Urban–rural differences in prevalence of coronary heart disease and its risk factors

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We surveyed 14,866 subjects (6614 men, 8252 women) between the ages of 25 and 65 years in Delhi and the neighbouring rural area to determine the prevalence of coronary heart disease (CHD) and select coronary risk factors. A five-fold higher prevalence of CHD was observed in the urban survey sample as compared to the rural population (31.9/1000 vs 5.9/1000 respectively). Hypertension, diabetes mellitus and obesity were more prevalent in the urban population. Paradoxically, low serum HDL-cholesterol and high serum triglycerides were more common in the rural sample. These findings have important implications for CHD prevention.

PUBLIC health planning in most developing countries has focused mainly on problems related to communicable diseases, which in the past have been responsible for high morbidity and mortality. The situation is different in developed countries, e.g. the United States, where coronary heart disease (CHD) is the leading cause of death[1]. However with changing lifestyle in developing countries like India, particularly in urban areas, chronic and degenerative diseases (e.g. CHD) are making an increasingly important contribution to mortality statistics of such countries[2].

Over the last three decades, substantial progress has been made in identifying the risk factors for CHD. Some of the preventive measures such as cessation of smoking, management of hypertension both by pharmacological and non-pharmacological means, control of diabetes mellitus, reduction in intake of dietary saturated fats and early diagnosis and management of hypercholesterolaemia have resulted in a significant decline in CHD mortality and morbidity in the industrialized world. Nevertheless, CHD continues to be the leading cause of death in many developed countries.

CHD is considered an important public health problem not only in the developed countries but also in developing countries like India. The WHO Expert committees on cardiovascular diseases and hypertension[3] recommended epidemiological surveys in as many countries as possible to analyse the coronary risk factors and to establish prevalence of the disease in different countries. During the past three decades, a few epidemiological studies have been conducted in India for evaluating the prevalence of CHD. A majority of these studies have been confined to hospital, select groups of population, or the sample size was too small, making it difficult to draw any valid conclusions about the prevalence of CHD and its risk factors.

Materials and methods

A community-based epidemiological study was conducted in adults (25–64 years) in the urban population of Delhi and rural areas about 50 km away from Delhi for prevalence of CHD and its risk factors. A cluster sampling methodology, using random house-to-house survey, was employed. The methods and the criteria for diagnosis of CHD have been reported earlier[4,5]. The epidemiological study was supported by ECG examination and analysis of fasting blood samples (for lipids) from clinically-detected CHD cases and asymptomatic adults free of clinically manifest disease in every 2nd and 5th household, respectively.

The present communication compares CHD risk factors in urban and rural populations. The risk factors studied are family history, socio-economic status, obesity, smoking, physical activity, hypertension, diabetes mellitus and hyperlipidaemia. In addition to these established risk factors, we included in our study dietary factors, e.g., total and saturated fat intake, dietary cholesterol, sodium intake and alcohol consumption.

The National Cholesterol Education Program Expert Panel on detection, evaluation and treatment of high blood cholesterol in USA[6] has defined 'desirable', 'borderline', and 'high' levels of blood lipids as; less than the 50th percentile, between the 50th–75th percentiles and above the 75th percentile values, respectively. For our study the cut off levels of blood lipids were based on the 50th percentile values which were obtained from our study population (Table 1). Hypertension was identified as a systolic blood pressure (SBP) of more than 160 mm Hg and/or a diastolic blood pressure (DBP) of more than 90 mm Hg or a history of current consumption of antihypertensive drugs. Isolated systolic hypertension was defined as SBP > 160 mm Hg and DBP < 90 mm Hg. Blood pressure was measured in a sitting position by the medical officers. The average of two measurements obtained at the interval of five

Table 1. 50th percentile values obtained from the study population

Blood lipids (mg/dl)	Urban	Rural
Total cholesterol	190	170
LDL-cholesterol	110	90
HDL-cholesterol	53	52
Triglycerides	120	138

minutes was used for this analysis. Phase V of Korotkoff sounds was taken for determination of DBP.

Body mass index (BMI), i.e. weight in kilograms divided by square of height in meters, was used as an indicator of obesity. The weight was measured by standard portable weighing machines to the nearest 0.5 kg with the subject wearing light clothing. The weighing machines were calibrated every week with a standard weight and necessary adjustments were made if required. The 75th percentile value of BMI in our study population was 25.4. Hence the empiric value of 25 was used as a cut-off point for obesity. Waist–hip ratio (WHR) was assessed by measuring the circumference of the waist at the umbilicus and dividing it by the circumference of hip at the maximum point of protrusion following the protocol of recommendations of Lohman *et al.*[7] Based on the 75th percentile value of WHR in the study population, 0.88 was taken as a cut-off point for this ratio to determine central obesity.

Diabetes mellitus was diagnosed based on clinical history supported by documentary evidence of antidiabetic therapy. For physical activity, the subjects were divided into three subgroups (light, medium and heavy) depending on the nature of their activities. Smoking status was evaluated by a self-report of cigarettes/bidis smoked or tobacco chewed. Persons who smoked 10 cigarettes/bidis or more, or chewed tobacco at least twice a day were categorized as smokers. Casual smokers were not included in the study.

A subsample for dietary survey was randomly selected from the same urban and rural population which was covered by initial epidemiological survey for prevalance of CHD and hypertension. A detailed urban and rural dietary profile was assessed by using a combination of 24 hours dietary recall and the weightment methods. The nutrient intake calculations for each subject were determined by reference to the *Manual of Nutritive Values of Indian Foods*[8]. A detailed dietary profile of the urban and rural study population has been reported earlier[9].

Statistical analysis. Data were pooled and computerized. Intergroup comparisons for risk factors were performed using analysis of variance (ANOVA) for numerical variables. The data were further analysed using t test and χ^2 test.

Results

In our community-based epidemiological study, the prevalence rate for CHD on clinical basis was significantly higher in the urban population (31.9/1000) than in the rural area (5.9/1000) (refs 4, 5). A similar trend in prevalence rate of asymptomatic CHD (diagnosed by ECG) was observed, 64.8/1000 adults and 21.2/1000 adults in the urban and rural populations, respectively. Prevalence of selected CHD risk factors in the two populations is given in Table 2. Prevalence of hypertension, diabetes mellitus, obesity and positive family history was significantly higher in urban men and women than in the rural population. Smoking was more prevalent among both rural men and women.

Mean values ± SD of blood lipids by age and sex are shown in Table 3. In urban men and women, the prevalence of high levels of total cholesterol and low density lipoprotein (LDL) cholesterol were higher than in rural men and women. High density lipoprotein cholesterol (HDL-C) level was lower in rural men and women in all age groups compared to that in urban men and women. Except in the age group of 55–64 years, triglyceride levels were higher in rural men than in urban men. In rural women triglycerides were higher in the age groups of 25–34 and 35–54 years than in urban women.

Percentage of adults with total cholesterol > 190 mm/dl in urban and rural men was 46.0 and 23.0 respectively. The corresponding percentages for urban and rural women were 50.1 and 23.9. HDL-cholesterol < 35 md/dl was found in 2.2% urban and 8.0% in rural men compared to 1.6% and 3.5% in urban and rural women respectively (Table 4).

Asymptomatic individuals with abnormal ECG patterns, e.g. 'Q wave' (Minnesota code 1-1.1 through 1-1.7 or 1-2.1. through 1-2.7) or 'ST-T' changes

Table 2. Prevalence of selected risk factors for coronary heart disease in Delhi and rural neighbourhood

Risk factors	Urban		Rural		Urban vs rural
	Men (*N*-5998)	Women (*N*-7136)	Men (*N*-616)	Women (*N*-1116)	'*P*' value
Hypertension	644 (10.8)	878 (12.3)	25 (4.1)	37 (3.3)	< 0.0001
Diabetes	97 (1.6)	117 (1.6)	3 (0.5)	–	<0.0001
BMI > 25.0	1222 (20.7)	2310 (32.6)	60 (9.7)	109 (9.8)	< 0.0001
Smoking	1755 (29.3)	128 (1.8)	492 (79.9)	324 (29.0)	< 0.0001
Family history of coronary heart disease	1296 (21.6)	1463 (20.5)	23 (3.7)	19 (1.7)	< 0.0001

Figures in parentheses indicate percentages. Height and weight measurements in urban population are available for 5898 men and 7081 women.

Table 3. Blood lipids by age and sex in urban and rural populations in and around Delhi (mean ± S.D.)

		Urban		Rural	
Blood lipids (mg/dl)	Age (yrs)	Men	Women	Men	Women
Total cholesterol	25–34	198 ± 39 (225)	194 ± 37 (309)	175 ± 36* (31)	171 ± 20** (93)
	35–44	202 ± 39 (158)	203 ± 40 (257)	194 ± 35 (NS) (25)	185 ± 31** (56)
	45–54	210 ± 43 (158)	215 ± 47 (282)	179 ± 22* (17)	186 ± 38** (47)
	55–64	211 ± 46 (140)	217 ± 42 (236)	170 ± 18** (27)	175 ± 20** (63)
Low density lipoprotein cholesterol	25–34	117 ± 38 (173)	109 ± 32 (235)	97 ± 36* (31)	96 ± 21** (93)
	35–44	119 ± 37 (135)	114 ± 37 (193)	116 ± 40 NS (25)	103 ± 30@ (56)
	45–54	121 ± 41 (121)	120 ± 44 (212)	99 ± 21@ (17)	104 ± 39@ (47)
	55–64	115 ± 42 (110)	121 ± 43 (166)	90 ± 18* (27)	94 ± 17** (63)
High density lipoprotein cholesterol	25–34	53 ± 12 (173)	58 ± 12 (235)	52 ± 12 NS (31)	51 ± 9** (93)
	35–44	54 ± 13 (135)	57 ± 13 (193)	50 ± 12 NS (25)	54 ± 9 NS (56)
	45–54	55 ± 12 (121)	57 ± 14 (212)	49 ± 11 @ (17)	52 ± 10 @ (47)
	55–64	58 ± 14 (110)	58 ± 15 (166)	50 ± 11* (27)	51 ± 10** (63)
Triglycerides	25–34	123 ± 28 (225)	108 ± 47 (309)	128 ± 29 (31)	120 ± 22 @ (93)
	35–44	133 ± 57 (158)	126 ± 51 (257)	142 ± 26 NS (25)	138 ± 24 NS (56)
	45–54	150 ± 57 (158)	154 ± 52 (282)	155 ± 34 NS (17)	151 ± 29 NS (47)
	55–64	168 ± 52 (140)	168 ± 53 (236)	152 ± 28 (27)	151 ± 26 @ (63)

Figures in parentheses indicate number of blood samples analysed.
P values – urban vs rural men, urban vs rural women.
$*P < 0.01$; $**P < 0.001$; @ $P < 0.5$; NS, not significant.

Table 4. Proportion of subjects with abnormal blood lipids in Delhi and rural neighbourhood

	Urban			Rural		
Lipids (mg/dl)	Men	Women	*P*-value	Men	Women	*P*-value
Total cholesterol > 190	300/681 (44.0)	543/1084 (50.1)	< 0.001	23/100 (23.0)	62/259 (23.9)	< 0.001
LDL-C > 110	264/539 (49.0)	377/806 (46.8)	< 0.001	29/100 (29.0)	70/259 (27.0)	< 0.001
Triglycerides > 120	340/681 (50.0)	369/806 (45.8)	< 0.001	75/100 (75.0)	174/259 (67.2)	< 0.001
HDL-C < 35	12/539 (2.2)	17/1084 (1.6)	< 0.001	8/100 (8.0)	9/259 (3.5)	< 0.05

Figures in parentheses indicate percentages.
LDL-C, low density lipoprotein cholesterol.
HDL-C, high density lipoprotein cholesterol.

(Minnesota code 4-1-1, 4-1-2, 5-1 and 5-2) are more prone to clinically manifest CHD. Such an abnormal ECG is therefore considered a risk factor for CHD. In asymptomatic urban adults, 1.7% men and 1.2% women showed 'Q wave' compared to 0.3% men and 0.4% women in rural population. The same trend was observed for electrocardiographic 'ST-T' changes in urban and rural asymptomatic adult population (Table 5). However, women showed higher prevalence of electrocardiographic 'ST-T' changes than men in both the populations.

Table 5. Abnormal ECG as a risk factor for coronary heart disease in asymptomatic adults

	Q wave		ST-T changes	
Sex	Urban	Rural	Urban	Rural
Males	45/2589 (1.7)	1/319* (0.3)	101/2589 (3.9)	1/319** (0.3)
Females	35/3032 (1.1)	3/728* (0.4)	195/3032 (6.4)	17/728** (2.3)
Total	80/5621 (1.4)	4/1047* (0.4)	296/5621 (5.3)	18/1047** (1.7)

Figures in parentheses indicate percentages.
*$P < 0.05$.
**$P < 0.01$.

Dietary risk factors for CHD are tabulated (Table 6). Average total caloric intake was higher in rural men and women than in the urban population. There were no appreciable differences in total dietary fat intake in the two populations, but saturated fat intake was higher in both rural men and women than the urban subjects. For this study, we calculated the percentage of subjects who consumed more than 15% calories as saturated fat and added this as another modifiable risk factor. The 15% cut-off point was selected as an indicator of high saturated fat intake. Surprisingly, 28.7% rural men and 27.7% women consumed more than 15% calories as saturated fats compared to urban men (8.2%) and women (8.5%). This may be due to a greater consumption of whole milk and milk products by the rural subjects in the area where the dietary survey was conducted.

Dietary cholesterol intake showed marginal differences in the two populations. The sodium intake was greater in both sexes in the urban population than rural population, which may be due to excessive intake of 'ready-to-eat' foods by the urban subjects. Such 'fast foods' are usually rich in sodium. The average daily alcohol consumption by urban men was 12.7 ml compared to 2.4 ml by rural men. The average daily alcohol consumption by urban women was 0.04 ml while none of the rural women consumed alcohol. The dietary survey lays bare the coronary paradox.

Discussion

The prevalence of CHD risk factors, e.g. hypertension, obesity, diabetes, family history and abnormal ECG (Q wave and ST-T changes) was significantly higher in urban than rural population. Smoking on the other hand, was more common among the rural men and women. Rural men and women work in agricultural fields and their work involves heavy physical activity. Most of the urban men and women have sedentary habits. Prevalence of hypertension, obesity and diabetes mellitus was higher in both sexes in the urban population than in rural areas. These differences in risk factors may be explained partly by the differences in lifestyles.

Gupta and Malhotra[10] conducted an epidemiological survey of CHD in almost similar ethnic groups. Prevalence of CHD was found to be almost two-and-a-half times more common in both sexes in urban than in the rural areas. A lesser degree of physical activity, a body weight on the higher side of normal and a higher prevalence of hypertension and diabetes mellitus in the urban population are some of the important risk factors responsible for such differences. We do not know of any other comparative epidemiological study for prevalence of CHD and its risk factors in urban and rural areas in our country.

Urban–rural differences were found in blood lipids, particularly the high-risk total cholesterol and LDL-cholesterol. Urban men and women have these blood lipids more than the 'desirable' levels. Low HDL-cholesterol is a risk factor for CHD and is usually associated with increased triglyceride level[11]. A higher prevalence of low HDL-cholesterol (< 35 mg/dl) and elevated triglyceride level (> 120 mg/dl) was found in the rural than urban population. This is contrary to the findings of Hannia Campos *et al.*[12] who reported higher prevalence of low HDL-cholesterol (< 35 mg/dl) in urban than rural areas of Puriscol, Costa Rica and Central America. These differences may be due to different dietary patterns and living styles in the two countries.

Populations which consume low fat and high-carbohydrate diets have low HDL cholesterol and high triglycerides levels[13–15]. In our study, rural–urban differences are marginal in the intake of total fat. Intake of saturated fat and carbohydrates as well as blood levels of triglycerides are significantly higher in rural than in the urban areas. These observations indicate that though low HDL-cholesterol levels may not enhance CHD risk, low HDL-cholesterol levels represent a marker for increased levels of other atherogenic lipoproteins, particularly LDL-cholesterol[16]. Alternatively the important determinant of atherosclerosis is the ratio of LDL–HDL-cholesterol. Low HDL-cholesterol levels are not a CHD risk factor in the presence of low LDL-cholesterol levels as observed in rural areas in our study.

There is an increasing awareness and interest in asymptomatic CHD. Postmortem angiographic studies indicate that severe CHD often occurs without symptoms. In the Framingham study, more than 25% of the patients with CHD were identified on the basis of routine periodic electrocardiographs[17]. ECG evaluation of 5021 asymptomatic persons, who formed the controls during the initial phase of the epidemiological study[4], revealed 376 abnormal ECG tracings that were consistent with a diagnosis of CHD. In the 3 year follow-up study of CHD, 297 of these individuals were revaluated[18]. Among them, 14 persons developed clinically manifest CHD. ECG evidence of left ventricular hypertrophy (LVH) is another important risk factor for CHD. In our

Table 6. Dietary risk factors for coronary heart disease in urban and rural populations in and around Delhi

Dietary factors	Urban			Rural		
	Men (*N*–400)	Women (*N*–506)	Total (*N*–906)	Men (*N*–87)	Women (*N*–188)	Total (*N*–275)
Total calories (K/cal)	1924 ± 524	1604 ± 456	1745 ± 512	2249 ± 636	1754 ± 533	1910 ± 612
Total fats (g)	61.4 ± 28.0	50.4 ± 23.2	55.3 ± 26.0	63.1 ± 37.5	48.1 ± 25.6	52.9 ± 30.7
SFA (g)	16.7 ± 13.0	13.6 ± 10.8	15.0 ± 11.9	34.9 ± 26.0	26.3 ± 13.7	29.0 ± 19.9
Cholesterol (mg)	95 ± 120	68 ± 93	80 ± 107	102 ± 90	71 ± 52	81 ± 68
PUFA linolenic acid (N–3) (g)	1.8 ± 1.3	1.4 ± 1.3	1.5 ± 1.3	0.9 ± 0.7	0.7 ± ± 0.5	0.8 ± 0.6
Linoleic acid (N–6) (g)	7.3 ± 5.5	6.0 ± 4.4	6.7 ± 5.0	5.5 ± 2.2	4.3 ± 1.9	4.7 ± 2.0
Sodium (mg)	2101 ± 1225	1799 ± 1002	1932 ± 1116	1569 ± 919	1309 ± 972	1391 ± 962

SFA, saturated fatty acids.
PUFA, polyunsaturated fatty acids.

follow-up study[18], out of the 4151 adults who were reexamined, 82 individuals had ECG evidence of LVH at the time of the initial survey. A total of 18 (22%) of these 82 individuals had CHD on reevaluation. This observation is consistent with the findings of the Framingham study[19].

It is important to consider the role of air pollution in the pathogenesis of CHD in the urban population group. According to available information, 1280 tonnes of pollutants are being emitted by vehicles every day in Delhi. It seems reasonable to speculate that at least a part of striking differences in prevalence of CHD in urban Delhi from its rural environs (about 50 km outside city) may be attributable to air pollution. The toxic compounds involved in air pollution, e.g. oxides of nitrogen, sulphur dioxide and suspended particles are powerful pro-oxidants which enhance the oxidation of lipoproteins. Oxidized lipoproteins, particularly LDL-C, are powerful inducers of atherosclerosis.

Other studies[20,21] have confirmed that risk factors such as obesity, hypertension, diabetes mellitus, increased intake of energy-rich foods and saturated fats, relatively sedentary occupation, increased stress, and high blood levels of LDL-cholesterol are more prevalent in urban than in rural areas. Our findings are similar except that the average daily saturated fat intake was found to be higher in rural than in the urban area. The trends are disturbing because it is estimated that more than 40% population in developing countries like India will be living in urban areas by 2000 AD with CHD risks similar to the industrialized/developed countries[22]. We do not have reliable mortality data in our country. It is presumed that mortality rates for CHD will definitely increase, particularly in urban areas with constantly increasing prevalence of risk factors. An increase in the prevalence of CHD risk factors is associated with an increased risk of CHD mortality[6].

One conclusion from the present study is that prevalence of CHD and its urban–rural differences cannot be related to any particular risk factor. It is necessary, therefore, to look beyond the conventional explanations for other factors to account for the striking urban–rural differences in CHD prevalence. The deleterious pro-oxidant burden imposed by air pollutants may be an important contributory factor for higher CHD prevalence in urban areas.

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