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Correspondence

Need for better anthropometric markers for prediction of cardiovascular risk in nutritionally stunted populations

Sir,

The article by Thakappan and colleagues in the January 2010 issue of the Journal, defines the enormous threat of non communicable diseases which the State of Kerala faces[1]. The results are aptly projected as the harbinger of what is going to happen in India and rest of the developing world. The study is all the more important for its finding that the anthropometric measures like body mass index (BMI) and waist circumference (WC) were only modest in predicting biochemical risk factors in this population. In this context, we would like to highlight the contribution of developmental origin of adult onset diseases and to project calf anthropometry as a marker to define the early life growth perturbation in this nutritionally stunted population. Though genetics and ethnic factors play role in the development of conventional risk factors, current literature is reassuring that lifestyle interventions can postpone the onset of adult onset diseases as a public health initiative[2].

The National Family Health Survey (NFHS) -3 data on malnutrition and stunting in Kerala done in 1992 and 2005-2006 show (Table) a significant nutritional stunting irrespective of the improvement in per-capita calorie intake (up to 400 Kcals/day)[3,4]. Obesity in adults in the NFHS-3 and the reported study is also around 30 per cent, reflecting the double burden of nutritional diseases[1,3].

In the absence of childhood health records and basic birth weights, the markers of nutritional stunting in adult life could reflect the developmental origin of adult onset diseases in them[5]. Persistence of nutritional stunting in children is unlikely to be due to failure of the supplementary programmes to reach the deserving, given the social equity and health awareness in Kerala[6]. The alternate possibility is that the nutritionally stunted children are failing to respond to the nutritional supplementation with adequate catch up growth or a newer pool of low birth weight babies are being born[7]. It is very likely that this trend will persist for at least for a few more generations because of the vicious cycle of childhood undernutrition and maternal health status[8]. Child development centre at Thiruvananthapuram, Kerala, has been following a cohort of low birth weight children, and normal weight children for 16 years[9]. At the age of 16, 15 per cent had BMI of more than 22 and low density lipoprotein cholesterol level of more than 130 mg/dl. More than 55 per cent had high density lipoprotein cholesterol levels less than 45 mg/dl concurring with the younger age onset of risk factors as noted in this study[1]. The lower birth weight predicted higher triglyceride levels, though their adolescent BMI levels were lower.

Table. Selected parameters of nutritional status in NFHS[1-3]

Nutritional staus	NFHS -3 (2005-06)			NFHS-2 1998-99	NFHS -1 1992-93
	Total	Urban	Rural		
Children <3 yr (stunted %)	26.5	27.3	26.0	28	32.8
Children <3 yr (wasted %)	15.6	9.1	18.8	13	13.7
Chil: <3 yr (underweight %)	21.2	15.3	24.0	21.7	22.1
Women (BMI < normal %)	12.5	9.1	14.3	18.7	na
Men (BMI < normal %)	11.9	11.2	12.3	na	na
Women (BMI > 25 as %)	34.0	40.1	30.9	20.6	na
Men with BMI > 25 as %	24.3	28.0	21.9	na	na

BMI and waist circumference are the anthropometric measures utilized as per the WHO non-communicable disease surveillance and both the parameters were just modest in predicting the step 3 risk factors in this study[1]. Large population survey done in Kerala has observed better body mass indices not to be associated with adult onset disease mortality[10]. Eighteen year follow up of the Monica cohort which adopted a variety of lifestyle modification also showed that increase in BMI was not associated with increasing diabetes or cardiovascular diseases[11]. For a similar change in body composition, nutritionally stunted population will have a higher BMI, given the lower values in denominator. But it is well known that Asians develop the risk factors at a lower BMI[12]. This is a clear indication that metabolic abnormalities are developing in Keralites at normal weight, which is referred to as the "metabolically obese normal weight individuals"[13]. Sarcopenic adiposity, *i.e.,* less muscle and more fat, is the common denominator for both nutritionally stunted adults, and thin fat Indian babies and is characterized by abnormal distribution of upper body and visceral fat[14]. For recognizing the visceral obesity in adults, waist circumference was used as a surrogate[15]. In study by Thankappan *et al*[1] the predictive utility of the waist circumference to the biochemical risk factors was modest, suggesting that by the time the adult cut-off values are reached the risk factors are already established. Similarly the recent registry for acute coronary syndrome created in Kerala registered 25,000 patients and 2/3rd had a waist circumference less than 90 cm, the cut-off value currently recommended for males of Asian origin (personal communication, Dr PP Mohanan for the Kerala ACS registry).Waist circumference may fare better if lower cut-off values or indexing to height is attempted[16]. The alternative surrogate is the fat free muscle mass for which calf muscle circumference or urinary creatinine can be used as surrogate markers[17]. Calf circumference has shown good correlation with carotid intima media thickness in adult life[18]. Fat free muscle mass measured by calf circumference and skin fold thickness at maximal calf circumference could be a good anthropometric marker to understand sarcopenia in adults. The subcutaneous fat at the calf also is a negative predictor of adult onset diseases, and by measuring the same we may be able to define the calf muscle mass better[18,19].

Waist circumference is low cost non laboratory marker for cardiovascular risk reduction[15]. As a self measured marker it's now a popular public health strategy in Japan[15]. Unlike body mass index, measures of abdominal obesity takes the risk factor assessment to early life experiences[14]. But by the time the recommended cut-off value is reached, the risk factors are well established in this traditionally stunted population. The other alternative marker of growth perturbation in early life is adult height which is a good indicator of cardiovascular and body weight scaling[5]. It will be interesting to see the data analyzed with height alone as a variable in these multivariate models. But studies done at Framingham have failed to demonstrate the utility of adult height alone as a risk predictor, given the time trends in increasing height of the population and the age related decline in vertebral height[20]. During the last three decades Kerala children also have shown a trend in increasing height[21].

Cohort studies done in the developed world and a few studies in the developing world have recognized short leg as a useful marker of early life growth perturbation, with adult onset cardiovascular diseases and risk factors[22,23]. Growth of the human newborn, proceeds in the cephalocaudal direction, in early infancy[24]. Both calf length and calf circumference are simple measurements which can be done in early adult life. If substantiated in epidemiologic studies, calf anthropometry, could turn out to be the non laboratory marker for adult cardiovascular risk reduction in populations suffering nutritional stunting. The calf measures could be the indirect markers of the contribution of early life growth perturbations to adult onset diseases in adult life. Essentially calf anthropometry defines the best possible growth for the most affected segment in growth perturbation and can be ascertained in early adulthood.

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References

1. Thankappan KR, Shah B, Mathur P, Sarma PS, Srinivas G, Mini GK, *et al*. Risk factor profile for chronic non-communicable diseases: results of a community-based study in Kerala, India. *Indian J Med Res* 2010; *131* : 53-63.
2. Vartiainen E, Laatikainen T, Peltonen M, Juolevi A, Mannisto S, Sundvall J, *et al*. Thirty-five-year trends in cardiovascular risk factors in Finland. *Int J Epidemiol* 2010; *39* : 504-18.
3. National Family Health Survey, data on Kerala available as Kerala.pdf. Available from: *http://www.nfhsindia.org/pdf/Kerala.pdf*, accessed on March 2, 2010.
4. Suryanarayana MH. Economic reform versus food security: Kerala's Gordian knot. *J Int Dev* 2001; *13* : 239-53.
5. Wannamethee SG, Shaper AG, Whincup PH, Walker M. Adult height, stroke, and coronary heart disease. *Am J Epidemiol* 1998; *148* : 1069-76.
6. Nath I, Reddy KS, Dinshaw KA, Bhisey AN, Krishnaswami K, Bhan MK. Country profile: India. *Lancet* 1998; *351* : 1265-75.
7. Raman Kutty V. Why low birth weight (LBW) is still a problem in Kerala (Open Library). Available from: *http://openlibrary.org/b/OL3332974M/Why_low_birth_weight_(LBW)_is_still_a_problem_in_Kerala*, accessed on April 12, 2010.
8. Bhattarai MD. Three patterns of rising type 2 diabetes prevalence in the world: need to widen the concept of prevention in individuals into control in the community. *JNMA J Nepal Med Assoc* 2009; *48* : 173-9.
9. Nair MKC, Nair L, Chacko DS, Zulfikar AM, George B, Sarma PS, *et al*. Markers of fetal onset adult diseases: a comparison among low birthweight and normal birthweight adolescents. *Indian Pediatr* 2009; *46* (Suppl.) : s43-7.
10. Sauvaget C, Ramadas K, Thomas G, Vinoda J, Thara S, Sankaranarayana R, *et al*. Body mass index, weight change and mortality risk in a prospective study in India. *Int J Epidemiol* 2008; *37* : 990-1004.
11. Krachler B, Eliasson M, Stenlund H, Johansson I, Hallmans G, Lindahl B, *et al*. Population-wide changes in reported lifestyle are associated with redistribution of adipose tissue. *Scand J Public Health* 2009; *37* : 545-53.
12. Yusuf S, Hawken S, Ounpuu S, Bautista L, Franzosi MG, Commerford P; Interheart study Investigations, *et al*. Obesity and the risk of myocardial infarction in 27,000 participants from 52 countries: a case-control study. *Lancet* 2005; *366* : 1640-9.
13. Romero-Corral A, Somers VK, Sierra-Johnson J, Korenfeld Y, Boarin S, Korinek J, *et al*. Normal weight obesity: a risk factor for cardiometabolic dysregulation and cardiovascular mortality. *Eur Heart J* 2010; *31* : 737-46.
14. Yajnik CS, Fall CH, Coyaji KJ, Hirve SS, Rao S, Barker DJ. Neonatal anthropometry: the thin-fat Indian baby. The Pune Maternal Nutrition Study. *Int J Obes Relat Metab Disord* 2003; *27* : 173-80.
15. Funahashi T, Matsuzawa Y. Metabolic syndrome: clinical concept and molecular basis. *Ann Med* 2007; *39* : 482-94.
16. Mombelli G, Zanaboni AM, Gaito S, Sirtori CR. Waist-to-height ratio is a highly sensitive index for the metabolic syndrome in a Mediterranean population. *Metab Syndr Relat Disord* 2009; *7* : 477-84.
17. Kuriyan R, Thomas T, Kurpad AV. Total body muscle mass estimation from bioelectrical impedance analysis & simple anthropometric measurements in Indian men. *Indian J Med Res* 2008; *127* : 441-6.
18. Debette S, Leone N, Courbon D, Gariepy J, Tzourio C, Dartigues JF, *et al*. Calf circumference is inversely associated with carotid plaques. *Stroke* 2008; *39* : 2958-65.
19. Tanko LB, Christiansen C. Can the obesity paradox be explained by the protective effects of peripheral adiposity? *Arch Intern Med* 2005; *165* : 1796-7.
20. Kannam JP, Levy D, Larson M, Wilson PW. Short stature and risk for mortality and cardiovascular disease events. The Framingham Heart Study. *Circulation* 1994; *90* : 2241-7.
21. Viswanathan B, Sharma V. Socio-economic differences in heights of adult Indian women. *J Dev Soc* 2009; *25* : 421-55.
22. Gunnell DJ, Davey Smith G, Frankel S, Nanchahal K, Braddon FE, Pemberton J, *et al*. Childhood leg length and adult mortality: follow up of the Carnegie (Boyd Orr) Survey of Diet and Health in Pre-war Britain. *J Epidemiol Community Health* 1998; *52* : 142-52.
23. Schooling CM, Jiang CQ, Heys M, Zhang WS, Lao XQ, Adab P, *et al*. Is leg length a biomarker of childhood conditions in older Chinese women? The Guangzhou Biobank Cohort Study. *J Epidemiol Community Health* 2008; *62* : 160-6.
24. Lampl M. Cellular life histories and bow tie biology. *Am J Hum Biol* 2005; *17* : 66-80.