Short Malnourished Children and Fat Accumulation With Food Supplementation

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BACKGROUND: In moderate acute malnutrition programs, it is common practice to not measure mid-upper arm circumference (MUAC) of children whose length is <67 cm. This is based on expert opinion that supplementation of shorter children with low MUAC and weight-for-height z score ≥ 2 may increase risk of excessive fat accumulation. Our aim was to assess if shorter children gain more fat than taller children when treated for moderate acute malnutrition diagnosed by low MUAC alone.

METHODS: In this observational study, we included children aged 6 to 23 months with a MUAC between 115 and 125 mm and a weight-for-height z score ≥ 2. On the basis of length at admission, children were categorized as short if < 67 cm and long if ≥ 67 cm. Linear mixed-effects models were used to assess body composition on the basis of deuterium dilution and skinfold thickness.

RESULTS: After 12 weeks of supplementation, there was no difference in change in fat mass index (−0.038 kg/m², 95% confidence interval [CI]: −0.257 to 0.181, P = .74) or fat-free mass index (0.061 kg/m², 95% CI: −0.150 to 0.271, P = .57) in short versus long. In absolute terms, the short children gained both less fat-free mass (−230 g, 95% CI: −355 to −106, P < .001) and fat mass (−97 g, 95% CI: −205 to 10, P = .076). There was no difference in changes in absolute subscapular and triceps skinfold thickness and z scores (P > .5).

CONCLUSIONS: Short children with low MUAC do not gain excessive fat during supplementation. With these data, we support a recommendation for policy change to include all children ≥ 6 months with low MUAC in supplementary feeding programs, regardless of length. The use of length as a criterion for measuring MUAC to determine treatment eligibility should be discontinued in policy and practice.

WHAT’S KNOWN ON THIS SUBJECT: Policy for national malnutrition programs in many African countries is to exclude shorter children with moderate acute malnutrition from treatment because of concerns they will accumulate fat and be at later risk of noncommunicable diseases.

WHAT THIS STUDY ADDS: We show that shorter children with low mid-upper arm circumference do not gain excessive fat during supplementation and thus should be offered treatment. This evidence should be integrated in guidelines by the World Health Organization.


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Dr Fabiansen conceptualized the research, conducted the research, acquired the data, performed statistical analysis, and wrote the first draft of the manuscript; Mr Phelan and Drs Yaméogo, Kurpad, Wells, Filteau, Briand, Christensen, Cichon, and Iuel-Brockdorff conducted the research and acquired the data; Dr Ritz conceptualized the research and design and performed statistical analysis and interpretation of data; Dr Shepherd conceptualized the research and contributed information regarding study design and analysis. All authors read and approved the final manuscript.

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Childhood malnutrition contributes to almost half the mortality in children <5 years. Moderate acute malnutrition (MAM) is currently defined as a weight-for-height z score (WHZ) between −3 and −2 (ie, moderate wasting) and/or a mid-upper arm circumference (MUAC) between 115 and 125 mm. Although the prevalence of MAM is unknown, moderate wasting alone affects 33 million children at any time and is associated with a threefold increased risk of death.

Protocols for management of acute malnutrition in many African countries (including Cameroon, Central African Republic, Chad, Guinea, Ivory Coast, Mali, Mauritania, Senegal, and Togo) instruct health personnel to measure the MUAC only of children with a length ≥67 cm when assessing eligibility for MAM or severe acute malnutrition (SAM) treatment programs. In Ethiopia, admission by MUAC alone for SAM treatment is restricted to children with lengths >65 cm. These shorter children are enrolled in treatment only if they meet WHZ criterion, which is less closely linked to the risk of death and misses children at high risk who would have been identified by MUAC. Hence, current practice excludes short children who would benefit from nutritional support.

The practice of restricting treatment admission by MUAC alone to children of lengths ≥67 cm is not supported by data and has never been formally endorsed by the World Health Organization (WHO). Two reasons can be identified to explain this exclusion. First, the inclusion of a length restriction for MUAC is likely a holdover from older versions of emergency nutrition guidelines that used length as a proxy for age (<6 months) because it can sometimes be difficult to ascertain age in a fast-moving emergency. WHO, for example, used a length of 60 cm as a proxy for an age of 6 months in publications from 1995 to 2000, whereas a length of 65 cm was used in older versions of guidelines from nongovernmental humanitarian organizations like Médecins Sans Frontières in 1995 and Valid International in 2006. It is important to identify children <6 months of age because they should be exclusively breastfed and treated as inpatients if in need of treatment. However, length is a poor proxy for age, and most often a caretaker can recall the month of birth for an infant.

Second, WHO now recommends against routine food supplementation of children with MAM because of a concern it may promote obesity and increase the risk of noncommunicable diseases later in life. Some experts worry that shorter children >6 months of age who only meet the low-MUAC criteria for MAM are most at risk for excessive fat accumulation during treatment. Although only based on expert opinion, this seems to be the reason why many national malnutrition protocols exclude shorter children from having their MUAC measured, thereby making children ineligible for treatment, unless they meet WHZ criteria. This concern led WHO to call for research to identify appropriate MUAC admission and discharge criteria for children <67 cm and >6 months for supplementary feeding programs.

We have previously shown that when supplemented, ponderal growth rates are similar in short and long children >6 months with a low MUAC. Niger dropped this length restriction in 2016, and its revised protocols now call for measuring MUAC in all children ≥6 months, regardless of length.

The extent to which there is a difference in fat accumulation during treatment has not been assessed. Our objective in this article is to assess if short children (<67 cm) gain more fat than long children (≥67 cm) when treated for MAM diagnosed by low MUAC (115–125 mm).

METHODS

As previously reported, a prospective cohort study was nested in a randomized nutrition trial (Treatfood) in which we investigated the effectiveness of 500 kcal/day supplements as either corn-soy blend (CSB) porridge or ready-to-use lipid-based nutrient supplements (LNSs). The LNSs provided almost 3 times more energy as fat than the CSB supplements (~57% vs ~21%). Assignment to 1 of the 12 supplements (6 CSBs and 6 LNSs) followed randomization stratified by site. The trial was approved by the Ethics Committee for Health Research in Burkina Faso (2012-8-059), and consultative approval was obtained from the Danish National Committee on Biomedical Research Ethics (1208204). Consent was obtained from caregivers, before inclusion, verbally and in writing (signature or fingerprints). All children were treated free of charge at the study sites, irrespective of study participation.

Participants

Data were collected in the Province du Passoré in the northern region of Burkina Faso at 5 research sites located at different governmental health centers and staffed by the nongovernmental organization Alliance for International Medical Action (Dakar, Senegal).

Children were screened in villages either by community health workers using MUAC tapes or by designated screening teams with the use of both MUAC and WHZ. Moreover, children could be referred from a health center or could present at site on a caretaker’s initiative. At the sites, the final assessment of eligibility for inclusion was performed.
For the main Treatfood trial, children aged 6 to 23 months with MAM (defined as a MUAC between 115 and 124 mm and/or a WHZ between −3 and −2), who were residents in the catchment area and whose caretaker consented to participate, were included. Children were not included if they had been treated for SAM or hospitalized within the past 2 months, if they were already in a nutritional program, or if they presented medical complications requiring hospitalization. Likewise, children with a severe disability limiting the possibility of investigations and children with a suspected allergy to milk, peanuts, corn, or soy were not included.

Of the 1609 children included in the trial, 468 had a MUAC between 115 and 124 mm and a WHZ ≥−2 and were included in the current study. In many settings, these children are excluded from treatment if they are also short (ie, <65 or 67 cm). Accordingly, children in this study were categorized, on the basis of baseline length, as short (<67 cm) or long (≥67 cm) to assess if short children gain more fat than long children when treated for MAM diagnosed by low MUAC.

Procedures and Study Visits

We previously described clinic visits, standard anthropometric measurements, and age determination. In the present article, we additionally report on indices of body composition and skinfolds assessed at baseline and at the end of intervention at 12 weeks. Total body water (TBW) was assessed by using the deuterium dilution technique to enable calculation of fat-free mass (FFM) and fat mass (FM). The method involved giving an oral dose of 5 g of deuterium oxide (D₂O) (99.8%; Cambridge Isotope Laboratories Inc, Andover, MA). The isotope was diluted in 5 g of bottled water (LAFI, Burkina Faso), with the dosing bottle weighed with 0.01 g precision (model CQT 202; Adam Equipment, United Kingdom) before and after administration of the dose. Predose saliva samples were obtained to assess background isotope levels in body fluids, and postdose saliva samples were collected after a 3-hour equilibration period as established during the pilot study. For each assessment, D₂O enrichment was measured in duplicate in the pre- and postdose saliva samples and in a diluted sample of the dose by using Fourier Transform Infrared Spectrometry (Agilent Technologies, Santa Clara, CA) at St. John’s Research Centre, Bangalore, India. Saliva samples required at least 60 μL of saliva for analysis. Deuterium dilution space was calculated as described previously and converted to TBW by using a factor of 1.044 to adjust for proton exchange. FFM was calculated as TBW divided by hydration by using age- and sex-specific hydration coefficients. FM was calculated as weight minus FFM. Data were cleaned for typographical errors and implausible TBW values on the basis of the association of TBW with FFM, weight, length, and skinfold thickness (both raw values and skinfold thickness-for-age z scores). By dividing FFM and FM by length squared, the indices are expressed as kg/m² independent of length, that is, fat-free mass index (FFMI) and fat mass index (FMI). In addition, fat was calculated as a percentage of total body weight.

Outcomes

Changes were evaluated in FM, FFM, weight, length, and skinfold thickness (both raw values and skinfold thickness-for-age z scores). The statistical analyses were conducted by using the statistical software package Stata version 12 (Stata Corp). Baseline and endline characteristics of children were summarized as mean (SD) or percentages, and categories were compared at baseline by using 2-sample t tests for continuous variables and χ² tests for categorical variables.

To evaluate differences in outcomes between groups during the 12-week supplementation intervention, a...
linear mixed analysis of covariance model was considered the main analysis, resulting in an estimated mean difference at the end of the study adjusted for differences at baseline. Specifically, in the analyses, we included adjustment for food intervention as well as a number of baseline characteristics: baseline measure of the outcome, age, sex, and month of admission (as fixed effects). Random intercepts were included in the model to adjust for variation between sites. In the adjusted analysis, weight was derived by adding FFM and FM. Additionally, for direct outcomes of fat (FM and FMI), we evaluated the 2-way interaction between length group and product group (CSB compared with LNS) to assess effect modification by type of product. Moreover, possible effect modification was also evaluated for stunting at admission in (1) 2 categories: greater than and less than a LAZ of $<$−2, or (2) 3 categories: severe stunting (LAZ $<$−3), moderate stunting (LAZ $\geq$−3 to $<$−2), or absence of stunting (LAZ $\geq$−2). The same adjustments were applied in these additional analyses as in the main analysis. Model checking was based on residuals and normal probability plots. A significance level of 0.05 was applied.

RESULTS

A total of 1609 children, of which 55% were girls, were randomly assigned to 12 weeks of supplementary feeding in the Treatfood trial. Of these, 50% (804) were included by both WHZ and MUAC, 21% (337) were included by WHZ alone, and 29% (468) were included by MUAC alone. Among the 468 children recruited by MUAC only, on whom this article is based, 230 (49%) were $<$67 cm (short), and 238 (51%) were $\geq$67 cm (long). During the intervention, 3 short and 11 long children dropped out before completion of supplementation at week 12. Body composition based on $^2$H2O dilution both at baseline and after 12 weeks were successfully determined in 195 (85%) of short children and 195 (82%) of long children (Fig 1). At baseline, there were substantial differences in age and sex distributions as well as in standard anthropometry between short and long, whereas there were no differences in allocation to the different experimental diets and season of inclusion (Table 1). Mean values of body composition variables determined at baseline and after supplementation are presented in Table 2. After the 12 weeks of supplementation, there was no difference of change in FMI (−0.038 kg/m$^2$, 95% CI: −0.257 to 0.181, $P$ = .74) or FFMI (0.061 kg/m$^2$, 95% CI: −0.150 to 0.271, $P$ = .57) in short versus long children. The short children actually gained both less FFM (−328 g, 95% CI: −355 to −205, $P$ < .001) and FM (−97 g, 95% CI: −205 to 10), although the latter was not significant ($P$ = .076). The differences in FM and FFM summed up to a 328 g (95% CI: 199 to 456, $P$ < .001)—lower weight gain in short compared with long children. There were no differences in subscapular and triceps skinfold thickness and z scores (all $P$ > .5).

For FM and FMI, there was no effect modification of product type (CSB or LNS) ($P$ > .30). Likewise, there was no effect modification by stunting at admission (present or nonpresent) ($P$ > .50) or stunting at admission stratified by severity (severe

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<tbody>
<tr>
<td>Enrolled by WHZ and MUAC n = 804</td>
<td>Enrolled by MUAC alone n = 468</td>
<td>Enrolled by WHZ alone n = 337</td>
</tr>
<tr>
<td>Short (≤67 cm) n = 230</td>
<td>Long (≥67 cm) n = 238</td>
<td></td>
</tr>
<tr>
<td>Completed 12 weeks n = 227</td>
<td>Completed 12 weeks n = 227</td>
<td></td>
</tr>
<tr>
<td>$^2$H2O complete n = 195</td>
<td>$^2$H2O complete n = 195</td>
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The data set used is available from the corresponding author on reasonable request.

**TABLE 1** Baseline Characteristics of Children With MAM Included by MUAC Only With Complete D0 Data (n = 390) by Length Category

<table>
<thead>
<tr>
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<th>Shorta</th>
<th>Longb</th>
<th>P</th>
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<tr>
<td></td>
<td>n = 195</td>
<td>n = 195</td>
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<tr>
<td>Girls, % (n)</td>
<td>84 (163)</td>
<td>72 (140)</td>
<td>.005</td>
</tr>
<tr>
<td>Age in mo, mean (±SD)</td>
<td>7.7 (1.6)</td>
<td>14.0 (4.3) &lt; .001</td>
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<tr>
<td>Wi in kg, mean (±SD)</td>
<td>6.012 (0.4)</td>
<td>7.487 (0.7) &lt; .001</td>
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<tr>
<td>Length in cm, mean (±SD)</td>
<td>65.9 (2.1)</td>
<td>72.0 (3.9) &lt; .001</td>
<td></td>
</tr>
<tr>
<td>MUAC in mm, mean (±SD)</td>
<td>121.0 (2.6)</td>
<td>122.2 (1.9) &lt; .001</td>
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<tr>
<td>WHZ, mean (±SD)</td>
<td>−1.5 (0.4)</td>
<td>−1.7 (0.3) &lt; .001</td>
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<tr>
<td>Wi-for-age z score, mean (±SD)</td>
<td>−2.4 (0.6)</td>
<td>−2.1 (0.6) &lt; .001</td>
<td></td>
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<tr>
<td>LAZ, mean (±SD)</td>
<td>−2.0 (1.0)</td>
<td>−1.7 (1.0) .002</td>
<td></td>
</tr>
<tr>
<td>LAZ &lt;−2, % (n)</td>
<td>49 (95)</td>
<td>35 (68) .006</td>
<td></td>
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<tr>
<td>Season at time of inclusion, % (n)</td>
<td></td>
<td></td>
<td>.17</td>
</tr>
<tr>
<td>Dry season</td>
<td>61 (118)</td>
<td>67 (131)</td>
<td></td>
</tr>
<tr>
<td>Rainy season</td>
<td>40 (77)</td>
<td>33 (64)</td>
<td></td>
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<tr>
<td>Food supplement, % (n)</td>
<td></td>
<td></td>
<td>.84</td>
</tr>
<tr>
<td>CSB</td>
<td>48 (94)</td>
<td>47 (92)</td>
<td></td>
</tr>
<tr>
<td>LNS</td>
<td>52 (101)</td>
<td>53 (103)</td>
<td></td>
</tr>
<tr>
<td>Site, % (n)</td>
<td></td>
<td></td>
<td>.009</td>
</tr>
<tr>
<td>0</td>
<td>12 (24)</td>
<td>27 (52)</td>
<td></td>
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<tr>
<td>1</td>
<td>15 (30)</td>
<td>11 (21)</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>21 (41)</td>
<td>18 (35)</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>51 (60)</td>
<td>26 (50)</td>
<td></td>
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<tr>
<td>4</td>
<td>21 (40)</td>
<td>19 (37)</td>
<td></td>
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<tr>
<td>Breastfeeding, % (n)</td>
<td></td>
<td></td>
<td>&lt;.001</td>
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<tr>
<td>0</td>
<td>100 (194)</td>
<td>91 (178)</td>
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a Short = <67 cm.
b Long ≥67 cm.

**TABLE 2** Body Composition Among Short and Long Children With MAM at Baseline and Endline, Without Adjustments

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Endline</th>
<th>Shorta</th>
<th>Longb</th>
<th>P</th>
<th>Δ</th>
<th>Short</th>
<th>Long</th>
<th>P</th>
<th>Δ</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n = 195</td>
<td>n = 195</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>n = 195</td>
<td>n = 195</td>
<td></td>
<td></td>
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<tr>
<td>FM in kg, mean (±SD)</td>
<td>1.098 (0.4)</td>
<td>1.218 (0.4) &lt; .001</td>
<td>1.123 (0.4)</td>
<td>1.246 (0.4) &lt; .001</td>
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<tr>
<td>FFM in kg, mean (±SD)</td>
<td>4.914 (0.5)</td>
<td>6.298 (0.8) &lt; .001</td>
<td>5.673 (0.5)</td>
<td>7.098 (0.9) &lt; .001</td>
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</tr>
<tr>
<td>Fat percentage (FM/WT x 100), mean (±SD)</td>
<td>18.3 (5.8)</td>
<td>16.4 (5.3) &lt; .001</td>
<td>16.3 (5.7)</td>
<td>15.0 (4.9) &lt; .015</td>
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<tr>
<td>FM in kg/m², mean (±SD)</td>
<td>2.689 (0.3)</td>
<td>2.364 (0.8) &lt; .001</td>
<td>2.489 (0.9)</td>
<td>2.295 (0.8) &lt; .006</td>
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<tr>
<td>FFM in kg/m², mean (±SD)</td>
<td>12.000</td>
<td>12.056</td>
<td>.53</td>
<td>12.640</td>
<td>12.723 (0.9)</td>
<td>.36</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Triceps skinfold in mm, mean (±SD)</td>
<td>6.8 (1.1)</td>
<td>6.5 (1.0)</td>
<td>.001</td>
<td>6.8 (1.1)</td>
<td>6.8 (1.2)</td>
<td>.85</td>
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<tr>
<td>Triceps skinfold-for-age z score, mean (±SD)</td>
<td>−1.5 (0.8)</td>
<td>−1.3 (0.8)</td>
<td>.04</td>
<td>−1.1 (0.8)</td>
<td>−0.8 (0.9)</td>
<td>.007</td>
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<tr>
<td>Subscapular skinfold in mm, mean (±SD)</td>
<td>5.6 (0.9)</td>
<td>5.1 (0.8)</td>
<td>&lt; .001</td>
<td>5.8 (1.0)</td>
<td>5.4 (0.8)</td>
<td>&lt; .001</td>
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<tr>
<td>Subscapular skinfold-for-age z score, mean (±SD)</td>
<td>−1.3 (0.9)</td>
<td>−1.5 (0.9)</td>
<td>.03</td>
<td>−0.9 (1.0)</td>
<td>−0.9 (0.9)</td>
<td>.96</td>
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a Short = <67 cm.
b Long ≥67 cm.

DISCUSSION

In response to WHO’s call for research to identify appropriate MUAC admission and discharge criteria for children <67 cm and ≥67 cm, we show here that short children with MUACs 115 to 125 mm but WHZs >−2 do not gain excessive fat during supplementation. Our results, therefore, can be used to challenge current policy and practice in several African countries of measuring MUAC only in children >6 months of age above a certain length threshold, thus excluding shorter children with only low MUACs from eligibility for MAM or SAM treatment programs.5–14

The exclusion of short children is based solely on expert opinion and reflects a concern that these children are just stunted rather than wasted, have slow catch-up growth, and may accumulate excessive FM after supplementation, thereby putting them at risk for noncommunicable diseases later in life. We previously reported that ponderal growth rates were similar in short and long children who received supplementation for MAM determined only by low MUAC,20 and a similar finding was reported in children with SAM.28

With this current study, we are the first to report direct evidence on fat accumulation after treatment of such children, and our data reveal there is no excessive fat gain in the short children compared with long children. In fact, weight gain in both groups overwhelmingly came in the form of FFM. These findings were independent of children having received LNS or CSB supplement. Because LNS contains a high level of fat, some experts were concerned that this food could lead to higher fat deposition in the malnourished child. Earlier we showed that LNS did not lead to high fat accumulation in the treatment of children for MAM.22 We now show that this concern is unwarranted even in shorter children enrolled in treatment solely by MUAC and,

stunting, moderate stunting, or non-present (P > .45).

months for supplementary foods,2 we show here that short children with MUACs 115 to 125 mm but WHZs >−2 do not gain excessive fat during supplementation. Our results, therefore, can be used to challenge current policy and practice in several African countries of measuring MUAC only in children >6 months of age above a certain length threshold, thus excluding shorter children with only low MUACs from eligibility for MAM or SAM treatment programs.5–14

The exclusion of short children is based solely on expert opinion and reflects a concern that these children are just stunted rather than wasted, have slow catch-up growth, and may accumulate excessive FM after supplementation, thereby putting them at risk for noncommunicable diseases later in life. We previously reported that ponderal growth rates were similar in short and long children who received supplementation for MAM determined only by low MUAC,20 and a similar finding was reported in children with SAM.28

With this current study, we are the first to report direct evidence on fat accumulation after treatment of such children, and our data reveal there is no excessive fat gain in the short children compared with long children. In fact, weight gain in both groups overwhelmingly came in the form of FFM. These findings were independent of children having received LNS or CSB supplement. Because LNS contains a high level of fat, some experts were concerned that this food could lead to higher fat deposition in the malnourished child. Earlier we showed that LNS did not lead to high fat accumulation in the treatment of children for MAM.22 We now show that this concern is unwarranted even in shorter children enrolled in treatment solely by MUAC and,
higher prevalence of stunting and had lower MUACs, and had were younger, more often girls, compared with the long children.

At baseline, the short children robust.

uncorrelated, but their results are techniques in which their error is imperfect. Here, we used 2 different all in vivo techniques are necessarily composition is cadaver dissection, Because the gold standard for body fat after supplementation in MAM.

More than 90% of the 1.5 kg–higher weight in long children at baseline was FFM, whereas short children had higher baseline FMI and fat percentage, and this was supported by greater baseline skinfold thicknesses. One possible explanation for this baseline difference is that, in the process of growing poorly, short children invested relatively more in fat reserves than long children, at a cost to their functional fat-free tissue. However, a simpler alternative explanation is that even in children with optimal growth conditions, such as those used to establish WHO 2006 growth standards, both triceps and subscapular skinfold thicknesses tend to decrease between 6 months and 2 years and more so in boys than girls. Hence, the fact that the short children were also ~6 months younger at baseline might explain the higher FMI at baseline. Supporting this, baseline skinfold thicknesses were thicker in short children, whereas their subscapular skinfold thickness was only greater by a z score of 0.2 compared with long children.

At the end of the intervention, the average skinfold thicknesses in this study were less than a z score of –1 in both groups, suggesting no excessive fat gain in either group.

At the end of the supplementation, relative measures of FMI, FFMI, and fat percentage were similar between the 2 groups, and likewise there was no difference between groups in skinfold thickness changes, whether expressed in absolute terms or as z scores. The short children gained less weight and FFMI and also showed a trend toward less FM gain.

In a recent study, it was found that children with MAM diagnosed solely on low MUAC experienced high rates of deterioration to SAM if left untreated. We are not aware of data on mortality specifically for short children with MAM based only on low MUAC. However, the low-MUAC short children in our cohort exhibited a high prevalence of additional anthropometric deficits. Half of the short children were stunted, and short children were more underweight compared with long children. Stunted and underweight children have a higher mortality risk and may, therefore, benefit most from nutritional interventions. MUAC is a simple way to target them for treatment.

WHO recently updated its Integrated Management of Childhood Illness and now recommends against routinely supplementing children with MAM because of a concern for obesity. We previously showed that there was no excess fat accumulation in children with MAM supplemented with LNSs or CSBs. In our findings here, we show that even the shorter children with MAM

**TABLE 3 Changes in Outcomes During Supplementation**

<table>
<thead>
<tr>
<th></th>
<th>Short*</th>
<th>Long*</th>
<th>Difference in Changec</th>
<th>P&lt;sub&gt;d&lt;/sub&gt; Δ</th>
</tr>
</thead>
<tbody>
<tr>
<td>FM in kg, mean (95% CI)</td>
<td>–0.022 (–0.088 to 0.045)</td>
<td>0.075 (0.010 to 0.140)</td>
<td>–0.097 (–0.205 to 0.010)</td>
<td>.057</td>
</tr>
<tr>
<td>FFMI in kg, mean (95% CI)</td>
<td>0.678 (0.604 to 0.752)</td>
<td>0.908 (0.634 to 0.982)</td>
<td>–0.230 (–0.555 to –0.108)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Fat percentage (fat-mass/wt * 100), mean (95% CI)</td>
<td>–1.8 (–2.6 to –1.0)</td>
<td>–1.5 (–2.3 to –0.7)</td>
<td>–0.3 (–1.8 to 1.1)</td>
<td>20</td>
</tr>
<tr>
<td>FFMI in kg/m(^2), mean (95% CI)</td>
<td>–0.186 (–0.309 to –0.034)</td>
<td>–0.128 (–0.271 to 0.015)</td>
<td>–0.058 (–0.257 to 0.181)</td>
<td>.74</td>
</tr>
<tr>
<td>FFMI in kg/m(^2), mean (95% CI)</td>
<td>0.684 (0.556 to 0.813)</td>
<td>0.624 (0.495 to 0.752)</td>
<td>0.061 (0.150 to 0.271)</td>
<td>.57</td>
</tr>
<tr>
<td>Triceps skinfold in mm, mean (95% CI)</td>
<td>0.39 (0.04 to 0.73)</td>
<td>0.35 (–0.002 to 0.70)</td>
<td>0.04 (–0.24 to 0.32)</td>
<td>.77</td>
</tr>
<tr>
<td>Triceps skinfold-for-age z score, mean (95% CI)</td>
<td>0.48 (0.25 to 0.71)</td>
<td>0.50 (0.26 to 0.73)</td>
<td>–0.02 (–0.22 to 0.185)</td>
<td>.88</td>
</tr>
<tr>
<td>Subscapular skinfold in mm, mean (95% CI)</td>
<td>0.32 (0.17 to 0.47)</td>
<td>0.24 (0.09 to 0.40)</td>
<td>0.08 (–0.15 to 0.30)</td>
<td>.52</td>
</tr>
<tr>
<td>Subscapular skinfold-for-age z score, mean (95% CI)</td>
<td>0.52 (0.36 to 0.67)</td>
<td>0.49 (0.34 to 0.65)</td>
<td>0.02 (–0.21 to 0.26)</td>
<td>.84</td>
</tr>
</tbody>
</table>

Data are mean and 95% CI calculated by using linear mixed models adjusted for food intervention, baseline measure of the outcome, age, sex, mo of admission, and site (random effects).

* Short = <67 cm.

* Long = ≥67 cm.

c Difference in change of short versus long children.

d P-value for difference in change from baseline to endpoint for short and long children.

furthermore, is not modified by stunting at admission.

Using the state-of-the-art D\(_2\)O dilution technique seconded by skinfold thickness measurements, we present comprehensive data on body fat after supplementation in MAM. Because the gold standard for body composition is cadaver dissection, all in vivo techniques are necessarily imperfect. Here, we used 2 different techniques in which their error is uncorrelated, but their results are similar, making our findings more robust.

At baseline, the short children compared with the long children were younger, more often girls, had lower MUACs, and had higher prevalence of stunting and underweight but were less wasted. More than 90% of the 1.5 kg–higher weight in long children at baseline was FFM, whereas short children had higher baseline FMI and fat percentage, and this was supported by greater baseline skinfold thicknesses. One possible explanation for this baseline difference is that, in the process of growing poorly, short children invested relatively more in fat reserves than long children, at a cost to their functional fat-free tissue. However, a simpler alternative explanation is that even in children with optimal growth conditions, such as those used to establish WHO 2006 growth standards, both triceps and subscapular skinfold thicknesses tend to decrease between 6 months and 2 years and more so in boys than girls. Hence, the fact that the short children were also ~6 months younger at baseline might explain the higher FMI at baseline. Supporting this, baseline skinfold thicknesses were thicker in short children, whereas their subscapular skinfold thickness was only greater by a z score of 0.2 compared with long children.

At the end of the intervention, the average skinfold thicknesses in this study were less than a z score of –1 in both groups, suggesting no excessive fat gain in either group.

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determined solely by MUAC, who were considered by some experts to be at the greatest risk for obesity from food supplementation, do not gain excessive fat. Such children should no longer be excluded from treatment eligibility.

We are not aware of any data on fat accumulation after treatment in short children with SAM by MUAC alone. Indeed, currently only data on short children with SAM by MUAC show that children with MAM are not put at risk for obesity after food supplementation, into its recently updated Integrated Management of Childhood Illness recommendations.

CONCLUSIONS

Short children with low MUAC do not gain excessive fat during supplementation. Admission by the same MUAC criteria (ie, <115 mm for SAM and 115–125 mm for MAM) should apply to any child 6 to 59 months of age, regardless of their length and irrespective of their WHZ. The use of length as criterion for measuring MUAC should be discontinued in policy and practice wherever such restrictions exist. It will also be important for WHO to integrate this evidence, in which we show that children with MAM are not put at risk for obesity after food supplementation, into its recently updated Integrated Management of Childhood Illness recommendations.

REFERENCES

2. World Health Organization. UNICEF, WFP and UNHCR Consultation on the Programmatic Aspects of the Management of Moderate Acute

ABBREVIATIONS

CI: confidence interval
CSB: corn-soy blend
D₂O: deuterium oxide
FFM: fat-free mass
FFMI: fat-free mass index
FM: fat mass
FMI: fat mass index
LAZ: length-for-age z score
LNS: lipid-based nutrient supplement
MAM: moderate acute malnutrition
MUAC: mid-upper arm circumference
SAM: severe acute malnutrition
TBW: total body water
WHO: World Health Organization
WHZ: weight-for-height z score


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