Hypertension epidemiology in India: lessons from Jaipur Heart Watch

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Cardiovascular diseases have emerged as an important health problem in India. High blood pressure (BP) is a major risk factor and better control can lead to prevention of 300,000 of the 1.5 million annual deaths from cardiovascular diseases in India. Epidemiological studies demonstrate that prevalence of hypertension is increasing rapidly among Indian urban populations and using the current definitions more than two-fifths of the Indian urban adult population has hypertension. The prevalence is lower in rural populations, but is increasing. Jaipur Heart Watch (JHW) is an ongoing cross-sectional epidemiological study in western India. Successive studies have been performed in rural (JHW-R, 1992–93) and urban locations (JHW-1, 1993–94; JHW-2, 1999–2000; JHW-3, 2002–03, and JHW-4, 2004–05). The studies evaluated adults ≥20 years using standardized methodology and in the present analyses subjects aged 20–59 years from these JHW studies [4102 men (1700, 1294, 469, 179 and 413) and 2872 women (1063, 655, 486, 195 and 433)] have been included. Prevalence of various cardiovascular risk factors: smoking/tobacco use, sedentary habits, overweight/obesity (body mass index ≥ 25 kg/m^2), central obesity (waist : hip ratio >0.95 men, >0.85 women), hypertension, dyslipidemias, metabolic syndrome and diabetes was determined. Trends were analysed using least squares linear analyses. Results show that mean systolic BP increased with age in all the study cohorts, while there was no significant difference in diastolic BP. Age-adjusted prevalence of hypertension in JHW-R, JHW-1, JHW-2, JHW-3 and JHW-4 studies in men was 21.6, 29.1, 29.6, 42.5 and 45.1% and in women it was 15.7, 21.7, 25.5, 35.2 and 38.2% (P for trend <0.05). There was a significant association of escalating hypertension with obesity and truncal obesity in both men (two-line regression analysis, unadjusted $r^2 = 0.91$ and 0.50 respectively) and women ($r^2 = 0.88$ and 0.57; $P < 0.05$). Increasing hypertension in India is related to increasing adiposity levels. Population and individual-based measures to prevent and control high BP should focus on measures to prevent obesity.

Keywords: Cardiovascular risk factors, hypertension, epidemiological studies.

Preamble

CARDIOVASCULAR diseases such as coronary heart disease and stroke are the largest causes of death in developing countries and are one of the main contributors to disease burden[1,2]. Age-standardized coronary heart disease death rates (per 100,000) in middle-aged subjects, 30–69 years, reveal low rates in developed countries such as Canada (120) and Britain (180) and high rates in developing countries like Brazil (320), China (280), India (405), Pakistan (400), Nigeria (410) and Russia (680)[1]. Stroke death rates also are high in developing countries (Nigeria 122, Tanzania 118, India 100, China 96, Pakistan 84 and Brazil 82) compared to less than 20 in the UK and Canada[1]. Between years 1990 and 2020 these diseases are anticipated to increase by 120% for women and 137% for men in developing countries compared to 30–60% in developed countries[3]. Moreover, in India about 70% of coronary heart disease-related deaths occurs in people younger than 70 years compared with 22% in the West, and 94% stroke deaths occurs in people less than 70 years in contrast to 6% in developed countries[2].

Control of the cardiovascular diseases will require modification of risk factors that have two characteristics. First, the risk factor must have high attributable risk or high prevalence or both, and secondly, most or all of the risks must be reversible cost-effectively. Blood pressure (BP) is directly associated with risks of several types of cardiovascular diseases, and the associations of BP with disease risk are continuous with large proportions of most populations having non-optimal blood pressure values[4]. Moreover, most or all BP-related risks appear to be reversible within a few years with inexpensive interventions. In India cardiovascular diseases cause 1.5 million deaths annually[2]. Hypertension is directly responsible for 57% of all stroke deaths and 24% of all coronary heart disease deaths. This fact is important because hypertension is a controllable disease and a 2 mm Hg population-wide decrease in BP can prevent 151,000 stroke and 153,000 coronary heart disease deaths[5]. This article summarizes epidemiology of hypertension in India and

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Table 1. Recent Indian hypertension prevalence studies in urban Indian subjects (BP ≥140/90)

Reference	Year	Age-group (yrs)	Place	Sample size		Prevalence (%)	
				Men	Women	Men	Women
12	1995	20–75	Jaipur	1415	797	29.5	33.5
24	2000	20–89	Thiruvananthapuram	76	130	31.0	41.2
26	2000	30–60	Mumbai	1521	141	34.1*	
25	2001	20–70	Chennai	518	657	21.1*	
27	2003	20–75	Jaipur	550	573	36.4	37.5
28	2004	18–60	Mumbai	40,067	59,522	43.8	44.5
29	2006	20–70	National	11,829	8039	29.3	25.2

*Gender-specific data not available.

reports on a study to evaluate trends in hypertension in an Indian urban population and its proximate determinants.

Hypertension in India

Reviews of studies on hypertension epidemiology in India have shown high prevalence in both urban and rural areas[6,7]. Indian urban population studies from the mid 1950s to late 1990s used the older WHO guidelines for diagnosis (known hypertension or BP ≥160 mm Hg systolic and/or ≥ 95 mm Hg diastolic). A significantly increasing adult prevalence of hypertension has been reported changing from 4.4% in Agra (1961)[8], 6.4% in Rohtak (1975)[9], 15.5% in Bombay (1980)[10], 14.1% in Ludhiana (1985)[11], 11.0% in Jaipur (1995)[12], 11.6% in Delhi (1997)[13], and 13.1% in Chandigarh (1999)[14] (χ^2 for trend = 5.99, P = 0.014). Although there is a lower prevalence of hypertension in rural Indian populations, there has been a steady increase over time here as well. Prevalence increased from 0.5% in Bombay (1959)[15], 2.0% in Delhi (1959)[16], 3.6% in Haryana (1978)[17], 5.4% in Delhi (1983)[18], 5.6% in Rajasthan (1984)[19], 4.0% in Maharashtra (1993)[20], 3.4% in Maharasthtra (1993)[21], 7.1% in Rajasthan (1994)[22] and 3.6% in Haryana (1998)[23] (χ^2 for trend = 2.75, P = 0.097). In Kerala rural subjects, that are almost urbanized, the prevalence has been reported to be as high as 17.8 (1993) and 12.5% (1994)[24].

Prevalence of hypertension using the current criteria (known hypertension or systolic BP ≥140 mm Hg and/or diastolic BP ≥90 mm Hg) has been reported among some urban Indian populations (Table 1). In population-based studies, Gupta *et al.*[12] reported hypertension in Jaipur in 30% men and 33% women aged ≥20 years, Joseph *et al.*[24] reported it in 31% men and 41% women in Thiruvananthapuram, while Mohan *et al.*[25] reported a crude prevalence rate of 21% in Chennai. Anand[26] reported hypertension in 34% middle-class executives in Mumbai, but after multiple BP measurements it was confirmed in 27% male and 28% female officers. Gupta *et al.*[27] reported its prevalence in 36% men and 37% women in Jaipur. Gupta *et al.*[28] reported hypertension in 44% men and 45% women in Mumbai. Reddy *et al.*[29] also reported high prevalence of hypertension in a study among industrial populations at multiple sites in India. These findings are in consonance with many developed countries where it has been reported that at any given time almost half of all individuals have high BP[4].

Jaipur Heart Watch studies and hypertension

Since 1992, we have been systematically collecting data regarding cardiovascular risk factors in Jaipur, western India. These population-based studies, collectively known as Jaipur Heart Watch (JHW), are multiple cross-sectional studies and report trends in multiple cardiovascular risk factors[30–34]. Here we describe the trends in the prevalence of hypertension and their determinants as observed in these studies.

Methods

All the studies were approved by the institutional ethics committee and supported financially by philanthropic institutions. In the first study in a rural population in central Rajasthan conducted during 1992–93, 3148 subjects (1982 men, 1166 women) aged ≥20 years were evaluated using total-community survey design (JHW-R)[30]. Of the target sample of 2188 men and 1968 women living in these villages, we could enroll 90.6% men and 59.2% women for the demographic questionnaire and physical examination. Among Jaipur urban subjects, the first study (JHW-1)[31] was conducted in 1993–94 and randomly selected 1608 men and 1392 women were targetted using stratified cluster sampling in six locations. Next, 2212 subjects (1415 men – 88.0%, 797 women – 57.2%) were evaluated for various cardiovascular risk factors. Attempt for fasting blood sample was made in 15% subjects in rural and urban studies. In the second urban study (JHW-2)[32] we targetted 960 men and 840 women in the same locations as in JHW-1, and could examine 550 men (57.3%) and 573 women (68.2%). The third (JHW-3)[33] and fourth (JHW-4)[34] urban studies were location-specific population-based studies targetted at a smaller population.

Table 2. Various Jaipur Heart Watch (JHW) studies and subjects in the present analyses

Study	Period of study	Target sample size		Study enrolled		Present study	
		Men	Women	Men	Women	Men	Women
JHW-Rural[30]	1992–94	2188	1968	1982	1166	1700	1063
JHW-1[31]	1993–95	1608	1392	1415	797	1294	655
JHW-2[32]	1999–2001	960	840	550	573	469	486
JHW-3[33]	2002–03	320	280	226	232	179	195
JHW-4[34]	2004–05	750	650	556	571	413	473

For the present analyses we present data for age-groups 20–59 years from JHW-R, JHW-1, JHW-2, JHW-3 and JHW-4 studies to increase comparability (Table 2). Here, 4102 men (1700, 1294, 469, 179 and 413) and 2872 women (1063, 655, 486, 195 and 433) from the respective studies have been included.

Methodological details have been reported[30–34]. A detailed proforma was utilized for data collection. Briefly, we collected information regarding demographic data, past history of major illnesses such as coronary heart disease, hypertension, diabetes or high cholesterol, and smoking or tobacco intake and alcohol intake. Brief questions were asked to evaluate physical activity and diet, but the results were considered inadequate and not included in the present analysis. Physical examination was performed to assess height, weight, waist and hip size and BP. Body mass index (BMI) was calculated as weight (kg) divided by squared height (m). Waist–hip ratio (WHR) was calculated. Fasting glucose was determined at a central laboratory using glucose peroxidase method and external quality control. Quality control measures were also followed for estimation of total cholesterol, high density lipoprotein (HDL) cholesterol and triglycerides. Low density lipoprotein (LDL) cholesterol was estimated[30].

Hypertension was diagnosed when systolic blood pressure was ≥140 mm Hg and/or diastolic blood pressure ≥90 mm Hg or a person was a known hypertensive[35]. All present and past smokers were included in the smoker category. Users of all other types of tobacco products were included in the other forms of tobacco use category. Obesity was defined as BMI ≥25 kg/m^2. Truncal obesity was diagnosed when WHR was >0.95 in males and >0.85 in females, according to the US National Cholesterol Education Program (NCEP) guidelines[36]. Dyslipidaemia was defined by the presence of high total cholesterol (≥200 mg/dl), high LDL cholesterol (≥130 mg/dl), low HDL cholesterol (<40 mg/dl) or high triglycerides (≥150 mg/dl), but data for hypercholesterolaemia are only presented[37].

Statistical analyses: The continuous variables have been reported as mean ± 1 SD and ordinal variables are in per cent. Prevalence rates have been reported in per cent. Age-stratified distribution of BP levels and hypertension prevalence rates are reported for decadal intervals from 20 to 59 years. Age-adjustment of various prevalence rates was performed[38] using direct method with the standard million Indian population of the year 1971. Significance of trends in study-specific prevalence rates was performed using least-squares linear trend analyses with GB-Stat for Windows® software, version 7.0 (Dynamic Microsystems Inc, Silver Spring, MD, USA) and reported as r^2 values. Comparison of trends in risk factors was performed using two-line regression analysis. P values less than 0.05 were considered significant.

Results

Age-specific mean BP levels in men and women are shown in Table 3. In all populations and genders there was a significant increase in BP with age along with greater variance. Mean BP levels were more in urban population groups and there was a greater age-associated escalation and greater variability (higher SD) in urban cohorts compared to their rural counterparts. Prevalence of hypertension also showed similar age-associated increase (Table 4). Prevalence was low in age-groups 20–29 years in all cohorts in men as well as women and increased significantly by age with high prevalence noted in age-groups 40–59 years in all the cohorts. More than 50% subjects were hypertensive beyond 50 years of age in recent urban cohorts (JHW-2 to JHW-4).

Age-adjusted prevalence of hypertension in JHW-R, JHW-1, JHW-2, JHW-3 and JHW-4 studies in men was 21.6, 29.1, 29.6, 42.5 and 45.1% and in women 15.7, 21.7, 25.5, 35.2 and 38.2% respectively. In both men and women significantly increasing trends were observed in age-adjusted prevalence of hypertension (men $r^2 = 0.93$, $P = 0.008$; women $r^2 = 0.98$, $P = 0.001$). Age-adjusted prevalence of some cardiovascular risk factors and trend analyses using least squares regression is shown in Table 5. Prevalence of generalized obesity as well as truncal obesity was significantly greater in the urban cohorts compared to the rural cohorts ($P < 0.05$). Age-adjusted prevalence of obesity in various cohorts, JHW-R, JHW-1, JHW-2, JHW-3 and JHW-4 respectively, in men was 9.4,

Table 3. Mean blood pressure (BP) levels in various age-groups

Risk factor	JHW-R	JHW-1	JHW-2	JHW-3	JHW-4
Men					
Systolic BP					
20–29	121.9 ± 9.1	118.6 ± 11.9	117.2 ± 11.9	121.7 ± 12.6	126.5 ± 10.7
30–39	124.7 ± 11.2	123.2 ± 13.6	119.0 ± 28.1	127.2 ± 47.4	126.5 ± 18.4
40–49	129.2 ± 13.0	128.7 ± 17.5	120.1 ± 21.8	129.2 ± 29.1	132.1 ± 12.6
50–59	130.1 ± 15.6	134.5 ± 20.0	127.7 ± 23.0	136.0 ± 34.0	132.2 ± 15.3
Diastolic BP					
20–29	78.1 ± 6.1	77.4 ± 6.7	76.6 ± 9.2	79.1 ± 8.5	82.5 ± 7.2
30–39	81.2 ± 7.3	80.1 ± 8.2	82.1 ± 19.2	81.0 ± 9.4	84.2 ± 8.9
40–49	83.4 ± 8.8	82.9 ± 10.1	80.6 ± 14.8	82.1 ± 16.7	87.8 ± 10.0
50–59	83.2 ± 9.1	86.1 ± 10.1	82.6 ± 14.1	91.2 ± 13.0	90.0 ± 11.9
Women					
Systolic BP					
20–29	119.1 ± 9.0	115.1 ± 12.0	106.3 ± 12.1	107.9 ± 9.8	120.8 ± 8.6
30–39	124.1 ± 11.4	117.2 ± 15.1	114.7 ± 16.9	118.1 ± 16.5	123.7 ± 10.2
40–49	126.7 ± 14.8	124.2 ± 14.9	121.6 ± 20.1	128.7 ± 18.3	125.2 ± 12.2
50–59	130.0 ± 18.2	137.2 ± 20.9	130.7 ± 22.2	138.9 ± 23.8	130.5 ± 14.8
Diastolic BP					
20–29	77.0 ± 6.0	75.1 ± 8.0	70.0 ± 10.0	73.2 ± 8.7	80.2 ± 5.8
30–39	78.9 ± 6.8	78.1 ± 10.0	75.2 ± 10.8	77.6 ± 10.2	83.1 ± 8.9
40–49	81.2 ± 8.2	79.0 ± 8.2	79.8 ± 13.8	85.2 ± 11.3	82.6 ± 8.5
50–59	83.0 ± 9.0	86.2 ± 12.1	81.4 ± 14.1	83.7 ± 18.0	85.9 ± 10.8

Values are mean ± 1 SD.

Table 4. Age-specific prevalence of hypertension in various JHW studies

Risk factor	JHW-R	JHW-1	JHW-2	JHW-3	JHW-4
Men					
Hypertension (history or BP ≥140/≥90)					
20–29	47/571 (8.2)	65/526 (12.4)	13/99 (13.1)	9/42 (21.4)	26/95 (27.4)
30–39	101/495 (20.4)	87/374 (23.3)	42/152 (27.5)	19/45 (42.2)	46/111 (41.4)
40–49	120/366 (32.8)	71/183 (38.8)	46/117 (39.3)	28/58 (48.3)	70/113 (61.9)
50–59	95/268 (35.4)	129/211 (61.1)	54/100 (54.0)	27/34 (79.4)	61/94 (64.9)
Women					
Hypertension (history or BP ≥140/≥90)					
20–29	27/382 (7.1)	8/136 (5.9)	6/90 (6.7)	3/40 (7.5)	22/148 (14.9)
30–39	52/342 (15.2)	24/157 (15.3)	36/151 (23.8)	18/62 (29.0)	37/106 (34.9)
40–49	48/212 (22.6)	66/211 (31.3)	54/132 (40.9)	33/49 (67.3)	67/108 (62.0)
50–59	37/127 (29.1)	94/151 (62.2)	62/113 (554.9)	32/44 (72.7)	78/111 (70.3)

Numbers in parentheses are percentages.

21.1, 35.6, 54.0 and 50.9% ($r^2 = 0.92$, $P = 0.009$) and in women was 8.9, 15.7, 45.1, 61.5 and 57.7% ($r^2 = 0.88$, $P = 0.018$). Prevalence of truncal obesity respectively in men was 3.2, 19.6, 39.6, 41.4 and 31.1% ($r^2 = 0.60$, $P = 0.124$) and in women was 10.1, 49.5, 42.1, 51.7 and 50.5% ($r^2 = 0.56$, $P = 0.146$). In both men and women trends of increase in generalized obesity correlated significantly with trends of increase in hypertension (two-line regression analysis unadjusted r^2, men 0.91 and women 0.88) as well as truncal obesity (unadjusted men $r^2 = 0.50$ and women 0.57; $P < 0.05$).

Comments

This study demonstrates that there has been a significant increase in prevalence of hypertension in this Indian population. The increasing hypertension correlates with increasing obesity as well as truncal obesity levels, which are used as physical markers of diet and physical activity levels. A low prevalence of hypertension in rural and high prevalence in urban cohorts was observed in the present study, especially in location-specific groups JHW-3 and JHW-4. These prevalence rates were similar to those

Table 5. Age-adjusted prevalence (%), 95% confidence intervals and trends in cardiovascular risk factors

	JHW-R	JHW-1	JHW-2	JHW-3	JHW-4	Least squares linear trend analysis r^2, P value
Men (*n*)	1700	1294	469	179	413	
Obesity	9.4 (8.1, 10.9)	21.1 (19.0, 23.4)	35.6 (31.2, 39.8)	54.0 (47.5, 60.4)	50.9 (46.0, 55.6)	0.92 (0.009)
Truncal obesity	3.2 (2.4, 4.1)	19.6 (17.6, 21.9)	39.6 (35.3, 44.2)	41.4 (34.9, 47.7)	31.1 (26.7, 35.6)	0.60 (0.124)
Smoking	50.0 (47.6, 52.4)	38.7 (36.1, 41.4)	35.7 (3.4, 40.0)	27.1 (21.6, 33.1)	59.9 (55.0, 64.0)	0.10 (0.872)
Hypertension	21.6 (19.7, 23.6)	29.1 (26.7, 31.7)	29.6 (25.7, 33.9)	42.5 (36.2, 49.0)	45.1 (40.3, 49.9)	0.93 (0.008)
Hypercholesterolaemia	19.1 (17.3, 21.1)	22.4 (20.2, 24.8)	36.0 (31.8, 40.5)	31.1 (25.3, 37.3)	28.5 (24.4, 33.1)	0.41 (0.241)
Diabetes	–	–	9.4 (7.1, 12.4)	10.4 (7.2, 15.3)	26.4 (22.4, 30.8)	0.79 (0.299)
Women (*n*)	1063	655	486	195	473	
Obesity	8.9 (7.4, 10.8)	15.7 (13.1, 18.7)	45.1 (40.7, 49.5)	61.5 (54.5, 68.1)	57.7 (53.2, 62.1)	0.88 (0.018)
Truncal obesity	10.1 (8.4, 12.0)	49.5 (46.7, 53.3)	42.1 (37.9, 46.6)	51.7 (44.8, 58.7)	50.5 (45.4, 54.4)	0.56 (0.146)
Hypertension	15.7 (13.6, 18.0)	21.7 (18.7, 25.0)	25.5 (21.8, 29.6)	35.2 (29.0, 42.3)	38.2 (34.0, 42.7)	0.98 (0.001)
Hypercholesterolaemia	5.3 (4.1, 6.8)	22.6 (19.6, 26.0)	35.3 (31.3, 39.7)	23.1 (17.7, 29.5)	27.8 (23.9, 31.9)	0.42 (0.234)
Diabetes	–	–	6.3 (4.5, 8.9)	6.8 (3.9, 11.1)	16.7 (13.6, 20.3)	0.79 (0.306)

95% confidence intervals calculated using modified Wilson's method.

observed in recent studies in South and western India (as reported in Table 1) and are therefore not an artifact. We did not assess major dietary determinants of hypertension such as calorie intake, fat intake and sodium intake. Physical activity was also not accurately assessed in the study and these are study limitations. However, obesity levels are a more true reflection of dietary intake and physical activity and as there is a strong correlation of obesity with increasing hypertension prevalence, it indirectly reflects the importance of diet and physical activity in increasing hypertension in India.

Meta-analyses such as the present study have been faulted on four grounds – reproducibility, precision, suitable extrapolation and fair comparison[39]. The present meta-analyses has combined results from five studies that have been performed using similar methodologies and epidemiological, clinical and laboratory protocols, and most of the criticisms have been negated. Data presented are age-specific as well as age-adjusted and show clear and significant trends in hypertension prevalence in various cohorts. Other study limitations relate to a low response rate in all the studies, small number of subjects in age-specific subgroups, and use of older cut-offs to diagnose truncal obesity (WHR >0.95 men, >0.85 women). However, the sample sizes have been determined using available recommendations for prevalence of cardiovascular risk factors in a community and are considered appropriate for inter-group comparisons[40]. This is also not a prospective study of a single cohort as a cohort-based prospective study is more powered to detect development of risk factors as well as hard outcome events. An other limitation of the study is low prevalence of hypertension in rural cohorts, which could have biased the trends. However, the study is a comparison of population obesity levels with increasing hypertension prevalence and inclusion of this group with low obesity prevalence is important to exactly evaluate the trends. Ideally prospective studies should commence in childhood and young adulthood to determine prognostic importance of various factors on development of hypertension and other cardiovascular risk factors.

Hypertension risk factors

Causes of the increase in hypertension in Indians are speculative[6,7]. Studies among the unacculturated societies have shown lower BP levels that are not influenced by age[41]. Data show that among the so-called less-culturated Indian rural populations, there is only a small increase in prevalence in hypertension over the years and the present studies show a smaller age-associated increase in systolic BP in rural populations (Table 3). On the other hand, in urban populations who are being exposed to stress of acculturation and modernization, the hypertension prevalence rates have more than doubled in the last few years and are now similar to the developed countries[4].

Important hypertension risk factors are genetic and environmental. There are a large number of genes that are responsible for hypertension. Single-gene related hypertension is, however, rare. Intermediate phenotypes are more important and prevalent than gene mutations[42]. These phenotypes are body-fat distribution, familial dyslipidemia, metabolic syndrome, insulin resistance, kallikrein deficiency, sodium sensitivity, nonmodulation of aldosterone and renal blood flow, abnormal cellular ion transport systems (Na, Li, K, H transport systems) and BP reactivity. The nature of genetic contribution to hypertension needs more studies among South Asians. Population and animal studies suggest a polygenic or oligogenic model for hypertension, wherein susceptibility imparted by any single gene is modest and quantitative[42,43]. Such gene variations would be expected to modulate response to environmental exposure and may only achieve signifi-

cance through cumulative integration of lifetime experiences. Although this scenario greatly complicates the task of genetic epidemiologist, major studies are currently under way and are likely to produce a list of common genes contributing to hypertension[44].

Essential hypertension may be considered the result of interactions between genes and the environment. The environmental effects are powerful and explain most of the BP differences between populations[43]. Obesity as well as truncal obesity are powerful determinants, as also shown in the present study. Other important environmental factors are smoking, alcohol intake, physical inactivity, dietary excess of sodium and fat and deficiency of potassium and fibre intake, and psychosocial stress[45]. Although we have not analysed association of these risk factors with hypertension in the present study, we speculate that the summation of these sociodemographic and lifestyle factors is accelerating the hypertension epidemic sweeping India.

Conclusion

Secular analyses of hypertension epidemiological studies in India in the present investigation have demonstrated that the prevalence is increasing exponentially in the country. Our studies demonstrate that increasing obesity and adiposity levels are driving this epidemic. There is an urgent need to develop suitable strategies for prevention of obesity in India using population-based approaches.

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