Urbanisation and Coronary Heart Disease Risk Factors in South Asian Children

R. Hakeem (Department of Food and Nutrition. RLAK Government College of Home Economics, Karach.)
J. Thomas (Department of Nutrition and Dietetics, Kings College.)
S. H. Badruddin (London, U.K., Department of Medicine/Community Health Sciences. The Aga Khan University . Karachi.)

Abstract

Background: Coronary Heart Disease (CHD) and other Non Communicable Diseases (NCDs) are increasing globally. Comparison of various sections of the South Asian populations living at different levels of urbanization can help in understanding the role of demographic transition in the increased prevalence of these diseases in urbanized populations.
Objective: To compare the prevalence of certain CHD risk factors in 10-12 year old school children living at different levels of urbanization.
Method: Differences in height, Body Niass index (BMI), Waist Hip Ratio (WHR), Fasting Blood Glucose (FBG) and Total Blood Cholesterol (TBC) were studied.
Subjects: Anthropometric and biochemical nieasurements of six groups of 10-12 year old children, representing various urbanization categories, were studied. Three groups of children were recruited front Punjab, Pakistan: rural, middle income urban and high income urban and they were assigned urbanization rank (UR) 1, 2 and 3. Another three groups of children were recruited from Slough, UK: British Pakistani, British Indian, and British Caucasian and they were assigned urbanization rank 4, 5 and 6 respectively.
Results: Proportion of children having high CHD risk increased with urbanization rank. Increase in BMI and TBC with urbanization status was steadier than the increase in FBG and WHR. Stunting which have been found to have a positive association with obesity and increased risk of CHD was higher among the less urbanized groups. BMI and TBC of the urbanized South Asian groups were lower, but FBG was higher than the British Caucasian, who served as controls.
Conclusion: These findings support the hypothesis that high CHD death rate among South Asians in UK may have its origin in the genetic predisposition to diabetes but are not likely to he solely due to this factor. The environmental factors like under nourishment in early life, adoption of urbanized life style or a combination of both could be the major determinants of CHD morbidity and mortality (JPMA 51:22; 2001).

Introduction

South Asians who migrated to more urbanized countries demonstrated a much higher CHD morbidity and mortality than the native inhabitants of those countries[1,2]. Comparison of migrants (South Asians) and natives (Caucasians) for classic risk factors like fat intake, obesity, blood lipids etc did not explain the difference in their CHD risk[1,3]. In 1985 Mckeigue suggested that nutritional transition, as the result of demographic shift could be responsible for high CHD risk among South Asians in UK[4]. Later Barker et al presented the theory of genetic programming that states that maternal and fetal under nutrition modify early fetal development and these adaptation may permanently alter adult metabolism in a way which is beneficial to survival under continued conditions of malnutrition but detrimental when

nutrition is abundant[5-12]. Higher prevalence of CHD in adults who were poor during childhood had been noted earlier also[13,15]. In a longitudinal study the highest death rates from CHD occurred in boys who were thin at birth but whose weight caught up so that they had an average or above average body mass from the age of seven years[16]. Thus poor nutrition in early life followed by over nutrition in later life seems to indicate a high CHD risk.
Urbanization is now increasing rapidly in South Asia and rates of maternal and fetal malnutrition as indicated by high maternal and infant mortality and low birth weights have been low for several decades[17,18]. Incidence of Coronary Heart Disease (CHD) is increasing worldwide and the rate of increase is expected to be higher in the urban areas of the developing world, including South Asia, in the coming decades[16,19,20]. Preventive measures in children and young adults are needed now, to check the spread of CUD epidemic in future.
In terms of prevention, assessment of CHD risk of children is very important. Seeds for CHD are sown in early life and differences in risk status are detectable during childhood and adolescence when behavior modifications are feasible. Risk status of children has been studied in several countries[21-28] and found to correspond with the risk factors found in respective adult population[26,29]. Risk factors track from childhood to adulthood[30-33] and are found to cluster[19,34] in some children. Positive results from early interventions have already been noticed[26,29,35,36] Comparing the risk status of more and less urbanized South Asian children can provide important clues for planning proper preventive strategies.

Methods

In order to study the association between urbanization and CHD risk, relative CF-ID risk status of six groups of l0-l2year-old school children, representing various urbanization categories was compared. Fasting blood glucose, total blood cholesterol, waist-hip ratio, body mass index and body height were used as risk indicators in this study. Anthropometric measurements were taken from a total of' 588 children and 457 children participated in blood tests. Group wise distribution of subjects is presented in Table 1.

Table 1. Sample size for anthropometric measurements and blood tests.

Measurement		Group						Total
		UR1 (RrP)	UR2 (MIP)	UR3 (HUP)	UR4 (BrP)	UR5 (BrI)	UR6 (BrC)	
Anthropometry								
	Girls	55	64	76	65	41	19	320
	Boys	64	67	39	51	33	14	268
	Total	119	131	115	116	74	33	588
Blood Test								
	Girls	46	49	45	54	35	17	246
	Boys	53	48	32	42	23	13	211
	Total	99	97	77	96	58	30	457

Subjects

Three groups of children were recruited from Ptinjab, Pakistan: rural (RrP), middle-income urban (MUP) and high-income urban Pakistani (HUP); and they were assigned urbanization rank (UR 1, 2 and 3). Another three groups were recruited from Slough, UK: British Pakistani (BrP), British Indian (Bri) and British Caucasian (BrC) and these were assigned urbanization rank 4, 5 and 6 respectively. Assessment of dietary habits and exposure to Western culture confirmed that ranking of Indian and Pakistanis in Britain was correct[37].

The recruitment of schools was purposive. The basic selection criterion was representation of particular demographic status. In UK, Slough (a city adjacent to London) was selected for the study because of the high concentration of South Asians. Out of the total of seven middle schools in Slough, the three schools that were willing to participate concurrently were recruited. In Pakistan the subjects were recruited from the province of Punjab because the majority (more than 90%) of the Pakistani immigrants in Slough come from that area. Six urban schools (from Lahore) and one rural school (from Kala-Shah-Kaku) were recruited to represent middle-income and high-income urban and rural groups. Initially the principals of several schools fulfilling the criterion of selection were contacted and the ones willing to participate at the same time were recruited. Information about rural or urban status of the area and general financial status of the students of particular schools was obtained from the association of private schools (Punjab). All the ten to twelve year old students studying in year 6 and 7 within each school were invited to take part in the study. Seventy-six to ninety-four percent of the eligible children from various schools participated in the study.

Anthropometric measurements

Height was measured with a portable stadiometer following standard procedure[38]. SOEHNLE digital personal weighing scale was used for weighing. All the subjects were weighed without shoes, in single layer of indoor clothing. For waist and hip measurements, subjects were asked to stand straight with feet together and head up. Waist circumference was measured by holding the non-stretchable measuring tape snugly around the waist at the midpoint between the bottom rib and tip of hipbone. Hip circumference was measured at the fullest part of the hips.

Blood test

Prior to testing blood, the fasting status confirmation sheet, signed by parents was received and subjects were also asked whether they had eaten or drank anything that morning. If they indicated eating or drinking anything except water they were excluded from the test. The Autoclix tool by Boehringer Mannheim was used for the finger skin prick. Children were asked to warm up their hands by exercising them so that blood flow was normal. The first finger of the left hand was selected for pricking, unless the child wanted the other hand or another finger. Accutrend GC meter was used for analyzing blood for fasting glucose and total cholesterol. This small portable instrument analyses the blood for glucose and cholesterol by using disposable strips and is found to be accurate and precise[39,40].

Results

Mean values for TBC and BMI increased with urbanization rank irrespective of genetic origin (Figure 1 and 2).

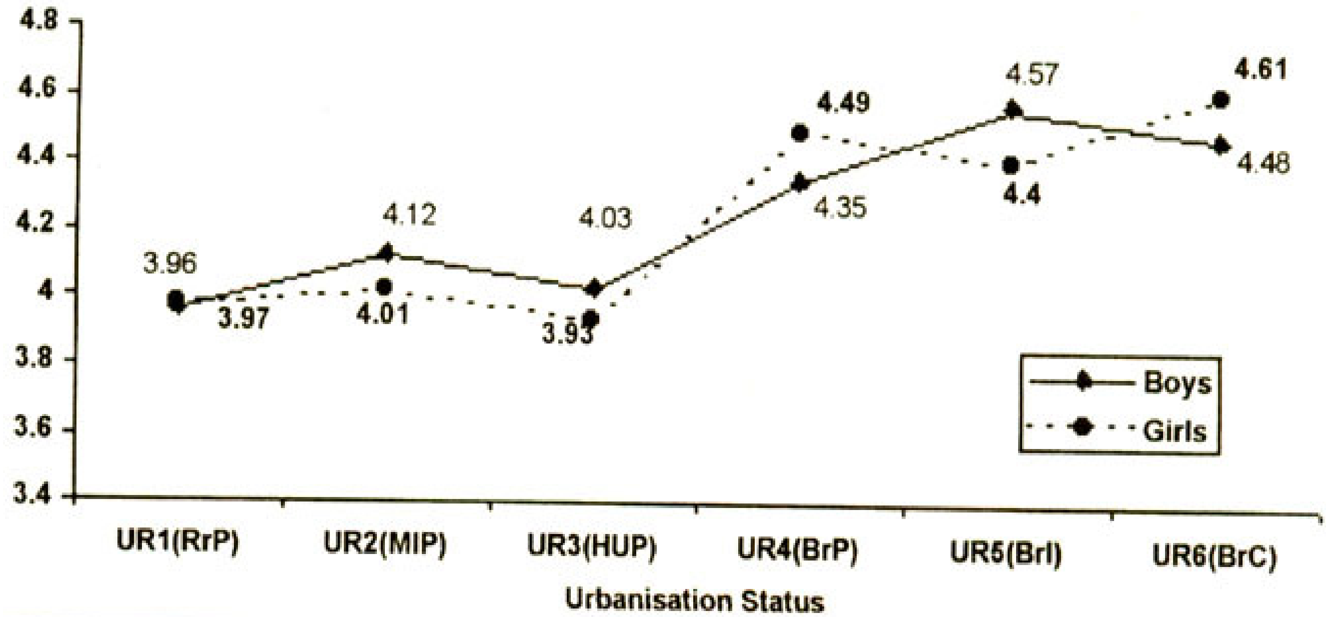

Figure 1. Mean Total Blood Cholesterol (TBC) of the six groups of children having different urbanization status

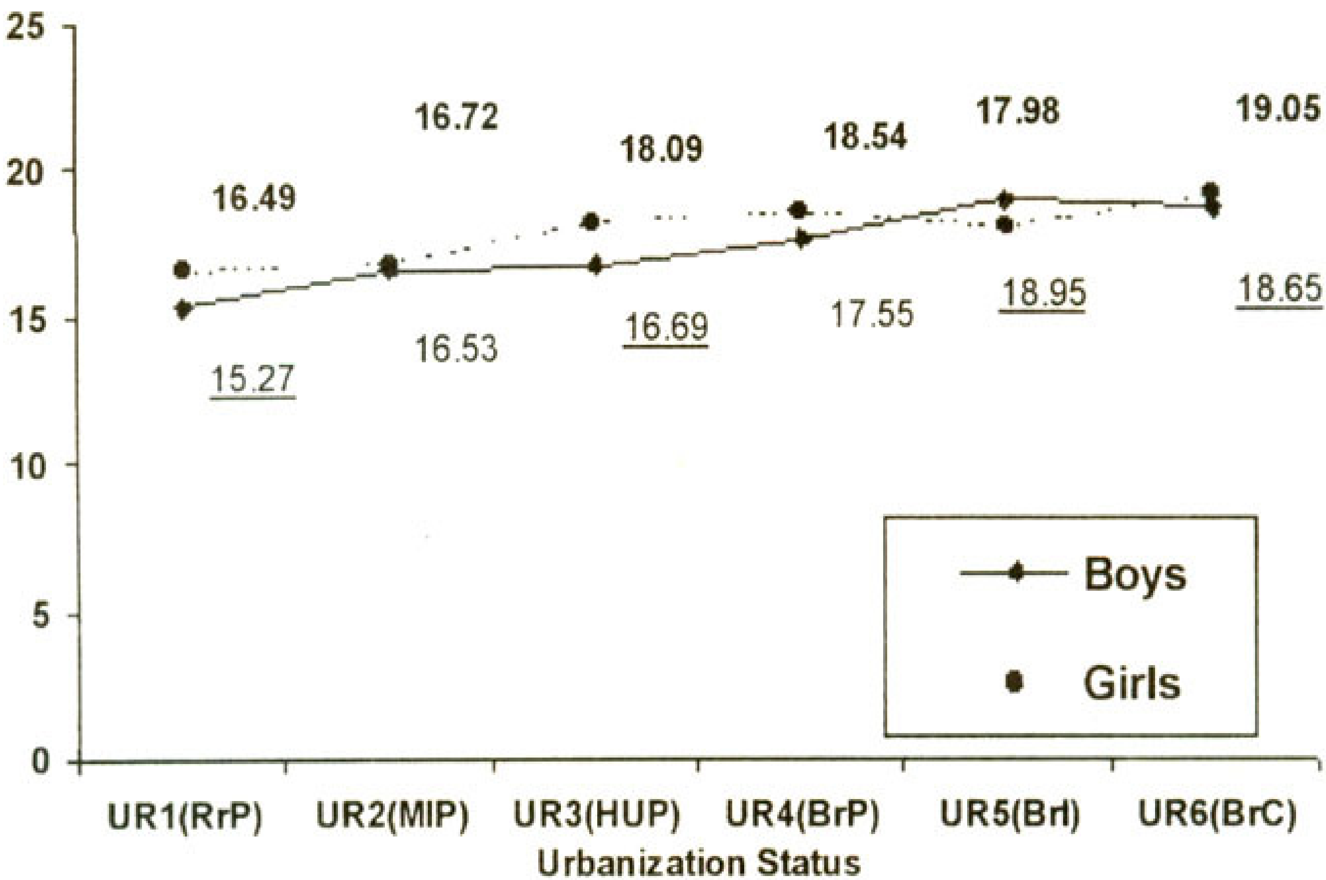

Figure 2. Mean Body Mass Index (BMI) of the six groups of children having different urbanization status

increment in FBG with urbanization rank occurred only within the South Asian group (Figure 3).

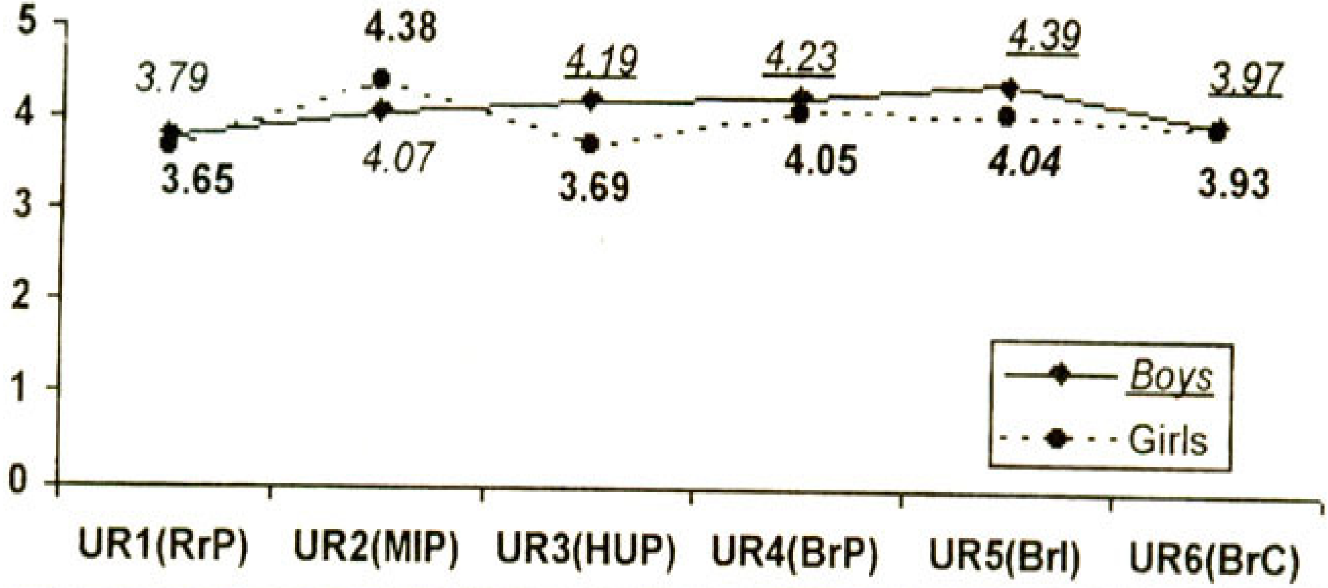

Figure 3. Mean Fasting Blood Glucose (FBG) of the six groups of children having different urbanization status

Increase in WHR occurred only through UR 3-5 for boys and tJR 2-4 for girls (Figure 4).

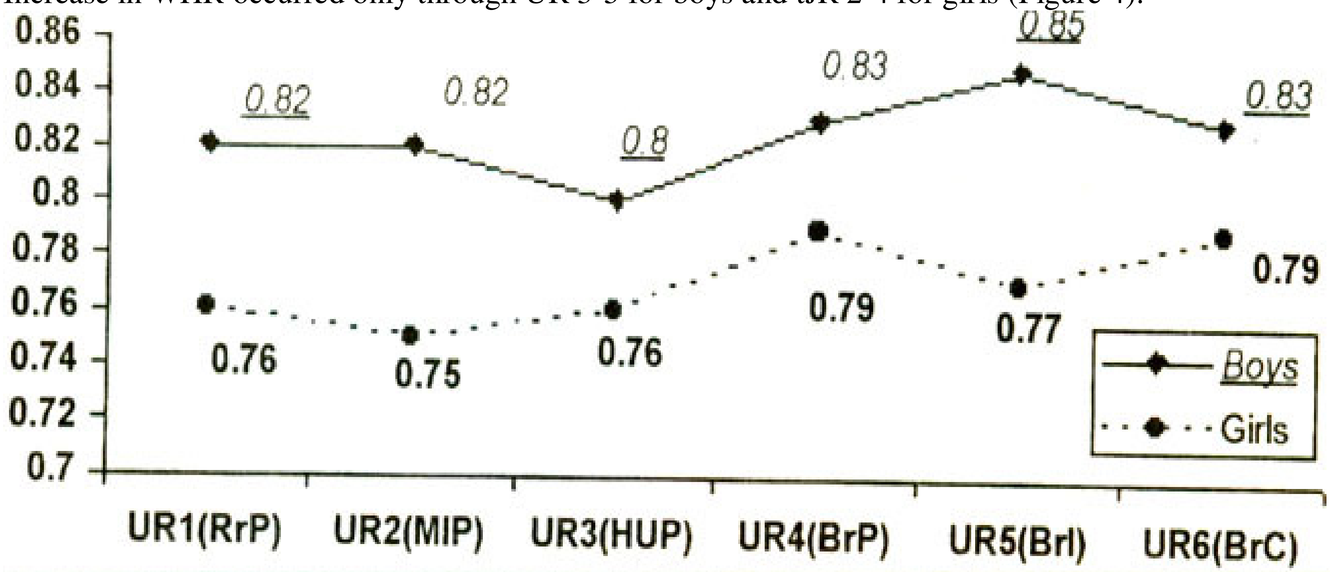

Figure 4. Mean Waist Hip Ratio (WHR) of the six groups of children having different urbanization status

Within the South Asian group (UR1 -5), FBG (r = 0.18, p = 0.000), TBC (r = 0.28, P = 0.000). WHR (r = 0.10, P=0.014) and BMI (r=0.27, P=0.000) had significant positive correlation (Pearson's r) with the urbanization rank. It could be noted that the associations between UR and TBC and UR and BM!, were stronger than the associations between UR and FBG, and UR and WHR. Inclusion of Caucasians (UR6) in the analysis made 0.17, P0.005) and BMI (r = 0.32, P = 0.000), but made it weaker for FBG (r = 0. 1 7, P = 0.000). These results while support the view that genetic susceptibility to diabetes and central obesity could be the underlying risk factors for CHD among South Asians. also indicate that the differences in diet and activity could be responsible for higher BMI and TBC.

Heights of children were compared with NCHS reference values with the help of a WHO sponsored software "ANTHRO'41. Height for age Z scores increased steadily with urbanization rank (Figure 5).

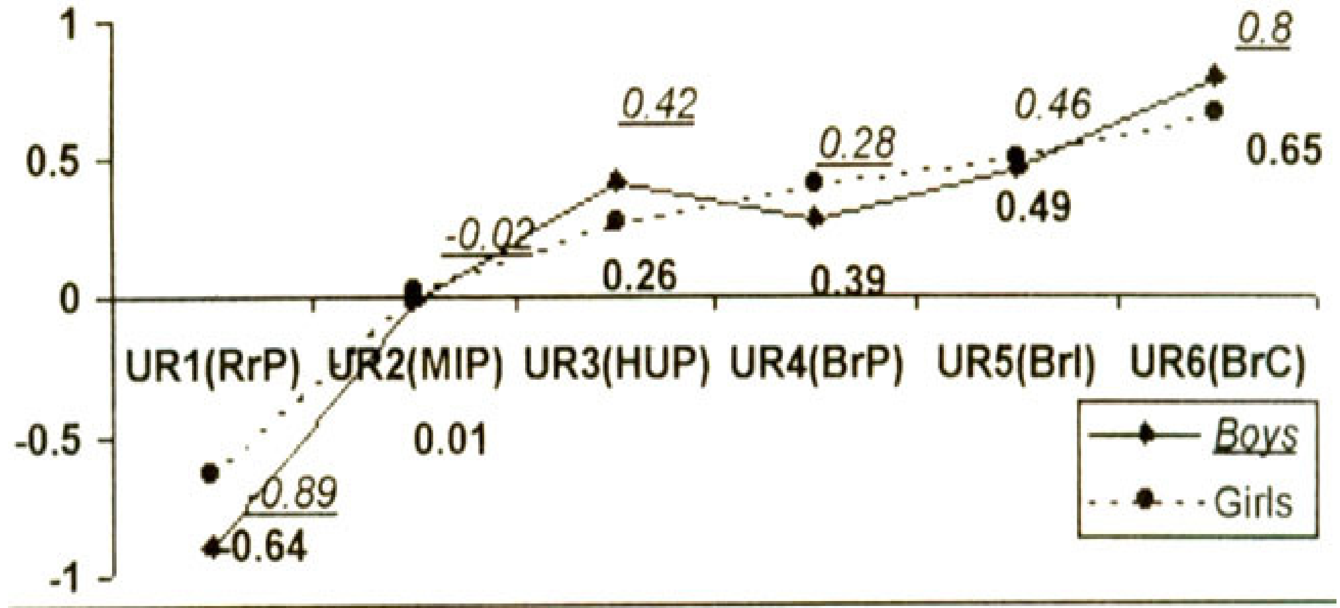

Figure 5 Mean height for Age Z scores (HAZ) of the six groups of children having different urbanization status

Clustering of Risk Factors

Cluster analysis procedure identifies relatively homogenous groups of cases based on selected characteristics, using an algorithm that can handle large number of cases. Cluster analysis (K means cluster) was performed on the whole sample (all groups combined) to see that whether groups of children were identifiable on the basis of selected CHD risk factors. The three clusters thus created by this technique had markedly different CHD risk status. There was significant difference in the central means of three clusters in relation to all the four variables entered. The cluster that had the least values was termed “low risk” and the one with the highest values was named “high risk” and so on (Table 2).

Table 2. Differences in Central Means of the Three Coronary Heart Disease Risk Clusters created by Cluster Analysis

Clusters	Central Mean of CHD Risk Factors			
	FBG	TBC	BMI	WHR
1 (low risk)	3.9	4.0	15.0	0.78
2 (moderate risk)	4.1	4.3	18.6	0.79
3 (high risk)	4.2	4.4	23.7	0.83
P value (chi sq.)	0.001	0.000	0.000	0.003

Proportion of children falling into high, middle or low risk category from each urbanization group was compared. Proportion of children having high CHD risk increased steadily with urbanization status.

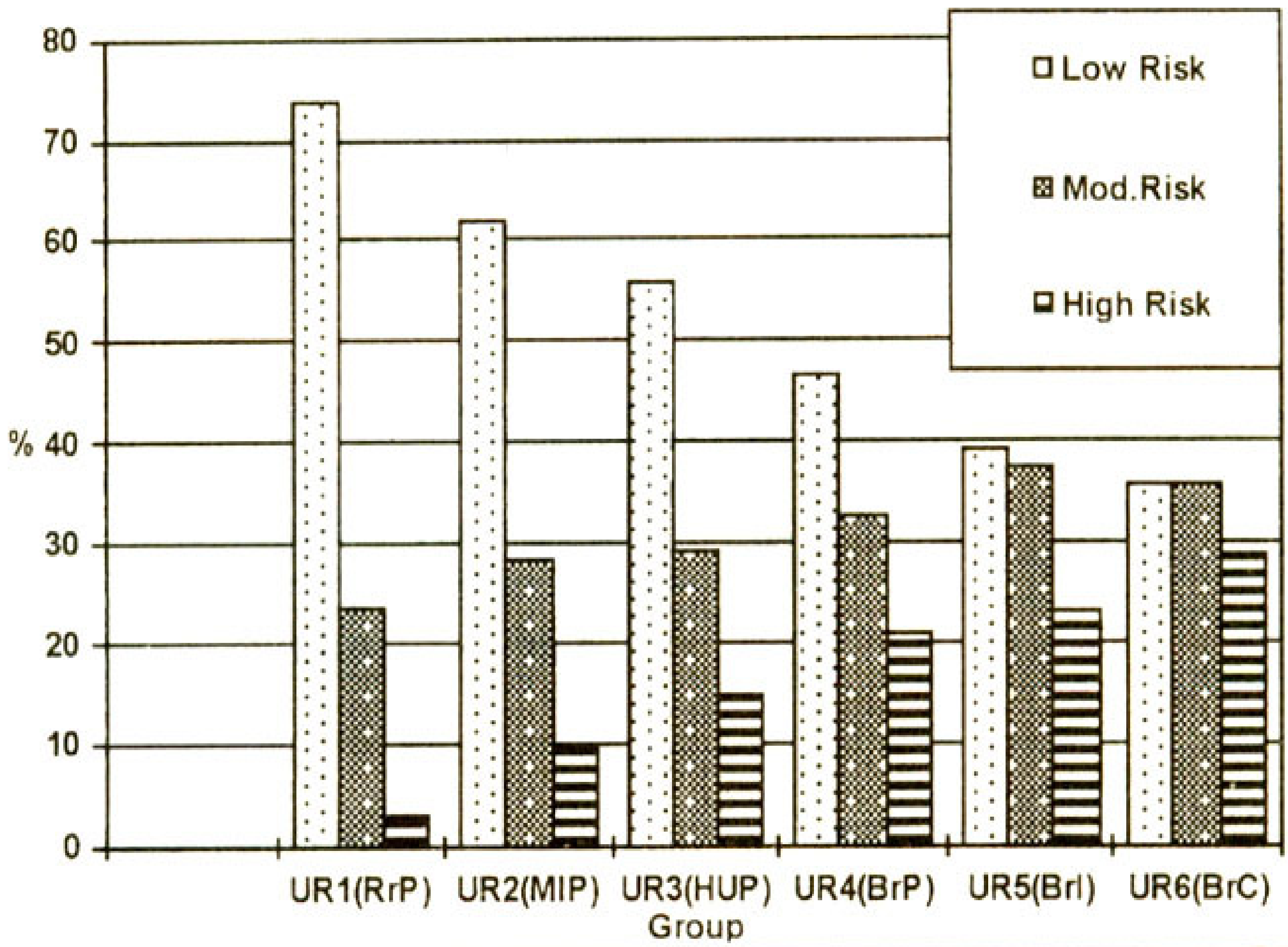

Figure 6 Proportion of children from each demographic group falling into Three CHD Risk Clusters

Figure 6 shows the percentage of children from each group falling into each risk cluster.
These findings indicate that even within the Pakistani population impact of urbanization was evident and thus support the hypothesis that the high CHD death rates among South Asians in UK are not solely due to genetic predisposition but environmental factors would also be playing their role. However the observation that the mean FBG level of British Caucasian children was lower than that of British Pakistani children points out the fact that there is some basis for genetic and/or innate susceptibility of Pakistanis to Type 2 Diabetes Mellitus.

Discussion

Differences observed in the relative CHD risk of the five groups of children in this study indicate the presence of an association between urbanization and CHD risk. The manifestation of this trend within the olispring of Pakistanis shows that the problem of higher rates of CHD in South Asians is not likely to be solely of genetic origin. Differences in CHD risk of more and less urbanized groups have been found in India also[24,42,43] Studies done at two different Limes in middle incorne[44] and more affluent[45] school children in Karachi showed that middle income children had lower blood cholesterol levels. Lower prevalence of CHD risk factors among low-income groups have been reported for Pakistani adults also[46]. The results of this study show that although the over nutrition related risk (TBC and BMI) increase with urbanization the undernutrition related risk (stunting) decrease with urbanization. Stunting is positively associated with obesity and other CHD risk factors[47-57]. High rates of stunting in less urbanized groups need to be considered a potential indicator of obesity and CHD in later life. According to current state of knowledge, the extent of demographic transition and incorporation of heart healthy food and activity habits in the modified life style would determine the ultimate outcome. This notion is supported by the findings in Bangladesh and Puerto Rico. In Bangladesh adjusting for age, sex and social class the prevalence of Type 2 Diabetes Mellitus among urban subjects did not differ significantly from that among rural subjects[58]. In Puerto Rico urban men who had always lived in the same area had an incidence rate as low as that for rural men whereas recent rural migrants to urban areas had the highest rates of all[59]. Thus changes in health related behavior, being brought by demographic transition needs to be checked and channeled in positive direction to prevent the hazardous outcome of urbanization. The price any nation pays for urbanization would be inversely proportional to alertness and promptness it shows in taking these measures.

Attempts to support urbanization, without making and implementing plans for providing health education and opportunities for physical activities could do more harms than good. While currently the most urbanized group indicates the highest CHD risk, the actual future risk would be determined by extent of over nutrition by various groups of children. In this regard undernourished children if they become more urbanized need to adopt a veiy prudent life style. Educational interventions are thus needed both for more and less urbanized groups of parents and children. Furthermore, with continued trends of migration, urbanization and prevalence of stunting, in future, the relative burden of CL-ID is likely to be higher among the urban poor as found in the developed countries.

Acknowledgements

Dr. R. Ilakeern was recipient of Commonwealth scholarship while doing Ph.D. and ISFE Switzerland provided additional financial support. The authors also wish to express thanks to the funding agencies and the students and staff of the participating schools for their cooperation in this study.

References

1.McKeigue PM, Miller GJ. Mannot MG. Coronary heart disease in South Asians overseas: a review J.Clin.Epidemiol., 1989; 42:597-609.
2.UKPDS. Ethnicity and cardiovascular disease. The incidence of myocardial infarction in white, South Asian, and Afro-Caribbean patients with type 2 diabetes (U.K. Prospective Diabetes Study 32). Diabetes Care, 1998; 21:1271-77.
3.McKeigue PM, Sevak L Coronary heart disease in south Asians overseas: a manual for health promotion. London, Health Education Authority, 1994.
4.McKeigue PM, Marmot MG, Adelstein AM, et al. Diet and risk factors for coronary heart disease in Asians in northwest London. Lancet, 1985: 2:1086 90.
5.Barker Di, Osmond C. Simmonds Si, et al. The relation of small head circumference and thinness at birth to death from cardiovascular disease in adult life. Br. Med. J., 1993, 306:422-26.
6.Ba kei Di. Outcome of low birthweight. Horm.Res, 1994; 42:223-230.
7.Barker DJ, Clark PM, Fetal undermitrition and disease in later life. RevReprod, 1997: 2.105-12.
8.Desai M, Hales CN. Role of fetal and infant growth in programming metabolism in later life. Biol.Rev..Camb.Philos,Soc., 1997; 72329-48.
9.Gallaher BW, Breier BH, Keven CL, et al. Fetal programming of insulin-like growth factor (IGF).1 and IGF-binding protein-3: evidence for an altered response to undernutrition in late gestation following exposure to periconceptual undernutrition in the sheep. J.Endocrinol , 1998; 159:501-08.
10.Godfrey KM. Maternal regulation of fetal development and health in adult life. Eur.J.Obstet.Gynecol Reprod.Biol., 1998; 78:141-50.

11.Langley-Evans SC, Gardner DS, Welham Si. Intrauterine programming of cardiovascular disease by maternal nutritional status. Nutrition, 1998; 14:39
12.Stanner SA, Bulmer K, Andres C. et al. Does malnutrition in utero determine diabetes and coronary heart disease in adulthood? Results from the Leningrad siege study, A cross sectional study. Br. Med. J., 1997; 315: 1342-48.
13.Arnesen E. Forsdahl A. The Tromso heart study: coronary risk factors and their association with living conditions during childhood. J.Epidemiol.Community.Health, 1985; 392 10-14.
14.Brunner E, Davey S, Marmot M, et al. Childhood social circumstances and psychosocial and behavioural factors as detcnninants of plasma fibrinogen. Laneet, 1996; 347:1008-13.
15.Notkola V. Punsar S. Karvonen Mi, et al. Socio-economic conditions in childhood and mortality and morbidity caused by coronary heart disease in adulthood in rural Finland. Soc.Sci.Med., 1985; 21:517-23.
16.Eriksson JG, Forsen T, Tuomilehto i, et al. Catch-up growth in childhood aitd death from coronary heami disease: longitudinal study. Br. Med. J.. 1999; 3 18:427-31.
17.UNICEF. State of the world's children. Focus on Nutrition., New York, Oxford University Press.. 1997.
18.Tinker AG. Improving Women's Health in Pakistan.. Washington DC, The World Bank, 1998.
19.Milligan RA, Thompson C, Vandongen R, et al. Clustci'ing of' cardiovascular risk factors in Australian adolescents: association with dietary excesses and deficiencies. J.Cardiovasc.Risk., 1995; 2 515-23.
20.WHO. World Health Report 1999, Making a difference. Geneva, World Health Organisation. 1999.
21.Purath i, Lansinger T, Ragheb C. Cardiac risk evaluation for elementary school children. Public Health Nurs 1995; 12:189-95.
22.Pninrose ED, Savage JM, Boreham CA, et al. Cholesterol screening and family history of vascular disease. Arch.Dis.Child , 1994; 71:239.42.
23.I wata F, Okada T, Harada K, et al. Coronary risk factors in school children in relation to their family history of coronary heart disease and hyperlipidemia. Acta Paediatr.Jpn., 1998; 40:30-34,
24.Gupta R. Lifestyle risk factors and coronary heart disease prevalence in indian men J.Assoc.Physicians.lndia 1996. 44:689-93.
25.Downey AM, Cresanta JL, Herensoit US. Cardiovascular health promotion in children: Heart Smart and the changing role of physicians. Ain.i.Prev.Med., 1989: 5:279-95.
26.Boreham C, Savage JM, Primrose 0, et al. Coronary risk factors in schoolchildren. Arch.Dis.Child., 1993; 68: 182-86.
27.Annstrong N, Balding J. Gentle P. et al. Estimation of coronary risk factors in British schoolchildren: a preliminary report. BrJ.Sports Med., 1990; 24:61-66,
28.Akerblom HK, viikari J. Rasanen L, et al. Cardiovascular risk in young Finns, results from the second follow-up study. Ann.Mcd 1989; 2 1:223-25
29.Friedman AG, Greene PG. Stokes T. Improving dietary habits of children: effects of nutrition education and correspondence training. J.Behav.Ther.Exp.Psychiatry, 1990: 21:263-68.
30.Mahoney LT, Lauer RM, Lee J, et al. Factors affecting tracking of coronary heart disease risk factors in children. The Muscatine Study Ann.N.Y.Acad Sci . 1991; 623:120-32.
31.Feinstein JA, Quivers ES. Pediatric preventive cardiology: healthy habits now, healthy hearts later. Curr.Opin.Cardiol., 1997; 12:70-77.
32.Andersen LB. Haraldsdottir J. Tracking of cardiovascular disease risk factors including maximal oxygen uptake and physical activity from late teenage to adulthood, An 8-year follow-up study. J.lnterii.Med., 1993; 234:309-15.
33.Andersen LB. Tracking of risk factors for coronary heart disease from adolescence to young adulthood with special emphasis on physical activity and fitness, A longitudinal study. Dan.Med.Bull., 1996; 43:407-18.

34.Raitakari OT, Porkka KV. Viikari iS, et at. Clustenng of risk factors for coronary heart disease in children and adolescents. The Cardiovascular Risk in Young Finns Study. Acta Paediatr., 1994; 83:935-40.
35.Viikari J, Akerbiorn HK, Rasanen L, et at. Cardiovascular risk in young Finns. Experiences from the Finnish Multicentre Study regarding the prevention of coronary heart disease. Acts Paediatr.Scand.Suppl., 1990: 365: 13-19.
36.Badruddin SH, Molla A, Khursheed M, et at. The impact of nutritional counselling on serum lipids, dietary and physical activity patterns of school children J.Pak.Med,Assoc. 1993: 43:235-37.
37.Hakeem R and Thomas J Exposure to Western culture and Acculturation in food Habits of South Asian Children. Eur,J.Clin.Nutr.. 1998:52:584.
38.WHO. Physical Status: The use and interpretation of anthropometry. Geneva: World Health Organisation., 1995.
39.del Canizo FJ, Froilan C. Moreira-Andres MN. Precision and accuracy of the measurement of total cholesterol using the retlectometer Aceutrend GC. Usefulness in primary care for diagnosis of hypercholesterolcnua. Aten.Primaria., 1996; 17:463.66.
40.Gottschling HD, Renter W, Ronquisi G, et al. Multicentre evaluation of a non-wipe system for the rapid determination of total cholesterol in capillary blood, Accutrend Cholesterol on Accutrend GC. Eur.J,Clin.Chem.Clin.Biochem. 1995: 33:373-81.
41.NCHS (National Center for Health Statistics: Center for Disease Control). NCHS growth curves for children birth-t8 years. United States. Washington DC. US Government Printing Office, 1978.
42.Chadha SL, Gopinath N, Shekhawat S. Urban-rural differences in the prevalence of coronary heart disease and its risk factors in Delhi. Bull,WHO. 1997; 75:31-38.
43.Singh RB, Sharma JP, Rastogi V. et at, Prevalence of coronary artery disease and coronary risk factors in rural and urban populations of north India. Eur.Heart J., 1997; 18:1728-35.
44.Badruddin SH, Molla A, Khurshid M, et at. Cardiovascular risk factors in school children from tow middle income families in Karachi, Pakistan. J.PakMcd.Assoc. 1994; 44:106-12.
45.Badruddin SH, Khurshid M, Molta A. et at. Factors associated with elevated serum cholesterol levels in well-to-do Pakistani schoolchildren. J.Trop.Med.H vg., 1991; 94:123-29.
46.Hameed K, Kadir M, Gibson 1, et at, The frequency of known diabetes, hypertension and ischacrnic heart disease in affluent and poor urban populations of Karachi, Pakistan. Diabetes. Med., 1995; 12:500-03.
47.Blumchen G, Jette M. Relationship between stature and coronary heart disease in a German male population. lnt.J.Cardiol., 1992: 36:35 1-55.
48.D'Avanzo B. La Vecchia C, Negri E. Height and the risk of acute myocardial infarction in Italian women. Soc.Sci.Med, 1994. 38:193-96.
49.Gunnell DJ. Davey S, Frankel S. et at. Childhood leg length and adult mortality: follow up of the Carnegie (Boyd On') Survey of Diet and Hcalth in Pre-war Britain, i.Epidemiol.Community.Health, 1998; 52:142-52.
50.Han TS, Hooper JP, Morrison CE, et al. Skeletal proportions and metabolic disorders in adults. Eur.J.Clin.Nutr., 1997; 51:804-09,
51.Hebert PR, Rich-I dwards JW, Manson JE, et at. Height and incidence of cardiovascular disease in mate physicians. Circulation, 1993; 88: 437-43.
52.Kannam JP, Levy D, Larson M, et at. Short stature and risk for mortality and cardiovascular disease events. The Framingham Heart Study. Circulation, 1994; 90:2241-47.
53.Kee F, Nicaud V. Tiret L, et at. Short stature and heart disease: nature or nurture? The EARS Group. lnt.J.Epidemiol., 1997; 26 748-56.
54.Nwasokwa ON. Weiss M, Gladstone C. et at Higher prevalence and greater severity of coronary disease in short versus tall men referred for coronary arteriography. Am.Heart J., 1997; 133:147-52.
55.Palmer JR. Rosenberg L, Shapiro S. Stature and the risk of mvocardial infarction in women. Am

.J.Epidemiol.. 1990; 1 3227-32.
56.Parker DR. t.apane KL. Lasater TM, et at, Short stature and cardiovascular disease among men and women from two southeastern New England communities. Int.J.Epidemiol . 1998: 27:970-75.
57.Rona RJ, Qureshi S. Chinn S. Factors related to total cholesterol and blood pressure in British 9 year olds. J.Epiderniol Community.Health, 1996: 50:512-18.
58.Abu S, Ali L, Hussain MZ, Rurni MA et at. Effect of soeiocconomtc risk factors on the difference in prevalence of diabetes between rural and urban populations in Bangladesh. Diabetes Care, 1997; 20:55 1-55.
59.Garcia-Palmieri MR, Costas RJ, Cruz-Vidal M, et al. Urban-rural differences in coronary heart disease in a low incidence area. The Puerto Rico heart study. Am.J.Epidemiol., 1978; 107:206-15.