

# Population-specific left ventricular hypertrophy in three groups from the northeastern region of India

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## ABSTRACT

**Background.** People living in the hills are continuously exposed to strenuous physical activity for their day-to-day work. Besides hypertension, left ventricular hypertrophy in different populations may be related to continuous physical activity.

**Methods.** Electrocardiogram, blood pressure and sociodemographic information of 12 252 subjects  $\geq 30$  years of age from three different population groups living in Mizoram (hilly) and Assam (plain) were recorded. Of them, 8058 were from Mizoram and 3180 and 1014 were indigenous Assamese and tea garden workers of Assam.

**Results.** Among the subjects from Mizoram the percentage of smokers (41.9%), mean (SD) BMI (21.9 [3.8]) and waist-hip ratio (0.87 [0.02]) were significantly higher than in those from other groups. Tea garden workers had a higher mean systolic blood pressure (145.2 [25.7]) and diastolic blood pressure (87.6 [13.6]). The prevalence of left ventricular hypertrophy was highest among tea garden workers (16.5%) followed by people from Mizoram (3.7%) and the indigenous Assamese (2%) people. In spite of a significantly higher prevalence of hypertension among the indigenous Assamese community than among those from Mizoram, left ventricular hypertrophy was found to be lower in the former.

**Conclusion.** High prevalence of left ventricular hypertrophy among tea garden workers was possibly related to a higher prevalence of hypertension but the higher prevalence of left ventricular hypertrophy among people from Mizoram might be related to more physical activity.

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## INTRODUCTION

Left ventricular hypertrophy (LVH) defined by electrocardiography (ECG) has been recognized as an independent risk factor for cardiovascular morbidity and mortality.<sup>1–4</sup> Although, prevalence of LVH among various population groups is dependent upon the

level of blood pressure, the influence of endurance sports and regular physical activity cannot be ignored. Endurance sports such as running, cycling, rowing and cross-country skiing may cause physiological hypertrophy of the heart to cope with the increased circulatory demand.<sup>5,6</sup> Regular physical activity can reduce pre-existing LVH by lowering the blood pressure.<sup>7</sup> However, data related to the combined effects of such variables on the development of LVH are scanty.

People residing in the hills need to work more physically for their daily needs because of the hilly terrain compared with those living in the plains. Also, people living in the plains might have a differential prevalence of hypertension.<sup>8,9</sup> We hypothesized that the prevalence of LVH in people living in hilly areas who are exposed to more physical activities will be different. We studied the population-specific variation in the prevalence of LVH (based on ECG) and associated risk factors in our study populations. ECG though less sensitive is convenient, cost-effective and feasible to use in the community.

## METHODS

We did this study in 2 states of the northeastern region of India between 2000 and 2006. Subjects from Assam (indigenous Assamese community and tea garden workers [TGW]) represented individuals dwelling in the plains and those from Mizoram represented the hill dwelling group. Mizoram is a hilly state in the northeastern part of India situated at an altitude of 2123.1 metres.

## SUBJECTS

**Indigenous Assamese population.** Five of the 23 districts of Assam were selected on the basis of geographical location and ethnic distribution. Villages from different districts were listed and stratified according to the population size. After stratification, villages were selected by simple random sampling and 25 villages (5 from each district) covering a population of 20 857 were included. A household list was collected from the revenue department and households were selected by systematic sampling.

**Tea garden workers.** A tea garden of Assam was taken to cover the required sample size and the garden was selected purposively on the basis of ethnic homogeneity, size of the population and operational feasibility. Every alternate family was selected from the list of TGW using systematic random sampling. In Assam, the majority of subjects from this community are engaged in the tea industry as manual workers. All the participants in this study were manual workers.

**Hill dwellers (Mizoram).** (i) Urban area: Aizawl, the capital town of Mizoram was selected purposively for the study. Of 69 villages/wards, 6 clusters were selected using probability proportionate to the size sampling method. Households from the

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selected localities were selected by systematic random sampling. A rural block was also selected randomly. The villages of the entire block were listed and stratified according to the population, e.g. 500–1000, 1000–1500, etc.; and 14 villages were selected randomly.

The required sample size for each population group was calculated on the basis of prevalence of hypertension in these communities observed in a pilot study.<sup>10</sup> All individuals  $\geq 30$  years of age from the selected households, who gave verbal informed consent for the study, were included. A total of 12 252 (men 5693; women 6559) subjects were included in the study. Of these, 8058 (rural 4416, urban 3642) were from Mizoram and 4194 were from Assam (plain dwellers). Subjects from Assam included the indigenous Assamese community (3180) and TGW (1014).

#### Collection of data and recording of ECG

Data on sociodemographic variables, smoking habit and alcohol consumption were recorded using a pre-designed proforma. A subject was categorized as a non-smoker if he/she was not a current or past smoker and a smoker if he/she was a current smoker irrespective of the number of cigarettes smoked. A subject was a non-alcohol user if he/she was not consuming alcohol at present and an alcohol user if he/she took alcohol. Height, weight, waist and hip circumference were measured by trained personnel following standard methods. Anthropometric rod and platform balance (SECA) were used for measurement of height and weight, respectively. The body mass index (BMI) was expressed as weight in kg divided by height in metre squared. The waist circumference was measured at the level of the umbilicus and hip circumference at the level of the greater trochanter using a non-stretchable metallic measuring tape. We presumed that the level of physical activity would be higher among hill dwellers than plain dwellers. We did not obtain any quantitative information on the physical activities of the study participants.

Blood pressure (BP) was measured using a mercury column sphygmomanometer following standard guidelines.<sup>11</sup> We took 3 readings for each participant and the average of the three was used for analysis—one reading was taken before the interview, one after the interview and the third after recording the ECG. This protocol was followed throughout the study. Participants who had eaten, smoked or taken alcohol were allowed to rest for 1 hour before recording the BP. Hypertension was defined as per the Joint National Committee (JNC-VI) guidelines.<sup>12</sup>

The ECG was recorded using a 12-lead electrocardiograph (BPL-Cardiart 408 or Schiller AT-2) in a comfortable environment and in the supine position. Smoking and heavy meals were avoided for 30 minutes before taking ECG. Particular attention was given to the correct and consistent positioning of the chest leads. A cardiologist read the ECG tracings and the results were coded by Minnesota coding, 1982 following the standard guidelines. All codes were translated to the clinical classification of disease. The criteria used for diagnosis of LVH were:

Minnesota code 3-1:

1. R-wave  $>26$  mm in  $V_5$  or  $V_6$  or
2. R-wave  $>20$  mm in I, II, III, aVF or
3. R-wave  $>12$  mm in aVL

Minnesota code 3-3 (when 3-1 is not present):

1. R amplitude  $>15$  mm but  $\leq 20$  mm in lead I or
2. R amplitude in  $V_5$  or  $V_6$ , plus S amplitude in  $V_1 >35$  mm

The institutional ethical committee of the Regional Medical Research Centre, Northeastern Region (Indian Council of Medical Research), Dibrugarh, Assam approved the study protocol.

#### Statistical analysis

All analyses were done using the Statistical Package for Social Science (SPSS-15.0) software. Comparison of different risk variables between hill and plain dwellers were done by chi-square test for categorical variables and independent samples *t*-test for continuous variables. The level of significance was determined at  $p < 0.05$ . Binary logistic regression model was constructed to determine the factors associated with LVH. For those with ECG evidence of LVH, this was taken as the dependent variable and age categories, sex, smoking, alcohol consumption, different BMI categories, blood pressure (hypertensive or normotensive) and population groups (Mizoram, Assamese and TGW) were taken as covariates.

#### RESULTS

The percentage of smokers (41.9%), mean age, BMI and waist-hip ratio were significantly higher in subjects from Mizoram than in the other groups. We also found a significantly higher percentage of men among TGW. Alcohol consumption by TGW (85.2%) and indigenous Assamese (36.3%) was significantly higher than of those from Mizoram (Table I). Consumption of extra salt (as a side dish) by those from Mizoram was significantly higher (84.1%) than the other groups (Assamese 53.6%, TGW 77.1%). The indigenous Assamese and TGW had significantly higher mean systolic and diastolic BP and prevalence of hypertension than those from Mizoram (Table II).

TABLE I. General characteristics of the study subjects ( $n=12\ 252$ )

Characteristic	Mizoram ( $n=8058$ )	Indigenous Assamese ( $n=3180$ )	Tea garden workers ( $n=1014$ )
Men (%)	3741 (46.4)	1441 (45.3)	511 (50.4)*
Mean (SD) age (years)	48.3 (13.8)	45.38 (13.19)†	44.0 (11.2)†
Smokers (%)	3380 (41.9)	398 (12.5)†	76 (7.5)†
Consumption of extra salt (%)	6779 (84.1)	1706 (53.6)*	782 (77.1)*
Alcohol users (%)	651 (8.1)	1155 (36.3)†	864 (85.2)†
Mean (SD) body mass index ( $\text{kg}/\text{m}^2$ )	21.9 (3.8)	19.8 (3.3)†	17.8 (2.0)†
Mean (SD) waist-hip ratio	0.87 (0.02)	0.86 (0.02)†	0.85 (0.02)†

Level of significance was determined with reference to subjects from Mizoram

\*  $p < 0.05$  †  $p < 0.001$

TABLE II. Prevalence of risk factors in the study subjects ( $n=12\ 252$ )

Characteristic	Mizoram ( $n=8058$ )	Indigenous Assamese ( $n=3180$ )	Tea garden workers ( $n=1014$ )
Mean (SD) systolic BP (mmHg)	120.6 (18.2)	134.4 (20.3)†	145.2 (25.7)†
Mean (SD) diastolic BP (mmHg)	78.1 (10.5)	82.8 (10.7)†	87.6 (13.6)†
Hypertension (%)	1614 (20.0)	1034 (32.5)†	596 (58.8)†
Treatment for hypertension (%)	511 (6.3)	226 (7.1)	33 (3.3)†
<i>Left ventricular hypertrophy (%)</i>			
Total	297 (3.7)	63 (2.0)†	167 (16.5)†
Male	182 (4.9)	23 (1.6)†	99 (19.4)†
Female	115 (2.7)	40 (2.3)	68 (13.5)†
Normotensive	192 (3.0)	17 (0.8)†	42 (10.0)†
Hypertensive	105 (6.5)	46 (4.4)*	125 (21.0)†

BP blood pressure Level of significance was determined with reference to

subjects from Mizoram \*  $p < 0.05$  †  $p < 0.001$

TABLE III. Risk factors for left ventricular hypertrophy using reduced multiple binary logistic regression analysis with backward elimination of non-significant determinants (cut-off=0.1)

Determinant	Adjusted odds ratio (95% CI)	p value
<i>Group</i>		
(Mizo) hill tribe	Reference	—
Indigenous Assamese	0.46 (0.34–0.61)	0.0001
Tea garden worker	3.42 (2.65–4.32)	0.0001
<i>Age group (years)</i>		
≤50	Reference	—
>50	1.38 (1.14–1.67)	0.001
<i>Gender</i>		
Female	Reference	—
Male	1.56 (1.30–1.87)	<0.0001
<i>Body-mass index (BMI)</i>		
Normal	Reference	—
Overweight	0.99 (0.73–1.35)	0.96
Obese	1.59 (0.95–2.7)	0.08
Underweight	1.33 (1.07–1.65)	0.01
<i>Hypertension</i>		
No	Reference	—
Yes	2.32 (1.96–2.91)	0.0001

Variables entered were age, gender, smoking, consumption of alcohol, BMI, status of hypertension, treatment for hypertension and type of community

TABLE IV. Risk factors for left ventricular hypertrophy in subjects stratified by status of hypertension. Reduced multiple binary logistic regression analysis with backward elimination of non-significant determinants (cut-off point=0.10)

Determinant	Adjusted odds ratio (95% CI)	p value
<b>Normotensive subjects</b>		
<i>Group</i>		
Mizoram	Reference	—
Indigenous Assamese	0.259 (0.16–0.43)	0.0001
Tea garden worker	3.473 (2.44–4.94)	0.0001
<i>Gender</i>		
Female	Reference	—
Male	2.0 (1.54–2.59)	0.0001
<b>Hypertensive subjects</b>		
<i>Group</i>		
Mizoram	Reference	—
Indigenous Assamese	0.71 (0.50–1.02)	0.062
Tea garden worker	1.79 (1.38–2.33)	0.0001
<i>Age group (years)</i>		
≤50	Reference	—
>50	1.79 (1.38–2.33)	0.0001

Variables entered were age, gender, smoking, consumption of alcohol, body-mass index, status of hypertension, treatment for hypertension and type of community

The overall prevalence of LVH was 2%, 3.7% and 16.5% among the native Assamese, those from Mizoram and TGW, respectively. In overall and group-wise analysis, hypertensive subjects showed significantly higher prevalence of LVH. Among the subjects from Mizoram, the prevalence of LVH in rural subjects was significantly higher than those from urban areas (4.8% v. 2.4%,  $p < 0.0001$ ).

We observed the effect of different predictor variables for the presence of LVH in overall and subgroups stratified by the level of BP. In the overall model we found that TGW, age >50 years,

male gender, underweight and hypertension were strong positive predictors for LVH. Assamese community was a strong negative predictor for LVH (Table III).

Analysis in the normotensive subgroup revealed male gender and TGW to be positive predictors whereas Assamese community was a strong negative predictor for LVH. In the hypertensive subgroup, only TGW and age >50 years were found to be significantly associated with LVH (Table IV).

## DISCUSSION

Electrocardiogram evidence of LVH has an acceptable sensitivity and specificity for diagnosis of LVH<sup>13</sup> and can predict increased risk of morbidity and mortality from cardiovascular diseases. It is also a useful tool in community studies in resource-constrained settings.

The prevalence of LVH based on ECG showed a 16.5% prevalence in TGW followed by those from Mizoram (3.7%) and the indigenous Assamese (2.0%) groups. We found a higher prevalence in all groups than the Second National Health and Nutrition Examination Survey (NHANES-II; 1.33%) conducted in representative samples of whites and blacks from the general population<sup>14</sup> or studies from metropolitan Reykjavik area (male 3.2% and female 0.5%).<sup>15</sup> However, the prevalence of LVH in hypertensive subjects from Mizoram (6.5%) and the Assamese community (4.4%) was significantly lower than that in studies reported from Mumbai, India (22.1%)<sup>16</sup> and DHCCP (Department of Health and Social Security Hypertension Care Computer Project) study (19%).<sup>14</sup> While the prevalence of LVH among hypertensive TGW (21%) was in conformity with the above studies, the prevalence among those with hypertension from Mizoram and Assam was found to be lower. This lower prevalence of LVH may be related to demographic, ethnic<sup>17</sup> or other factors.

Studies have shown that hypertension,<sup>18</sup> increasing age<sup>19</sup> and gender<sup>20</sup> are predictors for the development of LVH. Similarly, in our study multiple logistic regression analysis showed increasing age (>50 years), male gender and hypertension to be significant predictors of LVH. We also found underweight to be a predictor of LVH. This finding might have been influenced by the higher prevalence of hypertension and LVH among TGW, a majority of whom (65.3%) were underweight (BMI<18.5).

Multiple logistic regression analysis incorporating community as a risk factor identified the indigenous Assamese group as a strong negative predictor for LVH. This was evident in normotensive subjects but not in those with hypertension.

The groups studied were genetically heterogeneous and various gene–environment interactions may have resulted in the differences in prevalence of LVH. In addition, the role of physical activity cannot be ignored. Though we did not quantify the level of physical activity among people in the study groups, those living in a hilly terrain (those from Mizoram) are likely to be doing more physical activity. An earlier study<sup>21</sup> had shown that even in the absence of hypertension, exaggerated blood pressure responses during exercise testing and regular physical activities might cause physiological hypertrophy of the heart.<sup>5,6</sup> Continuous exposure of physical activity in the subjects from Mizoram might have a similar physiological effect on the cardiovascular system resulting in a higher prevalence of LVH in them.

A high prevalence of hypertension among TGW has been reported earlier.<sup>8</sup> This may be related to consumption of extra salt (salt as a side dish), non-smoked tobacco and alcohol among others.<sup>8</sup> Another notable finding was the high prevalence of LVH (10%) among normotensive TGW. Several non-haemodynamic

factors may play a role in the development of LVH<sup>22</sup> in TGW, such as consumption of alcohol (85.2%) and intake of extra salt (77.1%).

Our study has suggested some areas that need more research. Consumption of extra salt, non-smoked tobacco and consumption of alcohol were risk factors for the development of hypertension and LVH. Modifying these factors may prevent cardiovascular morbidity and mortality. Further, the low prevalence of hypertension in those from Mizoram might be related to the physical activity required for their daily activities. The limitations of our study include the use of the less sensitive ECG criteria for diagnosis of LVH and that we did not quantify the level of physical activity.

Hypertensive subjects associated with LVH are at greater risk than those without LVH for cardiovascular complications such as myocardial infarction, cerebrovascular events and congestive heart failure.<sup>13,23,24</sup> However, cardiovascular morbidity and mortality due to LVH induced by physical activity in the presence or absence of hypertension is not clear. As studies addressing this issue are presently not available, case-control studies with strict control of multiple risk factors in different communities may improve our understanding. Moreover, high prevalence of hypertension and LVH in normotensive participants from TGW warrants the need for in-depth genetic studies in this community.

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#### REFERENCES

- Kannel WB, Dawber R, Kagan A, Revotskie N, Stokes J 3rd. Factors of risk in the development of coronary heart disease—six year follow-up experience. The Framingham Study. *Ann Intern Med* 1961;**55**:33–50.
- Kannel WB, Gordon T, Offutt D. Left ventricular hypertrophy by electrocardiogram. Prevalence, incidence, and mortality in the Framingham study. *Ann Intern Med* 1969;**71**:89–105.
- Kannel WB, Gordon T, Castelli WP, Margolis JR. Electrocardiographic left ventricular hypertrophy and risk of coronary heart disease. The Framingham study. *Ann Intern Med* 1970;**72**:813–22.
- Kannel WB. Prevalence and natural history of electrocardiographic left ventricular hypertrophy. *Am J Med* 1983;**75** (3A):4–11.
- Maron BJ. Structural features of the athlete heart as defined by echocardiography. *J Am Coll Cardiol* 1986;**7**:190–203.
- Pelliccia A, Maron BJ, Spataro A, Proschan MA, Spirito P. The upper limit of physiologic cardiac hypertrophy in highly trained elite athletes. *N Engl J Med* 1991;**324**:295–301.
- Kokkinos PF, Narayan P, Collier JA, Pittaras A, Notargiacomo A, Reda D, *et al.* Effects of regular exercise on blood pressure and left ventricular hypertrophy in African-American men with severe hypertension. *N Engl J Med* 1995;**333**:1462–7.
- Hazarika NC, Biswas D, Narain K, Kalita HC, Mahanta J. Hypertension and its risk factors in tea garden workers of Assam. *Natl Med J India* 2002;**15**:63–8.
- Hazarika NC, Narain K, Biswas D, Kalita HC, Mahanta J. Hypertension in the native rural population of Assam. *Natl Med J India* 2004;**17**:300–4.
- Hazarika NC, Biswas D, Narain K, Phukan RK, Kalita HC, Mahanta J. Differences in blood pressure level and hypertension in three ethnic groups of northeastern India. *Asia Pac J Public Health* 2000;**12**:71–8.
- Rose GA, Blackburn H, Gillum RF, Prineas RJ. *World Health Organization: Cardiovascular survey methods*. 2nd ed. Geneva:World Health Organization; 1982.
- The sixth report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure. *Arch Intern Med* 1997;**157**:2413–46.
- Levy D, Garrison RJ, Savage DD, Kannel WB, Castelli WP. Prognostic implications of echocardiographically determined left ventricular mass in the Framingham Heart Study. *N Engl J Med* 1990;**322**:1561–6.
- Antikainen R, Grodzicki T, Palmer AJ, Beevers DG, Coles EC, Webster J, *et al.* The determinants of left ventricular hypertrophy defined by Sokolow–Lyon criteria in untreated hypertensive patients. *J Hum Hypertens* 2003;**17**:159–64.
- Thrainsdottir IS, Hardarson T, Thorgeirsson G, Sigvaldason H, Sigfusson N. Survival and trends of occurrence of left ventricular hypertrophy, gender differences, 1967–92: The Reykjavik Study. *J Intern Med* 2003;**253**:418–24.
- Lokhandwala Y, Damle A. Left ventricular hypertrophy in hypertensive patients in Indian primary care: Prevalence and effect of treatment with sustained release indapamide. *Curr Med Res Opin* 2004;**20**:639–44.
- Hanevold C, Waller J, Daniels S, Portman R, Sorof J, International Pediatric Hypertension Association. The effects of obesity, gender, and ethnic group on left ventricular hypertrophy and geometry in hypertensive children: A collaborative study of the International Pediatric Hypertension Association. *Pediatrics* 2004;**113**:328–33.
- Fox E, Taylor H, Andrew M, Han H, Mohamed E, Garrison R, *et al.* Body mass index and blood pressure influences on left ventricular mass and geometry in African Americans: The Atherosclerotic Risk In Communities (ARIC) Study. *Hypertension* 2004;**44**:55–60.
- Koren MJ, Devereux RB, Casale PN, Savage DD, Laragh JH. Relation of left ventricular mass and geometry to morbidity and mortality in uncomplicated essential hypertension. *Ann Intern Med* 1991;**114**:345–52.
- Antoniucci D, Seccareccia F, Menotti A, Dovellini EV, Prati PL, Rovelli F, *et al.* Prevalence and correlates of echocardiographic determined left ventricular hypertrophy in 2318 asymptomatic middle-aged men: The ECCIS project. *Epidemiologia e Clinica della Cardiopatia Ischemica Silente. G Ital Cardiol* 1997;**27**:363–9.
- Gottdiener JS, Brown J, Zoltick J, Fletcher RD. Left ventricular hypertrophy in men with normal blood pressure: Relation to exaggerated blood pressure response to exercise. *Ann Intern Med* 1990;**112**:161–6.
- Burnier M, Phan O, Wang Q. High salt intake: A cause of blood pressure-independent left ventricular hypertrophy? *Nephrol Dial Transplant* 2007;**22**:2426–9.
- Koren MJ, Devereux RB, Casale PN, Savage DD, Laragh JH. Relation of left ventricular mass and geometry to morbidity and mortality in uncomplicated essential hypertension. *Ann Intern Med* 1991;**114**:345–52.
- Brown DW, Giles WH, Croft JB. Left ventricular hypertrophy as a predictor of coronary heart disease mortality and the effect of hypertension. *Am Heart J* 2000;**140**:848–56.

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