BMI, body fat and waist-to-height ratio of stunted *v.* non-stunted Indian children: a case–control study

Mitravinda S Savanur* and Padmini S Ghugre

Department of Food Science and Nutrition, Sir Vithaldas Vihar, SNDT Women's University, Juhu Road, Mumbai – 400049, Maharashtra, India

Abstract

Objective: To compare the BMI, body fat and waist-to-height ratio (WHtR) of stunted and non-stunted children following different growth trajectories from low socio-economic strata in Mumbai, India.

Design: Cross-sectional, case–control study. Weight, height, skinfold thicknesses and waist circumference were measured. Information regarding the duration of breast-feeding, age at initiation of complementary feeding and income was obtained. Birth weight was obtained from records. BMI, body fat, WHtR and change in weight sd were calculated.

Setting: Children who were beneficiaries of *anganwadis*, Mumbai city, India.

Subjects: Three hundred and thirty children aged 2–4 years were selected in each of the stunted and non-stunted groups after matching for age and sex.

Results: After adjusting for birth weight, change in weight sd, duration of breast-feeding, age at complementary feeding initiation and income, stunted children had significantly higher body fat, WHtR and BMI than the non-stunted ($P < 0.01$). The stunted and non-stunted children were classified based on their change in weight sd. Stunted children with no change in weight sd had higher mean body fat, BMI ($P < 0.01$) and WHtR ($P < 0.05$) than their non-stunted counterparts. In the catch-up growth group, stunted children had higher BMI and WHtR than the non-stunted (both $P < 0.001$). In the catch-down growth group, stunted children had higher BMI than the non-stunted ($P < 0.001$).

Conclusions: Stunting was seen to increase the tendency of conserving body fat in young children. Such a tendency, if continued during later childhood and adolescence, can increase the risk of obesity and non-communicable diseases.

Globally, 171 million children (27 %) below 5 years of age were stunted in 2010[1]. The problem of stunting or chronic undernutrition is deeply rooted in poverty and deprivation, affecting mainly the developing and underdeveloped nations. Over the previous two decades, the prevalence of stunting in Asia has reduced by half from 48·6 % to 27·6 % in the years 1990 to 2010[1]. In comparison with the global rates, the prevalence of stunting in India is much higher (i.e. 48 %) with the rate in rural areas (50·7 %) exceeding that in urban ones (39·6 %)[2]. Stunting in early life is associated with short-term and long-term consequences. Stunting may hamper the structural and functional development of the brain, resulting in delayed cognitive function development or permanent cognitive impairment which may lead to poor academic achievement and low economic productivity[3–5]. Childhood stunting is directly related to short adult stature and may have intergenerational effects.

Maternal stunting increases the risk for intra-uterine growth restriction, perinatal mortality, maternal mortality and undernutrition in the offspring[6]. In addition to this, early stunting is viewed as a risk factor for overweight and adiposity later in life.

Much of the evidence to support the relationship between stunting and obesity emerges from some cross-sectional studies that point towards a possible risk of overweight/ obesity among stunted children[7–11] and adolescents[12–14]. Some studies have also shown that stunted children and adolescents have higher total body fat percentage (BF%) and central adiposity[14–18]. Central adiposity has been independently associated with high levels of serum TAG, non-HDL cholesterol, fasting insulin, homeostatic model assessment of insulin resistance and C-reactive protein in young children[19,20]. Further, Santos *et al.*[14] found that adolescents with mild stunting and overweight had significantly

Corresponding author: Email mitrasav@gmail.com

higher blood glucose, insulin and homeostatic model assessment of insulin resistance ($P < 0.05$) than normal-weight stunted ones. Also, Kruger et al.[17] showed that stunted girls had higher TNF-α levels compared with non-stunted girls ($P = 0.03$). These findings suggest that stunting at an early age can potentially increase the risk of over-weight, glucose intolerance and dyslipidaemia.

To the best of our knowledge, there are no studies that have explored the relationship of stunting and adiposity among Indian children. There is a need to explore this relationship given that India has a high prevalence of stunting in young children[2]. Additionally, Indian infants and children possess a 'thin–fat' phenotype[21,22]. Adiposity in stunted children depends on the rate of weight gain during the early years. Rapid weight gain during infancy and childhood is a major risk factor for higher BMI, total body fat and central adiposity in later life[23]. Accelerated weight gain in childhood can be attributed to the rapid nutrition transition and limited facilities for physical activity present in India. Kimani-Murage et al.[9] suggested that stunting and obesity may coexist in such transitional societies. All these factors in conjunction with each other predispose stunted children to overweight, thereby increasing the risk for non-communicable diseases.

We therefore conducted a case–control study with the objective to compare the BMI, BF% and waist-to-height ratio (WHtR) of stunted and non-stunted Indian children belonging to low socio-economic background following different growth trajectories.

Materials and methods

The study was approved by the Independent Ethics Committee (IEC no. 09122), Navi Mumbai, Maharashtra, India. Parents and/or guardians of the children received explanation about the study procedure in the local language and written informed consent was obtained from them.

Study design

We conducted a cross-sectional, case–control study in children aged 2–4 years belonging to low socio-economic sections in Mumbai city, India. Initially, we had conducted nutritional assessment of 1200 children aged 2–4 years from July 2013 to July 2014. From this initial group of children, we drew age- and sex-matched stunted (case) and non-stunted (control) children. Stunting was defined as height-for-age Z-score (HAZ) $\leq$ −2. The non-stunted or control group included children with HAZ $\geq$ −1·5. After matching for age and sex, the final sample included 330 stunted and 330 non-stunted children.

Participants

Children selected were beneficiaries of *anganwadis*, which are government-run mother–child care centres in India. Date of birth of the children was obtained from either the *anganwadi* records or their birth certificates. Age was calculated on the basis of their date of birth to the nearest one month. Birth weight of the participants was obtained from discharge card of the hospital where the child was born or the *anganwadi* register.

Measurements

All of the following measurements were taken by the researcher in triplicate.

Anthropometry

Weight and height of all children were measured using a standard procedure[24]. Children were weighed on a digital weighing scale (model MS8270; Dr Gene Health and Wellness), with an accuracy of 0·1 kg. Height was measured using a non-extensible, flexible measuring tape with an accuracy of 0·1 cm which was calibrated against the standard anthropometric scale. BMI was calculated.

Body fat

Harpenden callipers (Baty International, Burgess Hill, UK) were used to measure the skinfold thickness at triceps (TSF) and subscapular (SSF) sites[25]. BF% was calculated using the equation given by Slaughter et al.[26]. Slaughter's equation has been reported to predict BF% with high accuracy (95 %) and minimal bias, and has shown good precision compared with dual-energy X-ray absorptiometry in school-going children in Pakistan[27].

Measures of central fat

Waist circumference (WC) and WHtR were used as indicators of central body fat[28–30]. WC has been associated with biomarkers of cardiovascular risk in children[20,31]. WHtR correlates strongly with BF% and the distribution of body fat[32]. WC circumference was measured midway between the lowest rib margin (costal margin) and the superior anterior ileac crest with a non-extensible, flexible and accurate tape measure[33].

Change in weight SD

Change in weight SD was calculated as follows: change in weight SD = present WAZ – WAZ at birth, where WAZ is weight-for-age Z-score. Catch-up growth (CUG), catch-down growth (CDG) and 'no change in weight SD' were calculated[34]. If change in weight SD was >0·67, then it was considered that the child showed CUG. On the other hand, if change in weight SD was < −0·67, then it was considered as CDG. If the change in weight SD was between −0·67 and +0·67, then it was regarded as 'no change in weight SD'.

Child's feeding history

Information about breast-feeding – total duration of breast-feeding, whether the child was exclusively breast-fed,

duration of exclusive breast-feeding and the age at which complementary feeding was initiated – was obtained from the child's parent or guardian. In addition, details of income of all the family members were gathered to calculate the total family income.

Statistical analysis

HAZ and WAZ were computed using WHO Anthro software version 3·2·2. The data were analysed using the statistical software package IBM SPSS Statistics Version 20. Mean and SD were calculated for continuous variables and frequency distribution for the categorical variables. One-way ANOVA was performed to see if there were any differences in mean anthropometry, BMI, TSF, SSF, BF%, WC and WHtR between the two groups.

We carried out one-way ANCOVA to study the differences in BMI, BF%, TSF, SSF, WC and WHtR after adjusting the means for certain confounding variables that may potentially influence the growth and nutritional status of the children. The analysis was adjusted for the following covariates: birth weight, change in weight SD, duration of exclusive breast-feeding, total duration of breast-feeding, age at initiation of complementary feeding and total family income. The stunted and non-stunted children were classified into the three categories of change in weight SD: CDG, no change in weight SD and CUG. We performed one-way ANCOVA to study the differences in BMI, BF%, TSF, SSF, WC and WHtR between stunted and non-stunted groups who experienced CDG, no change in weight SD and CUG, independently, after adjusting the means for the studied covariates (birth weight, duration of exclusive breast-feeding, total duration of breast-feeding, age at initiation of complementary feeding and total family income).

Results

Sample profile

The characteristics of the sample are given in Table 1. Fifty-three per cent of the children were girls (n 350) and 47 % were boys (n 310) boys. The stunted group had a greater proportion of low-birth-weight children than the non-stunted group ($P = 0·001$). A higher percentage of stunted children experienced CDG, while more non-stunted children showed CUG and no change in weight SD ($P = 0·000$). In both groups, >90 % of the children received exclusive breast-feeding; however, the total duration of breast-feeding ($P = 0·008$) and that of exclusive breast-feeding ($P = 0·029$) were significantly higher among the stunted than the non-stunted children. The total family income of the stunted children was significantly lower than that of the non-stunted ($P = 0·008$).

Anthropometry, BMI, body fat percentage, waist circumference and waist-to-height ratio in stunted and non-stunted children

The stunted boys and girls had significantly lower mean birth weight, present weight, height, HAZ and change in

weight SD than their non-stunted counterparts (all $P < 0·05$; Table 2). Mean BMI, TSF, SSF, BF% and WHtR were similar between the stunted and non-stunted boys and girls. Only stunted girls had a lower mean WC than the non-stunted girls ($P < 0·05$).

After adjusting for the covariates (birth weight, change in weight SD, duration of exclusive breast-feeding, total duration of breast-feeding, age at initiation of complementary feeding and total family income), stunted boys and girls were seen to have significantly higher BF%, TSF, SSF, BMI and WHtR than their non-stunted counterparts (all $P < 0·001$; Table 3). Stunted girls had significantly higher WC than the non-stunted girls ($P < 0·05$). However, no such difference was noted in boys for WC.

BMI, body fat percentage, waist circumference and waist-to-height ratio in stunted and non-stunted children who had catch-down growth, no change in weight SD and catch-up growth

After adjusting for the covariates, stunted children who had no change in weight SD had significantly higher mean BF%, BMI (both $P < 0·001$) and WHtR ($P < 0·05$) than their non-stunted counterparts (Table 4). No differences were seen in mean WC measurements among them. In the CDG group, the stunted children had significantly higher BMI ($P < 0·001$) than the non-stunted children. No differences were seen in mean values for BF%, WC and WHtR. On the other hand, in the CUG group, stunted children had significantly higher BMI and WHtR (both $P < 0·001$) than the non-stunted children. Mean BF% was higher in the stunted compared with the non-stunted children but the difference did not reach statistical significance.

Discussion

Our study is unique because it represents the first time that the BMI, BF%, WC and WHtR of stunted children who experienced CUG, no change in weight SD and CDG in their early postnatal life have been compared with those of their respective non-stunted counterparts. In our study, almost 28 % of stunted and 16 % of non-stunted children had low birth weight. More than half of the stunted children experienced CDG while one-quarter showed CUG in their postnatal life. After adjusting for the covariates, the stunted children who experienced no change in weight SD had significantly higher BMI, BF% and WHtR than the non-stunted ones. In the CUG group, stunted children had significantly higher BMI and WHtR than the non-stunted ones. Stunted children who had CDG had significantly higher BMI than their non-stunted counterparts.

Our findings of higher BMI, BF% and WHtR in stunted children with no change in weight SD are consistent with previous studies carried out in stunted children[8,10,11,16]. Studies have reported that the higher body fat in stunted children could possibly be due to a reduction in the

Table 1 Characteristics of the sample of children aged 2–4 years from low socio-economic strata in Mumbai, India, July 2013–July 2014

Variable	Non-stunted (n 330)		Stunted (n 330)		χ^2	P
	%	n	%	n		
Sex						
Boys	47·0	155	47·0	155	0·000	1·000
Girls	53·0	175	53·0	175		
Age						
2–3 years	50·6	167	50·6	167	0·000	1·000
3–4 years	49·4	163	49·4	163		
Birth weight						
Low (<2·5 kg)	16·1	53	27·9	92	13·475	0·001
Normal (2·5–3·9 kg)	82·7	273	71·2	231		
High (>4·0 kg)	1·2	4	0·9	3		
Change in weight SD						
Catch-down growth (< −0·67)	20·0	66	58·5	193	109·492	0·000
No change (−0·67 to +0·67)	42·7	141	28·2	93		
Catch-up growth (>0·67)	37·3	123	13·3	44		
Exclusive breast-feeding						
Yes	91·5	301	92·4	305	0·670	0·383
No	8·5	29	7·6	25		
	Mean	SD	Mean	SD	F	P
Duration of exclusive breast-feeding (months)	4·9	2·7	5·5	3·7	4·767	0·029
Total duration of breast-feeding (months)	20·1	10·0	22·2	10·4	7·157	0·008
Age at the initiation of complementary feeding (months)	7·4	3·8	7·9	4·6	3·180	0·075
Total family income (Indian Rs)	14 475	10 263	12 523	8367	7·172	0·008

Table 2 Unadjusted mean anthropometric parameters, BMI, skinfold thickness, WC, WHtR, BF% and change in weight SD in stunted and non-stunted boys and girls aged 2–4 years from low socio-economic strata in Mumbai, India, July 2013–July 2014

	Boys					Girls					Total				
	Non-stunted (n 155)		Stunted (n 155)			Non-stunted (n 175)		Stunted (n 175)			Non-stunted (n 330)		Stunted (n 330)		
Parameter	Mean	SD	Mean	SD	F	Mean	SD	Mean	SD	F	Mean	SD	Mean	SD	F
Birth weight (kg)	2·87	0·48	2·59	0·59	19·848**	2·70	0·48	2·59	0·49	4·875*	2·78	0·50	2·59	0·50	22·334**
Weight (kg)	13·1	1·6	10·6	1·2	226·902**	12·2	1·8	10·2	1·2	166·836**	12·6	1·7	10·4	1·2	370·127**
Height (cm)	94·2	5·1	84·9	4·7	273·916**	92·5	5·2	83·9	5·0	239·196**	93·3	5·2	84·4	4·9	500·950**
HAZ	−0·41	0·70	−2·85	0·60	884·271**	−0·59	0·70	−2·79	0·66	903·860**	−0·51	0·73	−2·82	0·66	1777·056**
BMI (kg/m^2)	14·7	1·1	14·7	1·2	0·145	14·3	1·4	14·4	1·1	0·697	14·5	1·3	14·5	1·2	0·136
TSF (mm)	7·5	1·4	7·4	1·3	0·402	7·6	1·6	7·4	1·3	1·295	7·5	1·5	7·4	1·3	1632
SSF (mm)	5·2	1·0	4·9	0·8	5·075	5·4	1·4	5·2	1·0	2·942	5·3	1·3	5·1	0·9	7·324*
WC (cm)	48·4	2·6	46·4	6·6	32·085	47·6	6·2	44·5	8·1	16·328**	48·0	4·8	44·4	8·2	45·190**
WHtR	0·51	0·03	0·52	0·10	0·651	0·51	0·06	0·53	0·09	2·401	0·52	1·00	0·52	0·07	2·914
BF%	12·4	2·3	12·1	1·9	1·906	12·6	2·7	12·2	2·2	1·466	12·5	2·5	12·1	2·1	3·278
Change in weight SD	0·32	1·24	−0·69	1·35	47·812**	0·28	1·29	−0·88	1·32	70·345**	0·30	1·26	−0·79	1·33	117·743**

WC, waist circumference; WHtR, waist-to-height ratio; BF%, body fat percentage; HAZ, height-for-age Z-score; TSF, triceps skinfold; SSF, subscapular skinfold.
*$P < 0.05$, **$P < 0.001$.

oxidation of fat in these children[35,36]. The RQ indicates the substrate being used for energy production. In one study, stunted children had a significantly higher RQ (0·92 (SD 0·009)) than control children (0·89 (SD 0·007)). A higher RQ means that the chief substrate for metabolism is carbohydrate (RQ of carbohydrate = 1). Stunted children derived 25 % energy from fat oxidation while the controls obtained significantly higher energy (i.e. 34 %) from oxidation of fat[35]. Evidence also suggests that chronic starvation or undernutrition results in a reduction in total energy expenditure (i.e. RMR, sleeping metabolic rate and physical activity). This is believed to be because the depleted fat stores activate an autoregulatory feedback mechanism that suppresses thermoregulation[37,38]. Thus during prolonged starvation or periods of undernutrition the body adapts to lower energy expenditure which improves the metabolic efficiency. Further, Sawaya et al.[39] suggested that long-term undernutrition is accompanied by lower circulating levels of insulin-like growth factor-1. Low insulin-like growth factor-1 levels are associated with lower insulin levels and higher ratios of cortisol to insulin. High cortisol levels are associated with

Table 3 Adjusted mean BMI, BF%, skinfold thickness, WC and WHtR in stunted and non-stunted boys and girls aged 2–4 years from low socio-economic strata in Mumbai, India, July 2013–July 2014

	Boys					Girls					Total				
	Non-stunted (n 155)		Stunted (n 155)			Non-stunted (n 175)		Stunted (n 175)			Non-stunted (n 330)		Stunted (n 330)		
Parameter	Mean†	SE	Mean†	SE	F	Mean†	SE	Mean†	SE	F	Mean†	SE	Mean†	SE	F
BMI (kg/m^2)	13·7	0·079	15·7	0·079	211·112**	13·4	0·070	15·4	0·070	311·738**	13·6	0·052	15·3	0·052	529·860**
BF%	11·4	0·195	13·1	0·195	27·550**	11·4	0·192	13·4	0·192	40·194**	11·5	0·138	13·2	0·138	59·193**
TSF (mm)	6·9	0·129	7·9	0·129	22·392**	7·0	0·119	8·0	0·119	30·833**	7·0	0·087	8·0	0·087	49·006**
SSF (mm)	4·7	0·090	5·4	0·090	21·476**	4·8	0·092	5·9	0·092	54·675**	4·8	0·066	5·6	0·066	59·685**
WC (cm)	46·7	0·611	46·1	0·611	0·296	45·0	0·591	47·0	0·591	4·018*	45·8	0·424	46·6	0·424	1·343
WHtR	0·50	0·007	0·53	0·007	6·405**	0·49	0·007	0·55	0·007	29·982**	0·49	0·005	0·54	0·005	32·879**

BF%, body fat percentage; WC, waist circumference; WHtR, waist-to-height ratio; TSF, triceps skinfold; SSF, subscapular skinfold.
*$P < 0.05$, **$P < 0.001$.
†Means adjusted for birth weight, change in weight sd, duration of exclusive breast-feeding, total duration of breast-feeding, age at initiation of complementary feeding and total family income.

Table 4 Adjusted mean BMI, BF%, skinfold thickness, WC and WHtR across the change in weight sd categories in non-stunted and stunted children aged 2–4 years from low socio-economic strata in Mumbai, India, July 2013–July 2014

	CDG					No change in weight sd					CUG				
	Non-stunted (n 66)		Stunted (n 193)			Non-stunted (n 141)		Stunted (n 93)			Non-stunted (n 123)		Stunted (n 44)		
Parameter	Mean†	SE	Mean†	SE	F	Mean†	SE	Mean†	SE	F	Mean†	SE	Mean†	SE	F
BMI (kg/m^2)	13·9	0·103	14·3	0·068	8·176**	13·9	0·071	15·5	0·090	166·115**	14·7	0·124	15·8	0·232	14·249**
BF%	11·9	0·212	12·1	0·143	1·109	11·9	0·182	13·2	0·233	16·379**	12·6	0·243	13·4	0·454	1·801
TSF (mm)	7·2	0·127	7·3	0·085	0·703	7·4	0·120	8·0	0·154	9·278**	7·6	0·153	8·1	0·285	1·915
SSF (mm)	5·1	0·092	5·0	0·062	1·023	4·9	0·085	5·7	0·108	22·413**	5·5	0·131	5·8	0·245	1·501
WC (cm)	44·7	0·713	44·5	0·475	0·317	47·2	0·574	46·0	0·735	1·339	48·1	0·557	47·8	0·104	0·063
WHtR	0·51	0·008	0·52	0·006	1·296	0·51	0·007	0·54	0·009	4·333*	0·51	0·007	0·57	0·012	11·776**

BF%, body fat percentage; WC, waist circumference; WHtR, waist-to-height ratio; CDG, catch-down growth; CUG, catch-up growth; TSF, triceps skinfold; SSF, subscapular skinfold.
*$P < 0.05$, **$P < 0.001$.
†Means adjusted for birth weight, duration of exclusive breast-feeding, total duration of breast-feeding, age at initiation of complementary feeding and total family income.

central fat deposition. This may explain the higher WHtR found in stunted children than their non-stunted counterparts who experienced no change in weight sd in the present study. Thus, previously undernourished individuals develop a tendency to conserve more body fat particularly, abdominal fat.

We found that stunted children who experienced CUG had higher BMI and WHtR than the non-stunted ones. During recovery from starvation, owing to the improved metabolic efficiency, the body conserves energy and accumulates more body fat. This tendency is referred to as 'metabolic adaptation' or 'poststarvation obesity'[40]. In a prospective study, Grillo et al.[41] found that stunted children gained 6 kg/year while the non-stunted children maintained at 4 kg/year irrespective of both groups consuming similar diets. This was because during the follow-up period there was a lesser increase in RMR but a greater increase in the velocity of weight gain in the stunted group compared with the non-stunted group. The stunted girls had lower body fat at the beginning of the study but gained more fat than the non-stunted ones after 36 months. Thus, infants and children recovering from undernutrition disproportionately accumulate more fat than muscle mass during CUG. In the present study, the stunted children who exhibited CUG had higher mean BF% than the non-stunted group but the difference was not significant.

Stunted children who had CDG had higher BMI than their non-stunted counterparts. However, they did not show much difference in their body fat indices compared with the non-stunted. CDG represents growth faltering or a period of undernutrition. It appears that both stunted and non-stunted children who experienced growth faltering would have equally adapted to lower energy expenditure to improve metabolic efficiency. This may explain the similar BF%, WC and WHtR values in both these groups.

In addition to adaptation, body composition depends on the quality of the diet and the level of physical activity. Although we did not carry out a detailed dietary

assessment, surveys carried out in Indian urban low- to middle-income groups have shown an increase in the consumption of energy-dense foods among children[42–45]. Bentley et al.[45] found that in informal settlements of Mumbai, the diets of children under 5 years of age were marked by consumption of unhealthy snacks. This pattern was seen from infancy and increased during childhood years. The low per unit cost of the snacks made it an easier and a more convenient option for parents to include in their children's daily diet. Researchers have noted a similar unhealthy dietary pattern in overweight/obese stunted children[7,46,47]. Based on four large nationwide surveys from Russia, China, South Africa and Brazil in children aged 3–6 years and 7–10 years old, Popkin et al.[7] noted that stunted children were likely to become overweight or have abdominal obesity when exposed to energy-dense, high-fat foods along with moderate physical activity. Said-Mohammad et al.[46] observed that children who were only stunted and those who were stunted plus overweight spent more time in sedentary activities than the only overweight children. Also, the dietary diversity scores of the stunted plus overweight children were significantly lower than those of the only overweight group ($P < 0.01$). Li et al.[47] added that children who were stunted and stunted plus overweight consumed more energy-dense foods and less protein, PUFA, Fe, fruits and vegetables. Sawaya et al.[39] observed in mildly stunted girls that baseline fat intake level positively correlated with the rise in weight-for-height after 22 months ($P = 0.048$). This means that stunted girls gained more weight for the corresponding height compared with non-stunted girls.

Contrary to the findings of Sawaya et al.[39], Tanner et al.[48] reported that adolescents who were stunted at the age of 2 years were less likely to be overweight than those who were never stunted in a longitudinal study in 'Tsimani', an indigenous group residing in the Beni Department of Bolivia, South America. These participants belonged to families who earned their living primarily by agriculture and supplemented it with hunting and gathering. They depended wholly on crops grown at household level and domestic animals for food. Also, these children/adolescents actively participated in hunting, farming, fishing and assisting the elders in their work. Thus, diet and physical activity do influence the body composition in stunted children.

India is witnessing a rapid economic, social and nutritional transition. Urban areas are noticing a rise in the population density leading to poor living conditions. At the same time, there has been a shift from traditional foods to the consumption of energy-dense foods rich in fat and carbohydrates. This shift is evident across the socio-economic classes. Indians also possess a thin–fat phenotype from an early age that predisposes them to adverse metabolic outcomes[21]. Thus, impoverished living conditions coupled with easy availability of low-cost energy-dense foods and lesser scope for physical activity in areas with high prevalence of stunting elevate the risk of adiposity in Indians during recovery from undernutrition. However, CUG is imperative in stunted children. This CUG should be achieved with a healthy balance between adequate nutrition and physical activity.

Conclusions and recommendations

The present study is one of the first in India to have compared the body fat indices between stunted and non-stunted children belonging to low socio-economic background following different growth trajectories. Our findings suggest that stunted children with no change in weight, CDG and CUG have higher body fat indices than their non-stunted counterparts. Postnatal growth trajectory influences the change in body fat. Urbanization, nutrition transition and poor physical activity can potentially increase the risk of adiposity in stunted children as they grow older. Growth during childhood and adolescence is accompanied by changes in body composition. In view of this, longitudinal studies should be undertaken in India to map the changes in body composition during childhood/ adolescence in stunted children across the socio-economic strata. Also, there is a need for preventive measures to reduce the prevalence of stunting. Strategies to increase physical activity and improve dietary quality should be in place to achieve optimal growth without increasing the risk for adiposity in stunted children.

Acknowledgements

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