# Human Biology of Diet and Lifestyle Linked Chronic Inflammatory Non-Communicable Disease Epidemic – A Review

A. K. Pandey<sup>1</sup>, G. Pandey<sup>2</sup>, S.S. Pandey<sup>2</sup>, B.L Pandey<sup>3</sup>

Citation: Pandey AK, Pandey G, Pandey SS and Pandey BL. 2014. Human Biology of Diet and Lifestyle Linked Chronic Inflammatory Non-Communicable Disease Epidemic – A Review. Human Biology Review, 3 (1), 25-42.

Corresponding Author: Abhay Kumar Pandey, Department of Physiology, Institute of Medical Sciences, Banaras Hindu University, Varanasi 221005. Email: abhay.physiology@gmail.com

### **ABSTRACT**

Increased urbanized lifestyle implies diet transition, reduced physical activity, mental strains with tobacco and alcohol use. That in turn, increases obesity, raises blood pressure, sugar and lipids, the common risk factors of interrelated non-communicable diseases (NCDs). Individual susceptibility and chronic inflammation make huge area of biomedical understanding. Behaviour modifying interventions are prudent for control but food choices also depend on affordability and availability. The low and middle income groups are driven to cheap unhealthy energy dense foods. The scenario of socioeconomic and nutrition transition in India is mounting the non communicable diseases at alarming speed and magnitude. Cardiac death has become number one killer already. Demographic transition is making the poorer and rural population vulnerable too. Prevention programmes are warranted with urgency. Whole grains, legumes, fish, fresh fruits and vegetables constitute healthy old food pattern. Dietary education under strained finances may be ineffective. Policies of food availability made in exclusion of consultation with health sector undermine prevention of NCDs. Recognition of diverse perspectives in social life and environment call for multisectorial engagement with appropriate interventions to reverse the NCDs epidemic.

Keywords: Meta-inflammation, Chronic Inflammation, Nutrition transition, Metabolic Syndrome

<sup>&</sup>lt;sup>1</sup> Abhay Kumar Pandey, Department of Physiology, Institute of Medical Sciences, Banaras Hindu University, Varanasi 221005. Email: abhay.physiology@gmail.com

<sup>&</sup>lt;sup>2</sup> Garima Pandey, Department of Dermatology and Venerology, Institute of Medical Sciences, Banaras Hindu University, Varanasi 221005. Email: <a href="mailto:garimamsc@yahoo.com">garimamsc@yahoo.com</a>

<sup>&</sup>lt;sup>2</sup>S.S. Pandey, Department of Dermatology and Venerology, Institute of Medical Sciences, Banaras Hindu University, Varanasi 221005.Email: pandeyss@gmail.com

<sup>&</sup>lt;sup>3</sup> B.L Pandey, Department of Pharmacology, Institute of Medical Sciences, Banaras Hindu University, Varanasi 221005. Email: bhublp@gmail.com

#### 1. INTRODUCTION AND SCENARIO

Food and nutritional imbalance is most implicit in disease and deaths of present day world. Chronic non-communicable diseases typically associate overweight and obesity. These have rapidly raised in low and middle income countries. 20% of Indian population suffers NCDs and half of these suffer multiple NCDs. The poor and younger population under 45 year age is significantly afflicted (Planning Commission 2012). NCDs impose burden of disease, disability and death on individual and society, reducing productivity and development. The disability requires long term care burdening the health services. Prevention is far more cost effective in this context, for healthy aging with reduced morbidities.

## 1.1. Lifestyle and Nutritional Translation with Urbanization

Developing nations over past several decades are undergoing rapid urbanization with population migration from rural and remote areas to towns and cities. Current globalization has changed the lifestyle and dietary patterns, in the urbanizing lots. The nutritional transition reflects an increase in consumption of comfort foods. These are energy-dense comprising much sugar and fat. Comfort foods accompany mindless overeating under increased stress of adopted urban life. Mechanization and automation is fast spreading in work sectors, and even in tasks of household. This is replacing demand of physical work in daily life. In the emerging society, information and telecommunication gadgets heavily consume human wake-hours. Television is the ready means of entertainment for leisure time. Urban congestion restricts avenues of space for walks or play. Several mechanisms promote physical inactivity, by choice and/or compulsion. Increasing mental tensions and depression add to these. Alcohol, smoking or other forms of tobacco consumption, have increased to further worsen health perspective.

# 1.2. Consequences of Globalization

Integration of international markets for free trade strongly impacts regional agri-food system. Processing and trade of agricultural products via foreign direct investment is fast invading developing countries. An aggressive promotional advertisement through the proliferated info-tech channels is effectively altering quantities, cost and kinds of available food items. Consumption of vegetable oils highly processed fast foods, soft drinks and diverse sweetmeats has particularly risen. Consumerist urge is readily targeted in transforming middle income group. Low income class

suffers scarcities with inflation consequent to over consumption by others. A tiny wealthy class alone shares benefit of the liberalized trade, in a developing country. The scenario converges on to poor quality food pattern and increased risk of obesity and NCDs (Hawkes, 2006).

#### 2. NUTRITION- BASIC NEEDS

Adult nutritional needs comprise of energy resource, maintenance of tissue wear and tear and disease prevention. With progress of aging, order of needs is reverse. Less of fats, sweets and more of green vegetables and fruits had been the age old human food tradition. The same is simple and effective disease avoiding diet. Awareness of right balance of calories to pattern of physical activity and maintaining quantity of protein, vitamins and minerals is naturally implied.

# 2.1. Dietary pattern and Risk of NCDs

Examination of consumption pattern of food groups, individual food items and nutrients in victims of NCDs versus that of healthier population has revealed nutritional cause in these disorders. Disproportionate carbohydrate consumption puts in excess fructose-glucose mix in system. There is increased insulin secretion. Insulin promotes sugar utilization by tissues. Excess is driven by insulin to be stored as glycogen in liver and muscle, as well as diverted for triglyceride formation and deposition as fat around abdominal organs and under skin. Fat loaded tissues produce inflammatory cytokines which inflict disorder in system. That leads to failure of insulin to control blood sugar or the state of insulin resistance. Raised sugar in blood complexes with proteins and other molecules forming glycosylation end-products. These products increase production of reactive oxygen species. Antioxidant defence mechanisms of body (already weak due to faulty nutrition) fight to quench reactive oxygen species (ROS), but suffer exhaustion. Provocative diet without balanced replenishment of nutrient antioxidants (which include vitamins, phyto-chemicals and minerals) advances mechanisms of inflammation and premature ageing (Pandey and Pandey 2013). Conversion of sugar to fat involves increased formation of arachidonic acid, which is precursor of inflammatory prostaglandins. This simultaneously reduces anti-inflammatory prostaglandin formation due to deficient intake of n-3 fatty acid precursor. Dietary intake of excess of trans-fats (in hydrogenated oils) and saturated fatty acids (in butter etc) increase formation of inflammatory lipid mediators including prostaglandins. N-3 fatty acids present in fresh water fish and green vegetables are resource to synthesis of anti-inflammatory prostaglandins. N-3 fatty acids are deficient in prevailing urban dietary patterns.

The ROS, cytokines, prostaglandins and other mediators infringe upon mitochondria and genes, setting vicious circle of damage through activation of multiple bio-molecular pathways (Galland 2010). Nutrition transition accompanies several fold higher consumption of pro-inflammatory nutrients than in the old traditional diets. Countries like Japan and Korea defying transition and persisting with their traditional diets, have lowest NCDs prevaillance. Right mix of nutrients is needed to balance necessary pro-inflammatory mechanisms to deal with infections and irritants with efficient anti-inflammatory mechanisms to timely turn off inflammation process and allow repair and healing.

# 3. PHENOMENON OF NON SUBSIDING SILENT INFLAMMATION OR METAFLAMMATION IN PATHOGENESIS OF NCDS

Inflammation is initiated following sensing rapid disturbance of homeostasis or damage. The recognition system for unwelcome intruder reads molecular pattern of the intruder as well as bodily damage associated molecular patterns (DAMP). The DAMPs are released on perturbation of homeostasis under the state of stress. Special protein complexes termed "inflammosomes" recognize inflammatory stimuli and guide the formation of pro-inflammatory cytokines. Obesity is chronic state of metabolic alteration. Its nature is pro-inflammatory, promoting insulin resistance, causing dysfunction of pancreatic beta cells and reducing insulin production. There is promotion of fatty change in liver and atherosclerosis in blood vessels. Inflammosomes participate in inducing over activity of innate immune system and spread complications in multiple systems and organs. The maladaptive inflammatory response in obesity and consequent NCDs has metabolic basis and thus called "metaflammation" (Hotamisligii, 2004). Metaflammation, unlike normal inflammatory response, continuously perpetuates (even after causing NCDs). The basal metabolic rate of host in such chronic inflammation does not increase but deceases instead. The "inducers" of metaflammation are varied and produce intermediate innate immune reaction. Inducers persist and dys-metabolic state perpetuates leading to NCD. The strong link of obesity and metaflammation suggests that "inducers" of inflammation come from the metabolic excess or somehow based in the nutrients (Willet et al, 2006).

#### 3.1 Contribution of stress and anthropogenic inflammaging

Traditional biomedical view is simple that energy imbalance is causal to NCDs. Social and economic inequalities, insecurity, injustice and prejudice affect individuals as per personalities and culture impacting the lifestyle and aggravating the maladies of nutrition transition (Elovainia et al,

2010). Anthropogenic perspectives in biology and pathogenesis of NCDs thus bear relevance to prevention and control. Metaflammation characterizes the aging process termed inflammaging (Franischi, 2007). Raised standards in control of infection have prolonged life span. "Inflammaging" is therefore manmade. Socioeconomic deprivation contributes to metaflammation through aberrant methylation of DNA and increased NCD prevalence (McGuiness et al, 2012). Childhood stress and nutrient deprivations leave persisting epigenetic changes that increase vulnerability to disorders in adult life (Borghol et al, 2012). Epigenetic changes persist in chromatin through cell replication and adversely impact gene expression, unless diagnosed and rectified by therapeutic intervention (Choi and Frisco, 2010). Epigenetic basis underlies increased propensity of inflammation response genes to mount metaflammation on exposure to inducers in later life.

# 3.2 Pro and Anti-inflammatory contributors

Imbalance of dietary fat consumption pattern is potent determinant of metaflammation, i.e. Omega-3 fatty acids versus Omega-6, Trans and saturated fatty acids. These serve as precursors to protective or adverse prostaglandins and lipid mediators (Chen and Inui 2012). Processed fast foods and fried items, soft drinks and sweets carry precursors of metaflammation besides other harmful chemical ingredients. Deficiency states of B-complex, A, C, D and E vitamins and minerals zinc, selenium, magnesium and calcium increase risk of metaflammation. Poor nutritional status reduces immunity and increases subclinical infection. That is provocative to organ specific or generalized metaflammation. Emotional stress, sleep deprivation, sedentary lifestyle and aging accompany neuro-endocrine and immune disturbances and increasing tendency to metaflammation (Bray and Champaigne, 2004). Age old traditional rural diets comprised variety of fruits, vegetables, variety of raw grains. All are rich in fibre content and phytochemicals. Fat in nature fed fish or poultry are more of anti-inflammatory consequence. Transition to excess consumption of processed "comfort food" is alien to the system, and pro-inflammatory in constituents (Schumacher et al 2011). Gut micro flora suffers alterations by unnatural food consumption and acquires potential for serving adjuvant to aggressive metaflammation. Prebiotics and probiotics restore healthy gut flora and ameliorate this. Particular attention is deserved to risk of micronutrient deficiency in modern dietary change. Magnesium deficiency is found to profoundly increase metaflammation risk (US Dept of Agric, 1992).

#### 4. THE INDIAN CONTEXT

#### 4.1 Vulnerabilities of phenotype

Population in India and the subcontinent at large, has particularly vulnerable trait for developing obesity, metabolic syndrome and risk of non-communicable diseases and their sequele. Babies born to Indian mothers are among the lowest in birth weight average in world and a third of these are underweight below 2.5 Kg. This is linked to diagnosis of diabetes at age earlier by decade in Indians in contrast to those in west (Mckeigue et.al, 1991; Yajnik, 2001; Yajnik, 2004). The high average parity of Indian mothers causes poor maternal nutritional, cardiovascular and immunological profiles. These are linked to adverse epigenetic programming of fetus. The newborns, despite having lower birth weights, display tendency to abdominal fat accumulation, the hallmark of vulnerability to metabolic syndrome (Joshi et al, 2005). On average, maturity onset diabetes in Indians in contrast to westerners sets in a decade earlier in age. That causes greater loss of productivity and health risk (Bhargava et.al, 2004; Mehta et.al, 2009). At a given body mass index, Indians have greater body fat and tendency to abdominal fat accumulation (Misra et.al, 2006; Chandalia et.al, 2007; Raji et.al, 2007). Genetic polymorphisms in metabolism for higher risk of metabolic syndrome are being increasingly reported in sets of Indian population. Indians accumulate small-dense low density cholesterol, the more atherogenic lipid for cardiovascular complications (Kulkarni et.al, 1999). Deficit of protective high density lipoprotein cholesterol is similarly, more prevalent (Bhalodkar et.al, 2005; Chandalia et.al, 2008). Predictable greater risk for mounting systemic subclinical inflammation is confirmed in Indians at lower body mass indices and higher prevalence of childhood affliction (Isharwal et.al, 2009). Distinctive body phenotype, biochemical and procoagulant profiles and greater predilection to endothelial dysfunction makes Indians far more vulnerable compared to other nations, to risk and severity of metabolic syndrome and related non-communicable diseases (Joshi et.al, 2003; Misra and Khurana, 2011).

### 4.2 Nutrition and Lifestyle Transition

There is progressive urbanisation in South Asian countries with current 40% population in towns and cities likely to reach 50% by 2020. Rural population meets adverse change of environment and living, faulty food habits, stresses and restriction of physical activity. Free space for spending leisure time in exercise or play is rare with spread of television as alternative idle mode of pass time. Emphasis on academic and learning activities of children far more outweigh the playing activities. Outdoor security concerns and seclusive unitary families, particularly preclude physical activity of children and women folk where both husband and wife engage in jobs. Outdoor eating of fast energy-dense food items is rampant. Improved finances in urban jobs, makes traditional vegetarian dietary pattern more lacto-vegetarian. There is increased consuming of dairy products,

and foods made with lots of vegetable and hydrogenated oils. The latter are abundant in polyunsaturated and trans fatty acids. Consumption of non-vegetarian foods increases with more saturated fats. Salt, sweets, coca-colization and McDonaldization particularly find favour with youth. There is increased consumption of alcohol, smoking and other habituations (Govt of India 1998, National Nutrition Monitoring Bureau 1999; Misra et.al, 2001, 2011; Hayes et al, 2002; Deo et al, 2006; Isharwal et.al, 2009; Mitra et.al, 2009; Gupta et.al, 2002, 2003, 2010, 2012; Bhardwaz et.al, 2011).

The migrants reveres transmit urban cults of food items, entertainment and physical inactivity to their village creating new grounds for spread of metabolic syndrome (Chow et.al, 2006; Deo et.al, 2006; Mitra et.al, 2009). Smoking of bidis is common in rural areas which deliver thrice the nicotine and tar compounds then regular cigarette, sharpening teeth of dyslipidaemias to bite the cardiovascular health (Chaturvedi et.al, 2009).

#### 4.3 The Spreading Epidemic

#### (a) Obesity:

Population surveys around turn of the century are revealing of current trends. Urban obesity in 25-64 year age group was reported as 27.8% (Gopinath et.al, 1994) and that in slum dwelling rural migrants as 14% (Misra et.al, 2001). In contrast rural prevalence of obesity was 8% (Chadha et.al, 1997). Gopalan deciphered relation between affluence and obesity with the poor being mostly spared (Gopalan, 1998). Young adults in Mumbai were obese at 5.13 to 10.7% in different socioeconomic strata (Dhurandhar and Kulkarni, 1992).

Prevalence of obesity in urban males and females has shown steady rapid increase in surveys since carried out, e.g., This is 10% in males and 15.1% in females (Vikram et.al, 2003), 20.8% in males and 32.3% in females (Gupta et.al, 2004), overall 28% (Wasir et.al, 2007), 62.2% in males and 74.8% females in Delhi (Bharadwaj et.al, 2011). Childhood obesity prevalence increased from 16% in 2002 to 24% in 2006 in North India (Bharadwaj et.al, 2008), while in South India from 4.9% in 2003 to 6.6% in 2005, over mere 2 year gap (Raj et.al, 2007). Post pubertal adolescents had 16% prevalence of obesity in 2003 that rose to 24% in 2008 (Singhal et.al, 2010). Women above age of 35 in urban areas have 46.6% while in rural area 23.7% obesity prevalence in recent survey (Gupta et al, 2012). Rural South India shows trend of obesity catching up the urban rate one with 32.4% males and 41.4% female obese (Chow et.al, 2007).

### (b) Diabetes:

Earlier wide based survey across 108 locations in country revealed urban and rural prevalence of diabetes mellitus to be 4.7% and 2%, respectively (Sadilkot et.al, 2004). Rural migrants slum dwellers in urban area had diabetes in 11.2% males and 9.9% females with overall 10.3% prevalence (Misra et.al, 2001). Recent report based on all outdoor patients at Lucknow hospital states 19.5% people with impaired fasting glucose profiles (Arun et.al, 2013). A survey in Kerala found 12.4% urban vs 6.8% rural and remote area prevalence of diabetes (Menon et.al, 2006); while respective urban and rural diabetes prevalence was 7.3 % and 3.2% in Tamil Nadu (Padmini et.al, 2008). Much higher prevalence is reported by Madras diabetes foundation (Mohan et.al, 2006, 2008, 2011). An ongoing nationwide ICMR-INDIA survey estimates 8.1 to 14.6% diabetes prevalence in economically backward to more developed states in India (Anjana et.al, 2011), projecting 62.4 million diabetic and 77.2 million prediabetics in India. The observation far exceeds prediction of 69 million diabetics by 2025 in World Diabetes Atlas (Nathan et.al, 2007).

# (c) Cardiovascular complications:

Urban Delhi population samples between 25 to 64 year age range had coronary heart disease in 97 per thousand as opposed to rural 27 per thousand prevalence (Gopinath et.al, 1997). Later survey in urban Jaipur reported ECG evident coronary disease in 6.18% males and 10.12% females. Hypertension had near equal prevalence in the sexes being 36.4% in males and 37.5% in females. Prevalence of dyslipidaemia revealed as low levels of protective high density lipoprotein cholesterol in 54.9% males and 54.2% females. Hypercholesterolaemia prevailed in 37.4% males versus 41.1% females (Gupta et.al, 2002). During 1995 to 2002 the dyslipidaemia of lower HDL levels spanned from 43.2 to 54.2% and hypercholesterolaemia from 24.6% to 37.4% of random population sample (Gupta et.al, 2003). Multiple coronary disease risk factors were reported to be present in 20.8% males and 32.3% females (Gupta et.al, 2004). Postmenopausal women particularly have high marker levels of inflammation, insulin resistance and compound risk factors (Wasir et.al, 2007). Even rural population displays cardiovascular disease and risk factors in 32.4% males and 41.4% females (Chow et.al, 2007). The risk of cardiovascular events correlates to abdominal obesity in perticular. Recent survey of urban population found abdominal obesity prevalent in 62.2% of males and 74.8% of females in Delhi which is alarming (Bharadwaj et.al, 2011). As per 2006 assessment data by Indian Council of Medical Research, coronary heart disease victims increased from 18.6 million in 1998 to 22.4 million in 2004 in India. Stroke cases were 0.8 million in 1998 and rose to 9.3 million by 2004 (Upadhyay, 2012).

#### 4.4 The Burden

In 1990, 1.17 million deaths are estimated to have occurred in India due to coronary heart disease which was projected to be 2.03 million by 2010 (Ghaffar et.al, 2004). Indians get afflicted with coronary disease a decade ahead in age than global average. Substantial number of deaths occurs in working age group. The projected loss of years of productive life in India due to coronary deaths by 2030 is about 18 million (Leeder et.al, 2004). The diabetics in India were estimated as 19.3 million in 1995 with projected increase to 57.2 million in 2025 (King et.al, 1999). These are gross under estimates as suggested in findings of late surveys. Estimated 118 million hypertensives in 2000 are dreaded to increase to 214 million in 2025 (Kearney et.al, 2005). Analysis of 1,30,000 deaths occurring during 2001 to 2003 as part of Million Deaths study being carried out by Centre for Global Health research in collaboration with Registrar general of India and ICMR, reveal 42% of all deaths occurring in India are due to non-communicable disease. Every sixth death of the world occurs in India, amounting to annual 9.5 million. Only 0.4 million deaths get registered hence accessible to studies. Highest 25% deaths in Tamil Nadu state are by acute cardiac disease. Overall 32.8% urban deaths and 22.9% rural deaths have occurred due to cardiac disease. That currently makes it the single tallest killer in India (Sharma, 2010).

#### 4.5 Intervention perspective and NPCDCS

Urgency for corrective interventions by way of education for lifestyle and dietary perspects conducive to health is well recognised by the Govt and social sectors (Reddy et al, 2005). Mature age Indians bearing most of the NCDs also present cultural and religious difficulties to envisaged interventions (Mather, 1994). Globalization in Indian communities particularly spreads physical inactivity and there is major focus on evolving essential minimal physical activity guidelines suited to Indian contexts (Misra et al, 2011). Balanced intake of macronutrients avoiding excess of energy dense unhealthy diet is defined (Singh et al, 1997). Particular worth of popularizing old village diets with variety of ancient whole grains is recognised for reducing spread of NCDs in India (Dixit et al, 2011). On 8<sup>th</sup> July 2010 National programme for prevention and control of cancer, diabetes, cardiovascular diseases and stroke has been approved and it's various components are being implemented with rupees 500 crore financial out-lay for diabetes, heart disease and stroke and 730

crore for cancer interventions. 100 districts falling under 15 states are envisaged to get boost up of education programme for healthy lifestyle and NCD screening of people above 30 yrs age.

#### 5. PREVENTION STRATEGIES

# **5.1 Nature of challenge:**

Although obesity results from energy imbalance, the causal behaviours are shaped by broader contexts and processes of day to day life. Idea of prevention implies investigation on a social not individual problem. The big picture of causality spreads beyond changed lifestyle. It includes geography, ecology, macroeconomics and business. Variables such as food, physical activity, and adverse habits are rooted in as well as corrected through holistic ecological contemplation. Issues of family and its context to society, prevailing marketing, politics and public infrastructure and its functioning bear significant roles in the NCD paradigm (Kumanylka et al. 2007). Genetic, behavioural and environmental factors in pathogenesis of NCD require simultaneous interventions for control by health services, education, agriculture, energy, urban planning sectors. Policy instruments are necessary for such multisector collaboration.

# **5.2 Multisectoral engagement**

Health sector must undergo accommodative adaptation for leading the multisectoral collaboration for control of NCDs. Different levels of tasks and accountability have to be defined and assigned to individual sectors. Clear vision is required of what risk factor/s to be covered with what intervention/s; the sectors leading to the effort, scope of the intervention i.e. regional or national; whether the executives are from the government or the private sector/ NGOs and intensity of coordination necessary before hand (WHO, 2008).

#### **5.3 Innovative interventions:**

Comprehensive interventions are necessary, yet success is more likely in dealing with isolated risk factors. Comprehensive smoke free legislation by Uruguay, Argentina's legislation for reduced salt content of food items, and restriction on salt serving in restaurants; Samoa's curb on multinational industry dumping of turkey tail meat etc, met with exemplary success. Local laws by competent local authorities with similar intent can be effective. A creative intervention may provide example to follow at wider levels. As an example, closing of city roads to cars every Sunday on several routes in Bagota, Columbia, significantly increased physical activity of the city population, and reduced particulate pollution levels. The practice is adopted later by many cities in American

subcontinent. Architect of an educational complex building in New York provided lift that stopped only at 3 of the 8 levels, with provision of alternate wide staircases. This resulted in increased student's social interactions, besides benefit of regular physical activity. Command and control regulations to enable such measures, self propagates information and education. The targets focussed are well defined with multiple impacts on risk factor control. Scope for impact of actors beyond health sector is exemplified in these measures.

#### **5.4 Policies and administration of interventions:**

Such reforming initiatives may cause conflicts of economic interest among some involved parties. Apart from pious task of prevention of NCDs adequate incentives may be necessary to ensure cooperation and participation. High and comprehensive taxes are most cost effective measures to discourage key adverse consumption. Subsidization of public transport with fuel price hikes can selectively increase physical activity of sedentary upper class. Provision of health food items at variety of outlets with some supportive policy can promote healthy food choices of people. Buying-in agreements at mutually agreed prices may encourage cultivation of desired food items by farmers and local availability at reasonable prices. Law department can encourage and support with due initiatives.

Education and provision of information are often supported by government agency to promote public health. Media advertisements are handy corporate measures that affect attitudes of children and some grown ups. Additional policy measures can incentivise desired advertisement of healthy food options by preaching for their holistic benefits for acceptance and adoption. Public health sector's leadership role includes assessing the size of NCD problem and the nature of what drives it; initiation of dialogue with relevant actors and helping them develop priority interventions and monitoring and evaluating the outcomes (Metro-Lorenzo et al, 2011).

#### 6. EPILOGUE

Chronic inflammatory NCDs linked to diet and lifestyles are the greatest threat to health and survival world over. India is predicted to bear the severe brunt due to socioeconomic vulnerabilities. Concerted orientation, dedication and action of diverse functionaries with novel strategies matching the magnitude of the challenge, are the needs of 21<sup>st</sup> century. Empowerment of people to take charge of their health issues requires rational health belief, self efficacy and social learning. The contextual determinants of healthy behaviours and health are vital to appreciate. Special policies

and programmes need contemplation for addressing vulnerabilities and risks of the poor folk. All these are urgent as India risks to be the global NCD capital by 2020.

#### **REFERENCES:**

Anjana RM, Pradeepa R, Deepa M, Dutta M, Sudha V. 2011. Prevalence of diabetes and prediabetes in urban and rural India: Phase I results of the ICMR-India Diabetes (INDIAB) Study. *Diabetologia*. **54:**3022-3027.

Arun A, Sehgal S, Khan F, Khan MM, Gupta S, Tiwari S. 2013. Prediabetes: A study of its prevalence and association with anthropometric variables in adult population of Lucknow, a hospital based cross sectional study. J *Biological and Chemical Res.* **30:**329-346.

Bhalodkar NC, Blum S, Rana T, Kitchappa R, Bhalodkar AN, Enas EA. 2005. Comparison of high density and low density lipoprotein cholesterol subclasses and sizes in Asian Indian women with caucassian women from the Framingham offspring study. *Clin Cardiol*. **28:**247-251.

Bharadwaj S, Misra A, Khurana L, Gulati S, Shah P, Vikram NK. 2008. Childhood obesity in Asian Indians: a burgeoning cause of insulin resistance, diabetes and subclinical inflammation. *Asia Pacific J Clin Nutr.* **17:**172-175.

Bharadwaj S, Misra A, Misra R, Goel K, Bhatt SP, Rastogi K. 2011. High prevalence of abdominal, intra-abdominal and subcutaneous adiposity and clustering of risk factors among urban Asian Indians in north India. *PLOS One*. **6:**e24362.

Bhargava SK, Sachder HS, Fall CH, Osmond C, Lakshmy R, Barker DJ. 2004. Relation of serial changes in childhood BMI to impaired glucose tolerance in young adulthood. *New England J Med.* **350:**865-875.

Borghol N, Suderman M, McArdle W. 2012. Associations of early life socioeconomic position in adult DNA methylation. *International J Epidemiol*. **41:**62-74.

Bray GA, Champagne CM. 2004. Obesity and metabolic syndrome: Implications for dietetic practitioners. *J Am Diet Assoc.* **104**:86-89.

Chaddha SL, Gopinath N, Shekhawat S. 1997. Urban rural difference in the prevalence of coronary heart disease and risk factors in Delhi. *Bull WHO*. **75:**31-38.

Chandalia M, Lin P, Srinivasan T, Livingstone EH, Snell PG, Grandy SM, Abate N. 2007. Insulin resistance and body fat distribution in south asian men compared to caucassian men. *PLoS One*. **2:**e812.

Chandalia M, Mohan V, Adams-Huet B, Deepa R, Abate N. 2008. Ethnic difference in sex gap in high density lipoprotein cholesterol between Asian Indians and Whites. *J Investig Med.* **56:**574-580.

Chaturvedi M, Jindal S, Kumar R. 2009. Lifestyle modification in hypertension in the Indian context. *J Indian Acad Clin Med.* **10:**46-51.

Chen C, Inui A. 2012. What kind of polyunsaturated fatty acids should we eat to stay healthy. *Nutrition*. **28:**849-850.

Choi S, Friso S. 2010. Epigenetics: anew bridge between nutrition and health. *Advances Nutr.* **1:**8-16.

Chow CK, Raju PR, Raju R, Reddy KS, Cardona M, Celer-Majer DS. 2006. The prevalence and management of diabetes in rural India. *Diabetes Care*. **29:**1717-1718.

Chow C, Cardona M, Raju PK, Iyengar S, Sukumar A, Raju R. 2007. Cardiovascular disease and risk factors among 345 adults in rural India- the ARPHI study. *Int J Cardiol.* **116:**180-185.

Deo SS, Zantye A, Mokal R, Mithbawkar S, Rane S, Thakur K. 2006. To identify risk factors for high prevalence of diabetes and impaired glucose tolerance in Indian rural population. *Int J Diab Dev Countries*. **26:**29-33.

Dhurandhar NV, Kulkarni PR. 1992. Prevalence of obesity in Bombay. *Int J Obes Relat Metab Disord*. **16:**367-375.

Dixit AA, Azar KMJ, Gardner CD, Palaniappan LP. 2011. Incorporation of whole ancient grains in to a modern Asian Indian diet: practical strategies to reduce the burden of chronic diseases. *Nutr Rev.* **69:**479-488.

Elovainia M, Ferrie JE, Gimeno D. 2010. Organizational justice and markers of inflammation- The Whitehall 2 Study. *Occup Envir Med.* **67:**78-83.

Franeeschi C. 2007. Inflammaging as a major characteristic of old people: can it be prevented or cured? *Nutrition Rev.* **65**: $S_{173}$ - $S_{176}$ .

Galland L. 2010. Diet and inflammation. Nutr Clin Pract. 25:634-640.

Garima Pandey, Abhay Kuamr Pandey. 2013. Nutrition research perspectives in auto-inflammatory disorders. *Indian Journal of Rheumatology*. **8:** 30-36.

Ghaffer A, Reddy KS, Singhal M. 2004. Burden of non-communicable diseases in south Asia. *BMJ*. **328:**807-810.

Gopalan C. 1998. Obesity in the urban middle class. NFI Bull. 19:1-4.

Gopinath N, Chaddha SL, Jain P, Shekhawat S, Tandon R. 1994. An epidemiological study of obesity in adults in urban population of Delhi. *J Assoc Physicians India*, **42:**212-215.

Gopinath N 1997, Nutrition and chronic diseases: Indian experience. *South east Asian J Trop Med Pub Health*. **28:**113-117.

Govt. Of India. 2012. Report of the working group on disease burden for 12<sup>th</sup> five year plan WG-3(2): Non Communicable Diseases. Planning commission.

Gupta N, Shah P, Goel K, Misra A, Rastogi K, Vikram NK. 2010. Imbalanced dietary profile, anthropometry and lipids in urban Asian Indian adolescents and young adults. *J Am Col Nutr.* **29:**81-91.

Gupta R. 2004. Trends in hypertension epidemiology in India. J Hum Hypertens. 18:73-78.

Gupta R. 2008. Recent trends in coronary heart disease epidemiology in India. *Indian Heart J.* **60:**B4-18.

Gupta R, Gupta VP, Sarna M, Bhatnagar S, Thanvi J, Sharma V. 2002. Prevalence of coronary heart disease and risk factors in an urban Indian population-Jaipur Heart Watch-2 study. *Indian Heart J.* **54:**59-66.

Gupta R, Gupta VP, Sarna M, Prakash H, Rastogi S, Gupta KD. 2003. Serial epidemiological surveys in an urban Indian population demonstrate increasing coronary risk factors among the lower socioeconomic strata. *J Assoc Physicians India*. **51**:470-477.

Gupta R, Sarna M, Thanvi J, rastogi S, Kaul V, Gupta VP.2004. High prevalence of multiple coronary risk factors in Punjabi Bhatia community-Jaipur Heart Watch-2 study. *Indian Heart J*. **56:**646-652.

Gupta R, Agarwal A, Misra A, Gupta S, Pandey RM, Misra P. 2012. Migrating husbands and changing cardiovascular risk factors in wife: a cross sectional study in Asian Indian women. *J Epidemiol Comm Health*. **66:**881-889.

Hawkes C. 2006. Uneven dietary development: linking the policies and process of globalization with nutrition transition, obesity and diet related chronic diseases. *Globalization and Health*. **2:**4-21.

Hayes L, White M, Unwin N, Bhopal R, Fischbacher C, Harland J. 2002. Patterns of physical activity and relationship with risk markers of cardiovascular disease and diabetes in Indian, Pakistani, Bangladeshi and European adults in a UK population. *J Pub Health Med.* **24:**170-178.

Hotamisligii GS. 2006. Inflammation and Metabolic Disease. *Nature*. **444:**860-867.

Isharwal S, Misra A, Wasir JS, Nigam P. 2009. Diet and insulin resistance: a review of Asian Indian perspective. *Indian J Med Res.* **129:**485-499.

Joshi SR. 2003. Metabolic syndrome: emerging clusters of the Indian phenotype. *J Assoc Physicians India*. **51:**445-446.

Joshi NP, Kulkarni SR, Yajnik CS, Joglekar CV, Rao S, Koyaji KJ. 2005. Increasing maternal parity predicts neonatal adiposity-Pune maternal nutrition study. *Am J Obs Gynae*. **193:**783-789.

Kearney PM, Whelton M, Reynolds K, Munter P, Whelton PK, He J. 2005. Global burden of hypertension: analysis of worldwide data. *Lancet*. **365:**217-223.

King H, Anbert RE, Herman WH. 1998. Global burden of diabetes 1995-2025: prevalence, numerical estimates and projection. *Diabetes Care*. **21:**1414-1451.

Kulkarni KR, Markovitz JH, Nanda NC, Segrest JP. 1999. Increased prevalence of smaller and dense LDL particles in Asian Indians. *Atheroscler Thromb Vasc Biol.* **19:**2749-2755.

Kumanylka SK, Whitt-Glover MC, Gary TL. 2007. Expanding the obesity research paradigm to reach African American Communities. Preventing Chronic Diseases. **4:**07\_0067 htm.

Leeder SR, Raymond SU, Greenberg H, Lui H, Esson K. 2004. A race against time: the challenge of cardiovascular disease in developing countries. In, The Centre for Global Health and Economic Development New York. Columbia University.

Mather HM. 1994. Diabetes in elderly Asians. J Roy Soc Med. 87:615-621.

McGuiness D, McGlynn I, McIntyre A. 2012. Socioeconomic status is associated with epigenetic differences in the pSoBid cohort. *International J Epidemiol*. **41:**151-160.

Mckeingue PM, Shah B, Marmot MG. 1991. Relation of central obesity and insulin resistance with high diabetes prevalence and cardiovascular risk in south Asians. *Lancet.* **337:**382-386.

Mehta SR, Kashyap AS, Das S. 2009. Diabetes mellitus in India-The modern scourge. *MJAFI*. **65:**50-54.

Menon VU, Kumar KV, Gilchrist A. 2006. Prevalence of known and undetected diabetes and associated risk factors in central Kerala ADEP study. *Diab Res Clin Pract*. **74:**289-294.

Metro-Lorenzo M, Villatana TL, Harrit MN. 2011. Effective responses to non-communicable disease: Embracing action beyond the health sector. HNP Discussion Paper Sept, Washington DC. The World Bank.

Misra A. 2009. Prevention of type 2 diabetes- the long winding road. *Lancet.* **374:**1655-1656.

Misra A, Khurana L. 2009. The metabolic syndrome in south Asians: epidemiology, determinants and prevention. *Metab Syndr Relat Disord*. **7:**497-514.

Misra A, Khurana L. 2011. Obesity related non communicable diseases: south Asians versus white caucassians. *Int J Obes.* **35:**167-187.

Misra A, Pandey RM, Devi JR, Sharma R, Vkram NK, Khanna N. 2001. High prevalence of obesity and dyslipidaemia in urban slum population in northern India. *Int J Obes relat Metab Disord*. **25:**1722-1729.

Misra A, Sharma R, Pandey RM, Khanna N. 2001. Adverse profile of dietary nutrients, anthropometry and lipids in urban slum dwellers of northern India. *Eur J Clin Nutr.* **55:**717-734.

Misra A, Vikram NK, Gupta R, Pandey RM, Wasir JS, Gupta VP. 2006. Waist circumference cut off points and action levels for Asian Indians for identification of abdominal obesity. *Int J Obes*. **30:**106-111.

Misra A, Nigam P, Hills AP, Chaddha DS, Sharma V, Deepak KK. 2011. Consensus physical activity guidelines for Asian Indians. *Diabetes Technol Ther*. **14:**83-98.

Misra A, Singhal N, Sivakumar B, Bhagat N, Jaiswal A, Khurana L. 2011. Nutrition transition in India: secular trends in dietary intakes and their relationship to non communicable diseases. *J Diabetes*. **3:**278-292.

Mitra A, Basu B, Mukherjee S. 2009. Significance of different dietary habits in sections of Indian diabetics. *J Hum Ecol.* **26:**89-95.

Mohan V, Deepa M, Pradeepa R, Shantharani CS, Farooq S, Ganeshan A. 2006. Secular trends in the prevalence of diabetes and impaired glucose tolerance in urban south India-the CURES 17 study. *Diabetologia*. **49:**1175-1178.

Mohan V, Pradeepa R, Deepa M, Shukla DK, Menon GR. 2008. Urban rural differences in prevalence of self reported diabetes in India-the WHO-ICMR Indian NCD risk factor surveillance. *Diab Res Clin Pract.* **80:**159-168.

Mohan V, Shanthirani S, Deepa R, Premlatha G, Sastry NG, Saroja R. 2011. Intra-urban differences in the prevalence of the metabolic syndrome in southern India-CUPS 4 study. *Diabetes Med*. **18:**280-287.

Nathan DM, Davidson MB, Defronzo RA. 2007. Impaired fasting glucose and impaired glucose tolerance. *Diabetes Care*. **3:**753-758.

National Nutritional Monitoring Bureau. 1999. Report of second repeat survey in rural areas in NNMB states. NNMB Technical Report 18, NNMB, Hyderabad.

Padmini B, Komalamma N, Patel TG. 2008. A community based diabetes prevention and education programme in a rural village in India. *Diabetes Care*. **31:**1097-1104.

Raj M, Sundaram KR, paul M, Deepa AS, Kumar RK. 2007. Obesity in Indian children: time trends and relationship with hypertension. *National Med J India*. **20:**288-293.

Raji A, Seely EW, Arky PA, Simonson DC. 2001. Body fat distribution and insulin resistance in healthy Asian Indians and caucassians. *J Clin Endocrinol Metab*. **86:**5366-5371.

Reddy KS, Shah B, Vergese C, Ramados A. 2005. Responding to the threat of chronic disease in India. *Lancet.* **366:**1744-1749.

Sadilkot SM, Nigam A, Das A. 2004. Diabetes in India: the burden of diabetes and impaired glucose tolerance in India using WHO 1999 criteria: prevalence of diabetes in India study (PODIS). Diab Res Cli Pract. 66:301-307.

Schumacher M, Junker J, Schnekenburger M. 2011. Natural compounds as inflammation inhibitors. *Genes Nutr.* **6:**89-92.

Sharma D. 2010. India's no.1 killer-Heart disease, India Today 2010, April 12 (quoting- Office of the registrar general and centre for global health research report on cause of death in India 2001-2003, New Delhi, Govt. Of India, 2009).

Singh RB, Rastogi SS, Rao PV, Das S, Madhu SV, Das AK. 1997. Diet and lifestyle guidelines and desirable level of risk factors for the prevention of diabetes and its vascular complications in Indians. *J Cardiovasc Risk.* **4:**201-208.

Singhal N, Misra A, Shah P, Rastogi K, Vikram NK. 2010. Secular trends in obesity, regional adiposity, and metabolic parameters among Asian Indian adolescents in North India: a comparative data analysis of two selective samples five year apart 2003-2008. *Ann Nutr Metab.* **56:**176-181.

Laxmi B 2012, Diet in diseases of cardiovascular system in Sri Laxmi B ed DIETETICS- 5<sup>th</sup> Edn Revised, New Delhi, New Age International.

Upadhyay RP. 2012. An overview of the burden of non communicable diseases in India. *Iranian J Pub Health*. **41:**1-8.

US Dept. Of Agriculture-The food Guide Pyramid. Washington DC. GPO 1992:30 (Home and Garden Bulletin No 251).

Vikram NK, Pandey RM, Misra A, Sharma R, Devi JR, Khanna N. 2003. Non obese Asian Indians with normal waist circumference have high cardiovascular risk. *Nutrition*. **19:**503-509.

Wasir JS, Misra A. 2004. The metabolic syndrome in Asian Indians-the impact of nutritional and socioeconpmic transition in India. *Metab Syndr Relat Disord*. **2:**14-23.

Wasir JS, Misra A, Vikram NK, Pandey RM, Luthra K. 2007. C-reactive protein, obesity and insulin resistance in post menopausal women in urban slums of north India. *Diab Metab Syndr Clin Res Rev.* **1:**83-89.

WHO: 2008-2013 action plan for the Global Strategy for the prevention and control of Non-Communicable Diseases. Geneva WHO 2008.

Willet WC, Koplan JP, Nugent R. 2006. Prevention of chronic disease by means of diet and lifestyle changes. In, Jamisson DT et al. Editors, Disease control priorities in Developing Countries. 2<sup>nd</sup> Ed. Washington DC, World Bank.

Yajnik CS. 2001. The insulin resistance epidemic in India: fetal origins, later lifestyle or both. *Nutr Rev.* **59:**1-9.

Yajnik CS. 2004. Early life origins of insulin resistance and type 2 diabetes in India and other Asian countries. *J Nutr.* **134:**205-210.