IODISED SALT AND ARMED FORCES

Dear Editor,

Salt Iodine Monitoring in the Armed Forces (MJAFI 1999;55:187-8) by Maj MP Cariappa et al made very interesting reading. The quality control of lowly item like salt, though an important matter, is often left at the mercy of vendors. The authors have rightly brought out the need for all of us to be more vigilant. I would like to bring out certain related matters.

Iodine deficiency is not very widespread in India except for certain endemic areas [1]. Army personnel, the adult wandering population, with well-balanced diet, has extremely low risk of nutritional iodine deficiency. The iodised salt, therefore is not indicated for general consumption in the diet of average healthy soldier.

On the other hand, iodine toxicity due to forced administration of varying quantities of iodine through salt in diet is the real threat. Such toxicity could lead to hyperthyroidism [2]. In case of pregnant lady it could lead to congenital fetal abnormalities. Will such a disability, in the soldier as well as in her child, then be attributed to military service? Can an individual claim compensation for expos-

ing him/her and her unborn children to such unwarranted risk? The health planners and administrators should consider this aspect before it is too late. The logical step, therefore is to discontinue iodisation of salt supplied to the Armed Forces. Only the troops posted to endemic iodine deficiency areas should be issued iodised salt as a preventive measure.

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REPLY

I thank Lt Col Gokhale for having brought attention to an issue that is creating a roaring controversy in the community, both among the lay populace and the medical profession too. Contrary to widespread opinion, the threat that Iodine deficiency poses to India, is very real and not so subtle, if viewed under the spectrum of Iodine Deficiency Disorders. Sample surveys have been conducted in 25 states and 4 UTs of the country, which revealed that out of 2 75 districts surveyed so far, IDD was a major public health problem in 235 districts [1].

Pandav et al (1996) have found a high level of prevalence of IDD, even among school children in NCT of Delhi [9]. If endemicity is decided on the basis of presence of goitre alone, then the picture will appear rosy. However, the ICCIDD, with the definition "IDD's", paints a different scenario, and various authors have found Iodine deficiency to be prevalent in almost all part of the country which were hitherto considered non-endemic [2].

As brought by Lt Col Gokhale, the diet of an individual in addition to the iodised salt consumed, may supply the daily requirement of iodine (150 mcg per day). However, vegetables, staple foods, water, etc from an area which is environmentally deficient in iodine, will not provide a source of iodine for the individual. Iodine content of foods and of total diets differ appreciably and are influenced by geochemical, soil and cultural conditions which accordingly modify iodine intake [3].

Hence it would not be correct to assume that a seemingly balanced diet as consumed by our troops would supply essential micro-nutrients without the added input from iodised salt.

Concern has been expressed about the possible toxicity of Iodine through consumption of iodized salt, in perceived non-endemic areas. Through studies based on balance and excretion over 24 hr, it has been found that a safe level of iodine intake is 1000 mg [4].

If the salt supplied to the troops is iodized, and the consumption is in concordance with the national average for adult per day of 13.8 gm [5] then at 15 ppm (recommended level at consumer end), the Iodine intake from salt alone, would be 207 mg and if an additional

100-200 mg was absorbed from food and water, the total Iodine intake would still be within safe limits and not cause toxicity in a non-Iodine deficient individual. Thus apprehensions concerning the National Iodine Deficiency Disorders Control Programme policy of nationwide salt iodized are fundamentally baseless. Public health planners per se have to prioritize the good of the greater many at risk. For individuals found to have autonomous thyroid tissue, Joseph et al (1980) have reported that iodine intakes of less than 100 mg/day pose no risk [6]. Such individuals could be advised non-iodised salt or low salt diets.

Thus a soldier receiving iodized salt in his ration is at no risk for iodine toxicity. However, it must be reiterated that adequate quality control is maintained as proposed by Cariappa et al.

Iodine deficiency has been found to have an adverse effect on human reproduction and also child survival. Thilly and Hetzel found that the IMR for mothers given iodine supplements during pregnancy was significantly less than those who were not given iodine [7]. Iodine can in fact be considered as the unborn child's earliest benefactor!

Hence the ICC IDD and Public Health planners have rightly given an impetus to the goal of elimination of IDDs as an entirely preventable scourge.

The feasibility and relevance of selective iodisation is considered inappropriate at a national level and it is perceived that elimination can only be achieved by universal salt iodisation [8]. It may be interesting to note that the Govt of Maharashtra has not reversed its stand on the issue, despite widespread protests, keeping in view the long term health interests of the people.

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STUDY OF GLOMERULAR FUNCTION IN NEONATES

Dear Editor,

This is with reference to the original article on the subject of glomerular function in neonates [1]. The ultimate objective of any research should be to translate the benefits to the betterment of patient care. That neonates have a high plasma creatinine levels at birth and normalizing over period of time is a well-established text book fact. However, the riddle of the high plasma creatinine levels in term and particularly in preterms seems to be solved. Once the umbilical cord is severed, the perfect intrauterine maternal-fetal biochemical balance is disturbed. Thereafter, the already transferred exogenous adult level creatinine will rapidly disappear in the first urine specimens passed by the now autonomous newborn infant. A new steady state is achieved in due time, based on independent neonatal factors. One of the factors is the unusual occurrence of tubular creatinine reabsorption. The authors hypothesize that this latter temporary phenomenon is attributable to back flow of creatinine across leaky immature tubular and vascular structures. With time, maturational renal changes will impose a barrier to creatinine [2]. Secondly by suggesting that there is scanty Indian literature on the subject of renal functions in neonates, are the authors contemplating difference in different racial groups? Narang et al [3] have studied the maturation of neonatal glomerular function as evidenced by serum creatinine and creatinine clearance in 15 preterm small for dates infants (group 1) and compared with values obtained in 15 preterm appropriate for date babies (group 2) on 3rd, 7th and 14th postnatal days. Creatinine clearance was shown to be statistically less ($p < 0.005$ on day 3, $p < 0.001$ on day 7 and $p < 0.01$ on day 14 respectively) in group 1 as compared to group 2, thereby implying slower renal maturation in small for date preterm babies. This highlights the methodology error in the present report [1]. Thirdly

adaptation in neonatology of the once daily concept of aminoglycoside administration has been well studied. Langhendries JP et al [4] have been able to demonstrate after a trial period of single dose amikacin in 37 babies that minimum inhibitory concentration profiles tested in 43 successive bacterial strains remained adequate. A prospective randomized study compared the pharmacokinetics of once daily gentamicin regime in preterms to the conventional twice-daily regime. The observations suggested that once daily dosage regime guarantees initial peak serum concentration above the minimum inhibitory concentration of gram negative bacteria and trough concentration below potentially toxic levels of 2 µg/ml [5].

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REPLY

We thank the reader for expressing his views, which however, turn out to be his own assumptions but not based on the facts presented in the article. We, the authors, did not postulate any hypothesis that decrease in a serum creatinine in neonatal period is due to backflow of creatinine across leaky immature tubular and vascular structures as quoted by the reader but we stated that it is due to increase in Renal blood flow and redistribution of intrarenal pattern of glomerular blood flow as viewed by Arant BS Jr also [1]. Similarly mentioning the fact by the authors that "there is scanty Indian literature on this subject" is not expected to lead one to think (by any stretch of imagination) that racial differences are being postulated by the authors. The study is descriptive presentation of facts as observed in subgroups of neonates classified as per gestational age. Pointing out this to be a methodological error in the study is not justifiable and unwanted because various other authors [2,3] showed renal functional maturation is gestational age dependent

rather than birth weight dependent though the study by Narang et al as quoted by the reader narrates Renal function in SFD and AFD preterm babies.

Further, once daily dosage regimen of aminoglycoside in term guaranteeing initial peak serum concentration is a well known fact which was not at all countered by the authors. In the study [4] quoted by the reader the workers used Gentamicin in preterm neonates at a dosage of 4 mg/kg/24 hours when serum creatinine was less than 1 mg/dl. We recommended that dosage of aminoglycoside is to be adjusted as per glomerular functions (Serum creatinine and GFR) more so in preterms.

To reassure the reader, the authors feel that the purpose of research is very well served in the study, as it clearly reemphasises and draws the attention of one and all to be extremely cautious in the dosage of drugs to be administered to preterm neonates.

Thus, all the assumptions made by the reader are just unfortunate