Original Article

Dietary factors as determinants of hypertension: A case control study in an urban Indian population

Vijayalakshmi Kodali[1] PhD, Mallikharjuna Rao Kodavanti[1] MSc, Prasanna Krishna Tripuraribhatla[1] PhD, Thummala C Raghu Ram[1] MD, PhD, Parvathi Eswaran[2] PhD and Kamala Krishnaswamy[1] MD, FAMP, FASC, FNASc

[1]National Institute of Nutrition, Indian Council of Medical Research, Hyderabad, Andhra Pradesh, India
[2]Avinashilingam Deemed University, Coimbatore, Tamil Nadu, India

Hypertension, an important risk factor for coronary heart disease (CHD), is often associated with certain dietary habits which can either adversely affect or decrease blood pressure. Several Western studies have documented the role of diet, especially excess energy, both quality and quantity of fat and salt, in the aetiopathogenesis of chronic diseases such as hypertension, diabetes and CHD. Indians are particularly susceptible to these chronic diseases. A study was therefore undertaken to investigate the role of dietary factors in relation to hypertension. A total of 158 newly diagnosed cases were selected from the out-patient department of the 1000-bed Osmania General Hospital along with 172 age and gender-matched controls. A detailed diet history was collected and validated. An energy adjustment method was adopted by transforming the data on a log scale as all the nutrients depended upon the intake of energy. A total of 86 hypertensives and 79 controls participated in the study. Among those classified as hypertensives, men reported higher intakes of dietary fat and salt while women reported higher intakes of dietary protein and salt. Risk calculated by Odds ratio revealed that higher intakes of fat, protein and salt increase the risk for hypertension. Multivariate stepwise logistic regression identified salt as the risk factor in men and protein as the risk factor in women. These results suggest a role for dietary fat, protein and salt in hypertension.

Key words: hypertension, coronary heart disease, salt, protein, dietary fat, Coimbatore, India.

Introduction

While many Western countries are beginning to enjoy a decline in mortality from cardiovascular diseases, there are indications of an epidemic rise in diabetes, obesity and cardiovascular diseases in the developing world.[1] This could be because of the nutritional transition occurring in the developing world.[2]

Food balance data from the Food and Agriculture Organization (FAO) show that in Asian countries such as China, Indonesia, Malaysia and Thailand, the changes in energy intake are minimum but that there have been major changes in the consumption of animal products, sugars and fats.[2,3] Studies from India, including a survey conducted by the National Nutrition Monitoring Bureau (NNMB), have shown that there is a change in the quality of diet in Asian countries, particularly in urban India.[4–6]

The most prominent features of the changes in nutritional transition have been an increase in the dietary intake of fat and a decrease in the intake of complex carbohydrates and fibre accompanied by a significant decrease in physical activity. It has been suspected that this change in dietary habit and lifestyle is associated with certain chronic disorders such as diabetes, obesity and hypertension, thus enhancing the risk associated with coronary heart disease. However, there is a lack of systematic investigation relating to dietary pattern and these diseases in an Indian context. Hence, the present study was undertaken to assess the role of dietary factors in the risk assessment of hypertension.

Materials and methods

A case control approach was used to estimate possible risk factors for essential hypertension in a middle-income group of people attending the Government General Hospital at Hyderabad, Andhra Pradesh.

Subjects aged between 30 and 50 years attending the outpatient department of the hospital were classified as hypertensives (diastolic pressure above 95 mmHg on three consecutive occasions taken at an interval of 10 days) or normotensives (blood pressure (BP) < 140/90 mmHg). A total of 158 subjects were diagnosed as hypertensives and an equal number of normotensives were considered as controls. All subjects with known histories of hepatic or renal diseases and diabetes, along with those on any form of medication, were excluded from the study. Detailed information on personal habits, family history of chronic diseases and other relevant information was collected from the subjects. Dietary assessment was undertaken in a subsample of individuals (86 hypertensives and 79 controls) using the 24 h recall method.

Anthropometric measurements such as height, weight, waist-to-hip ratio and body composition were taken.[7,8] Lipid

Correspondence address: Dr Kamala Krishnaswamy,
Director, National Institute of Nutrition, Jamai-Osmania PO,
Hyderabad 500 007, Andhra Pradesh, India.
Tel: 91 40 701 8083; Fax: 91 40 701 9074
Email: icmrnin@ren.nic.in
Accepted 11 February 1999

profiles comprising cholesterol,[9] triglycerides[10] and high-density lipoprotein (HDL) cholesterol were estimated in fasting samples.[11] Glucose[12] and insulin were estimated in fasting as well as in post-prandial samples.[13] In the present paper, only the role of dietary factors in hypertension were explored.

Validation and energy adjustment

Home visits were undertaken and the dietary intake of each individual was assessed using the 24-h recall method.[14] A standardised instrument was used to elicit the recall of raw foods used for cooking and the volume of the cooked food for the entire family, as well as for collecting information on volume of cooked food consumed by the individual index person. The raw foods were then calculated from the recall using volumetric conversions. This 24-h recall method was validated against the average intake during 2 days in a week with the same recall method in 33 subjects.[15]

Twenty-four hour dietary intakes from the first and second days were assessed and intraclass correlations between the nutrients were calculated. The inter (S^2B) and intra (S^2W) individual variations were estimated from the two days of intakes using analysis of variance with a random individual effect and fixed effect model. Pearson's correlation coefficients were calculated between test-day intakes and 2-day average intakes.

The analysis of variance revealed that there was no significant difference between lst and 2nd day intakes. Hence, the data were pooled into a reference intake so that it could be compared with the test period intake. The intraclass correlations varied from 0.23 (lignin) to 0.56 (fat). The majority of the nutrients varied between 0.45 (magnesium) to 0.56 (fat), suggesting that the intra-individual variations were minimum and the intake of the test period was not significantly different from the reference period.

An energy adjustment method was adopted, as most of the nutrients are energy dependent, by transforming the data on a log scale.[16] The adjustment was done by replacing nutrient intake values with their respective residuals from the regression model, taking nutrient intake as the response and the total energy intake as the explanatory variable. The attenuation (correction for intra-individual variation) was done for the correlations of the adjusted nutrients between the 2-day period and the test period by multiplying by the factor $(1 + S^2W/S^2B)^{1/2}$ (Table 1). The majority of the nutrients when adjusted to energy varied from 0.45 to 0.68, which was considered a high correlation. When the correlations were corrected for attenuation they improved considerably.

Food composition tables were used to calculate the nutrient intakes of the subjects.[17] Total fibre and dietary fat were calculated from 'Dietary fibre in Indian diets and its nutritional significance' and 'Fats in Indian diets', respectively.[18,19]

Statistical analysis

All parameters including dietary intake were analysed separately for males and females. Because all nutrients are dependent upon energy, an energy adjustment method was adopted for the log transformed nutrients. The regression model was developed according to Puska *et al*.[20] The differences between cases and controls were tested using Student's *t*-test.

Table 1. Correlation coefficients between daily intake of nutrients between test period and reference period ($n = 33$)

Dietary variables (g)	Unadjusted	Energy adjusted	Corrected for attenuation
Energy (kJ)	0.64**	–	0.73
Protein	0.46*	0.28	0.33
Calcium	0.81**	0.68**	0.75
Iron	0.45*	0.32	0.38
Vitamin A	0.63**	0.47	0.56
Thiamin	0.32	0.004	0.005
Riboflavin	0.71**	0.57**	0.64
Niacin	0.44*	0.54**	0.65
Vitamin C	0.38	0.22	0.28
Fat	0.69**	0.41**	0.46
Magnesium	0.38	0.45*	0.54
Sodium	0.64**	0.52**	0.60
Potassium	0.45*	0.32	0.37
Fibre	0.56**	0.57**	0.65
Cellulose	0.63**	0.61**	0.71
Lignin	0.31	0.42*	0.54

* $P < 0.01$; ** $P < 0.001$.

Pearson's correlation coefficients were calculated to study the interrelationship between diet and clinical parameters, anthropometric and biochemical parameters. Odd's ratio was used to identify the risk factors and 95% confidence limits were calculated. Multivariate stepwise logistic regression was also undertaken to identify the risk.

Results

Dietary profiles of hypertensives and normotensives are given in Table 2. The intakes of milk and milk products, fats and salt were significantly higher in male hypertensives when compared with the respective controls. Similarly, significantly higher intakes of roots and tubers, flesh foods, milk and milk products, sugar and jaggery, and salt were observed among female hypertensives. However, there was no difference in the intakes of other food items among cases and controls of both genders.

No significant differences were observed in macro-nutrient intakes in either gender except for dietary fat ($P < 0.01$) among males and proteins ($P < 0.001$) among females (Table 3). In addition, the percentage of energy from protein was also significantly higher in female hypertensives than in controls. The intakes of saturated and monounsaturated fat were 46% and 50% higher, respectively, in male hypertensives when compared with the controls (Table 4). However, there was no difference in the polyunsaturated/saturated fatty acid (P/S) ratio and dietary cholesterol intake between cases and controls of both genders. Both hypertensives and normotensives were observed to derive equal amounts of energy from visible and invisible fat. A higher intake of fat energy was observed among male cases (22%) when compared with controls (18%) (Table 4).

The intake of total sodium was 24% higher ($P < 0.01$) in male hypertensives and 32% higher ($P < 0.01$) in female hypertensives when compared with the controls. No such differences were observed with respect to other minerals and vitamins (Table 5).

A significant correlation was observed between systolic and diastolic blood pressure and between dietary factors such

Table 2. Dietary profile of hypertensives and controls

Food groups (g)	Male		Female	
	Control (n = 35)	Case (n = 36)	Control (n = 44)	Case (n = 50)
Cereals	440 ± 24.8	406 ± 21.5	350 ± 16.4	364 ± 19.4
Pulses	47 ± 7.0	45 ± 8.3	41 ± 5.6	45 ± 7.4
Fruits	91 ± 15.5	108 ± 21.1	72 ± 12.6	90 ± 15.2
Green leafy vegetables	2 ± 1.6	7 ± 3.8	13 ± 4.7	18 ± 6.7
Roots and tubers	104 ± 18.4	149 ± 26.2	56 ± 9.6	90 ± 13.3*
Other vegetables	62 ± 15.6	43 ± 11.7	70 ± 12.6	91 ± 18.6
Nuts and oil seeds	1 ± 0.8	3 ± 1.7	3 ± 2.1	2 ± 1.2
Flesh foods	38 ± 9.7	72 ± 13.2*	33 ± 7.7	67 ± 10.6**
Milk and milk products	244 ± 27.1	266 ± 32.9*	184 ± 24.3	210 ± 25.8
Sugar and jaggery	25 ± 5.0	29 ± 6.6	31 ± 3.6	20 ± 2.4***
Fats and oils	25 ± 2.7	38 ± 5.1*	25 ± 2.3	23 ± 2.7
Salt	10 ± 0.9	13 ± 1.0*	7 ± 0.5	10 ± 0.8**

Values are mean ± SE. *P < 0.05; **P < 0.01; ***P < 0.001.

Table 3. Macronutrient intake of hypertensives and controls

Macronutrients	Male		Female	
	Control (n = 35)	Case (n = 36)	Control (n = 44)	Case (n = 50)
Energy (kJ)	10 460 ± 439	10 979 ± 686	8686 ± 343	9100 ± 435
Fat (g)	50 ± 3.3	68 ± 6.9**	45 ± 3.6	46 ± 4.1
Protein (g)	68 ± 3.8	72 ± 5.2	54 ± 2.6	67 ± 4.0***
Animal protein (g)	17 ± 1.9	23 ± 2.8	13 ± 1.6	19 ± 2.4
Vegetable protein (g)	52 ± 3.5	50 ± 3.3	41 ± 2.1	48 ± 2.7
Energy from protein (%)	11 ± 0.4	11 ± 0.3	10 ± 0.2	12 ± 0.3***
Fibre (g)	56 ± 4.6	52 ± 4.1	43 ± 2.7	49 ± 3.6

Values are mean ± SE; **P < 0.01 ***P < 0.001.

Table 4. Dietary fat intake of hypertensives and controls

Dietary fat	Male		Female	
	Control (n = 35)	Case (n = 36)	Control (n = 44)	Case (n = 50)
Total fat (g)	50.0 ± 3.3	68.0 ± 6.9**	45.0 ± 3.6	46.0 ± 4.1
Saturated fat (g)	13.1 ± 0.8	18.6 ± 1.7**	11.4 ± 0.9	12.7 ± 1.1
Mono-unsaturated fat(g)	17.7 ± 1.3	26.6 ± 3.0**	15.9 ± 1.2	18.0 ± 1.4
LA N6 (g)	11.2 ± 0.8	15.6 ± 1.8*	9.7 ± 0.6	11.2 ± 0.8
ALNA N_3 (g)	0.8 ± 0.1	1.1 ± 0.1*	0.7 ± 0.1	0.8 ± 0.1
PS Ratio	0.9 ± 0.1	0.9 ± 0.1	0.9 ± 0.1	1.0 ± 0.1
Invisible fat (%)	51.0 ± 2.7	46.0 ± 2.3	44.0 ± 2.4	49.0 ± 2.2
Visible fat (%)	49.0 ± 2.7	54.0 ± 2.3***	56.0 ± 2.4	51.0 ± 2.2
Fat energy (%)	18.0 ± 0.9	22.0 ± 1.1**	19.0 ± 1.1	18.0 ± 1.2
Cholesterol (mg)	107.2 ± 21.9	106.1 ± 14.0	74.8 ± 14.4	78.0 ± 14.7

Values are mean ± SE; *P < 0.05; **P < 0.01; ***P < 0.001; LA N6, linoleic acid; ALNA N_3, alpha-linolenic acid; PS, polyunsaturated/saturated fatty acid ratio.

as fat in males and protein and salt in females. However, no such associations were noted with energy or other dietary components in either gender (Table 6).

Similarly, correlations between dietary factors and anthropometric parameters revealed a significant association of energy intake with body weight, body mass index, waist-to-hip ratio and percentage body fat among males. However, among females such correlations were not found except for salt and protein intake (Table 7). In males, energy (P < 0.01), fat (P < 0.05) and salt (P < 0.05) intake showed a significant positive correlation with fasting glucose levels. Protein did not show a significant correlation with any of the biochemical parameters studied. Intakes of protein, fat and salt showed an inverse relationship with HDL levels. In females, the correlation between the dietary constituents and biochemical parameters were not consistent.

Energy and protein intake is significantly correlated with post-load insulin levels (P < 0.01); however, intakes of fat and salt were not found to correlate with any of the biochemical parameters studied. The absolute values for anthropometric and biochemical parameters have been reported earlier.[21]

Dietary factors examined by Odds ratio (OR) revealed a greater risk for hypertension with higher intakes of dietary fat (OR = 4.36) and salt (OR = 3.40) among males. However, among females higher intakes of protein (OR = 11.88) and

Table 5. Dietary intake of minerals and vitamins in hypertensives and controls

Minerals/vitamins	Male		Female	
	Control (n = 35)	Case (n = 36)	Control (n = 44)	Case (n = 50)
Minerals				
Sodium (mg)	4032±361	5004±392**	3023±212	3986±261**
Potassium (mg)	1325±140	1440±143	1042± 80	1292±122
Magnesium (mg)	587± 44	535±38	454± 28	508± 37
Calcium (mg)	725± 50	836±77	582± 47	676± 61
Iron (mg)	14±1.1	14±1.2	10±0.7	14±1.4*
Vitamins				
Vitamin A (µg) retinol	313±33	430± 80	338±66	370± 66
Thiamin (mg)	1.2±0.1	1.2±0.1	0.9±0.1	1.1±0.1
Riboflavin (mg)	0.9±0.1	0.9±0.1	0.7±0.1	0.8±0.1
Niacin (mg)	15±1.0	17±1.2	12±1.0	14±1.2
Vitamin C (mg)	67± 13	52±7	40±7	52±9

Values are mean ± SE; *P < 0.05; **P < 0.01.

Table 6. Correlation between dietary factors and blood pressure in males and females

	Male (n = 72)			Female (n = 94)		
	Systolic	Diastolic	Mean arterial	Systolic	Diastolic	Mean arterial
Energy	0.1528	0.0780	0.1113	0.1253	0.0943	0.1100
Fat	0.2776**	0.2599*	0.2729**	0.0895	0.0347	0.0584
Protein	0.0507	0.0199	0.0095	0.2943**	0.2441**	0.2724**
Salt	0.2114	0.2039	0.2113	0.2550**	0.2661**	0.2699**
Sodium : potassium ratio	0.0351	0.0201	0.0028	0.1530	0.1825*	0.1761*
Calcium	0.1302	0.0932	0.1109	0.0849	0.0829	0.0863

*P < 0.05; **P < 0.01.

Table 7. Correlation between anthropometric parameters and dietary factors in men and women

Anthropometric measurements	Male (71)					Female (94)				
	Energy	Protein	Fat	Salt	S : P ratio	Energy	Protein	Fat	Salt	S : P ratio
Weight	0.274**	0.131	0.167	0.100	0.257*	1.158[a]	0.251**	0.045	0.230**	0.032
BMI	0.251**	0.120	0.155	0.131	0.222	0.020	0.259**	0.050	0.261**	0.082
WHR	0.277**	0.148	0.231*	0.171	0.239*	0.075	0.174*	0.014	0.184*	0.029
Body fat (%)	0.395**	0.047	0.238*	0.266*	0.276**	0.163[a]	0.128	0.019	0.069	0.050

S : P, sodium : potassium ratio; BMI, body mass index; WHR, waist-to-hip ratio.
*P < 0.05; **P < 0.01; [a]0.05 < P < 0.10.

salt (OR = 3.48) were identified as risk factors for hypertension (Table 8). Multivariate stepwise logistic regression also identified salt and protein as the risk factors for hypertension in men and women, respectively.

Discussion

Dietary factors play an important and sometimes decisive role in cardiovascular diseases, especially hypertension. The findings of the present study suggest that protein, fat and salt are risk factors for hypertension. However, our study, in contrast to Puddey *et al.*, did not find an association between total energy intake and hypertension.[22] Differences in lifestyle factors such as physical activity could be one of the reasons for this variation.

In the present study, intake of excess fat was found to be an incriminating factor for men, while for women protein played a significant role. These findings support the observations made by Nara *et al.*, who suggested that a higher intake of protein results in an increase in blood pressure.[23]

Fat is an important factor, both quantitatively and qualitatively, as far as hypertension and coronary heart disease are concerned. Early studies from the West have shown that an increased intake of polyunsaturated fatty acids (PUFA) lowered the blood pressure to a significant extent.[20] Similarly, a study by Malhotra revealed that Indians from the north of India had a lower risk of hypertension when compared with those from the south of India because the diets of northern Indians tend to have a preponderance of short-chain fatty acids in comparison with the diets of southern Indians.[24]

In our study, Odds ratios calculated for risk analysis indicated that total dietary fat was a more prominent risk factor compared with other dietary macronutrients. Though saturated fat was higher by 5 g, the protective fatty acids such as monounsaturated fatty acid, linoleic acid (n = 6) and alpha linolenic acid (n = 3) also appeared to be higher, normalizing

Table 8. Odds ratio for hypertension in third tertile of dietary factors

Parameters	Male			Female		
	t_3	95% Confidence intervals		t_3	95% Confidence intervals	
		Lower	Upper		Lower	Upper
Energy	1.17	0.38	3.54	0.91	0.30	2.68
Protein	1.42	0.44	4.53	11.88	3.30	42.69
Fat	4.36	1.21	15.64	0.56	0.20	1.53
Visible fat (%)	1.91	0.64	5.63	2.02	0.68	6.00
Energy fat (%)	1.69	0.55	5.14	0.65	0.23	1.86
Salt	4.36	1.11	17.12	3.43	1.23	9.48
S : P ratio	3.27	0.70	15.29	2.33	0.79	6.84

Odds ratio > 1.5 is considered as risk. S : P, sodium : potassium ratio.

the P/S ratio. Hence, it is difficult to comment on the quality of fat in relation to hypertension in the present context. Significant differences in types of fat consumed, whether monounsaturated or saturated, were found to be related to the type of cooking oil used. For example, increased intakes of monounsaturated fats are mainly due to the use of groundnut oil and rice bran oil.

In Western studies, the consumption of cholesterol and serum cholesterol concentrations along with high blood pressure have been considered concurrent risk factors for the development of cardiovascular diseases.[25] Stamler suggested that the epidemiological associations between dietary lipids, serum cholesterol and incidence of coronary heart disease represent aetiologically significant relationships.[26] The present study, however, showed that cholesterol intake did not differ consistently between hypertensives and normotensives. It is likely that when the overall diet is vegetarian, which results in lower amounts of total fat and saturated fat consumed coupled with very low amounts of cholesterol, its ability to lower the blood cholesterol increases.

As already evidenced, the present study also revealed a positive association between salt intake and systolic, diastolic and mean arterial blood pressure.[27–30] However, there are reports indicating that in some populations with a more homogeneous diet a case control study would find that hypertensives and normotensives had a similar salt intake. In those populations, the distribution of hypertension would be explained by differences in genetic predisposition.[31]

Wright *et al.* showed that subjects with higher fibre intakes had significantly lower blood pressure than those with lower fibre intakes.[32] However, the present study revealed no differences in fibre intakes as the majority of those involved were vegetarians. Obviously, fibre in Indian diets is not causally related to the risk for hypertension. However, it is necessary to characterise the fibre and estimate the true intake of soluble fibre, which is an important component of Indian diets and has several beneficial effects. In conclusion, the present study identified dietary fat, protein and salt as risk factors for hypertension.

Acknowledgements. We thank Dr Vinodini Reddy, former Director, National Institute of Nutrition, for her keen interest and valuable suggestions. We also thank Mr A Nadamuni Naidu for his statistical expertise and Miss BVS Thimmayamma for valuable advice in dietary methodology. Our highest appreciation and acknowledgement is reserved for Mr P Krishnaswamy for his excellent technical assistance.

References

1. Wielgosz A. Behavioural change and nutrition programmes and poor nutrition and chronic disease – Part 1. Sub-committee on Nutrition News 1995; 13: 19–22.
2. World Health Organization. Obesity, preventing and managing the global epidemic. Report of a WHO Consultation on Obesity. Geneva, WHO/NUT/NCD/98.1, 1998.
3. Chen CM. Dietary guidelines for food and agricultural planning in China. Proceedings of the International Symposium on Food, Nutrition and Social Economic Development. Beijing: China Science and Technology Publishing House, 1991; 40–48.
4. National Nutrition Monitoring Bureau. Report of Repeat Surveys 1988–90. Hyderabad: National Institute of Nutrition, 1991.
5. Padmavati S. Epidemiology of cardiovascular disease in India. II. Ischemic Heart Disease. Circulation 1962; 25: 711–717.
6. Chada SL, Gopinath N, Shekhavat S. Dietary factors and urban rural incidence of coronary heart disease. Cardiothoracic J 1996; 2: 5.
7. Weiner JS, Lourie JA. Human Biology IBP Handbook No. 9. Oxford: Blackwell Scientific Publications, 1963.
8. Falson AR, Prineas RJ *et al.* Increased incidence of carcinoma of breast associated with abdominal adiposity in postmenopausal women. Am J Epidemiol 1990; 131: 794–803.
9. Zjatteis A, Zak B, Boyle AJ. A new method for the direct determination of serum cholesterol. J Lab Clin Med 1953; 41: 486–492.
10. Benstein M, Saneille J. Serum dosage rapid due to cholesterol like auxet B lipoproteins. Clin Chem Acta 1960; 5: 609.
11. Paster LB, Dunn RT. Stable reagents for determination of serum triglycerides by a colorimetric Hantzech Condensation Method. Clin Chem 1973; 19: 338–340.
12. Washko ME, Rice EW. Determination of glucose by an improved enzymatic procedure. Clin Chem 1961; 7: 542–548.
13. Loffler G, Weiss L. Radioimmunoassay of insulin in serum. In: Breker H, Hanel D, Krushkempu H, eds. Methods and hormone analysis. New York: John Wiley and Sons. 1976; 85–100.
14. Thimmayamma BVS. A handbook of schedules and guidelines in socio-economic and diet surveys. Hyderabad: National Institute of Nutrition, 1987.
15. Pietinen P, Hartman AM, Happa E *et al.* Reproducibility and validity of dietary assessment instruments. A self-administered food use questionnaire with a portion size picture booklet. Am J Epidemiol 1988; 128: 655–666.
16. Willet WC, Sampson L, Stamper MJ *et al.* Reproducibility and validity of a semi-quantitative food frequency questionnaire. Am J Epidemiol 1985; 122: 51–65.
17. Gopalan C, Ramasastry BV, Balasubramanian SC. Nutritive value of Indian foods. Revised and updated by BS Narasinga Rao, YG Deosthale and KC Pant. National Institute of Nutrition, Indian Council of Medical Research, Hyderabad, India, 1989.
18. Narasinga Rao BS. Dietary fibre in Indian diets and its nutritional significance. NFI Bull 1988; 9: 1–5.
19. Ghafoorunissa. Fats in Indian diets. NFI Bull 1989; 10: 1–5.
20. Puska P, Lacano JM, Nissimen A *et al.* Controlled randomised trial of the effect of dietary fat on blood pressure. Lancet 1983; 1: 1–5.

21. Vijayalakshmi K, Prasanna Krishna T, Raghu Ram TC, Mallikharjuna Rao K, Parvathi Eswaran and Krishnaswamy K. Abdominal adiposity and metabolic alterations in hypertension: A case control study. Asia Pacific J Clin Nutr 1997; 6: 180–185.
22. Puddey IB, Parker M, Beilin LJ *et al.* Effects of alcohol and caloric restriction on blood pressure and serum lipids in overweight men. Hypertension 1992; 20: 533–541.
23. Nara Y, Zhao GS, Huang ZD *et al.* Relationship between dietary factors and blood pressure in China. The Sino-Japan Cardiac Cooperative Res Group. J Cardiovasc Pharmacol 1990; 16 (Suppl. 8): S40–S42.
24. Malhotra SL. Dietary factors causing hypertension in India. Am J Clin Nutr 1970; 23: 1353–1363.
25. Glueck CJ. Dietary fat and atherosclerosis. Am J Clin Nutr 1979; 32 (Suppl.): 2703–2711.
26. Stamler J. George Lyman Duff Memorial Lecture. Lifestyles, major risk factors, proof and public policy. Circulation 1978; 58: 3–19.
27. Takahashi E, Sasaki N, Takeda J, Ito H. The geographic distribution of cerebral hemorrhage and hypertension in Japan. Hum Biol 1957; 29: 139–166.
28. Fodor JG, Abbott EC, Rusted IE. An epidemiologic study of hypertension in Newfoundland. Can Med Assoc J 1973; 108: 1365–1368.
29. Law MR, Frost CD, Wald NJ. Dietary salt and blood pressure. J Hypertens 1991; 9 (Suppl.): S37–S41.
30. Muntzel M, Drueke T. A comprehensive review of the salt and blood pressure relationship. Am J Hypertens 1992; 5: 1S–42S.
31. Altschul AM, Grommet JK. Sodium intake and sodium sensitivity. Nutr Rev 1980; 38: 393–402.
32. Wright A, Burstyn PG, Gibney MJ. Dietary fibre and blood pressure. BMJ 1979; 2: 1541–1543.