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'Micronutrient' Deficiencies
Public Health Implications

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During the last few decades there have been significant changes in the profile of nutrition-related diseases in several developing countries. While in parts of Africa, especially in the sub-Sahara, acute food shortages and near-famine conditions still persist, in countries of South and South-east Asia, severe calorie-protein malnutrition has declined. Also florid clinical manifestations of micronutrient deficiencies such as beri beri, pellagra and keratomalacia which once held the centre-stage have either been eliminated or have distinctly receded.

In these latter countries, the dominant public-health-nutrition problems, today, are widespread (chronic) diseases related to deficiency of micronutrients such as iron-folate deficiency anaemia, goitre and other iodine-deficiency disorders, and milder forms of vitamin A deficiency. These are perhaps not as spectacular and immediately life-threatening as some of the earlier manifestations, but they carry serious functional consequences and implications.

The possibility that micronutrient deficiencies may also be contributing, partly (or perhaps even substantially), to growth retardation, widely prevalent in developing countries, has to be borne in mind. Indeed, with the progressive elimination of energy-protein deficits in the dietaries of children, micronutrient deficiencies could emerge as the major bottleneck to growth.

It seems also possible that deficiencies of some micronutrients such as zinc and selenium may also demand increasing attention in the years ahead, from the point of view of public health.

It may be useful to briefly review some major factors which may have a bearing on the choice of appropriate strategies for the prevention and control of micronutrient deficiencies.

MICRONUTRIENTS' METABOLIC INTER-RELATIONSHIPS

There is growing evidence of metabolic inter-relationships between different micronutrients. While it may be true that the deficiency of a single micronutrient may play the dominant direct role in a given deficiency disease, deficiencies of other micronutrients could also play 'conditioning' roles in the pathogenesis of the same problem. Several examples of micronutrient inter-relationships may be cited but, for our present purpose, the following few will suffice:

- the evidence that selenium deficiency could impair the utilisation of iodine[1], selenium being a key component of the enzyme converting thyroxine to triiodothyronine
- the evidence that zinc deficiency could be a factor in the pathogenesis of hypovitaminosis A, zinc being involved in the transport of hepatic vitamin A to the target tissues[2]
- that excess iron medication in the absence of overall dietary improvement could depress the zinc nutritional status[3]
- that the bioavailability of iron from cereal-based diets can be considerably enhanced by ascorbic acid[4]
- that molybdenum could promote copper excretion[5] and thus aggravate copper deficiency.

CO-EXISTENCE OF MICRO- AND MACRONUTRIENT DEFICIENCIES

Apart from metabolic inter-relationships among micro- (and macro-) nutrients of the types mentioned above, it must also be remembered that in generally undernourished population groups, a micronutrient deficiency often tends to co-exist with other micro- and macronutrient deficiencies. Thus, for example, a child with clinical vitamin A deficiency is almost always stunted to some degree (Protein-energy malnutrition (PEM)) and in many cases is also anaemic (iron deficiency). In pregnancy in poor population groups, deficiencies of iron, folate and vitamin A often tend to co-exist.

These considerations must caution us against a policy of sole reli-

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ance on the supplementation of a single nutrient as the answer to a given micronutrient-deficiency-disease problem. While there is certainly a place for single nutrients, the ultimate logical approach obviously must consist in dietary improvement. Even where single nutrients may be employed to temporarily tide over a problem, they must be used as adjuncts to, and as reinforcements of, the basic approach of appropriate dietary improvement. Much of the failure of public health programmes for the control of micronutrient deficiencies may be traced to our failure to follow this guiding principle.

MICRONUTRIENT COMPOSITION OF FOODS

Micronutrients, like macronutrients, have to be ultimately derived from foods. Factors which affect the micronutrient composition of foods will therefore obviously have an important bearing on the pathogenesis of micronutrient deficiency diseases in humans.

Soil depletion: Modern agricultural technology, incidental to the propagation of high yielding grain varieties as part of the 'Green Revolution', has involved intensive irrigation and the heavy use of high-analysis chemical fertilisers. This has apparently resulted in substantial soil loss of S, Fe, Mn, Zn and Cu[2]. Apart from the actual content of micronutrients in the soil, the availability of micronutrients to plants is influenced by other factors such as soil pH, and relative concentrations of different micronutrients in the soil.

Varietal differences: Varietal differences in the abilities of plants to absorb different trace elements have also been demonstrated. Thus, different varieties of rye grass have been shown to accumulate varying concentrations of iodine from the same soil[6], different varieties of corn accumulate varied levels of zinc from the same soil[6]. It is theoretically possible that varieties selected for propagation on the basis of their good yield potential may be poor in some trace elements.

The richest sources of micronutrients are green leafy vegetables and fruits. There are striking locational and varietal differences with respect to the content of micronutrients in vegetables and fruits. While a great deal of effort has gone into the identification of high yielding varieties of food grains — mostly rice and wheat — horticultural research directed to the augmentation of the production of high nutritive value strains of vegetables and fruits has been mostly neglected, possibly because combating calorie deficiency through the augmentation of cereal production was perceived as the problem that must command high priority. It would appear that the very technologies that have been employed to bring about the augmented production of cereals in the 'Green Revolution' could have contributed, to some extent, to the erosion of the nutritive quality of foods with respect to micronutrients.

Plant genetic traits associated with high yield potential may not necessarily be associated with high micronutrient concentration potential. Genetic engineering programmes with respect to cereals (the main staple) are bound to be mostly governed, in future as in the past, by considerations of the need to identify high yielding varieties.

It is, therefore, obvious that for the purpose of ensuring a satisfactory overall micronutrient content, heavy reliance has to be placed on horticultural products, especially on green leafy vegetables. Future genetic engineering programmes with respect to horticultural products must be directed towards identifying and propagating high micronutrient concentration varieties.

It should be clear that any rational strategy for combating micronutrient deficiencies must be 'food-based' rather than 'drug-based'.

BIOACTIVE PHYTOCHEMICALS

Several phytochemicals have been found to play as important a role as some vitamins in the maintenance of health and in the prevention of diseases. Phytochemicals, such as sulforaphane and p-coumaric acid, act by boosting the activity of 'phase 2 enzymes' which detoxify carcinogens; and could, under certain circumstances, prove as important as vitamin E and β-carotene in cancer prevention. Indeed, these may all even act synergistically.

It is increasingly likely that micronutrient deficiencies in developing countries in the coming decades will coexist with ageing and degenerative states such as arteriosclerosis and cancer. Since phytochemicals are present in vegetables and fruits, this finding provides yet another powerful argument in favour of ensuring good health/nutrition through improved diets, which will provide both the desirable phytochemicals and vitamins, rather than through isolated medications with a single chemical or vitamin.

IRON/FOLATE DEFICIENCY

Anaemia due to iron deficiency is perhaps the most widespread clinical nutrition deficiency disease in the world today. Iron deficiency anaemia, now seen in millions of people of poor communities in developing countries, is largely due to dietary iron deficiency. The overall intake of iron in Asian dietaries is seemingly adequate on the basis of currently recommended dietary allowances. However, the poor bioavailability of iron in cereal-based diets seems to be the major factor responsible.

Implications: Anaemia, though not spectacular, has far-reaching public-health implications, such as increased maternal mortality and morbidity in pregnancy; intra-uterine growth retardation, low birth weight, increased neonatal mortality, depressed immunocompetence, and impaired learning ability in infants and children; and impaired work performance and behavioural changes in adults.

Efficacy of current strategy: In countries where moderate and severe anaemia in pregnancy is widespread, the current standard control programmes being undertaken by public-health agencies have largely consisted in the administration of iron/folate tablets (60 to 120 mg iron, 500 mcg folate) in the last 100 days of pregnancy as part of antenatal care. This is generally not accompanied by serious efforts towards improving diets in poor households and, even more importantly, towards improving diets of women during pregnancy.

That this strategy has been inadequate has been shown by reported experience. Thus, for example, a study by the Indian Council of Medical Research[7] (ICMR) in India showed that 17 per cent of pregnant women had Hb levels less than 9 gm/dl even to start with; while the compliance rate (with respect to the daily intake of tablets) was unsatisfactory in a con-

siderable proportion of cases.

What was disconcerting was that in as high a proportion as 38 per cent of cases of women who had consumed the tablets regularly for more than 90 days during the last trimester, Hb levels were less than 10 gm/dl, and in nearly 20 per cent less than 9 gm/dl at the end of pregnancy.

The message that stands out from this finding is that in cereal-eating populations, the control of anaemia will call for the effective convergence of three major approaches:

- dietary improvement
- iron/folate tablet administration
- fortification of appropriate food items with iron.

Dietary improvement: Diets, especially of women and children, will need to be improved. The bioavailability of iron in cereal-based diets can be improved substantially by ascorbic acid, the daily intake of which in diets of poor households is very low. It has been shown that the inclusion of ascorbic acid in the diet even at moderate levels of intake could double the bioavailability of iron. Green leafy vegetables supply not only iron but also vitamin C and folic acid. Despite an abundance of vegetables and fruits rich in iron, vitamin C, folic acid and β-carotene, South Asian countries have, so far, failed to optimally utilise these valuable sources. While half of their populations are anaemic, nearly one-third of the fruits and vegetables grown in these countries perish annually because of a lack of proper preservation and storage.

Iron/folate tablet administration — duration and periodicity: The present strategy of relying on iron/folate tablet administration in the last 100 days of pregnancy may need to be carefully reviewed. Apart from practical logistic problems in ensuring a regular daily intake of tablets, there is the fact that the majority of girls are anaemic even in their adolescence. Under the circumstances, even regular administration of iron/folate limited to the last 100 days may not succeed in achieving the required adequate Hb levels.

It has been suggested for this reason that iron/folate administration could start much earlier — in pregnancy or indeed even immediately after marriage — since a good proportion of adolescent girls are, in any case, anaemic[8]. It could be argued that with such a strategy, Hb could be raised to satisfactory levels even if compliance with respect to the daily intake is not achieved.

Viteri *et al*[9] have, in fact, suggested that intermittent iron administration may ensure better absorption than daily administration which could induce 'tiredness' of the intestinal mucosa — a point of view that has been contested by Hallberg (personal communication).

Viteri *et al*'s suggestions would favour a policy of spreading iron/folate administration over a longer period during pregnancy (longer than the last 100 days of pregnancy as at present).

Yet another argument in favour of initiating iron/folate administration early in pregnancy (or even in adolescence) is the claim that damage to the foetal brain arising from maternal anaemia takes place early in pregnancy[10], as also the claim that the deleterious effect of folic acid deficiency on neural tube development occurs within the first four weeks of pregnancy[11].

However, before extending the duration and intensity of the iron supplementation programme, due consideration must be given to possible side effects. There is evidence pointing to the inhibitory effect of inorganic iron on zinc absorption[3]. Iron therapy in doses generally prescribed in MCH care has been reported to have measurable adverse effects on maternal zinc status. The zinc nutritional status of poor population groups subsisting on cereal staples is already low. On top of it, evidence of impairment of the content and bioavailability of soil zinc and the poor zinc content of food grown on such soils is also forthcoming in areas where cereal is the main staple and where agricultural technology to augment the production of cereals is being undertaken.

That maternal zinc deficiency may have a deleterious effect on the foetus has also been reported. All these considerations have to be taken into account in formulating a revised policy for iron/folate tablet administration as an answer to the problem of iron-deficiency anaemia on a public health scale.

Fortification of foods with iron: An approach that is worth propagation is that of fortification of suitable food items with iron. The National Institute of Nutrition(NIN), Hyderabad (India), has successfully developed a technology for the fortification of common salt with iron[12], though this has not yet been used on a large scale. The fortification of fish sauce with iron is being attempted in Thailand[13], while an American group has suggested iron EDTA for food fortification[14].

IODINE DEFICIENCY DISORDERS

Disorders attributable to iodine deficiency are still a major public health problem in several parts of the world. The fortification of common salt with iodine is a widely used approach for combating the problem.

Neonatal hypothyroidism: Using radio-immunoassay techniques[15], it has now been shown that in goitre-endemic areas the incidence of neonatal hypothyroidism is several folds higher than what was once believed to be the case on the basis of the prevalence of frank cretinism.

New endemic areas: A disturbing finding in recent years is the emergence of new goitre-endemic areas in the irrigated plains of Asian countries such as India, Myanmar and Indonesia. The precise factors underlying this have not been identified; but it is being suspected that the modern practice of intensive agricultural technology could have resulted in the diminished bioavailability of soil iodine and the consequent diminished content of iodine in food and water.

The possible role of an excessive use of fertilisers, pesticides and food additives has also been suspected.

Goitrogens: The possibility that goitrogens may be involved in the emergence of new goitre-endemic areas is suggested by the finding of high levels of urinary excretion of thiocyanate in a significant proportion of subjects in these areas. Peroxidase-inhibiting goitrogens have been suspected to interfere with the effective utilisation of iodine by the thyroid gland. Such goitrogens could be either present in foods or as food contaminants.

The possible role of selenium deficiency in aggravating the goitre

problem also deserves consideration.

It is thus clear that problems of iodine deficiency are acquiring new dimensions in the context of intensive agricultural technology and ecological and environmental factors incidental to 'development'! In combating these new dimensions we may have to look for strategies other than (and over and above) the conventional approach of the fortification of common salt with iodine.

HYPOVITAMINOSIS A

Vitamin A deficiency has been a major public health problem in some countries of Asia — notably Bangladesh, Nepal, India, Indonesia, Myanmar, Sri Lanka and Thailand, and, to a lesser extent, in some other countries.

The earlier strategy: The earlier strategy for the control of vitamin A deficiency in many Asian countries (which is still operative) largely consists in the administration, at periodic intervals of six months, of a massive oral dose of 200,000 IU of vitamin A to children under three years of age in endemic areas.

This approach was originally envisaged purely as an *interim* measure, and as a temporary adjunct to dietary improvement, till such time as the obvious natural approach of dietary imrovement and the promotion of an increased intake of β-carotene-rich foods by the population at risk could be satisfactorily ensured.

The logical future approach: Developing countries, now confronted with the problem of vitamin A deficiency, are fortunately blessed with a wide array of inexpensive foods rich in provitamin A carotenoids. Apart from the well-known conventional green leafy foods, recent studies at NIN, Hyderabad, have shown that the leaves of a surprisingly large number of plants growing wild in the countryside (and which do not need specially tended 'kitchen gardens' for their cultivation) could also prove to be good sources of β-carotene. Studies from the same Institute have also recently highlighted the enormous — as yet untapped — potential that exists for the effective utilisation of a whole range of inexpensive locally available fruits including, specially, mangoes for combating vitamin A deficiency in poor tribal populations[16].

Spirulina, a rich source of provitamin A carotenoids, has been developed from the blue-green alga, *Spirulina fusiformis*[17]. With further research and improved technology, it should be possible to identify ways by which spirulina, which can be widely harvested, can be incorporated in a potent, inexpensive and acceptable form in the dietaries of developing countries.

There is also the possibility of augmenting the cultivation of red palm in India, Indonesia and Bangladesh. With further research designed to identify ways by which the provitamin A fraction of red palm oil could be utilised (instead of being discarded, as is unfortunately happening with the palmolein product being currently produced in Malaysia), it could become a powerful tool for combating vitamin A deficiency. The above foods can easily meet the vitamin A requirement.

It will thus be clear that developing countries have a vast, as yet, untapped potential for further augmenting the production of such foods. It will be extremely short-sighted and imprudent not to put these valuable, indigenous and inexpensive resources to proper use.

There is enough evidence that provitamin A carotenoids are at least as effective as (if not more than) synthetic vitamin A in the matter of combating vitamin A deficiency. Several green leafy vegetables are not only good sources of carotenoids, but they contain other nutrients as well, which can also contribute in some measure to better nutrition.

The claim of mortality reduction with vitamin A supplementation: There have been claims that periodic massive doses of synthetic vitamin A could bring about a significant reduction in child mortality[18,19,20].

These claims have not been borne out by independent studies by other groups of scientists[21,22]. Some basic defects in the designs of studies on which the claim of mortality reduction is based throw doubt on the validity of the claim[23].

GROWTH RETARDATION

As was pointed out earlier, growth retardation is widespread in many developing countries and this is now being generally considered as largely a reflection of protein-energy undernutrition. Indeed,'moderate' growth retardation in the classical Gomez scale is being considered as synonymous with moderate PEM. While this may still be largely true, there is the distinct possibility that, with the progressive elimination of PEM, micronutrient deficiencies could emerge as important bottlenecks to growth, and this could have implications both with respect to the prevention of growth retardation and with regard to the interpretation of its significance.

The results of studies of the effect of energy and/or protein supplementation on growth retardation are contradictory[24,25,26], possibly for the reason that a multiplicity of deficiencies may be involved in growth retardation; and the predominant nutrient deficiency bottleneck to growth may vary as between population groups. It has also been claimed that in some situations, linear growth faltering is seen among pre-school children subsisting on diets adequate in energy, protein and essential amino-acids[27], suggesting the possibility that deficiencies of micronutrients such as zinc, and possibly iron, copper, iodine and vitamin A, may be mainly involved in growth retardation, at least in some locations.

Mental retardation: There is yet another important dimension to this problem. Physical growth retardation in children has been shown to be associated with some degrees of mental underdevelopment. It cannot be assumed that all nutrient deficiencies which affect physical growth will also impair mental development to the same extent.

The relationship between physical growth retardation and mental underdevelopment in undernourished populations may well depend on the nature of the nutrient deficiency predominantly responsible for growth retardation in a given location. For instance, this relationship may not be the same in situations where PEM is the major causal agent on the one hand, and in others where some micronutrient deficiencies are primarly involved.

With the possibility that in the transitional stages of development, energy-protein deficits are likely to be progressively eliminated and micronutrient deficiencies may come to the fore, these questions could acquire

considerable practical importance in the future.

ZINC DEFICIENCY

Zinc in soils and plants has particularly emerged as a possible major factor in the wake of the intensive application of modern agricultural technology. Studies in Bangladesh[28] have revealed the possibility of poor zinc content in a wide range of foods, including fruits, vegetables, legumes, grains, grasses and fodder crops. Zinc deficiency has been particularly noticed in rice crops grown on alkaline, wet and water-logged soils. These findings could be of far-reaching importance to the nutrition of human populations in South-east Asian countries.

The possibility that the deficiency of zinc could have a bearing on three of the major nutritional deficiency problems of South-east Asia, namely PEM, hypovitaminosis A, and anaemia, has to be seriously considered. Zinc is a component of many key enzymes involved in protein synthesis. Zinc deficiency could, therefore, aggravate PEM and could be a factor in contributing to growth retardation.

Zinc deficiency could also induce the reduction of RBP (retinol-binding protein) in the plasma and liver, leading to the poor mobilisation of hepatic vitamin A. Thus, zinc deficiency may be a factor in the pathogenesis of hypovitaminosis A. The greater vulnerability of the rice crop to zinc deficiency could be reflected in the poorer zinc nutritional status of the rice-eating populations of Bangladesh and the eastern part of India. This could partly explain the more pronounced endemicity of vitamin A deficiency in these regions rather than in the 'wheat-eating' western and northern parts of India. The possible importance of the zinc nutritional status from the point of view of the management of iron deficiency anaemia has already been referred to.

SELENIUM MALNUTRITION

The only firmly established role of selenium in the body is as the prosthetic group of the enzyme glutathione peroxidase which is present in the cell cytosol and mitochondria, and functions to reduce hydroperoxide. This reaction has special significance in the protection of the polyunsaturated fatty acids located within the cell membranes. It apparently supplements the role of vitamin E as a primary antioxidant by scavenging reactive oxygen species and free radical intermediates of polyunsaturated lipid peroxidation. Substantial interactions exist between selenium and vitamin E. Deficiency symptoms associated with the inadequacy of one nutrient can be reversed by the supplementation of the other. Selenium, in common with sulphur, shares an affinity with heavy metals such as cadmium, mercury and silver. Supplements of selenium protect against the toxic effects of these metals.

Selenium levels in blood and tissues are very much influenced by dietary selenium intake. Selenium levels are very low (0.05-0.08 mcg/ml) in subjects from New Zealand, where the dietary intake is around 20-30 mcg/day[29]. In the selenium deficient areas of China, the blood levels are 0.009 mcg/ml[30].

Both the excess and deficiency of selenium have been shown to be associated with diseases in animals and humans as indicated below:

Excess (toxicity): 'Blind staggers' in animals[31]; 'Alkali disease' in horses[31]; and chronic dermatitis, loss of hair and brittle nails in humans[32].

Deficiency: Keshan disease (cardiomyopathy); Kaschinbeck disease — endemic osteoarthritis; a side-effect of total parenteral nutrition[31]; association with kwashiorkor[33]; association with increased risk of thrombotic episodes[34]; association with cancer[35,36]; and aggravation of iodine deficiency[1].

While selenium toxicity is reported in areas with highly alkaline soils, selenium deficiency has been reported from widely different areas of the world.

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Ecological and environmental degradation unleashed by developmental programmes is likely to generate problems related to micronutrient nutrition hitherto unsuspected.

They are also likely to influence the nature, dimension and course of community health problems related to micronutrients such as the emergence of new areas of goitre endemicity, or the aggravation of the problems of fluorosis in parts of Asia.

Research on micronutrition may, therefore, command a high priority in the Nutrition Research Agenda of the next two decades.

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Reviews & Comments

Lathyrism — A Historical Review

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Lathyrism, a disease associated with the excessive consumption of the pulse *Lathyrus sativus,* has a long and fascinating recorded history. *L sativus* is a native pulse of southern Europe and West Asia. It is still known to be cultivated in India, Bangladesh, Nepal, Pakistan, Ethiopia, Iran, the Middle East, southern Europe and South America.

HISTORY

Early history: Evidence is available of the consumption of *L sativus* leading to the causation of neurolathyrism from ancient times. As early as 400 BC, the famous Indian physician Charak[1] associated the consumption of 'Triputa' pulse (*L sativus*) with the development of 'Kalayakhanj' (lathyrism). Hippocrates[2] (460-377 BC) wrote that at Aions "all men and women who ate peas continuously became impotent in the legs and that the state persisted". *Bhave Prakash,* written in 1550[3], mentioned that, "Triputa pulse caused men to become lame and crippled, and irritated the nerves."

The first authentic description which clearly established the nexus between the consumption of *kesari dal* and the causation of lathyrism was given by General Sleaman in his book *Rambles and Recollections of an Indian Official*, published in 1844[4]. It was about an outbreak of lathyrism which occurred in 1829-31, affecting cattle and humans in the district of Saugor of central India. Another instance which appears to be an accidental human experiment happened during the Second World War in a German concentration camp on the Ukrainian border. From September 1942, a diet containing 400 gm of *L sativus* and 200 gm of bread made out of barley and grass was served to about 600 newly-inducted and 600 other inmates of the camp; by December 1942, more than 800 of these inmates developed lathyrism[5].

Recent history: The Indian Council of Medical Research (ICMR) carried out extensive studies on lathyrism which clearly established the association between the consumption of *L sativus* and the occurrence of lathyrism[6]. A neuro-excitatory toxin p-N-oxalyl-L-x-p-di amino propionic acid (ODAP/BOAA) was isolated and identified from *L sativus*[7,8]. Spencer *et al*[9] demonstrated, in an experiment on monkeys, the neurotoxic activity of *L sativus* and its toxin BOAA.

In vitro field studies to develop toxin free strains remain inconclusive[10]. The precise nature of the mechanism controlling the bio-synthesis of βODAP in *L sativus* is still not known. Conventional breeding methods have so far failed to evolve stable low-toxin varieties safe for human consumption. Several factors are believed to influence the rate of synthesis and concentration of neurotoxin in *L sativus*[11]. Thus, there has been evidence of a twofold increase of the toxin level in seeds of *L sativus* under osmotic stress[12]. The toxin appears to be a natural protector, ensuring the survival of the plant under drought conditions or osmotic stress. Intensive efforts over the last three decades directed towards developing a stable, safe variety of *L sativus* have not met with success.

From available evidence, it would appear that epidemics of the large scale that used to occur periodically in the earlier years have not been reported since 1974[13]. However, sporadic cases do occur from time-to-time. The probable reason for this change in the profile of the disease will be discussed later in this article.

LEGAL PROVISIONS

Several punitive measures have been taken from time-to-time to control the problem. As early as 1671, the Duke of Wurtemberg issued an edict by which the use of flour of *L sativus* was prohibited for making bread because of its paralysing effect on legs. Several edicts were issued by his successors between 1705 and 1714[14]. In France, the prohibition of *L sativus* was recommended by Des Paranches[15] in 1829, and in Algeria[14] in 1881. In Russia, its cultivation was banned in

1945, following an outbreak of lathyrism in southern Russia; the outbreak resulted from the devastation of other less hardy crops during the Second World War and the consequent reliance on lathyrus.

In India, the cultivation of *L sativus* was prohibited in Allahabad[14] in 1870, because of successive epidemics reported by Irving[16]. Buchanan[14], who investigated lathyrism in the Central Provinces during 1896-1902, wrote: "Hon'ble Mr Fuller has informed me that a Deputy Commissioner in Seoni (now a district of Madhya Pradesh) has a 'Malguzar' imprisoned after the outbreak of lathyrism in Seoni." He further wrote, "It is not likely that a criminal prosecution will be necessary; but if there is no doubt about knowledge, then Section 328 of the Indian Penal Code, which makes it a criminal offence 'to administer or cause to be taken any unwholesome drug or thing with intent and knowing it would hurt' could be brought into force."

The late Maharaja of Rewa State (now a district of Madhya Pradesh), Shri Venkat Raman Singh passed an order on November 29, 1907, prohibiting the cultivation of *'Matra'* (*L sativus*). The Maharaja noted: "The paralysis chiefly affects the labour class, more particularly, those of lower caste and specially those who are given their wages in food ('*Harwaha*'). The paralysis is due to eating *'kesari dal'*, locally called *'Butterha'* which is cheap in famine times." He, therefore, forbade the cultivation of this vetch and placed restrictions on the trading of the grain. However, as the vetch was the staple food of the population, it was impossible to prevent its cultivation in outlying villages. In a year or so, the order was forgotten and the dal continued to be cultivated without any restraint[17].

On the basis of Patwardhan's suggestion in 1948[18], the Government of the Central Provinces ordered that *L sativus* shall not be sold unless in a mixture with other food grains in which its proportion shall not exceed 33 per cent. Around the same time, the Government of Madras had also completely banned the import and sale of *L sativus* within the Madras province.

Under the Prevention of Food Adulteration Act (PFA) of India[19], foods which are considered injurious to human health are prohibited for human consumption. Owing to the potential danger to health associated with the consumption of *L sativus*, lathyrus has been included among foods to be prohibited under the above Act. This is in consonance with measures undertaken by several other countries to discourage the consumption of *L sativus.*

Rule 44 A included in the PFA Act 37 of 1954 states that "no person in any State shall, with effect from such date as the State Government concerned may, by notification in the Offical Gazette specify in this behalf, sell or offer or expose for sale or have in his possession for the purpose of sale under any description or for use as an ingredient in the preparation of any article intended for sale — *kesari* (*L sativus*) and its products, etc"[19].

This was further reinforced in 1981 with the Planning Commission's recommendation of the prohibition of the cultivation of *kesari dal* (*L sativus*)[20].

The Supreme Court of India ordered (order dated March 5, 1982, in writ petition number 153 of 1982 under Article 32 of the Constitution) an enquiry into the incidence of lathyrism in bonded labour and its relationship to the practice of paying wages to the agricultural workers in the form of kesari dal (*L sativus*) by the employers. The enquiry, conducted by the Collector of Rewa (MP), concluded that all the affected persons under the survey belonged to the agricultural worker families (or *'Harwaha'* families) and as such, the probable reason could be that the members of such families were paid their wages in *kesari dal* (*L sativus*); and kesari dal was a major component of their diet (in the form of *chapati* as well as dal). It was concluded that "the assumption that the regular consumption of *kesari dal* (*L sativus*) in whatever form, has a crippling effect on the lower limbs of the body, appears to be true"[21].

In view of the above, the Government of India recommended that the State (Madhya Pradesh) Government should take action to ban the cultivation of the *L sativus* crop and for the enforcement of Rule 44 A under the PFA Act for banning its sale. The Government of MP, which is producing 40 per cent of the total produce of *L sativus* in India and has the majority of lathyrism cases, passed an order on December 15, 1983, banning the payment of wages in the shape of *L sativus* to the farm labourers as they are the most vulnerable section of the population at risk of developing the disease[22].

COMMENTS

It will thus be seen that lathyrism caused by the consumption of *L sativus* is a subject of antiquity. Certain facts stand out and it will be useful to recall these.

- There is clear epidemiological, biochemical and experimental evidence to support the cause-and-effect relation between the consumption of *L sativus* and the occurrence of lathyrism[23].

- Efforts to evolve zero-toxin strain or a low-toxin level stable strain of *L sativus* have thus far proved futile.

- On the basis of present knowledge, the enforcement of legal provisions to discourage the production and consumption of *L sativus* by the population, irrespective of its economic and social background, is a logical step. India's food grain stock situation is fortunately satisfactory; and there cannot be any justification for relaxing the ban on the cultivation and consumption of foods of potential harmful nature on the ground of possible acute food shortages.

- Unfortunately, the implementation of measures for banning the cultivation and sale of *L sativus* by the Government of India and by different state governments — particularly those producing *L sativus* — have been halfhearted, possibly because of political reasons. As a result, despite all the laws, the total production of *L sativus* in the country has shown no decline, and is steadily maintained at around 12,614,600 tonnes (1989-90).

- Since legal provisions and media support have created a strong impact in the minds of the consumer, causing the rejection of *L sativus*, the 'producers' and unscrupulous traders have sought other outlets for their product. Adulteration of the most popular pulse, red gram (*Cajanus cajan*) and of the flour of Bengal gram (*Cicer arietinum*) with *L sativus* has become a flourishing practice in some parts of this country. This 'outlet' has diverted the bulk of *L sativus* from the rural to the urban scene and from Madhya Pradesh to other parts of the country, especially South India. As a result, the earlier epidemic character of lathyrism has now given place to the sporadic oc-

currence of cases which apparently do not excite the same attention and escape detection. Though low levels of intake of *L sativus* as an adulterant in urban populations may not result in frank paraplegia of lower limbs, the toxin ODAP may continue to cause motor-neuron deficits as there is evidence based on animal experiments that BOAA selectively inhibits mitrochondrial NADH-dehydrogenase[24].

Gopalan drew attention to this question in his paper entitled 'A Revisit to Rewa'[25] in which he compared the situation with respect to lathyrus consumption in 1961 and 1981. He noted that while lathyrus consumption by the poor of Madhya Pradesh was being discouraged, the use of lathyrus for the purpose of adulteration of other pulses by unscrupulous traders was gaining momentum. He warned that if this trend was not checked, neurolathyrism, so far localised to some parts of Madhya Pradesh and Bihar, may well become a national problem.

Recent ill-conceived attempts to relax the ban on the cultivation of lathyrism must be resisted. To relax the ban on cultivation at this stage, when a 'profitable' outlet (far more profitable than the earlier practice of the use of lathyrus as a payment of wages to poor agricultural labourers) has been developed by unscrupulous traders, will be to open the floodgates. The ban and its rigorous enforcement have, if anything, become even more relevant and necessary today than in the past.

All efforts should be made to develop a zero or low-toxin level stable strain of *L sativus*. Till such time that these efforts succeed, vigorous efforts should continue in order to ensure that the pulse is not utilised for human consumption. The pulse may perhaps be used as an animal feed but even here, it will be necessary to ensure that lathyrus contributes a small fraction of the total animal feed. Now that a 'profitable outlet' in the form of adulteration has been devised by unscrupulous traders, any relaxation of the ban on cultivation and human consumption at this stage could further worsen the problem.

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FOUNDATION NEWS

Organisation of a National Health Scout Movement: A Task Force meeting on 'Mobilisation and Training of Youth for a Health Scout Movement' was held at New Delhi on May 18. Drs Saroj Jha, Sharda Jain, Shanti Ghosh, Saramma T. Mathai, Bhagvan Prakesh, A.K. Govila, K.S. Sundaram, C. Gopalan,.Ms L. Krishnamurthy, Mr K.S. Krishnamurthy, Ms Deepa Gupta and Ms Harvinder Kaur participated.

In the follow-up meeting at Gwalior the protocol for the project was prepared and finalised. The participants were Drs Saroj Jha, N.R. Bhandari, Saramma T. Mathai, A.K. Govila, S.Upadhyay, S.S. Kushwaha, Y.D. Badgayan, Ranjana Tiwari, Vasundhara Aras and Ms Deepa Gupta.

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