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Rising Incidence of Chronic Degenerative Diseases – A Health Hazard of Developmental Transition

C. Gopalan

Several reports in the last two decades indicate a disturbing escalation in the incidence of chronic degenerative diseases, especially in India and in countries which are now in various stages of developmental transition¹⁻¹¹. Thus, the prevalence of Type II diabetes mellitus in urban Indian adults has been reported to have increased from <3 per cent in 1970 to >12 per cent in 2000². It is also reported that the prevalence of coronary heart disease (CHD) has increased from <2 per cent to ~ 10 per cent³. Studies carried out at the Foundation⁴ showed that the prevalence rate for "over weight" (BMI>25) in the middle class population was 32 per cent in men and 50 per cent in women; the prevalence of abdominal obesity in the middle class was 29 per cent in men and 46 per cent in women. While the accuracy of these estimates may be debatable, it is reasonable to conclude that there has been an escalation in the incidence of these diseases in the last two decades. The major messages, which emerge from these reports, are the following.

- There has been a steady increase in obesity, Type II diabetes mellitus and CHD in India and in several other developing countries during the last two to three decades. If the present trend continues, these diseases may well become the major public health problems of these countries.

- While these chronic degenerative diseases continue to be seen, as in the past, among the elite sections as well as the poor, the recent escalation

in the incidence of these diseases is largely accounted for by increasing incidence of these diseases in the middle class⁸.

- Unlike in the past, relatively younger sections of the population seem to be the major victims⁸.

- The incidence of these diseases is much higher in urban than in rural areas¹.

It is important to understand the reason for this escalation. It has been suggested that Indians may be genetically more prone to these diseases than the Caucasians^{12,13}. But this higher genetic proneness cannot explain the sudden escalation in recent years.

The countries, in which a significant increase in the incidence of chronic degenerative diseases is being observed, are undergoing developmental transition affecting the socio economic status, life styles and dietary habits of vast sections of the population. The possible role of ongoing developmental transition in causing the escalation in the incidence of chronic degenerative diseases needs to be considered.

DEVELOPMENTAL TRANSITION

The factors in developmental transition, which could be possibly related to the changing health and nutrition problems, are briefly discussed below.

- **Transition from "poverty" to affluence:** The "poverty line" in India has been defined on the basis of the

amount of food required to meet the energy requirements of an average person in the rural areas (2400 KCal/day) and urban areas (2100 KCal/day) areas of the country. On the basis of this criterion, 54.9 per cent of India's population was below the poverty line in 1973-74. This had declined to 26.1 per cent by 1999-2000¹⁴ (Table 1). If the poverty ratio had persisted at the same level as in 1973-74 (54.9 per cent) we would have had over 500 million persons below the poverty line as against approximately 250 million now.

It is clear that in the last three decades, several millions of people have crossed the poverty line and now constitute a considerable part of the Indian "middle class". They may be termed the "first generation affluent". These are people who were born and reared in poverty and who no longer face economic constraints in their adulthood. According to some estimates there may be about 200 million of such "neo-affluents" in India today.

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TABLE 1: Poverty in India

Year	Poverty Ratio (%)	Number of poor (million)
1973-74	54.9	321.3
1977-78	51.3	328.9
1983	44.5	322.9
1987-88	38.9	307.1
1993-94	36	320.3
1999-00	26.1	260.3

Source: Tenth Five Year Plan

● **Child Survival:** During the last three decades, as a part of developmental transition, there has also been a steep decline in infant and under-five mortality rates¹⁵ (Figure 1). Several million infants and children who might have died in earlier years are now “surviving” because of effective child-survival strategies instituted by public health agencies: universal immunization, oral rehydration and improved hospital care of children. On the other hand, the incidence of low birth weight deliveries has shown no appreciable decline and remains at 25-30 per cent of births especially in the poor income groups¹⁶.

Unfortunately, programmes for improvement of child health have not always gone hand in hand with programmes for ensuring child survival. Several millions of children with low birth weights and reared in poverty are now surviving, with their physical and intellectual development permanently impaired. While several thousands of these survivors continue to languish in poverty, considerable proportions of them have succeeded in escaping from the poverty trap and have achieved *relative affluence* in their adulthood. These are the “first generation affluent” or the “neo-affluent”.

● **Rural-urban migration:** A major feature of developmental transition has been the migration of large numbers of the rural population to the urban areas. Thus, the urban population which was 17.3 per cent in India in 1950, exceeded 28.3 per cent in 2003⁵. Millions of the rural poor have migrated to urban areas in search of better employment opportunities. It is reasonable to presume that some of these migrants to the urban areas include a high proportion of the “first generation affluent”, while many urban slum dwellers are still quite poor.

● **Changes in life styles:** Ascent up the socio economic scale, changing occupations, and urbanization, contribute to changes in lifestyles result-

ing in a drastic reduction in the quantum of daily physical exercise. Bicycles, which were once in wide use for commuting to work places, are being replaced progressively by public transport and private vehicles. The energy expenditure from physical work is considerably reduced.

● **Changes in dietary practices:** Developmental transition has also brought about changes in dietary practices in households. The intake of millets, which are rich sources of dietary fibres, has now considerably reduced, with rice and wheat having become the preferred food grains¹⁷.

The overall intake of food grains has declined (Table 2). The overall nutritional quality of household diets has not shown significant increase. The intake of pulses and legumes, which help to improve the protein quality of vegetarian diets, has also considerably reduced¹⁷ (Table 3). Pulses are not only a good source of the amino acid lysine, but, also supply essential nutrients such as riboflavin and n-3 fatty acids, which are poor in Indian diets. The intake of fruits and vegetables, which play an important part in ensuring health and nutritional well being continues to be low, being around 40-50 gms per person per day (as against 450 gms recommended in western diets). There has been a marginal increase in the intake of milk and a significant increase in the intake of edible oils and sugar.

The intake of fats (edible oils) has shown an increase (Table 4). The “average figures” for fat intake in the table would be misleading because of wide variations in the level of fat

intake by different income groups. Studies by the National Nutrition Monitoring Bureau (NNMB) showed that over 17 per cent of our rural households do not consume any visible fat (fat in the form of edible oils) while in 54 per cent, the per capita consumption of fat is less than 10 gms daily¹⁸. In an earlier publication¹⁹, I had computed that 75 per cent of all currently available fat in the country was being consumed by the urban well to do (urban middle class and the rich). Since Indian diets also provide fair amounts of ‘invisible fat’ (fats present in foods and not as oil), the total fat intake among the urban middle class could well exceed 100 gms per person per day.

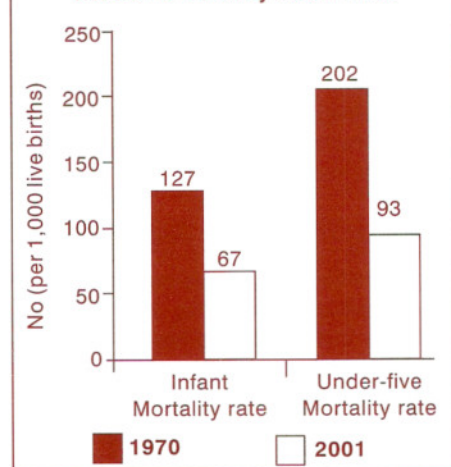
These dietary practices largely induced by developmental transition are not conducive to health and nutritional well-being.

HEALTH HAZARDS OF DEVELOPMENTAL TRANSITION

● **Vulnerability of the “neo-affluent”:** The classical work of Barker and colleagues²⁰ showed that impairment of foetal development due to maternal under-nutrition could result in irreversible metabolic changes in the offspring leading to increased susceptibility to chronic degenerative diseases in adult life. Yajnik^{21,22} in his elegant studies on ‘Foetal origins of chronic degenerative diseases’ demonstrated an inverse relationship between low birth weight and risk of diabetes and metabolic syndrome. (It should, however, be remembered that babies born with very high birth weights also run the risk of diabetes).

The basis of the theory of the ‘fetal origin’ of adult chronic diseases is that nutritional deprivation of the foetus during the critical intra-uterine developmental phase induces adaptive survival strategies resulting in the offspring, being “programmed” to grow and develop in a substandard growth trajectory. If the subject continues to live in a state of poverty and deprivation, such adaptation may prove beneficial. However, when the subject acquires affluence in later life, the “thrifty phenotype” becomes detrimental. The “thrifty phenotype” is an adaptation for survival in a poverty-stricken state. However, when the subjects ascend in the socio-economic ladder scale, “adaptation” apparently breaks down, leading to degenerative dis-

FIGURE 1: Infant mortality rate and under-five mortality rate in India



Source: HD Report 2003 (UNDP)

TABLE 2: Time trends in cereal consumption (kg/month)

	Rice + Wheat	Coarse cereals	Total
1972-73	10.5	4.8	15.3
1999-2000	11.3	1.4	12.7

Source: NSSO 2000

eases like diabetes and syndrome X. This is the essence of the message that emerges from numerous recent publications on the subject of the foetal origin of adult chronic diseases.

● **The “double burden”:** The actual biochemical mechanisms involved in the ‘adaptation’ leading to “thrifty phenotype” and the “dysadaptation” following affluence are still to be elucidated. Subjects suffering from such dysadaptation are apparently more vulnerable to obesity, syndrome X and Type II diabetes mellitus²¹⁻²². It has been claimed that one of the mechanisms involved in such dysadaptation could be associated with impaired fat oxidation, leading to obesity among stunted adults and adolescents in developing countries²³. The “double burden” of childhood stunting and chronic degenerative diseases like obesity and Type II diabetes mellitus in adulthood, which is now being observed in some communities in several developing societies would seem to be partly explained by the foetal origin hypothesis.

The BMI cut-off points (BMI \geq 30 kg/m² for obesity and BMI \geq 25 kg/m² for over weight) which are now in international use are being increasingly considered to be inappropriate for Indian subjects. This is because studies have shown that for a given BMI, Indians have more body fat and less lean body mass than Caucasians^{12,13}. Indians also have high levels of central obesity associated with higher plasma non-esterified fatty acids (NEFA), triglycerides, hyperinsulinemia and insulin resistance²⁴. Apparently the recommended levels of dietary fat intake, which may be appropriate for Caucasians, may be too high for Indians.

All these observations would suggest that the recent escalation in the incidence of chronic degenerative diseases is to a considerable extent attributable to the poverty-affluence transition, and to the lifestyle and dietary changes associated with such transition. The recognition of

this fact should guide our public health policies regarding control of chronic degenerative diseases.

PREVENTION AND CONTROL

● **Adults:** The adult population must be helped to adopt healthy lifestyles and dietary practices in order to avoid overweight and obesity. Our attempt must be to promote the message that the BMI of adults should not exceed 23. In the maintenance of optimal health and freedom from obesity, physical exercise plays an important part as a dietary discipline. Physical exercise helps in the avoidance of obesity along with sustaining and improving the lean body mass.

A vigorous programme of health education should include nutrition education designed for healthy balanced diet, for lifestyle modification and for adequate exercise, to combat and correct some of the recent unhealthy trends; this is an urgent national necessity. Appropriate health and nutrition education at work places and through the media designed to bring about desirable changes must get top priority.

Prevalence of diabetes and Cardiovascular Diseases (CVD) are higher among the urban and affluent segments of the population. However, in terms of numbers, the overall contribution of large rural (70 per cent of Indian population) and not very affluent population to diabetes and CVD is quite high. There is some evidence that because of lack of ready access to health care for early detection and treatment, morbidity and mortality rates may be higher in these population groups. So nutrition and health education aimed at lifestyle changes should be provided to all segments of population both in urban and rural areas.

● **Combating intra-uterine growth retardation:** The message that stands out from recent publications is that

the battle against the escalation of chronic degenerative diseases in developing societies has to be fought and won at the prenatal stage of “intra-uterine” development. In a developing society like ours, unless we take vigorous steps to curb the incidence of low birth weight deliveries, we will continue to have large numbers of “first generation entrants” into the “vulnerable” ranks. If children of the poor escape intra-uterine retardation and have appropriate birth weights, their subsequent ascent from poverty to affluence may not render them vulnerable to metabolic diseases. The poverty-affluence transition may then not pose any health hazard.

Apart from the possible role of intra-uterine growth retardation in increasing the vulnerability to chronic degenerative diseases in adulthood, much of the stunting in childhood that we are seeing today, and poor health in the important years of childhood are also related to intra-uterine growth retardation. There are therefore, very solid grounds for according the highest national priority to combat the low birth weight problem.

However, during the last fifty years Indian public health agencies have made little progress in combating the low birth weight problem. The conventional methods of antenatal care, which are currently in vogue, have been largely ineffective in preventing the incidence of low birth weight deliveries. The National Programme for the distribution of iron and folic acid tablets to expectant mothers during the last 100 days of pregnancy has had some limited success with respect to control of anaemia, but has apparently not been effective in bringing about a reduction in the incidence of low birth weight. Bold new strategies are obviously called for.

The important question is, “What are the inexpensive practical ways by which maternal nutrition can be improved in order to prevent intra-uterine growth retardation in the foetus”?

Studies on the effect of additional supplementation of calories, protein, and micronutrients in pregnancy have been shown to be ineffective in combating the low birth weight problem²⁵⁻²⁹. On the other hand, there are observations, showing that n-3 fatty acid supplementation during pregnancy improves birth weight³⁰⁻³³.

TABLE 3: Pulses (g/month) consumption rural

Rural	Income Levels		
	Lowest income	Middle income	Highest income
Years			
1972-73	20	120	750
1977-78	20	100	490
1999-2000	10	20	90

Source: NSSO 2000

	Rural	Urban
1972-73	24	36
1983	27	37
1993-94	31.4	42
1999-2000	36.1	49.6

Source: NSSO, 2000

Studies at the Nutrition Foundation of India have confirmed these observations and shown that a simple measure like administration of 15 ml of soya oil along with 100 mg of elemental iron and 500 mg of folic acid to pregnant women from the 22nd week of gestation till delivery results in a significant increase in the mean birth weight of infants and a reduction in the incidence of low birth weight deliveries³⁴. Indian diets are low in n-3 fatty acids and the n-6 to n-3 ratio is abnormally high³⁵.

Green leafy vegetables	Spinach, Amaranth, Mint, Fenugreek leaves and Drumstick leaves
Cereals	Bajra, Wheat
Pulses and legumes	Rajma, Soya bean and Black gram
Oils	Mustard / Rape seed, Soya bean, Linseed and Canola

Source: Reference 5

The accompanying table (Table 5) contains a brief list of foods that are rich in n-3 fatty acids³⁵. These foods are traditionally acceptable and inexpensive and their inclusion into household diets should not pose any problems. What we now need is a vigorous programme of nutrition education to bring about desirable changes in maternal diets. It is also important that instead of continuing along the ineffective beaten track, we should now opt for a National Antenatal Care Programme which will include supplementation of n-3 fatty acids food sources along with iron and folic acid supplementation. Such a measure should go hand in hand with education programmes for promoting adequate intake of n-3 fatty acids in maternal diets.

The author is President, Nutrition Foundation of India.

References

1. Chadha S.L., Gopinath N. and Shekhawat S.: Urban-rural differences in the prevalence of coronary heart disease, Bulletin of the World health

Organization, 75(1): 31-38, 1997

2. Ramachandran A., Snehalata C., Kapur A., Vijay V., Mohan V., Das A.K., Rao P.V., Yajnik C.S. Kumar KMP and Nair J.D.: High prevalence of diabetes and impaired glucose tolerance in India: National Urban Diabetes Survey.

3. Gupta R. and Gupta V.P.: Metaanalysis of coronary heart disease prevalence in India, *Ind Heart J*, 48:241-245, 1996

4. Obesity in the urban middle class in Delhi, Scientific Report 15, Nutrition Foundation of India, 1999

5. Ramachandran A.: Epidemiology of diabetes in India—three decades of research.: *JAPI*, 53:31-38, 2005

6. McKieuge P.M., Miller G.J., Marmot M.G.: Coronary artery disease in South Asians: a review. *J Clin Epidemiol*: 42:597-609, 1989

7. Ramachandran A., Snehalata C., Latha E., Satyavani K., Vijay V.: Clustering of cardiovascular risk factors in urban Asian Indians, *Diabetes Care*, 21, 6, 1998

8. Reddy K.S.: Cardiovascular diseases in India. *Wild Hlth Stat Q*, 46:101-107, 1993

9. Enas E.A., Yusuf S., Mehta J.L.: Prevalence of coronary artery diseases in Asian Indians. *Am J Cardiol*, 70:945-949, 1992

10. Popkin B.M., Doak C.M.: The obesity epidemic is a worldwide phenomenon. *Nutr Rev*, 56:106-14, 1998

11. Gupta R., Prakash H., Majumdar S., Sharma S. and Gupta V.P.: Prevalence of Coronary heart disease and coronary risk factors in urban population of Rajasthan. *Ind Heart J*, 47:331-8:1995

12. Banerji M.A., Faridi N., Atluri R., Chaiken R.L. and Lebovitz H.E.: Body composition, visceral fat, leptin and insulin resistance Asian Indian men. *J Clin Endocrinology. Metab.* 84:137-144, 1998

13. Deurenberg P., Deurenberg Y. and Guricci S.: Asians are different from Caucasians and from each other in their body mass index/body fat percent relationship. *Obes. Rev.* 3:141-146, 2002

14. Tenth Five year Plan, Planning Commission, Government of India, 1997-2002

15. Human Development Report 2003, United Nations Development Programme, Oxford University Press, New York, USA, 2003

16. Sharma A.: Low birth weight In Indian Infants, *NFI Bulln*, 22(4), 8, 2001

17. National Sample Survey organization, 2000

18. National Nutrition Monitoring Bureau, 1979

19. Gopalan C.: Edible oils: Needs and wants, *NFI Bull* vol 8(1), 1987

20. Barker D.J.P., Hales C.N., Fall C.I.L., Osmond C., Phipps K., Clark P.M.: Type II (Non-insulin dependent) diabetes mellitus, Hypertension and hyperlipidemia (Syndrome X) : relation to reduced fetal growth. *Diabetologia*, 36:62-7, 1993

21. Yajnik C.S.: Interactions of perturbations in intrauterine growth and growth during childhood on the risk of adult-onset disease, Proceedings of the Nutrition Society, 59:257-265, 2000

22. Yajnik CS.: Obesity epidemic in India : intrauterine origins?, Proceedings of the Nutrition Society, 63,(3): 387-396, 2004

23. Hoffman D.J., Sawaya A.L., Verreschi I., Tucker K.L. and Roberts S.B.: Why are nutritionally stunted children at increased risk of obesity? Studies of metabolic rate and fat oxidation in shantytown children from Sao Paulo, Brazil, *Am J Clin Nutr*, 72:702-7, 2000

24. McKeigue P.M., Shah B. and Marmot M.G.: Relation of central obesity and insulin resistance with high diabetes prevalence and cardiovascular risk in South Asians, *Lancet* 337: 382-386, 1991

25. Iyengar, L.: Effects of dietary supplement in late pregnancy on the expectant mother and her newborn. *Ind J Med Res*, 55:85-89, 1967

26. Bhatnagar, S., Dharamshakti, N.S., Sundaram,

K.R. and Seth, V.: Effect of food supplementation in the last trimester of pregnancy and early postnatal period on maternal weight and infant growth. *Ind J Med Res*, 77:366-372, 1983

27. Adair, L.S. and Pollitt, E.: Outcome of maternal nutritional supplementation. A comprehensive review of the Bacon Chow Study. *Amer J Clin Nutr*, 41:948-973, 1985

28. Lechtig, A., Habicht, J.P., Delgado, H., Klein, E., Yarbrough, C. and Martorell, R.: Effect of food supplementation during pregnancy on birth weight. *Pediatrics*, 56:508-520, 1975a

29. Rush, D., Stein, Z. and Susser, M.: A randomized controlled trial of prenatal nutritional supplementation in New York City. *Pediatrics*, 65:688-697, 1980

30. Leaf A.A., Leighfield M.J., Costeloe K.L. and Crawford M.A.: Long chain polyunsaturated fatty acids and fetal growth. *Early Human Development*; 30:183-191, 1992

31. Olsen S.F., Hansen H.S., Sorensen T.I.A. et al. Intake of marine fat, rich in (n-3)-polyunsaturated fatty acids may increase birth weight by prolonging gestation. *Lancet II*; 367, 1986

32. Sanders T.A.B.: Marine oils metabolic effects and role in human nutrition. *Proc. Nutr. Soc*; 52:457-472, 1993

33. Olsen S.F., Sorensen J.D., Secher N.J., Hildegaard M., Henriksen T.B., Hansen H.S., and Grant A.: Randomized controlled effect of fish oil supplementation on pregnancy duration. *Lancet*; 339: 1003-7, 1992

34. Gopalan S., Patnaik R., and Ganesh K.: Feasible strategies to combat low birth weight and intrauterine growth retardation, *Journal of Pediatric Gastroenterology and nutrition*. Vol 39(1), S37, 2004

35. Ghafoorunissa, Polyunsaturated fatty acids in Indian diets and their clinical implications. pp464, Proceedings, IX Asian Congress of Nutrition, Nutrition Goals for Asia 2020, 2003

FOUNDATION NEWS

● Study Circle Meetings

April 20, 2005: Dr S B Agnihotri (Commissioner cum Secretary, Women & Child Development, Government of Orissa, Bhubaneswar) spoke on "Planning and Monitoring in Social Sectors".

May 30, 2005: Dr Sarath Gopalan (Executive Director, CRNSS; Senior Consultant: in Paediatric Gastroenterology & Clinical Nutrition) delivered a talk on "Nutritional Management in Liver Disease & Celiac Disease".

June 28, 2005: Dr Mahtab Bamji, (Emeritus Scientist, Dangoria Charitable Trust, Hyderabad) shared her experiences on "Lessons on Health and Nutrition from Field Studies in Andhra Pradesh."

● The new website address of NFI is www.nutritionfoundationofindia.res.in and all the correspondence can be mailed to: nfi@nutritionfoundationofindia.res.in