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Original Article

Upper arm fat and muscle in stunted and non-stunted children aged 0-24 months

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Abstract

Background The prevalence of stunting in Indonesia is high, with particularly negative impacts on health during childhood as well as adolescence. Stunting impacts the health of children as well as adults, especially with regards to future obesity. Therefore, evaluating body composition of stunted children before 2 years of age is necessary.

Objective To compare upper arm fat and muscle measurements in stunted and non-stunted children aged 0-24 months of age.

Methods We analyzed secondary data of the Division of Nutrition and Metabolic Disease, Department of Child Health, Universitas Gadjah Mada Medical School, Yogyakarta which were collected using cluster random sampling from the Yogyakarta Special Province. We compared upper arm fat area (UFA), including the upper arm fat area estimate (UFE) and the upper arm fat percentage (UFP), as well as upper arm muscle area (UMA) and upper arm muscle area estimate (UME), among stunted and non-stunted children aged 0-24 months.

Results We analyzed 2,195 children. The prevalence of stunting was 354/2,195 (16.1%). The UFA, UFE, and UFP among stunted children were significantly lower compared to non-stunted children aged 7-12 months [UFA: 4.48 *vs.* 5.05 cm2 (P <0.001), respectively; UFE: 4.88 *vs.* 5.55 cm2 (P <0.001), respectively; and UFP: 30.82 *vs.* 32.58% (P = 0.03), respectively]. The UMA in children aged 7-12 months was also significantly lower in stunted than in non-stunted children [11.31 *vs.* 11.79 cm2 (P = 0.02), respectively], as well as in children aged 13-24 months [11.05 *vs.* 11.75 cm2 (P <0.001), respectively]. In addition, the UME in children aged 13-24 months was significantly lower in stunted compared to non-stunted children [10.50 *vs.* 11.18 cm2 (P <0.001), respectively].

Conclusion The UFA in children aged 7-12 months is smaller in stunted than in non-stunted children, whereas UMA in children aged 7-12 months and 13-24 months was smaller in stunted compared to non-stunted children. **[Paediatr Indones. 2017;57:252-61; doi: http://dx.doi.org/10.14238/pi57.5.2017.252-61]**.

Keywords: *stunting; overweight; obesity; upper arm fat area; upper arm muscle area; upper arm fat percentage*

Prevalence of stunting was high in developing countries (39.7% in 1990), but predicted to decrease to 21.8% in 2020.[1] In Indonesia in 2013 the prevalence was 37.2%.[2] Stunting impacts the health of children as well as adults, especially with regards to future obesity.[3] Therefore, evaluating body composition of stunted children before 2 years of age is necessary.

Previous studies have shown that upper arm anthropometry, i.e., fat mass measured as upper arm fat area (UFA) and upper arm fat area estimate (UFE), as well as muscle mass measured as upper arm muscle area (UMA) and upper arm muscle

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estimate (UME) could be representive of a child's body fat and muscle mass.[4-5] The simple methods of upper arm anthropometry to assess fat and muscle mass are clinically beneficial, since the standard methods of assessing body composition, i.e., dual X-ray absorptiometry, isotope dilution, hydrostatic weighing, bioelectrical impedance analysis, air displacement plethysmography, and total body electrical conductivity[6] are not generally available in primary health facilities.

Unfortunately, few studies have been done on fat and muscle mass of stunted compared with non-stunted children less than 24 months of age in Indonesia. Studies on the significance of upper arm anthropometry as representative of the child's body composition are important as a practical method of promoting health in primary care settings. Stunted children less than 2 years of age should be monitored regularly to identify increased fat, using upper arm anthropometry. Children usually experience stunting during the first 2-3 years, and they will have difficulty catching up their growth if they remain in a poor environment.[7]

The aim of the study was to evaluate fat and muscle mass, measured as UFA, UFE, UMA, UME, and UFP, in stunted compared with non-stunted children 0 to 24 months of age in Yogyakarta Special Province, Indonesia.

Methods

We analyzed secondary data from the Division of Nutrition and Metabolic Disease, Department of Child Health, Universitas Gadjah Mada Medical School, Yogyakarta. The study population was children 0 to 24 months of age from Yogyakarta Special Province who were recruited using a multi-stage cluster sampling method from the primary health center (posyandu) as the sampling unit. We randomly selected 6 posyandu (351 children) from Yogyakarta Municipality, 10 posyandu (573 children) from Bantul Regency, 7 posyandu (688 children) from Sleman Regency, 7 posyandu (338 children) from Gunung Kidul Regency, and 7 posyandu (255 children) from Kulon Progo Regency, for a total of 37 posyandu (2,205 children) .

Children who fulfilled the inclusion criteria were 0 to 24 months of age and had mothers who consented to participate. We excluded children who were ill at the time of data collection. Anthropometric data [weight, length, middle upper arm circumference (MUAC), and triceps skinfold thickness (TS)] were measured. Demographic data of the family included parental education and occupation, number of children, and child's birth weight. We added exclusion criteria for data analysis when anthropometric data of the registry were missing.

Weight, length, MUAC, and TS were measured by a trained research assistant. All measurements were taken in triplicate and mean values calculated. Children were weighed using a *GEA®* Baby Scale, and length was measured using a locally produced wooden board. *Harpenden Skinfold Caliper®* (*Baty International RH15 9LR* England) was used to measure TS and an upper arm measuring tape (Indonesian Ministry of Health) was used to measure MUAC. Measurement techniques were based on standardized methods.[8-9]

The minimum required sample size was calculated for two unpaired samples with nominal scale of dependent variable and estimating a difference of two proportion.[10] We assumed that the value of $\alpha = 1.960$, proportion of overweight and/or obese among stunted children from literature was 0.1,[11] proportion of overweight and/or obesity among stunted children was 0.075 (researcher's judgment calculated from secondary data), and absolute precision 0.05 with 95% confidence interval of 0.05. Therefore, the minimum required sample size was 245 children.

We defined stunting to be height-for-age z-score (HAZ) < - 2 SD, based on the 2006 *WHO Child Growth Standard*.[12] A child with weight-for-age z-score (WHZ) > 2 SD was classified as overweight or obese. The UMA, UFA, and total upper arm area (TUA) were calculated using the following formulas: UMA = [MUAC – (TS x π)]2 / (4 π) (cm2); UFA = TUA – UMA (cm2); and TUA = MUAC2 / 4π (cm2).4 The UFE and UME were calculated using the following formulas: UFE = MUAC x (TS/2) (cm2) and UME = TUA – UFE (cm2). We included UFE and UME instead of UFA and UMA, since both UFE and UME were well correlated with MRI which precisely measured fat as well as muscle areas.5 Upper arm fat percentage (UFP) was calculated by the formula UFE x TUA x 100%.[4,5]

We analyzed the prevalence difference of

overweight or obesity between stunted and non-stunted children according to age groups by Chi-square test. The mean differences of UFA, UFE, UMA, UME and UFP between stunted and non-stunted children were tested using unpaired T-test. We set the statistical significance at $P < 0.05$, and analyzed data using the *Statistical Package for the Social Science (SPSS) version 15.0* (*SPSS Inc*., Chicago, IL, USA) software. This study was approved by the Research and Health Ethics Committee of Gadjah Mada University Medical School/Dr. Sardjito Hospital, Yogyakarta, Indonesia.

Results

Figure 1 shows the trial profiles. The highest prevalences of stunting in girls (42%) and boys (52%) were at 22 and 21 months of age, respectively (**Figure 2**). **Table 1** shows the basic characteristics of study subjects, where subjects were grouped by age (0-6, 7-12, and 13-24 months). **Table 2** indicates that in total, the prevalence of overweight or obesity in non-stunted children was significantly higher compared to stunted children (72.5% *vs.* 27.5%, respectively, P=0.005). The prevalences of overweight or obesity in non-stunted compared to stunted children were significantly different in two age groups: 0-6 months (5.38% *vs.* 17.65%, respectively, P=0.001), and 7-12 months (2.81% *vs.* 9.45%, respectively, P=0.002).

Figure 2. The proportion (%) of stunting (indicated by numbers on rows of boys and girls) according to age (0-24 months) among boys and girls.

Stunted children aged 7-12 months had significantly lower UFA, UFE, and UFP means than non-stunted children of the same age group [UFA: 4.48 *vs.* 5.05 cm^2, respectively, (P<0.001) (**Table 3**); UFE: 4.88 *vs.* 5.55 cm^2, respectively, (P<0.001) (**Table 4**); and UFP: 30.82 *vs.* 32.58% (**Table 5**), respectively, (P=0.03)].

We created scatter plots to identify trends in UFA, UFE, and UFP from birth to 24 months of age.

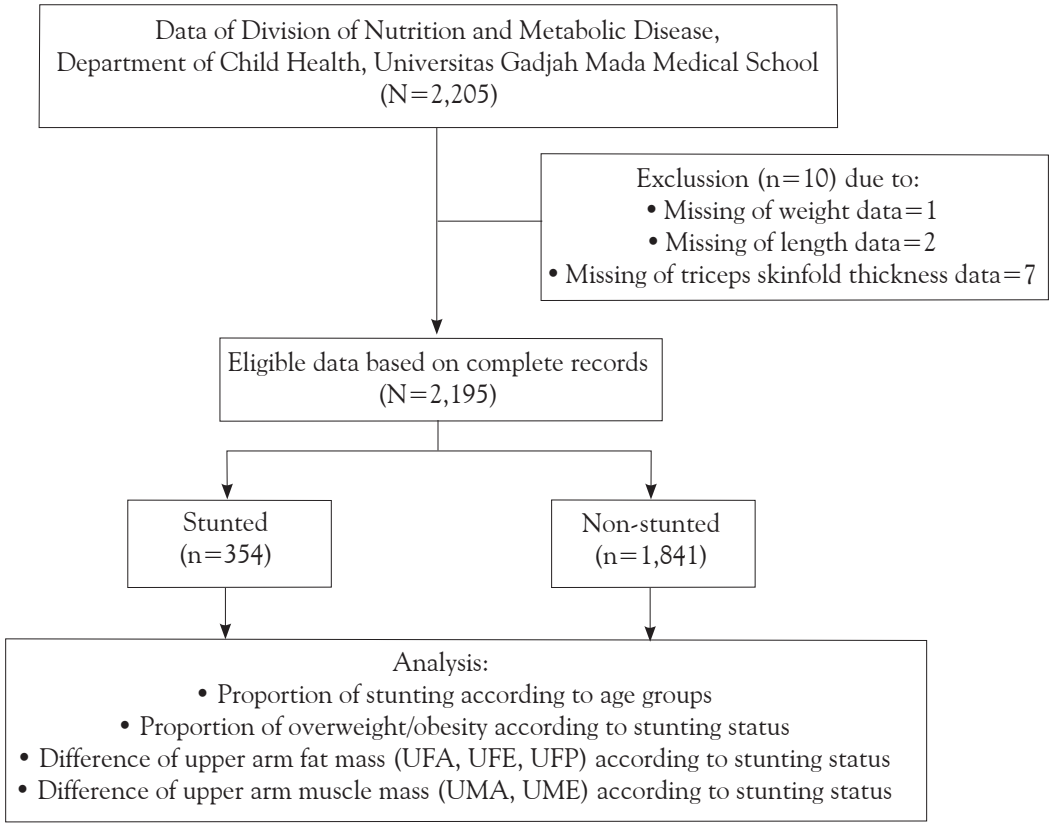

Figure 1. Trial profile

Table 1. Basic characteristics of subjects (N=2,195)

Characteristics	n(%)
Gender	
Male	1,137 (51.8)
Female	1,058 (48.2)
Age groups	
0-6 months	885 (40.3)
7-12 months	677 (30.8)
13-24 months	633 (28.8)
Number of children in family	
1	1,040 (47/4)
2	706 (32.2
3	297 (13.5)
4	94 (4.3)
>4	55 (2.5)
Stunted	
Yes	354 (16)
No	1,841 (84)

Figures 3, 4, and **5** show that mean UFA, UFE, and UFP appeared lower in stunted children aged 4-12 months, 4-12 months, and 8-11 months, respectively, compared to non-stunted children of the same respective age groups.

Non-stunted children aged 7-12 months and 13-24 months also had significantly higher mean UMA compared to stunted children of the same age groups, where the mean differences were 0.48 cm^2 (P=0.02) and 0.70 cm^2 (P< 0.001) (**Table 6**) respectively. Mean UME in the 13-24 month age group was also significantly higher in non-stunted children than in stunted children, with a mean difference of 0.68 cm^2 (P< 0.001) (**Table 7**).

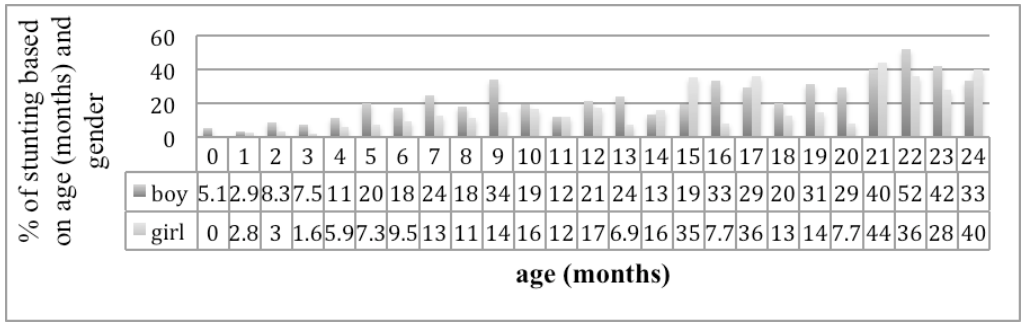

Figure 2. The proportion (%) of stunting (indicated by numbers on rows of boys and girls) according to age (0-24 months) among boys and girls

Table 2. Prevalence of overweight or obesity among non-stunted and stunted children according to age groups

Age groups and overweight/obesity status	Stunted	Non-stunted	P value
0-6 months, n (%)			
Overweight/obese	12 (21.8)	43 (78.2)	0.005
Not overweight/obese	54 (6.5)	776 (93.5)	
7-12 months, n (%)			
Overweight/obese	12 (46.2)	14 (53.8)	0.001
Not overweight/obese	112 (17.2)	539 (82.8)	
13-24 months, n (%)			
Overweight/obese	1 (10)	9 (90)	0.446
Not overweight/obese	163 (26.2)	460 (73.8)	
Total, n (%)			
Overweight/obese	25 (27.5)	66 (72.5)	0.005
Not overweight/obese	329 (27.5)	1,775 (84.4)	

Table 3. Mean UFA in stunted and non-stunted children according to age groups

Age groups	Stunted		Non-stunted		Mean difference, cm^2	95%CI	P value
	Mean UFA, cm^2	SD	Mean UFA, cm^2	SD			
0-6 mo	4.84	1.38	4.79	1.30	-0.05	-0.38 to 0.28	0.76
7-12 mo	4.48	1.36	5.05	1.73	0.57	0.29 to 0.85	<0.001
13-24 mo	5.26	1.70	5.45	2.04	0.19	-0.16 to 0.54	0.28
Total	4.91	1.56	5.04	1.66	0.13	-0.06 to 0.32	0.17

Table 4. Mean UFE in stunted and non-stunted children according to age groups

Age groups	Stunted		Non-stunted		Mean difference, cm^2	95%CI	P value
	Mean UFE, cm^2	SD	Mean UFE, cm2	SD			
0-6 mo	5.36	1.62	5.29	1.49	-0.07	-0.45 to 0.31	0.72
7-12 mo	4.88	1.58	5.55	2.03	0.67	0.36 to 0.99	<0.001
13-24 mo	5.81	2.01	6.01	2.41	0.20	-0.17 to 0.59	0.28
Total	5.40	1.84	5.56	1.95	0.16	-0.07 to0.37	0.18

Table 5. Mean UFP between stunted and non-stunted children according to age groups

Age groups	Stunted		Non-stunted		Mean difference, cm^2	95%CI	P value
	Mean UFP, cm^2	SD	Mean UFP, cm^2	SD			
0-6 mo	36.53	9.21	36.47	7.16	-0.06	-1.90 to 1.78	0.95
7-12 mo	30.82	7.78	32.58	9.08	1.76	0.19 to 3.34	0.03
13-24 mo	35.10	9.00	34.28	10.03	-0.82	-2.57 to 0.92	0.35
Total	33.87	8.91	34.74	8.71	0.87	-0.12 to 1.87	0.08

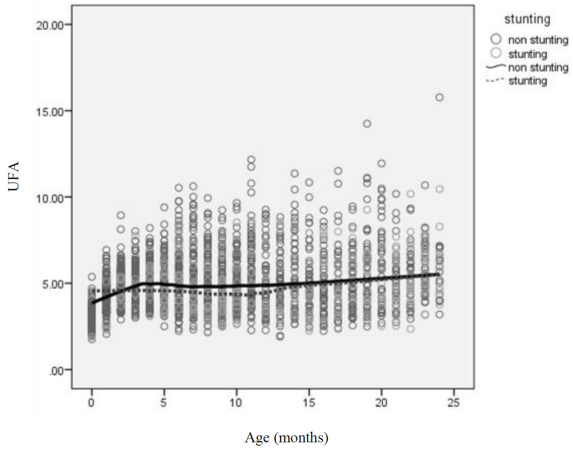

Figure 3. Scatter plot for UFA of stunted and non-stunted children according to age in months, showing the trend from birth to 24 months of age

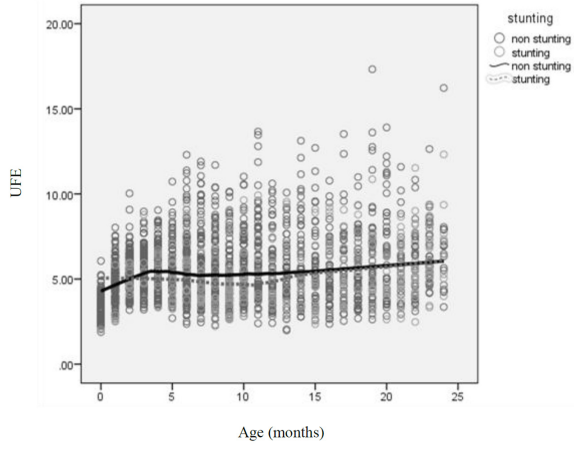

Figure 4. Scatter plot for UFE in stunted and non-stunted children according to age, showing the trend from birth to 24 months of age

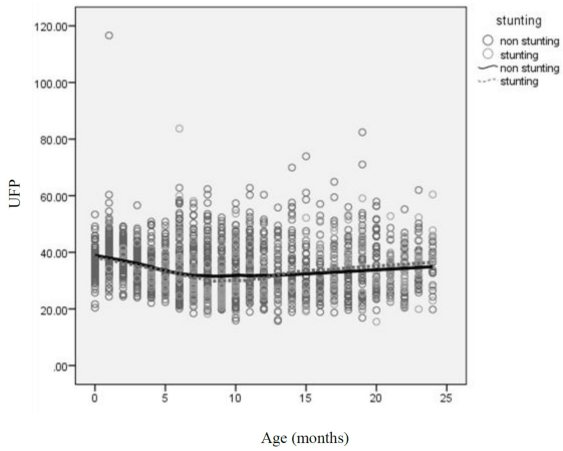

Figure 5. Scatter plot for UFP in stunted and non-stunted children according to age, showing the trend from birth to 24 months of age

Table 6. Mean UMA differences between stunted and non-stunted children according to age groups

Age groups	Stunted		Non-stunted		Mean difference, cm^2	95%CI	P value
	Mean UMA, cm^2	SD	Mean UMA, cm^2	SD			
0-6 mo	9.88	2.47	9.78	2.35	-0.10	-0.69 to 0.49	0.74
7-12 mo	11.31	2.25	11.79	2.12	0.48	0.07 to 0.91	0.02
13-24 mo	11.05	1.77	11.75	2.04	0.70	0.35 to 1.05	<0.001
Total	10.92	2.14	10.89	2.42	-0.03	-0.31 to 0.23	0.79

Table 7. Mean UME differences between stunted and non-stunted children according to age groups

Age groups	Stunted		Non-stunted		Mean difference, cm^2	95%CI	P value
	Mean UME, cm^2	SD	Mean UME, cm^2	SD			
0-6 mo	9.37	2.51	9.28	2.33	-0.09	-0.67 to 0.51	0.78
7-12 mo	10.90	2.28	11.30	2.19	0.4	-0.03 to 0.83	0.07
13-24 mo	10.50	1.83	11.18	2.12	0.68	0.32 to 1.05	<0.001
Total	10.43	2.19	10.37	2.44	-0.06	-0.31 to 0.19	0.06

The mean UMA and UME also appeared lower in stunted children aged 3-24 months compared to non-stunted children, as shown in the scatter plots (**Figures 6** and **7**).

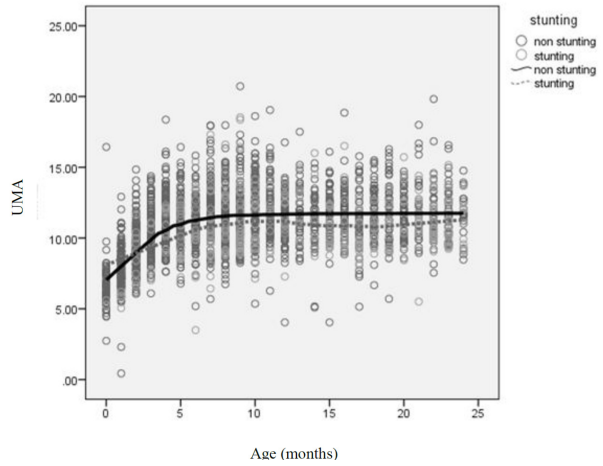

Figure 6. Scatter plot for UMA in stunted and non-stunted children according to age, showing the trend from birth to 24 months of age

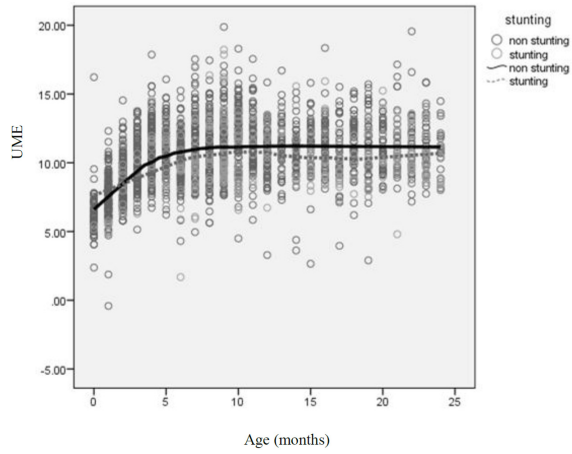

Figure 7. Scatter plot for UME in stunted and non-stunted children according to age, showing the trend from birth to 24 months of age

Discussion

In our study, stunted children aged 7-12 months had less upper arm fat (UFA, UFE, and UFP) than non-stunted children. In addition, upper arm muscle area, represented by UMA at 13-24 months and UME at 7-24 months of age, was significantly lower in stunted than non-stunted children. Therefore, stunted children less then 2 years of age had less fat and muscle area than non-stunted children, at certain ages.

Yemeni children aged 0-7 years indicated similar results, in that stunted children had significantly smaller UFA and UMA compared to non-stunted children at the same age.[13] There was also a significant correlation between upper arm muscle mass and height-for-age (HAZ) Z-scores in Kenyan children. The HAZ values were higher among children aged 9 months who had bigger upper arm muscle mass compared to those with smaller mass (-1.044 *vs.* -1.917, respectively; P <0.001) as well as at 24 months of age (–1.114 *vs.* –1.301, respectively; P=0.04).14 In Bolivia, stunted children aged 2-10 years had lower UMA Z-scores compared with non-stunted children (-0.43 *vs.* –1.23, respectively; P<0.001).[15]

Lipsberga *et al.* reported that among Latvian children aged 5-7 years, UFA and UMA correlated with body mass index (BMI) (in boys: r=0.35; P<0.05 and r=0.53; P<0.01, respectively; and girls: r=0.41; P<0.05 and r=0.48; P<0.01, respectively). However, correlation between fat and muscle mass with BMI should be interpreted cautiously, since taller children with normal fat may have lower BMI, in contrast to children with more muscular and bigger body frame having higher BMI.[16]

Slower fat and muscle growth may be explained by insufficient intake of energy and essential nutrients, and subsequent growth failure of long bones in stunted children. By 6 months of age, most children consume complementary food, therefore, food quality as well as quantity influences children's growth and development. Briend *et al.* suggested that energy deficient children without infection fulfill their energy requirement by fat mobilization. In this situation, the brain is prioritized, whereas other organs such as kidney, liver, thymus, and muscle receive lower energy supplies. Moreover, levels of hormones, such insulin and glucagon, as well as enzymes, are adjusted such that the organism enters an energy-sparing mode. When glucose is limited, brain and erythrocytes are supplied with energy from ketone bodies of fat tissues. However, glucose should always be available for brain and erythrocytes, Hence, the glucose should be supplied from other sources, i.e., glycerol breakdown from triglycerides, as well as amino acids alanine and glutamine breakdown in liver and kidney. The source of these two amino acids is muscle breakdown. Although the loss of amino acids from muscle in children with deficient energy and essential nutrients

but without infection is minimal, protein metabolism for muscle growth may be affected. The fat area of stunted children aged 7-12 months and muscle area at 13-24 months are smaller that those who are non-stunted, possibly indicating poor muscle growth after fat loss at younger ages.[17]

After 6 months of age, children often experience infection, especially diarrhea and respiratory tract infection. Children face a double burden of health problems due to limited energy and essential nutrient intake leading to anorexia, increased protein requirement for synthesis of an acute phase protein, glutathione, and to improve immune function. In the case of negative nitrogen balance, amino acids from muscle mass will be mobilized.[17]

The majority of muscle mass is located at the lower extremities, whereas muscle mass of the upper arm is considered to be representative of body muscle mass. Stunted children have diminished length of legs and arms, therefore, their muscle mass is also diminished. Growth faltering mainly occurs before 24 months of age, and length growth occurs at the lower part of the body. Before 6 months of age, children receive breast milk as the main source of nutrients, therefore, nutrient deficiencies and infection during this period of time is minimal. This phenomenon differs from the 7-24 month period, in which stunted children in our community probably received poor complementary food, leading to poor growth of muscle mass.[17]

Hormones may contribute to lower fat and muscle mass in stunted children, because fat regulates bone mass and linear growth. Fat and bone are considered to be endocrine organs that produce hormones to interact with the other organs, including the brain. Leptin is a hormone that affects bone density and catch-up growth. Wasted children may experience catch-up growth if their leptin levels are normal. However, this argument is still debated, since there was a case report of two children with congenital leptin deficiency with normal height. This phenomenon could be explained by multiple causes of stunting. A community report described a high prevalence of stunting among children with low prevalence of wasting. As we know, wasted children have smaller body fat mass. Another factor that causes stunting is dysentery. Moreover, stunting is probably accompanied by overweight, i.e., stunted-overweightness, and if stunted may stimulate linear growth, this fact could not be satisfactorily explained.[17]

Another essential nutrient for child growth and development is fatty acids. Fatty acids are an energy source and have a physiological role in cellular membrane structure, vision, skin integrity, wound healing, cardiac health, cognitive function, and the immune response. Essential fatty acids, such as linoleic acid or fatty acid n-6, and α-linolenic acid or fatty acid n-3, should be sufficiently provided in the food, because humans lack Δ-12 and Δ-15 desaturases, enzymes which synthetize essential fatty acids. Insufficient essential fatty acids in the diet affects growth, including fat and muscle mass. Linoleic acid-rich foods are sunflower seeds, nuts, soy oil, corn, and canola oil; whereas flaxseed, walnuts, and soy are rich in α-linolenic acid. Sources of essential fatty acids in animal foods are salmon, trout, egg, and poultry. Children with insufficient intake of fatty acids have higher risk of stunting with poor fat and muscle growth.[18] Tanzanian children aged 2-6 years with low intake of fatty acid n-6 had a high prevalence of stunting, however, increased intake of fatty acid n-9 correlated with stunting. Fatty acid n-9 is a non-essential fatty acid, which can be synthetized from fatty acid n-3 and n-6 using Δ-5 and Δ-6 desaturases, as well as elongases.[18]

Our study shows that smaller fat and muscle area in stunted children is clinically important. Decreased muscle mass is related to high mortality among children. Stunting and wasting indicate changes in body composition. Severely decreased fat and muscle mass diminishes the energy reserve that is imperative for vital organs, such as the kidneys, liver, heart, gut, and immune system. Previous studies have demonstrated a higher mortality among adults suffering from malnutrition with comorbidities of liver cirrhosis, cancer, or chronic lung disease. Children have smaller muscle mass compared to adults (23% in neonates *vs.* 43% in adults). But a child's brain is relatively larger for their body weight, therefore, children need more glucose. The glucose source for brain is muscle, therefore, reduced muscle mass effects higher mortality in children compared to adults.[17]

Stunted and wasted children with lower fat and muscle mass have a higher mortality risk. Therefore, health programs should prioritize children with combined stunting and wasting. Younger children

with infection are also important targets, as well as children born small-for-gestational age.[17]

A limitation of our study was the cross-sectional research design, as we were unable to identify changes in individual growth and nutritional status from birth to 24 months of age, nor could we describe factors causing differences in overweight and obesity prevalence, as well as differences in fat and muscle mass in stunted *vs.* non-stunted children. Also, in order to understand the role of leptin on fat and muscle mass in stunted and non-stunted children, the measurement of leptin levels is recommended. Leptin levels may be used as an indicator of whether extra calories given to stunted children will result in normal growth without any excess fat accumulation. A strength of our study was that the study population consisted of young children randomly selected from a whole province.

Based on the study results, we recommend a future longitudinal study from birth to 24 months of age to identify the change of overweight and obesity status, fat and muscle mass, and potential risk factors, especially with regards to food intake and infection. Leptin should be measured in order to precisely calculate the additional calories needed, in order to prevent obesity in stunted children. The differences in body composition among stunted and non-stunted children are indicative of their different nutritional requirements.

In conclusion, the prevalence of overweight or obesity among stunted children is significantly lower compared with non-stunted children. The highest prevalence of stunting is at 22 months of age in boys and 21 months of age in girls. Upper arm fat mass, as represented by UFA, UFE, and UFP, is lower among stunted than non-stunted children at 7-12 months of age. Upper arm muscle mass, as represented by UMA, is lower in stunted children aged 7-24 months, whereas UME is lower in stunted children aged 13-24 months.

Conflict of Interest

None declared.

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