NFI BULLETIN

Bulletin of the Nutrition Foundation of India

Volume 33 Number 1 January 2012

Evolutionary aspects of diets in the context of current chronic diseases

Kamala Krishnaswamy

The science of nutrition is a dynamic area with several challenging issues for nutritionists, public health scientists, clinicians and food technologists. From time immemorial, the relationship between the quality and quantity of diets on the one hand and human health and well being on the other has been well recognized. The rising burden of noncommunicable diseases (NCDs) is due to the health transition that is a characteristic of the current era all over the world. Most of the epidemiological evidence suggests that chronic diseases probably operate on a continuum from birth to adulthood, with both genetic and environmental factors playing significant roles in their inception, progression, and manifestations. This article captures the prehistoric hunter-gatherer (HR) diets, and examines the changing scenarios in respect of life styles, particularly diet and physical activity, and their association with disease patterns, with specific reference to India.

It is likely that we are better adapted physiologically to the diets of our ancestors during the millions of years of Hominid evolution than to the diets we have been eating since the dawn of the agricultural revolution 10,000 years ago and subsequently after the industrial revolution only 200 years ago[1]. Chronic degenerative diseases characterised by atherosclerosis, inflammation, and insulin resistance reflect discordance between ancient genes and current diets. The dietary guidelines are similar for most of these disorders with a few exceptions. There is a need for a unifying dietary strategy[1]. Recently, foetal/ metabolic programming in-utero in response to poor maternal diets, resulting in "metabolic syndrome" (a cluster of risk factors, which leads to chronic diseases in later life) has become well recognized. The central pathogenic mechanism is insulin resistance due to both thrifty genotype of our ancestors and thrifty phenotype due to metabolic programming.

Our ancestral genes, foods and nutrients

Genes

The genus Homo belongs to the paleolithic era, dating back about 2 million years. It was only about 10,000 years ago that early humans started to cultivate plants (predominantly cereals) and domesticate animals. During the earlier period, food had been a limiting factor for population growth. Paleolithic (Stone Age) diets of the hunter-gatherer society had sculpted the human gene for diets which generally favored fat preservation because their lifestyle consisted of alternate feasting and famine periods, and vigorous physical activity with far more energy expenditure than what we currently encounter. During the Stone Age, the insulin-resistant genotype evolved to confer survival and reproductive advantage. Researchers in human genetics and evolutionary theorists[1] have come to a consensus that the current generation of humans is almost identical, genetically, to our ancestors, given that evolution at the molecular level is highly conservative[2]. Paleoanthropologists have shown that there are similarities in the genetic make-up of Finns and Australian aborigines, although they live on opposite sides of the world[2]. The genomic pattern has not changed much since the emergence of Homo sapiens sapiens about 40,000 years ago. While the Paleolithic Age lasted for two million years of our evolutionary experience, cereals, which form the bulk of current diets, have interacted with the human genome only for the past 10,000 years, which apparently is too short a period (<500 generations) for any genetic adaptations to have evolved[3]. The development of agriculture 10,000 years ago has had a minimal influence on genes[4].

Foods and Nutrients

The prehistoric foods of hunter-gatherers, also referred to as the "Stone Age/Cave Man" diet, consisted of terrestrial mammals (wild animals), fish and other aquatic foods, birds, and wild plants. After branching out in the evolutionary tree from the chimpanzees, our prehistoric ancestors (Homo erectus) obtained meat through foraging, scavenging and hunting. Simultaneously, they developed simple stone tools for digging, and were able to add roots and tubers to their diets. Such diets were far healthier and provided the much needed micronutrients which increased their life spans and reduced infant mortality. During this period, their

CONTENTS

heights and brains sizes also increased, high-quality animal foods being probably one of the major driving factors for brain growth (encephalisation). In addition, they experienced alternating periods of abundance and scarcity; this led to the adaptive metabolic trait of quick insulin release and lipid synthesis to provide energy during periods of starvation and for reproductive performance. The nomadic lifestyle, followed later by agricultural activity, called for vigorous physical activity. This adaptive metabolic trait has turned into a pathological pitfall due to the sharp reduction in physical activity in current lifestyles.

The nutrient content of our ancestors' diets was high in animal protein and relatively low in carbohydrates, but provided adequate fibre and limited animal fat. Large quantities of animal remains are still found in human habitations dating back 1.8-1.6 million years. In those early periods, meat actually accounted for more than 50% of the diets. With the changing climate, meat protein in diets began to be slowly replaced by wild plants. Humans lived on a variety of uncultivated plants. Tools such as mortars, pestles, and grinding stones were developed to process vegetable matter. The strontium levels in bone in human fossils indicate consumption of vegetable matter. The hunter-gatherers moved from place to place and cultivated only virgin soils; hence plants were rich in micronutrients. The phytic acid content was low and hence the bioavailability of micronutrients was better, and the early humans were tall, muscular, and robust[5]. Currently information is available on as many as 329 different wild plants and game animals consumed by early humans[6]. The existence of fossilized fruit pits, nuts, fish bones, and shells bears evidence of the eating patterns of the humans of those times.

The dawn of agriculture introduced cereals as sources of energy and protein. However, the time line of cereal consumption represents less than 1% of Homo evolutionary time[7]. After the advent of agriculture, our ancestors were shorter, had skeletal manifestations of suboptimal nutrition, and experienced high infant mortality. It follows that modern Man's nutritional requirements reflect evolutionary experience and adaptations over millions of years. As humans progressed, protein intake again went up due to sufficiency of supply, and they became as tall as their ancestors on meat diets had been. Studies of many of the hunter-gatherer societies that have survived to the present day suggest that our ancient ancestors obtained about 30% of dietary energy from proteins, 35% from fats, (with saturated fats contributing about 7.5% of total energy) and 35% from carbohydrates. Trans-fatty acids were negligible in the diet, being present mainly in mother's milk and in the meat of certain herbivores. Even these were metabolically converted to conjugates of linoleic acid, which is anti carcinogenic and antiatherogenic[8]. Polyunsaturated fat intake (including long-chain PUFAs) was high, with the n-6:n-3 ratio approaching 2:1. Cholesterol consumption was about 400-500 mg/d from lean meat. The nutritional quality of their animal food was totally different from that of modern-day meat from domesticated animals.

The latter contains more fat (25-30% vs 3.9%) including more saturated fat, because of cattle feeding and rearing practices, with long-chain fatty acids being rare whereas wild animal meat had about 4% of long-chain polyunsaturated fats, providing fewer calories and more protein per unit weight[7]. Similarly, the wild vegetable foods provided more protein, while domesticated plants have more starch. Again, potassium content of the early diets was high while sodium was low, the ratio of potassium to sodium being 16:1; calcium content represented an intake of 1500 mg/day, and vitamin C was also high. There were hardly any refining techniques at that time, and calculations reveal that fiber consumption would have been in the range of 45-47g. These diets were more base-yielding than current diets.

The carbohydrate intake of these early humans was only 35% of the total energy intake, with honey contributing 2-3%[9].It is evident that the current diets are very different in composition and quality, with no corresponding changes in metabolic and physiological processes. Macro-, micro- and phytonutrients in human diets have significantly changed with the advent of agriculture and animal husbandry, leading to changes in foods, feeds, and lifestyles. Sedentary lifestyles (resulting from mechanisation) have reduced obligatory physical exertion, while the intake of salty, sugary, and fatty processed foods results in rapid glucose release and positive energy balance. Thus, whereas evolutionary enrichment of thrifty genes enhanced the chances of survival in the past when food supplies were meagre and available only intermittently, it has become detrimental in contemporary situations of greater availability of food and sedentary lifestyles.

Investigations on Paleolithic diets

In order to generate more robust scientific data, experimental intervention studies were carried out on simulated Paleolithic diets. In a metabolically controlled study in non-obese subjects with stable body weights (albeit with sedentary lifestyles), two diets were compared, namely, their habitual diets (3 days) and a paleotype diet (15days)[10]. The usual diet had a calculated K/Na ratio of 0.6±0.3;18% of the calories were from protein, 44% from carbohydrates, and 38% from fats. The Paleolithic diet had a K/Na ratio of 4.3 and contained 30% of calories from proteins, 38% from carbohydrates, and 32% from fat (mainly unsaturated). Meat, fish, poultry, eggs, fruits, vegetables, tree nuts, canola oil, mayonnaise, and honey were included in the Paleo phase of the diet study. Dairy products, legumes, cereals grains, sugar, juices, and potatoes were excluded. The Paleolithic diet resulted in:

- a significant decrease in the mean diastolic blood pressure (BP) and brachial artery reactivity.

- reduction in total cholesterol and low low density lipoprotein (LDL) cholesterol by 18 and 22%, respectively.

A 3-month crossover study in diabetic individuals compared a Paleolithic diet with a currently recommended diabetic diet. The Paleolithic diet resulted in lower fasting blood glucose, HbA1c, triglycerides, systolic and diastolic pressure, weight, and waist circumference, and higher high density lipoprotein (HDL), as compared to the recommended diabetic diet[11]. Similar observations were made in patients with ischemic heart disease in comparison with Mediterranean diets[12]. Three weeks of a Paleolithic diet significantly reduced weight, BMI, waist circumference, systolic blood pressure (SBP) and plasminogen activator inhibitor-1 (PAI-1)[13]. Deficiency of potassium base in modern diets increases the net systemic acid load. Loss of bone is a consequence of metabolic acidosis which, with age-related decline in kidney function, can impact bone mass (osteoporosis) and muscle mass. The Paleolithic diet can apparently be an excellent preventive prescription even for osteoporosis[14].

Chronic diseases in India

Epidemics of diet-related chronic diseases are presently emerging as serious health disorders in most of the

developing countries. Demographic changes, changing economies, urbanization/migration, mechanization, and industrialization, developments in science and technology (modernisation), rapid changes in dietary habits, and globalization have fuelled the rising epidemics of chronic disorders, collectively resulting in the current "health transition". The prevention and control of these disorders is becoming a public health challenge. A total of 57 million deaths occurred in the world during 2008 of which 36 million (63%) were due to NCDs[15]. The number of deaths from cardiovascular diseases (CVD) annually in India is projected to rise from 2.26 million in 1990 to 4.77 million in 2020[16]. Overweight, obesity and physical inactivity are important determinants of metabolic abnormalities in urban and rural India, leading to increase in blood pressure, abnormal lipid patterns and enhanced resistance to insulin[17]. The metabolic patterns raise the risks of coronary heart disease, stroke, diabetes mellitus and some forms of cancer.

Coronary heart disease (CHD) and diabetes occur at an earlier age in Indians than in the populations in Western Countries[18,19]. In adults over the age of 20 years, the estimated prevalence of CHD was about 3-4% in rural areas and 8-10% in urban areas, representing a two-fold rise in rural areas and a six-fold rise in urban areas between the years 1960 and 2002[20], with the risks becoming relevant at a younger age[21]. A meta-analysis of studies on stroke indicated a prevalence rate of 1.54/100022. The proportion of strokes in younger adults is also high. The prevalence rate for hypertension is 164/1000 in urban areas and157/1000 in rural areas[22]. India is known as "the diabetes capital of the world". Over the next decade the number of diabetic patients is expected to reach 200 millions.

The incidence rate for cancer in males is 71/100,000 and in females it is 83/100,000. Prevalence rates, mortality, DALYS and disease burden relating to several of the chronic diseases appear to warrant the attention of policy makers, public health workers, primary care providers, industry, and the civil society. The triad of (i) low birth weight and stunting due to malnutrition in early life, (ii) availability of energy-dense foods at a later age in life, and (iii) sedentary habits result in escalation of obesity and changes in body composition, specifically, in respect of fat deposition, insulin resistance, and diet-related chronic diseases. The risk factors for chronic disorders are high blood pressure, high blood cholesterol (LDL) and triglycerides, low HDL cholesterol, overweight and central obesity, physical inactivity, inadequate intake of vegetables and fruits, and the use of tobacco and alcohol. In addition, high homocysteine concentrations impair endothelial function, increase oxidative stress, impair methylation reactions, and alter protein structure. Deficiencies of micronutrients (such as vitamin B_{12}) and omega fatty acids may exaggerate endothelial dysfunction.

Metabolic syndrome

Indian mothers, especially rural ones, are small in stature, having been undernourished over generations. Indian babies are among the smallest in the world. It is now well recognized that low birth weight (due to foetal programming in response to maternal malnutrition) and unsatisfactory growth in the early years of life may contribute to the development of chronic diseases in adult life[23]. Metabolic syndrome is widely prevalent in Asia (India accounting for 30%) and is associated with a higher risk of CVD and diabetes[24]. Skeletal muscle mass also plays an important role in insulin resistance / sensitivity[25]. South Asians have lower muscle mass and higher percentage body fat and central obesity as compared to their Western counterparts[26,27].

The foetal genes are apparently programmed by the environment in utero. Maternal undernutrition mimics starvation syndrome and, through epigenetic mechanisms, alters gene expressions. The current diabetes epidemic is attributed to a "thrifty genotype"[28]. However, the theory of the developmental origin of health and disease attributes metabolic syndrome or insulin resistance (IR) to foetal adaptations to inadequate intrauterine nutrition; this is known as the "thrifty phenotype" hypothesis[29,30]. During pregnancy and again during critical windows of development, the foetus is susceptible to the effects of maternal undernutrition. The "thin-fat" Indian babies, with enhanced adiposity and insulin resistance at birth, are predisposed to diabetes in later life[31]. Children born to mothers with high folate and low Vitamin B_{12} concentrations had the highest insulin resistance[32] at 6 years of age. The epigenetic mechanisms that determine the phenotype are DNA methylation and acetylation of the histones. The thrifty genotype and thrifty phenotype hypotheses, though not mutually exclusive, are certainly inadequate to explain the IR which characterizes several conditions and populations. Given that brain growth is preserved (to maximize function) in preference to muscle mass, it has been suggested[33] that insulin resistance is more likely to have evolved as "a switch in reproductive and sustenance strategies than energy homeostasis alone". Irrespective of hypotheses, it is evident that low birth weight predisposes to adiposity, IR and cardiometabolic risk factors earlier in adult life[34,35,36].

These relationships may be more complicated than envisaged: rapid weight gain/BMI during infancy, childhood, and adolescence is strongly related to metabolic syndrome, whereas impaired glucose tolerance is associated with rapid BMI gain in later childhood and adolescence, but with lower BMI in infancy[35,36]. Birth weight and BMI gain during infancy and early childhood predict adult lean mass more strongly than adult adiposity. Greater BMI gain in late childhood and adolescence predicts increased adult adiposity and central obesity. Subjects from a Delhi cohort, for whom anthropometric data had been recorded serially, were studied at a later date as young adults. Nearly two-thirds were overweight as per the Asian cutoff value. Even at the relatively young age of 30 years, 15.2 percent had impaired glucose tolerance and 4.4% had diabetes.

The highest prevalence of impaired glucose tolerance and diabetes was among subjects who had been in the lowest tertile of BMI at the age of 2 years and the highest at the age of 12 years[36]. Children with the earliest adiposity rebound (5 years of age or younger) had the highest body-mass index in later childhood, and this difference was maintained even in adulthood[37]. Indians are also more prone to hypertriglyceridemia, an independent risk factor for coronary heart disease that is related to rapid weight gain and BMI during infancy, childhood, and adolescence[38]. Data from cohort studies in Brazil, Gautemala, the Philippines, and India, showed that undernutrition in mothers (short stature) results in growth retardation in their offspring, both intrauterine and in the early post-natal period. The height in adulthood is positively related to birth weight and length. Impaired foetal nutrition results primarily in long-term deficits in lean mass rather than in fat mass[39]. It is essential to understand critical periods of development of fat mass and lean tissue and be aware of the negative consequences of rapid weight gain in late childhood. A recent study from South

India indicated that nearly half of urban adults were obese (including central obesity) and were potentially at risk of developing diabetes and cardiovascular diseases[40]. Insulin resistance syndrome evolves continuously over an individual's lifetime. A recent study in the Delhi cohort indicated that the prevalence of cardiovascular risk factors is very high between the ages 29-36 years[41]. Protein-calorie supplements given to pregnant women and children in a public health programme had a positive impact on the offspring, studied at 13-18 years of age. The participants from the intervention villages were 14 mm taller than the controls (P=0.007). These children had more favourable measures of insulin resistance and arterial stiffness[42]. In order to tackle the rising prevalence of cardiovascular disease and IR in India, it is important to use interventions to improve foetal / early child nutrition to promote growth upto 2 years of age.

Diet and chronic diseases-the Indian scenario

Cardiometabolic risk factors

Diet in relation to chronic diseases has not been well investigated in India. There are very few prospective studies on diet and disease. Dietary habits in India are diverse and need to be studied both in relation to risk factors and disease prevalence. In a recent study on diet and cardiometabolic risk factors across several regions in India, interesting observations were made[43]. The investigators tried to arrive at dietary patterns and explore multivariate-adjusted associations between dietary patterns and abdominal adiposity, hypertension and diabetes. The data, adjusted for key demographic and lifestyle confounders, indicated that, across all regions, diets that were characterized by consumption of dairy foods, fried snacks and sweets, appeared to be positively associated with abdominal adiposity, whereas those characterized by intake of vegetables and pulses, were inversely related to diabetes and hypertension. Another study showed that diets rich in vegetables and mustard oil contributed to lowering the risk of IHD among Indians[44]. The prevalence of risk factors was high in rural India as well [45]. In the lower socio-economic groups, the use of tobacco and alcohol were high and the intake of fruit and vegetables was low. Risk factors were more prevalent among South Indians than among North Indians. Rural-to-urban migration was associated with enhanced intake of fat, sugar, and dairy products, but also of vegetables and fruits[46].

Diabetes

The prevalence of type 2 diabetes parallels the increase in obesity rates among Indians. This may be attributed to increasingly sedentary lifestyles and consumption of foods high in calories, particularly refined carbohydrates and fats. Among Chennai urban adults, white rice is the major cereal in the diet (76% of the total refined cereals, mean 253 g/day) and represents almost one-half of the daily calorie intake and 66% of the total dietary glycemic load (GL). In South India, refined grains are mainly polished rice, refined wheat flour (white flour), semolina, and a little ragi (finger millet) flour[47,48]. Traditional diets with unrefined foods, whole grains, handpounded and unpolished brown rice and whole wheat flour could possibly explain the lower rates of CVD[49] and type 2 diabetes mellitus in India in the 1960s-70s[50]. It is observed that high-carbohydrate South Indian diets, predominantly derived from refined grains (64% of total carbohydrates), are associated with higher triglycerides, lower HDL-C and the risk of metabolic syndrome and Asian Indian phenotype, characterized by higher glucose intolerance, dyslipidemia and central obesity[51]. The urban component of the Chennai urban / rural epidemiological study had also shown that higher fruit and vegetable intake was inversely associated (48% protection) with cardiometabolic risk factors such as high waist circumference, BMI, total cholesterol, and LDL cholesterol, independent of age, sex, and smoking and alcohol habits[52,53]. However, the mean intake of fruits and vegetables was lower (265g/day) than the recommended intake of 400g. Similarly, fish consumption was also low (20g/day) in this population. In a recent study, PUFA intake was observed to be significantly higher in the hyperinsulinemic group than in the normal insulinemic group (p = 0.021) [54]. Higher intake of PUFAs was associated with higher fasting serum insulin levels (OR: 2.2, CI: 1.2–4.1). When omega 6 fatty acid intake was less than 3 energy percent, there was no hyperinsulinemia.

Salt and hypertension

Salt intake promotes sodium retention and extracellular fluid volume expansion, thereby increasing blood pressure. Recent studies show that salt intake of more than 5-6 g/day elevates blood pressure in both urban and tribal populations[55,56].

Cancer

There is mounting scientific evidence to indicate that unhealthy diets and physical inactivity play an important role in causing cancers[57]. There are very few studies in India on the role of diet in cancer. A study from Bombay indicated that cabbage-eaters of both sexes had a 50% reduction in the risk of colorectal cancers as compared to those who did not eat cabbage[58]. Sprout eaters also had a 30–50% reduction in this risk. There was a 1.6-fold higher risk among men who ate 'dry fish' as compared to those who did not eat dry fish (OR = 1.6), whereas eaters of fresh fish had a 40–70% lower risk as compared to those who did not eat fresh fish. Studies on the dietary correlates of breast cancer have yielded inconsistent results. Vegetarianism has not been shown to protect against cancer. However, in Kerala, a non-vegetarian diet was associated with an increased risk of breast cancer(OR 1.82) [59]. Some early studies relating to a limited number of cases [60,61] with upper aero digestive tract cancers suggested a protective role for vegetables and fruits[61]. A prospective case–control study on stomach cancers identified rice, spicy food, chillies and hot food (high temperature) as risk factors[62]. The worldwide literature on diet and physical activity clearly indicate that obesity, body fatness, central obesity, and the consumption of red meat, alcohol and moldy grains are aetiologically related to several cancers, whereas plant foods such as whole grains, pulses, vegetables, fruits, micronutrient-rich foods and physical activity (1.6 PAL) are protective, along with salt intake limited to less than 6 gm/day. Other protective measures suggested are exclusive breast feeding for the first 6 months of life] and complementary feeds after 6 months.

Economic aspects

In India the year 2005 alone, the economic impact of premature deaths due to heart disease, stroke and diabetes on national income was estimated to be 9 billion dollars, and was projected to increase to 237 billion dollars (1.5% of GDP) by 2015[63]. The out-of-pocket health expenses incurred by households increased from 31.6% in 1995 to 47.3% in 2004. Modelling studies have estimated that, if non-communicable diseases (NCDs) were completely eliminated in India, the estimated annual GDP would be 4-10%[64]. NCDs result in losses to national productivity and push several millions of people below the poverty line. The

deaths of individuals in mid-life, apart from representing economic loss, have a devastating effect on families.

Prevention

The prevention and control of NCDs, 'the invisible" epidemic, is a major public health challenge for India in the twenty-first century[65]. Given the magnitude of the problem, the public health responses should be strong, committed and focused both on primary and secondary prevention. It is necessary to address the changes in lifestyles and behaviours. However, developing countries including India are still grappling with undernutrition and its impact on human growth and development, and also with infectious diseases. Extending health care facilities throughout an individual's life span, with emphasis on the reproductive phase in women, can ameliorate diseases at both ends of the spectrum. The prevention of childhood/adolescent obesity should be a priority.

A systematic approach to prevention and control is the need of the hour. Comprehensive and integrated approaches cutting across common risk factors and across the spectrum of disorders can impact the pathogenesis of chronic ailments. It is said that 80% of heart disease, strokes and diabetes can be prevented. WHO, therefore, urges investment in prevention of chronic diseases, possibly preventing 36 million premature deaths in the next 10 years, mostly in those under the age of 70 years[15]. Population-wide interventions are likely to reduce risk factors, and early detection and timely treatments can impact mortality and morbidity. Therefore, it is important to address both physical and metabolic abnormalities using population-based and risk-based approaches. Unhealthy dietary practices, physical inactivity, positive energy balance, overweight and obesity, and the use of tobacco and alcohol are very much amenable to prevention. Even the metabolic risk factors can be controlled with diet, physical activity and drugs. Dietary and physical activity guidelines for prevention of diseases, and adequate primary and secondary health care are effective approaches for tackling NCDs.

The guidelines on assessment and management of cardiovascular risk for medical officers developed under the Government of India – WHO Collaborative Programme [66] (2008-2009) document the steps for prevention and control of NCDs. With the help of the WHO/ISH risk prediction chart developed for the Indian population, decisions can be taken for appropriate prevention and management of these disorders even in primary health centres. The guidelines help to identify those at high risk of cardiovascular disease, and to motivate persons to change their life styles and, where appropriate, to take drugs. Dietary counselling must emphasize the following:

- maintain BMI between 18.5-23
- shift from saturated fats and transfats to polyunsaturates (mono and omega three fats),
- change from sugars and carbohydrates with high glycemic index (refined grains) to high-fibre and complex carbohydrates (whole grains), and
- increase the consumption of fish, nuts, vegetables and fruits (phytonutrients).

Inclusion of 400-500g of vegetables /fruits per day will reverse the potassium:sodium ratio in the diets, reduce blood pressure, and improve bone health. It has been reported that the impact of dietary approaches to stop hypertension (DASH), namely diets low in salt, dairy fat and sugar and high in fruits and vegetables, whole grains, poultry, fish, and nuts, is comparable to the impact of antihypertensive drugs[67].

Convergent and coherent policies and programmes with consensus on strategies essential for an enabling environment include:

- appropriate policies on health, food, agriculture, nutrition, education, industry and commerce, urban and rural development and social welfare with strong political commitment;
- advocacy for decision making and to alter public perceptions;
- appropriate legislation relating to food pricing, subsidies, and food labeling;
- infrastructural and other facilities for promoting physical activity;
- the food industry's willingness to promote healthy food choices;
- school-based health programmes and incentive-based workplace interventions;
- primary healthcare facilities, human resource development and capacity building;
- harnessing available resources for management of disease risks; and
- the setting up of a public health cadre with career opportunities, and restructuring of the health care system.

Conclusions

Epidemiological transition brought about by several developments has affected all walks of life. NCDs further enhance social inequity. Existing evidence suggests that a mismatch between our Paleolithic genome and our modern diets and lifestyles is playing an important role in health transition, resulting in obesity, cardiovascular diseases, hypertension, diabetes and cancer. Foetal / metabolic programming due to maternal undernutrition compounds the issue further. Availability and accessibility of energy-dense refined foods, coupled with reduced physical activity predisposes to obesity/central obesity, a forerunner of several chronic disorders. . Pharmacological and technological advances cannot replace healthy diets and physical activity as the means to safeguard against both under- and overnutrition. The 21st century offers both a challenge and an opportunity for developing countries as they embark on the task of controlling modifiable risk factors and tackling the emerging epidemic of chronic diseases.

The author is Former Director, National Institute of Nutrition, Hyderabad and Former President, Nutrition Society of India. This article is based on the C Ramachandran Memorial Lecture that she delivered on 29th November 2011 at NFI.

References

1 Eaton SB. The ancestral human diet: what was it and should it be a paradigm for contemporary nutrition? Proc Nutr Soc. Feb;65(1):1-6, 2006.

2: Neel JV. When some fine old genes meet a 'new' environment. World Rev Nutr Diet. 1999;84:1-18.in Evolutionary Aspects of Nutrition and Health: Diet, Exercise, Genetics and Chronic Disease. World Review of Nutrition and Dietetics, vol. 84. Edited by A. P. Simopoulos. Basel: Karger. 1999.

3: Eaton SB, Eaton SB 3rd. Paleolithic vs. modern diets—selected pathophysiological implications. Eur J Nutr. Apr;39(2):67-70, 2000.

4. Klein RG. The Human Career. Human Biological and Cultural Origins The

Quarterly Review of Biology Vol. 65, No. 2, pp. 257-258, 1990.

5. Smith P, Bloom RA, Berkowitz J. 1984a. Diachronic Trends in Humeral Cortical Thickness of Near Eastern Populations. Journal of Human Evolution 13: 603-611, 1984.

6. Eaton SB, Eaton SB 3rd, Konner MJ et al. An evolutionary perspective enhances understanding of human nutritional requirements. J Nutr. Jun;126(6):1732-40, 1996.

7. Eaton SB, Konner M. Paleolithic nutrition. A consideration of its nature and current implications. N Engl J Med. Jan 31;312(5):283-9, 1985.

8. Food and Nutrition Board, Institute of Medicine. Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids. Washington, DC: National Academy Press; 2002.

9. Cordain L, Miller JB, Eaton SB et al. Plant-animal subsistence ratios and macronutrient energy estimations in worldwide hunter-gatherer diets. Am J Clin Nutr. 2000 Mar;71(3):682-92, 2000.

10. Frassetto LA, Schloetter M, Mietus-Synder M et al. Metabolic and physiologic improvements from consuming a paleolithic, hunter-gatherer type diet. Eur J Clin Nutr. Aug;63(8):947-55, 2009.

11 Jönsson T, Granfeldt Y, Ahrén B et al. Beneficial effects of a Paleolithic diet on cardiovascular risk factors in type 2 diabetes: a randomized cross-over pilot study. Cardiovasc Diabetol. 16;8:35, 2009.

12. Lindeberg S, Jonsson T, Granfeldt Y et al. A Palaeolithic diet improves glucose tolerance more than a Mediterranean-like diet in individuals with ischaemic heart disease. Diabetologia; 50(9):1795-1807, 2007.

13. Osterdahl M, Kocturk T, Koochek A et al. Effects of a short-term intervention with a paleolithic diet in healthy volunteers. Eur J Clin Nutr ; 62(5):682-685, 2008.

14: Frassetto L, Morris RC Jr, Sellmeyer DE et al. Diet, evolution and aging--the pathophysiologic effects of the post-agricultural inversion of the potassium-to-sodium and base-to-chloride ratios in the human diet. Eur J Nutr.; Oct;40(5):200-13, 2001.

15. WHO. Global status report on noncommunicable diseases, Geneva,WHO, 2011.

16. Murray CJ, Lopez AD. Alternative projections of mortality and disability by cause 1990-2020: Global Burden of Disease Study. Lancet; 24;349 (9064):1498-504, 1997.

17. Enas EA, Mohan V, Deepa M et al. The metabolic syndrome and dyslipidemia among Asian Indians: a population with high rates of diabetes and premature coronary artery disease. J Cardiometab Syndr.; 2(4):267-75, 2007.

18. Xavier D, Pais P, Devereaux PJ et al. CREATE registry investigators. Treatment and outcomes of acute coronary syndromes in India (CREATE): a prospective analysis of registry data. Lancet. Apr 26;371(9622):1435-42, 2008.

19. Ramachandran A, Snehalatha C, Latha E et al. Rising prevalence of NIDDM in an urban population in India. Diabetologia. Feb;40(2):232-7, 1997.

20. Gupta R, Joshi P, Mohan V et al. Epidemiology and causation of coronary heart disease and stroke in India. Heart; 94: 16–26, 2008.

21. Ajay VS, Prabhakaran D. Coronary heart disease in Indians: Implications of the INTERHEART study Indian J Med Res; 132 (5): 561-566, 2010.

22. ICMR , Assessment of burden of noncommunicable diseases in India. New Delhi, 2006.

23. Barker, DJP. Mothers, Babies and Health in Later Life. 2d ed. Edinburgh, UK: Churchill Livingston, 1998.

24. Misra A, Misra R, Wijesuriya M et al. The metabolic syndrome in South Asians: continuing escalation & possible solutions. Indian J Med Res.;125(3):345-54, 2007.

25. Kurpad AV, Ramprakash S, Pai P et al. Role of muscle in fat/ glucose metabolism and Insulin resistance. Nutrition foundation of India bulletin; 28,1-6, 2007.

26. Banerji MA, Faridi N, Atluri R et al. Body composition, visceral fat, leptin and insulin resistance in Asian Indian men. J Clin Endocrinol Metab;84:137– 44, 1999.

27. Chandalia M, Abate N, Garg A et al. Relationship between generalized and upper body obesity to insulin resistance in Asian Indian men. J Clin Endocrinol Metab; 84:2329 –35, 1999.

28. Neel JV. Diabetes mellitus: a "thrifty" genotype rendered detrimental by "progress"? 1962. Bull World Health Organ.; 77 (8):694-703; discussion 692-3, 1999.
.

29. Hales CN, Barker DJP. Type 2 (non-insulin-dependent) diabetes mellitus: the thrifty phenotype hypothesis. Diabetologia 35:595-601, 1992.

30. Yajnik CS, Deshmukh US. Maternal nutrition, intrauterine programming and consequential risks in the offspring. Rev Endocr Metab Disord.;9(3):203-11, 2008.

31. Yajnik CS, Lubree HG, Rege SS et al. Adiposity and hyperinsulinemia in Indians are present at birth. J Clin Endocrinol Metab.;87(12):5575-80, 2002.

32. Yajnik CS, Deshpande SS, Jackson AA et al. Vitamin B12 and folate concentrations during pregnancy and insulin resistance in the offspring: the Pune Maternal Nutrition Study. Diabetologia.;51(1):29-38, 2008.

33. Watve MG, Yajnik CS. Evolutionary origins of insulin resistance: a behavioral switch hypothesis. BMC Evol Biol. 17;7:61, 2007.

34. Bavdekar A, Yajnik CS, Fall CH et al. Insulin resistance syndrome in 8-year-old Indian children: small at birth, big at 8 years, or both? Diabetes.;48(12):2422-9, 1999.

35. Fall CH, Sachdev HS, Osmond C et al. New Delhi Birth Cohort. Adult metabolic syndrome and impaired glucose tolerance are associated with different patterns of BMI gain during infancy: Data from the New Delhi Birth Cohort. Diabetes Care.;31(12):2349-56, 2006.

36. Bhargava SK, Sachdev HS, Fall CH et al. Relation of serial changes in childhood body-mass index to impaired glucose tolerance in young adulthood. N Engl J Med. 26;350(9):865-75, 2004.

37. Sachdev HS, Fall CH, Osmond C et al. Anthropometric indicators of body composition in young adults: relation to size at birth and serial measurements of body mass index in childhood in the New Delhi birth cohort. Am J Clin Nutr.;82(2):456-66, 2005.

38. Ramakrishnan L, Sachdev HS, Sharma M et al. Relationship of APOA5, PPARγ and HL gene variants with serial changes in childhood body mass index and coronary artery disease risk factors in young adulthood. Lipids Health Dis.; 8;10:68, 2011.

39. Victora CG, Adair L, Fall C et al. Maternal and Child Undernutrition Study Group. Maternal and child undernutrition: consequences for adult health and human capital. Lancet.; 26;371(9609):340-57, 2008.

40. Deepa M, Farooq S, Deepa R et al. Prevalence and significance of generalized and central body obesity in an urban Asian Indian population in Chennai, India (CURES: 47). Eur J Clin Nutr.;63(2):259-67, 2009.

41. Huffman MD, Prabhakaran D, Osmond C et al. New Delhi Birth Cohort. Incidence of cardiovascular risk factors in an Indian urban cohort results from the New Delhi birth cohort. J Am Coll Cardiol.; 26;57(17):1765-74, 2011.

42. Kinra S, Rameshwar Sarma KV, Ghafoorunissa et al. Effect of integration of supplemental nutrition with public health programmes in pregnancy and early childhood on cardiovascular risk in rural Indian adolescents: long term follow-up of Hyderabad nutrition trial. BMJ.; 25;337:a605. doi: 10.1136/bmj.a605, 2008..

43. Daniel CR, Prabhakaran D, Kapur K et al. A cross-sectional investigation of regional patterns of diet and cardio-metabolic risk in India. Nutr J.; 28;10:12, 2011.

44 Rastogi T, Reddy KS, Vaz M et al. Diet

and risk of ischemic heart disease in India. Am J Clin Nutr.;79 (4):582-92, 2004.
45 Kinra S, Bowen LJ, Lyngdoh T et al. Sociodemographic patterning of non-communicable disease risk factors in ruralIndia: a cross sectional study. BMJ. 27;341:c4974. doi:10.1136/bmj.c4974, 2010.

46. Bowen L, Ebrahim S, De Stavola B et al. Dietary intake and rural-urban migration in India: a cross-sectionalstudy. PLoS One.;6(6):e14822. Epub 2011, 2011.

47. Radhika G, Van Dam RM, Sudha V et al. Refined grain consumption and the metabolic syndrome in urban Asian Indians (Chennai Urban Rural Epidemiology Study 57). Metabolism.;58(5):675-81. PubMed PMID: 19375591, 2009.

48. Mohan V, Radhika G, Sathya RM et al. Dietary carbohydrates, glycaemic load, food groups and newly detected type 2 diabetes among urban Asian Indian population in Chennai, India (Chennai Urban Rural Epidemiology Study 59). Br J Nutr. 2009 Nov;102(10):1498-506. Epub 2009 Jul 9. Erratum in: Br J Nutr.;103(12):1851-2, 2010.

49. Padmavati S. Epidemiology of cardiovascular disease in India. II. Ischemic heart disease. Circulation.;25:711-7, 1962.

50. Ahuja MM. Epidemiological studies on diabetes mellitus in India. In: Ahuja MM, editor. Epidemiology of diabetes in developing countries. New Delhi: Interprint; pp. 29–38, 1979.

51. Deepa R, Sandeep S, Mohan V. Abdominal obesity, visceral fat and type 2 diabetes: "Asian Indian Phenotype." In Type 2 Diabetes in South Asians: Epidemiology, Risk Factors and Prevention. 1st ed. Mohan V, Rao GHR, Eds. New Delhi, India, Jaypee Brothers Medical Publishers, p. 138–152, 2006.

52. Radhika G, Sudha V, Mohan Sathya R et al. Association of fruit and vegetable intake with cardiovascular risk factors in urban south Indians. Br J Nutr. 2008 Feb;99(2):398-405, 2008.

53. Radhika G, Sathya RM, Ganesan A et al. Dietary profile of urban adult population in South India in the context of chronic disease epidemiology (CURES-68). Public Health Nutr. Apr;14(4):591-8, 2011.

54. Isharwal S, Arya S, Misra A et al. Dietary nutrients and insulin resistance in urban Asian Indian adolescents and young adults. Ann Nutr Metab.;52(2):145-51, 2008.

55. Radhika G, Sathya RM, Sudha V et al. Dietary salt intake and hypertension in an urban south Indian population--[CURES - 53]. J Assoc Physicians India.;55:405-11, 2007.

56. NNMB Tribal survey 2008/2009. Personal communication.

57. World Cancer Research Fund/American Institute for Cancer Research. Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective. Washington, DC: AICR; 2007.

58 Ganesh B, Talole SD, Dikshit R. A case-control study on diet and colorectal cancer from Mumbai, India. Cancer Epidemiol. 2009 Oct;33(3-4):189-93. Epub. PubMed PMID: 19717354, 2009.

59. Toral Gathani T, Ali R, Dame Valerie Beral et al. Risk Factors for Breast Cancer in India: an INDOX Case-Control Study. http://indox.org.uk/research/epidemiology/casecontrol

60. Prasad MP, Krishna TP, Pasricha S et al. Esophageal cancer and diet--a case-control study. Nutr Cancer.18(1):85-93, 1992.

61. Krishnaswamy K, Newer roles of vegetables in the prevention and control of problem of over nutrition and chronic degenerative diseases in Nutrition and Vegetables. Prem Nath, P.B.Gaddagimath and O.P.Dutta (editors), Dr.Prem Nath Agricultural Science Foundation, Bangalore, pp- 162-180 2004.

62. Mathew A, Gangadharan P, Varghese C et al. Diet and stomach cancer: a case-control study in South India. Eur J Cancer Prev. 9(2):89-97, 2000.

63. WHO. Preventing chronic diseases: A vital investment. Geneva, 2005.

64. Mahal A, Karan A, Engelgau M. Health, Nutrition and Population (HNP) Discussion Paper. The Economic implications of non-communicable diseases for India. Washington, DC: The World Bank; 2010.

65. Srinath Reddy K, Shah B, Varghese C et al. Responding to the threat of chronic diseases in India. Lancet; 12;366(9498):1744-9, 2005.

66. WHO- National Programme for Prevention and Control of Diabetes, Cardiovascular Disease and Stroke. Developed under the Government of India Collaborative Programme 2008-2009

67. Sacks FM, Svetkey LP, Vollmer WM et al. DASH-Sodium Collaborative Research Group. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. DASH-

FOUNDATION NEWS

- **Annual Foundation Day**

The Annual Foundation Day 2011 of Nutrition of Foundation of India was held on 29th November, 2011. Dr. Kamala Krishnaswamy, Former Director of National Institute of Nutrition and Former President Nutrition Society of India delivered the C. Ramachandran Memorial Lecture on "Evolutionary aspects of diets in the context of current chronic diseases".

- **Symposium**

The ICMR brought out the revised Recommended Dietary Allowances for Indians, at a time when the country is gearing up to fight the dual nutrition burden. The mother child protection card jointly brought out by the Ministry of Women and Child Development and Ministry of Health and Family Welfare incorporates the WHO Child Growth Standards and from now on given to every child born in the country. The card makes it possible for the frontline workers in both the ministries to plot the child's growth and detect early growth faltering. Early detection and effective management of growth faltering holds the key for prevention of stunting and under-nutrition. This is especially important in the context of finding that under-nutrition in childhood can predispose to over-nutrition and increase the risk of non communicable diseases in adult life .

The Working Groups of the Ministry of Health and Family Welfare, Ministry of Women and Child Development and Ministry of Human Resource Development have reviewed the status of ongoing intervention programmes under their ministries and have formulated strategies and programmes to improve ambit, content and quality of the ongoing interventions to combat dual nutrition burden during the Twelfth Plan.

NFI organised a one day symposium on "Nutrition interventions in the new decade" on 30th November 2011 at Nutrition Foundation of India to discuss some of the newer developments and also the proposed interventions to improve nutritional status of vulnerable groups in the Twelfth Five Year Plan. The symposium was very well attended and the presentations were followed by excellent discussions.

The programme of the symposium is given below:

Dr B Sesikeran: RDA for Indians.

Dr Prema Ramachandran: WHO growth standards: importance in the dual nutrition burden era.

Ms Ashi Kholi Kathuria : Overcoming stunting.

Dr S K Bhargava: Undernutrition in childhood and adult health.

Dr Anjana Saxena: Health interventions to combat nutritional problems (undernutrition and micronutrient deficiencies) in pregnant women.

Dr Ajay Khera: Health interventions to combat nutritional problems (undernutrition and micronutrient deficiencies) in children.

Dr K Kalaivani: CAB component of AHS for district based mapping of nutrition problems.

Dr Amarjit Singh: MDM in the 12th Plan.

- **Study Circle Lecture**

Dr Suparna Ghosh-Jerath (Assistant Professor, IIPH, Delhi) delivered a lecture on "Dietary intervention in inborn errors of metabolism – a formidable challenge" on 17th October 2011.