QUALITATIVE DIMENSIONS OF THE POPULATION GROWTH

By

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The Society is fortunate to be invited to hold its 36th Annual Conference in this premier Agricultural University of Maharashtra. I have had the good fortune of following the progress of research at this University for the last 12 years—first under the then Vice-Chancellors Drs M.S. Pawar and Dr. A.B. Joshi and now under the dynamic personality of the Vice-Chancellor Prof. D.K. Salunkhe. I have no hesitation in saying that this University has made a solid break-through in increasing agricultural productivity of plants and animals. Furthermore, several other advances are in the making. I am deliberately using the word 'making,' because breeding for crops and animals is a slow process. The agricultural technology which this University has evolved is suited to meet the needs of small and marginal farmers in the drought prone parts of Maharashtra. However, technology by itself cannot be adequate to ensure that the needs will be met in practice. A small farmer has little or no resources; he has necessarily to depend upon administrative services for the inputs and implements he needs such as bullock power, fertilisers and pesticides on time to sow and harvest. It is usually here that delay occurs and he suffers as a result. Given the timely help and continuity in policy of research, I am confident that this University can solve the problem of Maharashtra. I also look forward to the days when a small farmer can be helped with technology that can be incorporated in the seed itself. I refer to advances like fixation of N and tissue culture in the development of new plant breeding methods which permit multiple copies of productive plants grown to be without waiting for the selected plant to flower and reproduce sexually. I would urge the delegates to go round and acquaint themselves with the work done at this place.

2. As you know Dr. M.S. Swaminathan was the President of the Society until April this year when he left India to take up the

^{*}Presidential Remarks at the 36th Annual Conference of the Society held in January, 1983 at Mahatma Phule-Krishi Vidyapeeth, Rahuri (Maharashtra).

most prestigious position in agricultural sciences in the world as Director General of International Rice Research Station, Manilla. I am merely acting in his place until a new president is elected by the General Body. The Society could not have had a more distinguished scientist to preside and guide its deliberations. Nowhere in the world agricultural statistics in its application to research and planning has made more progress than in India. This in large measure is due to the encouragement which the Society got from Dr. M.S. Swaminathan. Let us place on record our great admiration and appreciation of this brilliant scientist for the services he has rendered to the Society and express the hope that his guidance will be available again after he returns to India.

3. I have been away from India for the last 10 weeks, I therefore did not have enough time to think over the theme for my remarks. However, on reaching here last Friday, my attention was drawn to controvertial territory described under the topic 'Qualitative Dimensions of the Population Growth that was discussed at the last Population Conference in Delhi on December 23. What was said at this Conference has apparently attracted considerable attention in the press, both in India and outside. The key-note address by C. Gopalan contains pointed reference to my own work. I owe it to the press, the public and to you to clarify my position and this is what I will be primarily doing today.

What did Gopalan say in his key-note address to the population conference? It was said that malnutrition is a major problem in India, some 50% of our people are so poor that they cannot afford a diet which meets their minimum energy needs and some 85% of the children born develop into people of sub-standard quality. 'If current trends continue, this steady and relentless undermining of the most valuable of all our resources will pose a far greater threat than any threat of aggression from external agencies'. It does not surprise me that such strong words should cause a furore in the press.

4. What is the basis of these observations? The basis as I understand it is that more and better food increases rate of growth and adult size. Man cannot be an exception to this rule that is observed in animals. The U.S. median weights and heights are a demonstration of the growth that can be achieved in practice with more and better food. Unless our intake is brought to their level, we will not realise our genetic potential for growth. Because the intake is low, our children adapt themselves to low body weights; because body weights are low, the work output is low and because the work

output is low, we get poorer still. This is the vicious circle in which we find ourselves today with no option other than of adapting ourselves to low intake, meaning that our children have to come to terms and reconcile themselves with what they get. Gopalan continues "it is like a person with high blood pressure adapting himself to his condition through hypertophy of his heart". Gopalan expresses his surprise that I should recommended a yardstick of mean energy requirement minus two times the standard deviation instead of the published figure for mean requirement itself and observes that even after doing it, the problem of undernutrition cannot be wiped out-'at least on paper' if not in reality.

One after another Gopalan has put in my mouth statements which he interprets in his own way. Thus, I have never spoken of adaptation in the sense of coming to terms with a bad lot; what I have spoken about is adaptive regulation of variance. The two concepts of adaptation to a bad lot and of adaptive regulation of variance are totally different from each other. They are as different as south is different from the north. Let me illustrate. It is well-known that a healthy active individual will usually have a concentration of glucose in his blood in the fasting state between 60 and 120 mg per 100 ml but we do not speak of a healthy active individual adapting to a fixed level of either 60 or 220 mg and coming to terms with it. What we speak about is whether the value observed lies within the normal range of intra individual variation. Likewise there is ample evidence to show that a healthy active man can do a given amount of work on a range of intakes. This is not a new result. It has been repeatedly brought to our attention by Widdowson since 1947. She observed that in a very age-sex group of healthy active individuals—there are two in twenty, one of whom is found to eat twice the amount of the other and yet all are engaged in similar work. The data on energy intake in young army recruits reported by English physiologists led by Edholm show that energy cost of the same work tasks varies widely in the same individual. Edmundson gave a convincing demonstration in several villages in Java and in Katavi village near Pune to show how our villagers are able to work harder using less food energy than overnourished westerners by their improved metabolic efficiency. He showed that the BMR of subjects who ate more was large by 80% than the BMR of those who ate in the lower range of intakes. If one visits Indira Community Kitchen, Pune, one will find much the same result as that observed by Edmundson with some women eating only 1200 calories per day and others having an intake nearly twice as large and yet the work output of everyone is about the same, viz.

one bhakari per minute. One has to visit and see for oneself the sight to appreciate the intensity of work involved in the kitchen. It means that the body simply works more efficiently at the lower range of intake. Usually I give my own example in illustrating this. I am 70 years old I weigh 45 kg or even less, and eat about 1200 Kcals a day though I work for over 10 hours a day. According to current nutrition standards a healthy active man of my age and engaged in sedentary activity ought to have some 1700 Kcals. Evidently, my efficiency of work output is not affected by my low intake.

6. Let me digress a little. It is true that my body weight is low but that I would ascribe largely to childhood morbidity, particularly diarrhoea. Longitudinal surveys on children in villages confirm that diarrhoea and related diseases limit the growth of a child. frequency of diarrhoeal episodes is so large in the first two years of life that a child has hardly time to recover long enough to continue normal growth that comes from adequate food. By the time a child is two or three years old, he has already lagged behind in growth on average upto 10 cm in height and 3 kg in body weight compared to his counterparts in U.S.A. It is not surprising that infant mortality in the country should be high under these conditions. By contrast observations on children of Indian origin born and brought up in U.S.A. show that they grow better even though they continue to take much the same diet as in India. However, the same children are found to suffer set backs to their health and growth when they visit India for short period. A good diet by itself will be of little avail in initiating a process of normal growth, reduction of infant mortality and increase in life expectancy in the environment as it exists in villages and slums unless simultaneous measures are taken to reduce the incidence of morbidity. The experience of World Bank expert narrated by Dr. Lincoln Chen is worth mentioning here.

He went to Bangla Desh with a conviction that a well-nourished person, that he was would be immune to diarrhoea. Within days of his travelling in the villages around Dacca, a message came 'Emergency, Emergency'! And lo! he was literally pouring protein when he reached Headquarters. He even forgot to take the mixture of salt and gur in boiled water as he dashed to Dacca.

One would have thought that despite early set backs to body weight school meal programmes would restore the growth but none of the school meal programmes examined confirms this. Apparently the organisation of the feeding programmes leaves a lot to be desired. On the other hand, children from the well-to-do societies, although

bigger in height and weight than those in the poor areas continue to fall short of the U.S. heights and weights by large margin. One could well argue that the genetic potential is different, but that would imply that feeding would only act to accelerate growth rate at earlier ages to reach maturity earlier than otherwise. Whether accelerating growth rate without improving adult size has any advantage is more than doubtful, since inverse relationship between early growth rate and longevity appears to have a strong genetic basis. Unless these matters are further investigated, one cannot accept Gopalan's statement based on NNMB data that 85% children are not able to develop full genetic potential for lack of nutrition. The fact is that intake of the majority of these children more than meets their physiological needs for energy but they are not able to utilise what they eat for lack of conducive environment; potable water, hygiene and sanitation. This is the crux of the problem.

7. Again Gopalan refers to my method as mean requirement minus twice the standard deviation, but does not specify the nature of the standard deviation. What he refers to is interindividual variation, but I am talking of intra-individual variation, not inter-Then again Gopalan is talking of adaption to low values of intake, whereas I am talking of interaction between the two components of the phenotypic trait, viz. the genotype (g) and the environment (e), given by the covariance term in the variance of (g+e) over time in the same individual within the framework of ontogenic growth. Nutrition literature mentions either genotypic or environmental variance but ignores the covariance term. Consequently, people talk either in terms of inheritance through natural selection that they read in the first component V(g) or the inheritance of acquired characteristics associated with the second term V(e). Actually development of man, his intellect his mind and his conscious thinking is made possible by the covariance term. If you and I are what we are, it is due to the change resulting from the covariance term over and above what we are born with and inherit from parents. This term emphasises the importance of appropriate environment and education as a tool of achieving a way of life suited to our culture, needs and resources. The underlying theory is given in the book 'Newer Concepts in Nutrition and their Implications for Policy' (1983) published by MACS and edited by me. Modern science cannot talk of traits like intake or I.Q. as wholly determined either by nature or nurture, ignoring the third component altogether. To do so is to admit that we are either racists or fatalists or alternatively 'prisioners' of culture. We are neither of these two.

Covariance, its nature, magnitude and determinants are as basic to understanding social change and development of man as rules of additions, subtraction, multiplication and division are, for the development and understanding of algebra.

- 8. Gopalan writes that my views have been forcefully challenged and rebutted by several members of statistical fraternity-let alone physiologists and nutritionists. Evidently, he refers to the current debate in EPW (1981, 1982). Space and time do not allow me to elaborate but I must say that I do not feel surprised. Agreement or disagreement from individuals who refuse to understand how nature and nurture interact to produce a stable variance means little to me.
- 9. He quotes results of NNMB surveys as proof of his statement that 85% of the people are malnourished even after adopting my method. I have dealt with both these questions at length in my book on Newer Concepts in Nutrition. It will only make one observation that NNMB has misused my method in evaluating their data. At the same time, I feel happy to report that NIN has decided to set the matter right. NIN has not only asked for my help, but have also sought the help of NSS and CSO. When NNMB data are reanalysed, it will be found that the order of under-nutrition in children is 25 per cent and not 85 per cent. Field surveys reveal it to be even less, but I will not enter into these details here.
- 10. Again Gopalan says that we are being told that small is healthy. None of us have ever said this or even remotely hinted that it is better to remain small and adapt oneself to low intake. The phrase that came to be formulated and is due to Seckler is 'Small but Healthy' to express the finding that wight deficit in children down to 35 per cent of the Harvard standard, made little difference to the susceptibility to childhood diseases. In other words, 65 per cent of the Harvard median standard constituted the lower homeostatic limit. Below this no one can call a small child as healthy. I wish Gopalan had not gone that far to destort the original statement and ascribing it to me. Again the statement that there is little difference in susceptibility to childhood diseases over a range of body weights must not be understood to mean that no further efforts are needed to reduce infant mortality and improve life expectation. The latter objective may be irrelevant for animals but not for humans.
- 11. That a healthy active man is able to adapt on intakes below the mean requirement down to some 35 per cent of mean

without adversely affecting his work output or body weight is due to the fact that intake is under partial biological control and can be influenced by environment. Take for instance the addition of drink to our diet. Many of us take a glass or two of wine and beer, many more prefer stronger drinks. The energy that these give is not...dictated by physiological need or hunger. One simply enjoys taking a drink and the larger amount of food such as fish and chips or steak that is automatically consumed with it. It is natural for man to indulge in more food when food is tasty and one can afford Fortunately, body has a mechanism to dissipate what one eats in excess of body needs. If the body did not possess this mechanism none of us would be able to indulge in eating more the way we do. But we also know that when indulgence is carried too far, i.e. over the upper limit of homeostasis, we pay dearly in the form of obesity, blood pressure, heart and other diseases. Homeostatic range is an expression of the interaction between the biological and environment at components of the behavioural trait in the individual. It is the genetic basis implied in the interaction that explains the wide range of intake that we enjoy without detriment to our health and work output.

- 12. Gopalan argues forcefully in favour of massive intervention programmes for achieving the genetic potentials. I have nothing against these programmes but surely we must enquire as to why feeding programmes fail in the first instance. I have evaluated several of these programmes carefully. Take for instance the programmes organised by CARE in the state of Kerala. It was perhaps the most extensive among the feeding programmes very well organised by CARE for over 10 years in 8000 and odd schools in the State. Soman's evaluation of this programme leaves little doubt that the programme failed. I will commend to your attention the Kerala experience described by him.
- 13. I am the last man to advocate restricting food intake Visit our Indira Community Kitchen. Although drawn from the poorest of the poor, the workers are free to eat as much and whatever they wish from amongst the foods prepared and sold by the Kitchen. I am not even against distribution of food-grains such as they have in Kerala and Sri Lanka provided this is feasible.

What I am again is to serve so called nutritious foods to children from 6 months to 6 years standing or sitting in a row. Apart from the fact that the sight is dehumanising, no food can be universally suitable for all children since food habits differ from family to family. Secondly while it may satisfy hunger for the time being, it can do little to initiate a process that will contribute to alleviating the basic cause of hunger. Such interventions will only increase helplessness and begging attitude among children.

- 14. Finally Gopalan says no physiologist or nutritionist can accept my yardstic based on the hypothesis that nature interacts with nurture to create a fundamental source of Intra individual variance. He does not mention the reasons but I can guess them. His difficulty appears to be that each of us is born with a fixed genetic code and therefore, genetically programmed to achieve the growth potential with adequate intake. That we cannot change the genetic code with which we are born is true but that does not mean that the genetic potential for growth is fixed and is the same for all. The function of the genetic code is simply to direct the assembly of amino acids released from food in a precise sequence to make the protein that body needs. This is done by a sequence of three out of 4 nucleotides of DNA. But although the precise sequence of amino acids is under the control of DNA, the number of proteins to be made and the time and conditions under which they are to be made are not under the control of DNA. Experimental evidence shows that a large number of DNA sequences are never transcribed into messenger RNA and suggest that it is the function of the information contained in the untranscribed DNA to control the speed of metabolism so that the body throws out disruptive forces of variation and assimilate the random variation into the pattern of hierarchical variation as part of man's progressive development. All the data that I have analysed confirm this hierarchical associative nature of intake in man engaged in fixed tasks. This is the reason why daily requirement is found to be dynamic and distributed with stationary variance. The variance of the energy requirement cannot therefore decrease inversely as the length of the period but decrease slowly to a point where it assumes a stable value. It is easy to show that this stable value for the variance arises from the interaction of genetic entities in an individual with the environment.
- 15. Repetition, variation and integration at each step of internal differentiation is the way man grows. This is how for example a child picks up speaking. He would construct, test and reconstruct a progressively more adequate set of words and rules by perception of symbols and relationship between them. He gropes for correct syntax and phonetics, gets it after trial and error and assimilates it.

Man has an innate capacity to learn and to incorporate his learning experience into traces of hierarchical pattern. The answer to the question then that appears to bother nutritionists is that traits which are under partial biological control are not hereditary in the sense in which height or colour of the eyes are hereditary but they have an heritable basis which can be traced to cognitive faculties in man which are known to be served by billions and trillions of neurons the neural pathways in controlling the action of genes. The fact that daily intake for given workoutput has stationary variance implies that man can direct his daily intake in almost innumerable ways to correspond to stored neural messages. It is in this sense that man regulates his intake without modifying the germplasm by determining what portion of DNA to transcribe and when and where while maintaining his health and level of work output.

- 16. Much of the experimental evidence referred to above is very recent. My role has been to show its relevance to the concept of stationary variance. It may be concluded that while man is a product of heredity, he is even more importantly a product of education and upbringing and the culture surrounding him. Obviously, the assumption that the genetic potential for anthropometric growth is the same for all cannot be valid. It follows too that Gomez classification for evaluating the incidence of malnutrition would exaggerate the size of the problem. The inference that any deviation from U.S. median standards for weight and height beyond 10% is due to deficiency in energy intake, and quality of diet is even less justified. There just cannot be any universal fixed standards as assumed in nutrition literature.
- 17. How then did these assumptions came to be accepted by the nutritionists when all available data contradicted them? This is a question more for political economy than science to answer. Morris (1982) has given a very incisive analysis of the problem. Work on human nutrition standards according to him was influenced by the earlier results in animal nutrition. Since animals grow faster and larger on protein-rich feeds and supplements like bonemeal and ash, nutritionists directed their attention to isolating factors responsible for growth. Their efforts were richly rewarded first by the discovery that N was the decisive element in determining the growth and hence the nutritional quality of diet and secondly, vitamins and minerals if not present in adequate quantities led to specific deficiency diseases. The former led to the rapid and increased consumption of animal products and the latter to the measures of alleviating ailments through promotion

and sale of vitamins and minerals. The use of food technology in processing and synthesis gave further stimulus to the live stock industry not only in increasing the domestic demand, but also catering to international markets. The attraction were so large that the more important issues like reduction of infant mortality and increase in life expectation were by-passed. These issues were irrelevant for securing faster and better growth of animals, but they are vitally important for humans. However, it was believed that these issues are more appropriately tackled through the development of drug industry. Even the manly vigour, superiority of military strength and industralisation began to be ascribed to superior western diet. To drive home, urgency of action, it was further stated that low productivity in the poor countries was the result of malnutrition and unless it was corrected by extensive nutrition intervention programmes, progress in combating poverty was bound to be slow.

Experiments on animals with human diets reported British workers in India, more particularly by Maccarison, Macay and Aykroyd gave a respectable basis to this line of thinking. As an example, they observed that rats grew better on the diet eaten by the vigorous Sikhs than on the diet eaten by the South Indians. They conclude that the nutritional value of the Sikh diet was higher than of the South Indian diet. No one however asked why rats fed on English diet did poorly and why were these results not reported. Aykroyd went a step further. He fed poor children with nutrition supplements and showed how supplements in the form of milk and animal products improved height, weight, health and sleekness. Evidence marshelled on these lines natural appealed to politicians and was an important factor in the nutrition scientists setting aside basic source of variation and its implication for efficiency of work output. For the same reason, the fact that infant mortality is smaller and life expectation longer, despite low food intake in Kerala has received no attention. Much the same attitude is evident with respect to the results reported by Lincoln Chen that it makes little difference to susceptibility to diarrhoeal diseases and rate of mortality even if body weights of children are lower than those in the U.S. down to 35%. This is the main reason why scientists in the developing countries and especially in India brought up in the conviction that our diet is deficient in good quality protein and in vitamins and minerals are busy emphasising the need and role of feeding programmes for the poor and the distribution of vitamins tablets as part of the health set-up. But as Morris puts it, those who work in this way have been mostly trained in the western countries and to use his

own expression 'are so sold to their philosophy that they work more as agents than on their own. The trend continues. One has only to examine the field experiments on feeding reported from Gautemala. On the face of it, the design is unsound and the interpretation of results even more so. What came to be ascribed to better food was partially confounded with differences in the social status of children. The story of Farangwal study, supposedly undertaken to remove the defects in the field experiments reported by Gautemala, is even more depressing. I have dealt with it elsewhere. There would be little point on my expanding this list. Govalan's effort in fanning the feelings of the people by describing the younger generation as of sub-standard quality for lack of nutrition is the latest in this series. As long as we are not prepared to take a fresh look and keep science out of the arena of political economy, we are unlikely to make a headway in identifying the factors responsible for population growth and nutrition status and in according their rightful priority in our planning.