Place of residence (urban)	1.9 (1.4–2.6)	1.6 (1.2–2.2)	0.7 (0.5–0.8)	1.4 (1.1–1.8)	1.6 (1.3–2.0)	1.4 (1.1–1.9)	1.5 (0.8–2.7)	1.1 (0.9–1.4)	1.6 (1.2–2.1)	1.6 (1.3–2.1)
Possessions score (fifths)										
1 (Lowest) (Reference)										
2	1.2 (0.6–2.1)	1.4 (0.8–2.4)	0.9 (0.7–1.3)	1.3 (0.7–2.4)	1.3 (0.9–1.8)	1.0 (0.7–1.6)	0.8 (0.3–2.3)	1.2 (0.9–1.8)	0.6 (0.4–0.9)	0.8 (0.6–1.3)
3	1.9 (1.1–3.4)	1.5 (0.8,2.5)	0.8 (0.6–1.1)	2.4 (1.3–4.3)	1.5 (1.1–2.1)	1.0 (0.6–1.6)	0.6 (0.2–1.9)	1.9 (1.3–2.7)	0.5 (0.3–0.7)	0.8 (0.6–1.3)
4	2.9 (1.7–5.1)	1.9 (1.2–3.4)	0.6 (0.4–0.8)	3.7 (2.1–6.7)	1.7 (1.2–2.5)	1.8 (1.2–2.8)	1.7 (0.6–4.4)	2.1 (1.4–3.0)	0.5 (0.4–0.9)	1.0 (0.7–1.5)
5(Highest)	4.1 (2.3–7.3)	2.5 (1.5–4.4)	0.4 (0.3–0.6)	6.4 (3.5–11.6)	2.4 (1.6–3.5)	2.1 (1.3–3.3)	1.3 (0.5–3.8)	2.8 (1.9–4.1)	0.4 (0.2–0.6)	0.8 (0.5–1.3)
Education[a]										
I (Reference)										
II	0.9 (0.6–1.8)	0.9 (0.6–1.7)	0.8 (0.5–1.1)	1.3 (0.7–2.7)	1.0 (0.7–1.6)	0.9 (0.5–1.5)	0.3 (0.1–0.9)	0.8 (0.6–1.2)	0.8 (0.5–1.4)	1.3 (0.7–2.3)
III	1.0 (0.6–1.9)	1.1 (0.6–1.9)	0.8 (0.5–1.1)	1.4 (0.7–2.8)	1.0 (0.7–1.7)	0.6 (0.4–1.1)	0.5 (0.2–1.4)	0.7 (0.5–1.0)	0.5 (0.3–0.9)	1.1 (0.6–1.9)
IV	0.9 (0.4–1.8)	1.1 (0.5–2.1)	0.6 (0.3–0.9)	1.4 (0.6–2.9)	1.1 (0.7–1.8)	0.8 (0.4–1.5)	0.7 (0.2–2.6)	1.0 (0.6–1.6)	0.2 (0.1–0.4)	0.6 (0.3–1.2)
Paternal education[a]										
I (Reference)										
II	0.9 (0.6–1.3)	1.1 (0.8–1.6)	1.1 (0.9–1.4)	1 (0.7–1.4)	1 (0.8–1.7)	0.9 (0.7–1.3)	1.2 (0.6–2.6)	0.9 (0.8–1.2)	0.9 (0.7–1.1)	0.8 (0.7–1.2)
III	1.1 (0.6–1.7)	0.9 (0.5–1.5)	0.9 (0.7–1.4)	0.9 (0.6–1.5)	1.2 (0.9–1.7)	0.9 (0.6–1.4)	1.2 (0.6–3.2)	0.9 (0.6–1.3)	1.1 (0.7–1.6)	0.9 (0.6–1.4)
IV	3.1 (1.3–7.4)	1.3 (0.4–3.9)	1.6 (0.6–4.6)	2.4 (1.1–5.3)	0.6 (0.3–1.4)	0.9 (0.3–2.4)	2.4 (0.5–10.4)	0.8 (0.4–1.7)	2.9 (1.1–8.0)	1.6 (0.5–5.0)

I: No education (0 years); II: Primary and middle school (1–8 years); III: High and higher secondary school (9–12 years); IV: >12 years of schooling.
[a]Educational status.

was associated with lower tobacco use. Having a father who had >12 years of education was associated with higher odds of obesity, abdominal obesity and tobacco use. However, there were no trends across all categories of paternal educational status. Most risk factors were higher in urban than in rural participants independently of SES indicators; exceptions were hypertension and glucose intolerance.

Discussion

This study examined the prevalence of risk factors for CVD in rural and urban men and women living in South India, and their associations with three measures of SES (household possessions score, adult educational status and paternal educational status). We found that cardiovascular risk factors were higher in the urban than in the rural population. All three indicators of SES were positively related to most of the CVD risk factors, including overweight and obesity, dyslipidaemia, hypertension and abnormal glucose tolerance. The exception was tobacco use which showed an inverse relationship with all three indicators of SES. The household possessions score (an indicator of wealth and ability to purchase consumer goods) showed stronger positive associations with CVD risk factors than the other SES indicators, and in a concurrent analysis of all three indicators together (Table 6), it was the only indicator independently associated with risk factors.

The nutrition transition is predicted to lead to changing relationships between SES and CVD risk. The nutrition transition is defined as changes in dietary intake patterns because of the adoption of 'modern' lifestyles due to social and economic development. As a consequence, disease patterns initially shift towards nutrition-related chronic diseases like CVD. In the early stages of such a transition, risk factors tend to be concentrated among the high SES groups and urban dwellers, who have earlier access to these 'modern' lifestyles.[23–25] Experience in high-income settings has shown that as the transition progresses, people of higher SES start to change their behaviour and adopt healthier lifestyles, probably due to multiple factors (greater awareness, greater self-efficacy, better access to healthy diets), leading to a lowering of their risk, while the burden of disease shifts to lower SES groups. A number of studies in India have reported, as in our study, higher levels of risk factors in urban compared with rural populations,[12,26,27] and in higher socio-economic groups.[28,29] Studies conducted in industrialized populations in Chennai (Southern India) revealed a higher prevalence of CVD risk factors compared with the general population.[30] In North India, the prevalence of diabetes and hypertension was found to be positively associated with social class (assessed using a composite score).[28] In a study of women in five cities of India, social class was found to be directly associated with all risk factors for CVD and undernutrition was negatively associated with social class.[29] The prevalence of dyslipidaemia was found to be more common and severe among the middle-income group compared with the low-income group.[31] Our findings are also consistent with a recent study of the socio-economic patterning of CVD risk factors among rural populations selected from four Indian cities (Lucknow, Nagpur, Hyderabad and Bangalore). This reported a higher prevalence of risk factors among higher SES groups (measured using a composite SES score), with the exception of tobacco and alcohol use, which was found to be more common among lower SES groups.[11] We expected evidence of a more advanced transition in the urban sample (a shift towards greater risk in lower socio-economic groups) and the reverse in the rural group. Our results showed, however, similar socio-economic patterning (positive associations between SES and most risk factors) in both the urban and rural populations, suggesting that the nutrition transition is at an early stage in both these Vellore populations.

The nutrition transition might produce a 'disconnect' between associations of risk factors with indices of wealth compared with indices of educational status. Greater material and financial wealth enables the purchase of healthier food and access to better quality health care, but it may also be associated with unhealthy lifestyle choices.[23] Economic development combined with modernization can lead to an increase in the consumption of processed foods, animal fats and a shift to a more sedentary lifestyle.[32] Previous studies have shown that higher wealth and income are associated with diets rich in animal fats, and there is evidence of this in India.[25] Education could counteract these trends.[33] More educated people will tend to have better knowledge about the relationship of lifestyle to health, and may also have greater 'self-efficacy', the concept that they can influence their health by the choices they make. On the other hand, educated people may be more status conscious, and 'more modern' foods and more sedentary living habits may carry status value. In our study, we found that education level, like possessions score, was a positive predictor of most risk factors. After adjusting for material wealth as indicated by the possessions score, there were no significant associations between education level and most risk factors. The index of material wealth was an apparently stronger risk factor than educational status, but the associations were in the same direction. We thus found no evidence that better education was protective in our population. These findings are consistent with a cross-sectional study of risk factors for CVD conducted in highly urban, urban and peri-urban regions of India, which found that SES, as measured by level of education, was positively associated with risk factors for CVD (except for tobacco and alcohol use).[8] In contrast, in another study, an inverse graded

relationship of education with tobacco use, diabetes and hypertension was observed, though this was not true for other CVD risk factors.[34]

The possessions score and education level were positively correlated in our study ($\tau = 0.44$), though not so strongly that they could be considered to be measuring exactly the same thing. It is difficult to say whether the stronger associations observed with possessions score than with educational level indicate a genuine difference in the influence of these two aspects of SES on cardiovascular risk, or whether this is an artefact due, for example, to clumping of the educational data (and thus some loss of statistical power), or to the fact that education is free in government schools in India up to the 12th standard (age 17 years), and thus education may not reflect socio-economic differences as strongly as purchased goods. We attempted to examine this further using risk factors cross-tabulated according to high and low categories of both possessions score and education level (data not shown). However, these tabulations had to retain the male/female and rural/urban stratification, because of the strong effects of gender and place of residence on education level, and, along with the fact that these two socio-economic variables were correlated, the numbers of participants in discordant cells (for example high possessions score but low education level) were too small to see meaningful patterns.

We used paternal education level as an indicator of childhood SES, to determine whether exposure to higher parental education from childhood might have a protective effect on CVD risk factors. However, as with the individual's own education, paternal education tended to have positive (adverse) associations with most risk factors and in a simultaneous model with possessions score, little independent effect.

Tobacco use showed a different socio-economic pattern from the other cardiovascular risk factors. In contrast to overweight/obesity, blood pressure and the biochemical risk factors, higher tobacco use was associated with lower SES (all three indicators). There is strong evidence of social patterning of smoking or tobacco use in developing countries. Our results are consistent with other studies from India that have shown a reversal of the social gradient for tobacco use.[35–37] Although the causes for this are not clear, it could reflect better knowledge about the adverse effects of tobacco[38] and/or greater family, peer and workplace pressure against tobacco use.[39,40] Cultural factors are also important, as indicated by the near zero prevalence of smoking among women.

Our study illustrated the so-called 'double burden' of coexistent under- and overnutrition in both the rural and urban Vellore populations. Alongside a high prevalence of overweight and obesity in our sample, ~40% of the rural and 20% of the urban sample were underweight. This too showed strong socio-economic patterning, with lower levels of underweight among those with higher possessions score and education level. This is consistent with a recent cross-sectional study of a nationally representative sample, which also demonstrated the coexistence of undernutrition with obesity, and showed a positive association between SES and obesity and a negative association of underweight with SES.[41] This is likely to reflect the ability to purchase food, combined with occupational energy expenditure; lower status jobs tend to involve more physical work. Perhaps a surprising finding was that there were substantial levels of underweight even in the most affluent groups, for example 16% of urban men in the top fifth of the possessions score, and 8% of those in the highest education category (Tables 3 and 4).

Our study has a number of limitations. The most important is that it is causally uninformative. Given that SES indicators and CVD risk factors were assessed at the same point in time, it is not possible to comment on the temporal relations between these factors. Further, like many other birth cohort studies there was considerable attrition due to migration and mortality, which could introduce bias. Loss of the families whose mothers delivered in their native villages is likely to have removed the more 'traditional' families. Out-migration of cohort members in later life is likely to have led to the loss of a mixture of individuals—both well-educated and poorly educated and unskilled men and women seeking better opportunities elsewhere. Our study sample was young compared with much of the previous research on CVD risk factors from India[4,8,11,30] which has been conducted on middle-aged adults. We would not expect the socio-economic patterning of CVD risk factors to vary greatly by the participants' age. Results from a recent study in India shows that there is no evidence for interaction between individual standard of living index and women's age on the risk of being obese.[42] Another limitation relates to the merging or collapsing of categories (smoking and tobacco use) due to small numbers in the specific sub-categories, which could have blurred important differences. Finally, we could not calculate NFHS Standard of Living (SLI), which came into widespread use after our study and has come to be accepted as the best composite SES scoring system for urban and rural populations in India.[43] It includes questions on education of the head of the household, family type, house type, number of people per room, sources of drinking water and light, availability of toilet facilities and ownership of house, land, farm animals and household possessions. Although there is considerable overlap with the data we collected, we are not able to calculate the exact SLI score for our cohort. Our study sample comprises a population-based sample of adults living in urban and rural regions of Vellore, Tamilnadu. It is not intended to be nationally representative and estimates of the prevalence of CVD risk

factors are not generalizable to the whole Indian population. However, there is no a priori reason to believe that our estimates of associations between SES indicator and CVD risk factors are very different from studies based on nationally representative samples. Given these limitations, our study has the advantage of a population based birth cohort which is rare in developing countries like India. It also represents both urban and rural areas, with detailed measurements of various risk factors for CVD and social indicators.

Conclusion

Our study adds to scant existing information on social patterning of CVD risk factors in urban and rural residents from a developing country. We found that obesity and underweight coexist, but remain socioeconomically segregated, suggesting that the population is still at an early stage of nutrition transition. Our findings clearly indicate that most risk factors for CVD are associated with greater material wealth in both rural and urban settings. We conclude that public health strategies to control and prevent CVD should consider the rural–urban differentials and the presence of socio-economic disparities.

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KEY MESSAGES

- Among the SES indicators, possessions score was most strongly associated with CVD risk factors in this Indian population.
- People with higher possessions scores tended to have higher levels of CVD risk factors, with an exception of tobacco use.
- The associations between possessions score and CVD risk factors were consistent in both urban and rural populations.

References

[1] Reddy KS, Yusuf S. Emerging epidemic of cardiovascular disease in developing countries. *Circulation* 1998;**97:** 596–601.
[2] Sobal J, Stunkard AJ. Socioeconomic status and obesity: a review of the literature. *Psychol Bull* 1989;**105:**260–75.
[3] Langenberg C, Hardy R, Kuh D, Brunner E, Wadsworth M. Central and total obesity in middle aged men and women in relation to lifetime socioeconomic status: evidence from a national birth cohort. *J Epidemiol Commun Health* 2003;**57:**816–22.
[4] Gupta R, Gupta VP, Ahluwalia NS. Educational status, coronary heart disease, and coronary risk factor prevalence in a rural population of India. *BMJ* 1994;**309:** 1332–36.
[5] Yu Z, Nissinen A, Vartiainen E, Hu G, Tian H, Guo Z. Socio-economic status and serum lipids: a cross-sectional study in a Chinese urban population. *J Clin Epidemiol* 2002;**55:**143–49.
[6] Lawlor DA, Batty GD, Morton SM, Clark H, Macintyre S, Leon DA. Childhood socioeconomic position, educational attainment, and adult cardiovascular risk factors: the Aberdeen children of the 1950s cohort study. *Am J Public Health.* 2005;**95:**1245–51.
[7] Batty GD, Leon DA. Socio-economic position and coronary heart disease risk factors in children and young people. *Eur J Public Health.* 2002;**12:**263–72.
[8] Reddy KS, Prabhakaran D, Jeemon P *et al*. Educational status and cardiovascular risk profile in Indians. *Proc Natl Acad Sci U S A* 2007;**104:**16263–68.
[9] Ebrahim S, Smeeth L. Non-communicable diseases in low and middle-income countries: a priority or a distraction? *Int J Epidemiol* 2005;**34:**961–66.
[10] Kaur P, Rao TV, Sankarasubbaiyan S *et al*. Prevalence and distribution of cardiovascular risk factors in an urban industrial population in south India: a cross-sectional study. *J Assoc Physicians India* 2007;**55:**771–76.
[11] Kinra S, Bowen LJ, Lyngdoh T *et al*. Sociodemographic patterning of non-communicable disease risk factors in rural India: a cross sectional study. *BMJ* 2010;**341:** c4974–74.
[12] Das M, Pal S, Ghosh A. Rural urban differences of cardiovascular disease risk factors in adult Asian Indians. *Am J Hum Biol* 2008;**20:**440–45.
[13] Monteiro CA, Conde WL, Popkin BM. Independent effects of income and education on the risk of obesity in the Brazilian adult population. *J Nutr* 2001;**131:**881S.
[14] Antonisamy B, Raghupathy P, Christopher S *et al*. Cohort profile: The 1969-73 Vellore birth cohort study in South India. *Int J Epidemiol* 2009;**38:**663–69.
[15] Raghupathy P, Antonisamy B, Geethanjali FS *et al*. Glucose tolerance, insulin resistance and insulin secretion in young south Indian adults: relationships to parental size, neonatal size and childhood body mass index. *Diabetes Res Clin Pract* 2010;**87:**283–92.

[16] Vyas S, Kumaranayake L. Constructing socio-economic status indices: how to use principal components analysis. *Health Policy Plann* 2006;**21:**459–68.
[17] Yarows SA, Brook RD. Measurement variation among 12 electronic home blood pressure monitors. *Am J Hypertens* 2000;**13:**276–82.
[18] Anon. *WHO/IASO/IOTF The Asia Pacific perspective: redefining obesity and its treatment*. Melbourne, Australia: Health Communications Australia Pty LTD, 2000; 18.
[19] Ramachandran A, Snehalatha C, Satyavani K, Sivasankari S, Vijay V. Metabolic syndrome in urban Asian Indian adults—a population study using modified ATP III criteria. *Diabetes Res Clin Pract* 2003;**60:**199–204.
[20] Chobanian AV, Bakris GL, Black HR *et al*. The seventh report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure: The JNC 7 Report. *JAMA* 2003;**289:**2560–71.
[21] Expert Panel on Detection E. Executive summary of the third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III). *JAMA* 2001;**285:**2486–97.
[22] Alberti KG, Zimmet PZ. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus provisional report of a WHO consultation. *Diabetic Med* 1998; **15:**539–53.
[23] Popkin BM. The nutrition transition and its health implications in lower-income countries. *Public Health Nutr* 1998;**1:**5–21.
[24] Popkin BM. The shift in stages of the nutrition transition in the developing world differs from past experiences! *Public Health Nutr* 2002;**5:**205–14.
[25] Shetty PS. Nutrition transition in India. *Public Health Nutr* 2002;**5:**175–82.
[26] Chadha SL, Gopinath N, Shekhawat S. Urban-rural differences in the prevalence of coronary heart disease and its risk factors in Delhi. *Bull World Health Organ* 1997;**75:** 31–38.
[27] Kumar R, Singh MC, Ahlawat SK *et al*. Urbanization and coronary heart disease: a study of urban-rural differences in northern India. *Indian Heart J* 2006;**58:**126.
[28] Singh RB, Singh V, Kulshrestha SK *et al*. Social class and all-cause mortality in an urban population of North India. *Acta Cardiol* 2005;**60:**611–17.
[29] Singh RB, Beegom R, Mehta AS *et al*. Social class, coronary risk factors and undernutrition, a double burden of diseases, in women during transition, in five Indian cities. *Int J Cardiol* 1999;**69:**139–47.
[30] Mohan V, Deepa M, Farooq S, Prabhakaran D, Reddy KS. Surveillance for risk factors of cardiovascular disease among an industrial population in southern India. *Natl Med J India* 2008;**21:**8–13.
[31] Pradeepa R, Deepa R, Rani SS, Premalatha G, Saroja R, Mohan V. Socioeconomic status and dyslipidaemia in a South Indian population: the Chennai Urban Population Study (CUPS 11). *Natl Med J India* 2003;**16:**73–78.
[32] Popkin BM, Horton S, Kim S, Mahal A, Shuigao J. Trends in diet, nutritional status, and diet-related noncommunicable diseases in China and India: the economic costs of the nutrition transition. *Nutr Rev* 2001;**59:**379–90.
[33] Rosengren A, Subramanian S, Islam S *et al*. Education and risk for acute myocardial infarction in 52 high, middle and low-income countries: INTERHEART case-control study. *Heart* 2009;**95:**2014.
[34] Gupta R, Gupta VP, Sarna M, Prakash H, Rastogi S, Gupta KD. Serial epidemiological surveys in an urban Indian population demonstrate increasing coronary risk factors among the lower socioeconomic strata. *J Assoc Physicians India* 2003;**51:**470–77.
[35] Gupta R. Smoking, educational status & health inequity in India. *Indian J Med Res* 2006;**124:**15.
[36] Subramanian SV, Nandy S, Kelly M, Gordon D, Davey Smith G. Patterns and distribution of tobacco consumption in India: cross sectional multilevel evidence from the 1998-9 national family health survey. *BMJ* 2004;**328:**801.
[37] Sorensen G, Gupta PC, Pednekar MS. Social disparities in tobacco use in Mumbai, India: the roles of occupation, education, and gender. *Am J Public Health* 2005;**95:** 1003–08.
[38] Thankappan K, Sharma I, Sarma P. Awareness, attitude and perceived barriers regarding implementation of the cigarettes and other tobacco products act in Assam, India. *Indian J Cancer* 2010;**47:**63.
[39] Viswanath K, Ackerson LK, Sorensen G, Gupta PC. Movies and TV influence tobacco use in India: findings from a national survey. *PLoS ONE* 2010;**5:**e11365.
[40] Mishra GA, Majmudar PV, Gupta SD, Rane PS, Uplap PA, Shastri SS. Workplace tobacco cessation program in India: a success story. *Indian J Occup Environ Med* 2009; **13:**146–53.
[41] Subramanian SV, Perkins JM, Khan KT. Do burdens of underweight and overweight coexist among lower socioeconomic groups in India? *Am J Clin Nutr* 2009;**90:** 369–76.
[42] Subramanian SV, Davey Smith G. Patterns, distribution, and determinants of under- and overnutrition: a population-based study of women in India. *Am J Clin Nutr* 2006; **84:**633–40.
[43] Anon. *International Institute for Population Sciences (IIPS) & ORC MACRO*. National Family Health Survey (NFHS-3), 2005-06: Mumbai, India, 2007, 1.