European Journal of Clinical Nutrition
https://doi.org/10.1038/s41430-019-0439-4

ARTICLE

Food and health

Stunting is not a synonym of malnutrition

C. Scheffler[1] · M. Hermanussen[2] · B. Bogin[3] · D. S. Liana[4] · F. Taolin[5] · P. M. V. P. Cempaka[5] · M. Irawan[5] · L. F. Ibbibah[5] · N. K. Mappapa[4] · M. K. E. Payong[4] · A. V. Homalessy[4] · A. Takalapeta[4] · S. Apriyanti[4] · M. G. Manoeroe[4] · F. R. Dupe[4] · R. R. K. Ratri[4] · S. Y. Touw[4] · P. V. K[4] · B. J. Murtani[4] · R. Nunuhitu[4] · R. Puspitasari[6] · I. K. Riandra[7] · A. S. Liwan[7] · P. Amandari[7] · A. A. I. Permatasari[7] · M. Julia[8] · J. Batubara[9,10] · A. Pulungan[10,11]

Received: 10 October 2018 / Revised: 14 April 2019 / Accepted: 5 May 2019

Abstract

Background WHO documents characterize stunting as, "…impaired growth and development that children experience from poor nutrition, repeated infection, and inadequate psychosocial stimulation." The equation of stunting with malnutrition is common. This contrasts with historic and modern observations indicating that growth in height is largely independent of the extent and nature of the diet.

Subjects We measured 1716 Indonesian children, aged 6.0–13.2 years, from urban Kupang/West-Timor and rural Soe/West-Timor, urban Ubud/Bali, and rural Marbau/North Sumatra. We clinically assessed signs of malnutrition and skin infections.

Results There was no relevant correlation between nutritional status (indicated by skinfold thickness) and height SDS (hSDS). In total 53% of boys, and 46% girls in rural Soe were stunted, with no meaningful association between mean of triceps and subscapular skinfolds ($\bar{x}$SF) and height. Skinfold thickness was close to German values. Shortest and tallest children did not differ relevantly in skinfold thickness. The same applied for the association between hSDS and mid-upper-arm circumference (MUAC) using linear mixed effects models with both fixed and random effects. In total 35.6% boys and 29.2% girls in urban Ubud were overweight; 21.4% boys and 12.4% girls obese, but with mean hSDS = −0.3, still short. Relevant associations between hSDS and $\bar{x}$SF and MUAC were only found among the overweight urban children confirming that growth is accelerated in overweight and obese children. There were no visible clinical signs of malnutrition or chronic infection in the stunted children.

Conclusion The present data seriously question the concept of stunting as prima facie evidence of malnutrition and chronic infection.

✉ M. Hermanussen
michael.hermanussen@gmail.com

1 University of Potsdam, Human Biology, 14469 Potsdam, Germany

2 Aschauhof, 24340 Altenhof, Germany

3 Health and Wellbeing Global Challenge, School of Sport, Exercise & Health Sciences, Loughborough University, Leicestershire LE11 3TU, UK

4 Medical Faculty Nusa Cendana University, Kupang-East Nusa Tenggara, Kupang, Indonesia

5 East Nusa Tenggara Indonesian Society of Pediatrician, Kupang-East Nusa Tenggara, Kupang, Indonesia

6 School of Medicine, University of Sumatera Utara, Medan, Sumatera Utara, Indonesia

7 Child Health Department Udayana University Sanglah Hospital, Bali, Indonesia

8 Department of Child Health, Faculty of Medicine, Public Health and Nursing, Universitas Gadjah Mada, Yogyakarta, Indonesia

9 Department of Child Health, Medical School, University of Indonesia, Jakarta 10430, Indonesia

10 Indonesian Pediatric Society, Malang, Indonesia

11 Department of Child Health University of Indonesia, Dr. Cipto Mangunkusumo Hospital, Jakarta 10430, Indonesia

Published online: 29 May 2019

Introduction

The clinical audience is wedded to the idea that stunting is nutritional. "There is convergence [also] among the nutrition community on the use of length-for-age as the indicator of choice in monitoring the long-term impact of chronic nutritional deficiency" [1]. The conventional definition of height stunting is, "…impaired growth and development that children experience from poor nutrition, repeated infection, and inadequate psychosocial stimulation" [https://www.who.int/nutrition/healthygrowthproj_stunted_videos/en/]. We accept that malnutrition can result in stunting, but question the reverse equation, stunting = malnutrition. Stunting is frequent among children of low- and middle-income countries (LMIC) [2]. But it is not specific for poverty, poor health, and nutrition. Short stature when compared to modern references has been a frequent feature also among the healthy and wealthy European societies of the past. The pediatric journals of the late 19th and early 20th century reported that breastfed European infants and children, independent of social strata, grew far below WHO standards. Some 15–30% of adequately-fed historic European children would today be classified as stunted by the WHO standards [3]. Historic literature explicitly states that "…growth in height is largely independent of the extent and nature of the diet" [4]. Such statements are even more surprising when considering that the authors of these words were the pediatricians and school doctors of the starving German children raised during and after World War I. These pediatricians were not only aware of the physical effects of starvation on growth, but also aware of the effects of nutrition interventions in starving children. Bloch and Abderhalden [5] described exceptional catch-up growth in height of 3–5 cm within 6–8 weeks during refeeding. These outcomes significantly differ from the results of modern nutrition intervention studies in the LMIC [6]. Repeated infections and poor sanitation have also been claimed to be responsible for the shortness of stature in the LMIC. Yet, recent systematic reviews of water–sanitation–hygiene, and educational interventions to prevent or treat stunting find little evidence for beneficial effects or effects so small as to fall within the measurement error of body length/height [7]. We feel that the failure of many of the modern interventions is not due to some general insensitivity to improvements in nutrition and living conditions, but it is due to a misconception of the interpretation of short stature.

In this context, we note especially the articles by Seoane and Latham [8] and by Waterlow [9, 10]. These articles provided a medical classification of malnutrition based on height-for-age that served as the basis for the modern understanding of the causes of short stature. The reasoning of these papers originated from observations and classification of Gomez et al. [11] who, when drawing up their classification, were assessing the prognosis of malnutrition according to the weight on admission to a hospital in Mexico City in the early 1950s. The significance of these observations was questioned later [12], and even Seoane and Latham criticized a classification based on single parameters. Nevertheless, the basic strategy of associating nutrition and height remained and became entrenched in the literature by the publications of Waterlow who described a deficit in height-for-age due to undernutrition over a long period as, "…*nutritional growth failure*, but for the sake of brevity I shall call this condition *stunting*" [10]. This purely anthropometric definition of nutritional status was discussed in detail in a World Health Organization 1971 report [13] and was broadly accepted after publication of a Nestlé Nutrition Workshop in 1988 [14].

These papers have become the corner stones for the 21st century diagnosis of undernutrition. The terms "stunting", "malnutrition", and "undernutrition" are used as synonyms in the epidemiological, medical, and scientific literature. Prendergast and Humphrey [15] summarized: "Linear growth failure is the most common form of undernutrition globally". We question this statement.

Considering that the history of the word "stunting" originates from observations in hospitalized Mexican children almost 70 years ago, and was used with weight cutoff points at 90%, 80%, and 70% of "expected weight for height", and 95%, 87.5%, and 80% of "expected height for age" based on "Boston 50th percentiles" and that this approach was presented again by Waterlow in 1973 [10] by the example of "two hypothetical children", rather than empirical data, we feel justified to question if the terminology and limited geographical criteria are still valid for global use and classification of undernutrition.

Starvation inhibits growth. This statement cannot be questioned, and there is ample evidence also in historic studies that this is the case. Keys et al. [16, p. 1000–1001] explicitly state that "there can be no doubt from the evidence in the literature that the growth of children can be and is influenced by a restriction in the food intake." But reversing the line of argument by connecting short stature with shortage of food, lacks substantiation [17]. Scheffler et al. [18] reanalyzed cross-sectional growth studies of middle-class school children performed in Kolkata, India, and failed to detect an association between nutritional status (as indicated by skinfold thickness) and body height [19]. Even in socioeconomically disadvantaged children with below-average skinfold thickness, when nutritionally supplemented, the net effect of nutrition on body height is generally small [20]. Out of 22 nutrition intervention studies in urban areas of LMICs only 6 interventions had a small positive effect on length- or height-for-age, often within the 0.3 cm measurement error, weight, and fat increased in 9,

and all other studies showed no beneficial effect on body height or body mass [6]. Even when nutrition interventions on nutritional status were integrated with a child development component, Grantham-McGregor et al. [21] summarized: "There was generally little benefit of at-scale programs to nutritional status", and reported an effect size on height-for-age *z*-score of 0.23 SD. Though significant statistically this is a small biological effect compared with the historic observations on refeeding reported by Bloch and Abderhalden summarized by [5] with effect sizes of more than 1.0 SD. Based on the reviews cited here and others with similar findings we propose that though widely used for assessing the effectiveness of health and nutrition intervention programs, stature has falsely been adopted as the tool of choice for detecting undernutrition.

We measured Indonesian children, with particular focus on West-Timor, one of the poorest provinces of Indonesia, with a prevalence of stunting of up to 50% [22]. Skinfold thickness is a valid indicator of body fat stores and reflects the nutritional status as already recognized historically by Keys et al. [16]. We used the association of skinfold thickness as an indicator of the nutritional status and mid-upper-arm circumference (MUAC) and elbow breadth as a proxy for skeletal frame size and physical activity [23], and based on the conventional definition of height stunting, and the well-known association of parental education with childhood undernutrition in LMIC [24, 25] we hypothesize:

stunted children are characterized by depleted fat stores, measurable by decreased triceps and subscapular skinfold thickness [16, 26].

Better nutrition leads to less stunting.

1. Fat stores of less stunted children are less depleted, measurable by positive correlations between height standard deviation scores (hSDS) and skinfold thickness.

2. Positive correlations between hSDS and the educational level of the parents assuming a priori that better parental education might minimize the risk of child malnutrition [24, 25].

3. Stunted children exhibit one or more visible clinical signs of malnutrition [26].

We question that "Linear growth failure is the most common form of undernutrition globally" [15], especially when stunting is prevalent in wealthy populations of LMICs. We present such data for Indonesia.

Subjects and methods

We measured 1716 elementary school children, age between 6.0 and 13.2 years, from three Indonesian provinces in February and March 2018. Indonesia is not a poor country, it ranks 7th out of 190 countries in the World Bank list of GDP [27], but Indonesian children are short in stature, and officially considered malnourished. A total 35.6% of Indonesian children were stunted in 2010. The prevalence of stunting increased to 37.2% in 2013 [28]. With a global hunger index of 22, Indonesia is considered "seriously" affected by starvation [29]. This view, however, is strongly questioned by Indonesian pediatricians with direct clinical experience.

1. West-Timor, belonging to East Nusa Tenggara, is the poorest province of Indonesia with a nominal per capita GDP of US$1288 [30]. The population of this island is among the shortest of Indonesia [22], with an exceptionally high rate of stunted children. We measured in two representative elementary schools in urban Kupang/West-Timor, and one representative school in rural Soe/West-Timor. Kupang and Soe are situated 110 km apart from each other, connected by one asphalted road, driving time ~3 h by private car. Kupang has some 330,000 inhabitants, a university, an airport, and a harbor. Rural Soe has some 40,000 inhabitants, there is very little ecotourism and no remarkable industry. The population of West-Timor is comparably homogeneous. Except for refugees from nearby East-Timor, there was little migration in recent history. The people are very short, physical contact with modern tourists is virtually absent, but the pleasures of modern communication, such as television, internet, and cellular phones are ubiquitously present in the young generation.

2. The "tourist island" Bali, is densely populated and economically more prosperous with a nominal per capita GDP of US$3791. Balinese people are the tallest Indonesians [22]. We studied elementary school children of the city of Ubud.

3. We studied elementary school children of rural North Sumatra, near Marbau that was selected because its nominal per capita GDP with $3588 is close to that of Bali, but the region is known for high stunting rates. Marbau has some 2500 inhabitants surrounded by rural settlements along the roads and is strongly involved in modern agro-industry.

Parental informed consent was given. Ethical approval was provided by the Medical and Health Research Ethics Committee, Faculty of Medicine, Gadjah Mada University, Yogyakarta, Ref. no. KE/FK/0175/EC/2018. We excluded one child with trisomy 21, one child with hydrocephalus, one child with club feet, and one child with gait disorder. Six children refused being measured.

All measurements were performed in the presence of the children's teachers, and supervised and accompanied by 26 local physicians, pediatricians, and medical residents. Birth date and information on parent education was obtained from school records. Parent education was expressed as the total number of school years of both parents including university education. We measured body height (technical error 2.5 mm), weight (technical error 0.15 kg), triceps (technical error 1.5 mm), subscapular skinfolds (technical error

2.0 mm), and clinical signs of malnutrition (hair, skin, and general appearance [13]). The children were lightly dressed and measured without shoes. Weight of the school uniforms was found to be close to 200 g in children below age 10 years, and about 300 g in children above age 10 years, and was subtracted from the weight measurements. Body height was determined by digital laser rangefinder GLM Professional® Bosch 250 VF [31] to the nearest millimeter, weight by digital scales (Soehnle, Nassau, Germany, Style Sense Compact 100) to the nearest 100 g, and skinfold thickness by caliper (Holtain, Ltd Crosswell, Crymych, UK) to the nearest 0.2 mm. All measurements were taken under standardized conditions [32]. The study included traveling of some 900 km by car, and extensive walking through residential areas surveying housing conditions, food markets, and sanitary facilities.

To estimate fatness, energy balance, and nutritional status we used the average of three measurements of triceps and three measurements of subscapular skinfold thickness ($\bar{x}$SF). Skinfold thickness changes with age and the distribution of fat depends on sex [33–35]. By averaging two skinfolds, we tried to avoid possible confounding due to age, sex, SES, and ethnic influences, and rather focused on the association between height, body fat, and external skeletal robusticity.

hSDS and body mass index (BMI_SDS) were calculated according to WHO references [3]. To test whether particular effects only occurred in the very thin, or in the overweight/obese group of children, we focused on children with "normal BMI" (BMI_SDS < −1.28; and BMI_SDS > 1.28). "Thinness" was defined as BMI below the 10th centile, i.e., BMI_SDS < −1.28. The terms "overweight" and "obesity" were defined in the usual way with BMI_SDS > +1 or >+2, respectively.

To further estimate nutritional status and external skeletal robusticity, we measured MUAC and elbow breadth and calculated the frame index (FI, elbow breadth/height [36–38]). The latter has been used as a proxy for everyday physical activity levels [19].

Statistical analysis

We performed correlation analyses and linear mixed effects models with both fixed and random effects for hSDS, MUAC, and $\bar{x}$SF, we then plotted the samples and fitted with LOWESS. LOWESS, also referred to as robust locally weighted regression, is a method for fitting a smooth line through x–y data points [39]. Calculations were performed using SPSS version 25 (IBM SPSS Statistics, Armonk, NY), Statistica version 13.2, and with the programming language "R" (R-version 3.5.1 2018).

Results

Nutritional status as indicated by $\bar{x}$SF and hSDS do not correlate (Fig. 1).

Indonesian children lack relevant associations between $\bar{x}$SF and hSDS (Table 1). Rural boys of Soe were shortest (hSDS −2.08), and thinnest (BMI_SDS −1.41), closely followed by the girls (hSDS −1.90, BMI_SDS −1.25). A total 53% of the Soe boys, and 46% of the Soe girls were stunted. $\bar{x}$SF (boys 5.50 mm, girls 6.72 mm) was significantly less than in the children of Kupang (boys 7.65 mm, girls 8.75 mm). Even though the very thin children of Kupang were slightly shorter, they were still significantly taller than the children of Soe. The correlation between $\bar{x}$SF and hSDS ranged from $r = 0.12$ to $r = 0.38$,

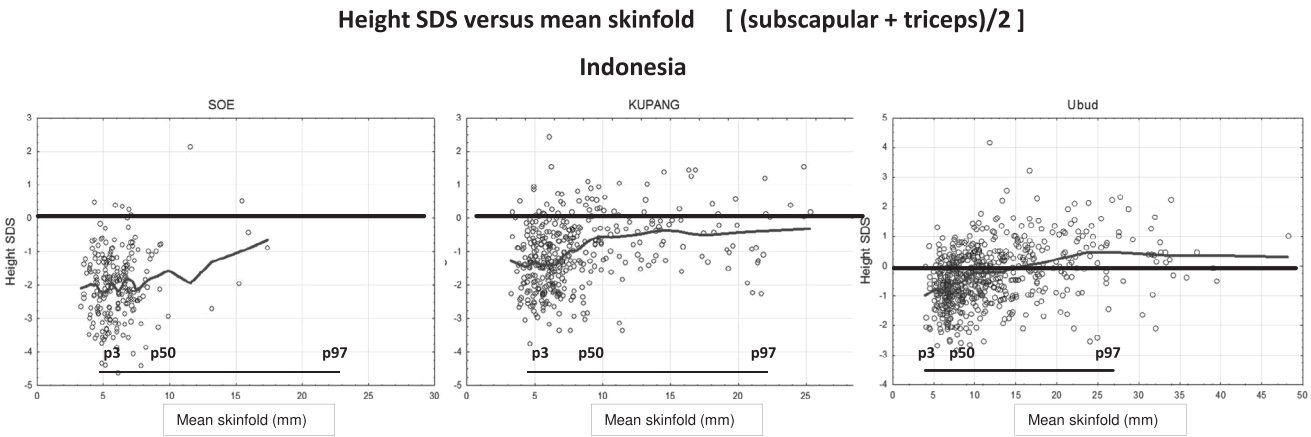

Fig. 1 hSDS and mean skinfold thickness [(subscapular + triceps)/2] of 206 children from urban Kupang/West-Timor, Indonesia, 107 children from rural Soe/West-Timor, Indonesia, and 591 children from urban Ubud/Bali, Indonesia. Age and sex averaged centiles (p3, p50, and p97) of mean subscapular and triceps skinfold thickness of German children [40] (bars) are added for comparison. hSDS and $\bar{x}$SF were plotted and fitted with LOWESS to better highlight the overall shape of the relationship between the *x* and *y* variables. Please note that the scale of the Ubud children differs due to the exuberant prevalence of obesity

Table 1 Anthropometric data and parental education of 908 boys and 808 girls from elementary schools in Ubud (Bali), Kupang (two schools, West-Timor), Soe (West-Timor), and Marbau (two schools, North Sumatra)

	Ubud	Kupang	Soe	Marbau
Boys (6–13 years old)				
n	317	206	107	278
n-normal body mass (BMI_SDS ± 1.28)	186	93	46	196
%-BMI between SDS ± 1.28	58.7	45.2	43.0	70.5
%-hSDS < −2 (% stunted)	5.4	21.8	53.3	25.2
%-BMI_SDS < −1.28 (% thinness)	9.2	40.8	55.1	19.8
%-BMI_SDS > 1 (% overweight)	35.6	15.0	1.9	11.5
%-BMI_SDS > 2 (% obese)	21.4	11.6	0.0	4.7
Mean hSDS	−0.34	−1.10	−2.08	−1.33
SD for hSDS	1.00	1.00	0.93	0.99
Mean BMI_SDS	0.54	−0.69	−1.41	−0.37
SD for BMI_SDS	1.61	1.78	1.06	1.19
Mean skinfold (SF [mm])	12.01	7.65	5.50	8.67
SD for skinfold [mm]	7.86	4.66	1.50	4.60
MUAC [cm]	21.4	18.4	16.6	20.4
SD for MUAC [cm]	2.1	1.9	1.3	1.7
Frame index	38.6	39.0	40.9	39.8
SD for Frame index	2.8	2.4	2.1	1.8
Coeff corr SF × hSDS	0.38	0.38	0.12	0.32
Coeff corr SF × hSDS (BMI_SDS ± 1.28)	0.20	0.12	0.09	0.13
Coeff corr MUAC × hSDS	0.51	0.35	0.25	0.43
Girls (6–13 years old)				
n	274	197	113	224
n-normal body mass (BMI_SDS ± 1.28)	182	106	53	156
%-BMI between SDS ± 1.28	66.4	53.8	46.9	69.6
%-hSDS < −2 (% stunted)	4.0	17.8	46.0	23.7
%-BMI_SDS < −1.28 (% thinness)	8.8	34.0	53.3	18.4
%-BMI_SDS > 1 (% overweight)	29.2	13.7	4.4	8.9
%-BMI_SDS > 2 (% obese)	12.4	5.1	0.0	3.6
Mean hSDS	−0.27	−1.00	−1.90	−1.27
SD for hSDS	0.97	1.02	1.11	0.94
Mean BMI_SDS	0.30	−0.65	−1.25	−0.41
SD for BMI_SDS	1.26	1.49	1.01	1.10
Mean skinfold (SF [mm])	12.33	8.75	6.72	10.34
SD for skinfold [mm]	5.40	4.06	2.15	4.31
MUAC [cm]	20.9	18.4	17.0	20.0
SD for MUAC [cm]	1.8	1.7	1.4	1.7
Frame index	37.3	37.4	39.0	38.2
SD for Frame index	2.6	2.3	1.8	1.7
Coeff corr SF × hSDS	0.33	0.24	0.21	0.12
Coeff corr SF × hSDS (BMI_SDS ± 1.28)	0.27	0.07	0.13	−0.04
Coeff corr MUAC × hSDS	0.37	0.35	0.19	0.34
Parental education—both sexes				
Paternal education (years)	12.28	13.33	11.15	9.18
Maternal education (years)	12.40	13.05	10.73	8.62
Coeff corr hSDS × paternal education	0.04	0.13	0.04	0.12
Coeff corr SF × paternal education	0.00	0.15	0.10	0.10
Coeff corr hSDS × maternal education	0.04	0.09	0.06	0.13
Coeff corr SF × maternal education	−0.07	0.06	0.16	0.10

SD scores were obtained using WHO references [3]

explaining a maximum of 14.4% of the hSDS variance. The average number of parental school years was less in Soe (fathers 11.15 years, mothers 10.73 years) than in Kupang (fathers 13.33 years, mothers 13.05 years, $p < 0.01$). But the correlation between parental education and anthropometry was weak and only explain some 6.5% of the variance in hSDS and some 3.5% of the variance in $\bar{x}$SF.

To minimize the effects of overweight and thinness on growth, we eliminated the extremes and repeated the calculations with "nutritionally normal" children (BMI between ± 1.28 BMI_SDS [3]). Doing so, the weak correlations between $\bar{x}$SF and hSDS disappeared completely (Table 1).

We also studied the thin children from West-Timor, with $\bar{x}$SF below the 10th centile for mean subscapular and triceps skinfold thickness of German children [40]. One hundred and twenty-four boys and 115 girls from Kupang, and 86 boys and 98 girls from Soe belonged to this sample. In these children, hSDS was slightly lower than the average hSDS of the respective groups of all West-Timor children. Yet, the association between hSDS and $\bar{x}$SF was insignificant. In the children of Kupang, the regression analysis showed insignificant betas of −0.015 (95% CI −0.246, 0.207; boys) and 0.048 (95%CI −0.120, 0.204; girls), and in the children of Soe, the regression analysis showed insignificant betas of −0.065 (95% CI −0.190, 0.351; boys) and −0.136 (95%CI −0.349, 0.067; girls).

On the other side, many children particularly from urban Ubud were obese though still shorter than the WHO reference (mean hSDS = −0.3). The correlation between hSDS and skinfold thickness was negligible (Table 1).

Using linear mixed effects models with both fixed and random effects, we further studied the association between

hSDS, MUAC, and x̄SF. When considering the whole sample, MUAC and x̄SF together explained 25.5% of the hSDS variance in boys, and 18.5% of the hSDS variance in girls. But the associations markedly decreased when considering the two parameters, and each population separately (Table 1). It is of particular interest that the shortest population, the children from rural Soe with stunting rates of 53.3% (boys) and 46.0% (girls), showed no meaningful association between hSDS, MUAC, and x̄SF. The hSDS variance explained by both parameters together was 6.5% ($p = 0.03$) in boys and 4.8% ($p < 0.01$) in girls. The FI of the frequently stunted rural children of Soe, with 40.9 (SD = 2.1) mm (boys) and 39.0 (SD = 1.8) mm (girls) was highest among the various regions investigated. The Frame Index (FI) of the frequently obese urban children of Ubud, however, was lowest with 38.6 (SD = 2.8) mm (boys) and 37.3 (SD = 2.6) mm (girls). Previous research reports that FI is positively correlated with physical activity levels and uncorrelated with body fatness [41]. On this basis the high level of physical activity of the Soe children rather contracts the vision that the short stature of these children results from malnutrition or infection.

It is of interest to note that the associations between hSDS and both skinfold thickness and MUAC were least in the stunted rural children of Soe, and greatest in the urban overweight children of Ubud.

We found no visible clinical signs of PEM such as edema, irritability, apathy, or decreased social responsiveness, and anxiety, nor glossitis and nail changes (iron deficiency), goiter (iodine deficiency), hair changes (vitamin A deficiency), nor signs of diminished immune response such as multiple skin infections and poor wound healing [26]. We rather met happy, vivid, and very interested children in apparently good health. We incidentally found an almost 100% prevalence of untreated dental caries, indicating very poor dental hygiene in the rural children of Soe and Marbau. A systematic review of longitudinal studies reported that, "Evidence of the association between anthropometric measurements and dental caries is conflicting and remains inconclusive" [42]. Less than one percent of the children wore eyeglasses indicating underdiagnosed visual impairments.

Discussion

The present analysis rejects the three hypotheses proposed here, all of which are based on the conventional definition of height stunting as due primarily to nutritional inadequacy: (1) stunted children are not uniformly characterized by depleted fat stores; (2) fat stores of less stunted children are not less depleted and better parental education does not minimize the risk of child undernutrition; (3) stunted children do not exhibit visible clinical signs of PEM. Quite in contrast to common expectations, the associations between growth and parameters reflecting nutritional status such as skinfold thickness, FI, and MUAC were least in the stunted rural children of Soe, and greatest in the urban overweight children of Ubud. Small subcutaneous fat depots and narrow-upper-arm circumferences do not appear to inhibit growth. The data rather confirm that growth is accelerated in overweight and obese children. The investigation in 1716 Indonesian school children with stunting rates up to 53% in rural Soe, thus, does not support the modern concept of stunting as an indicator of malnutrition.

Arguments have been raised that observing signs of adequate or even overnutrition in stunted school age children does not exclude undernutrition at younger age, as stature is the summary of all previous height increments. Such arguments are compelling at first view. But growth is plastic. Long periods of childhood starvation are usually followed by catch-up growth [43] and complete recovery. This has repeatedly been shown in school children born during and shortly after wars [44, 45]. The effect of refeeding was particularly well documented in historic studies of severely undernourished children raised during World War I [4, 5] with average catch-up growth rates of 3–5 cm within 8 weeks of refeeding. We found no evidence in the historic European pediatric literature that supports the view that intermittent infant undernutrition due to war and postwar food shortages result in permanent height deficits.

Thus, we cannot share the opinion expressed in the quotation in our "Introduction" that length-for-age is the indicator of choice to monitor chronic nutritional deficiency [1]. We appreciate that this is the working agenda of public health specialists, governments, the food industry, and funding bodies. In recent communication with the German Federal Ministry for Economic Cooperation and Development (*Bundesministerium für wirtschaftliche Zusammenarbeit und Entwicklung*) we were told that this Ministry, "…follows the international definition that stunting results from chronic malnutrition—so-called "hidden hunger". Affected people often receive a sufficient (energy-rich) but not a balanced diet" (email communication to MH, 09 May 2018). The belief that height growth depends on an ideal intake of food and essential nutrients is intuitive. But intuition may not necessarily be true [46]. We are aware that the estimates of the global prevalence of stunting for under 5 years old is close to 155 million [47]. We do not question that these children are short in stature, but we question that all these children are undernourished [17]. Nearly 100 years ago, after World War I, the German pediatrician and school physician Schlesinger summarized that *"the child's longitudinal growth is largely independent of the extent and nature of the diet …"*. Very similar data were published by other pediatricians [4, 5].

Our new Indonesian study consisted of extensive ethnographic observations of residential areas and was supervised and accompanied by 26 local physicians, pediatricians, and medical residents, with direct clinical experience of urban and rural communities. All of us failed to detect visible clinical signs of PEM and essential nutrient deficiencies. Instead we detected obesity except for Soe children. A positive energy balance has mild stimulatory effects on developmental tempo and growth [24], but the obese children were not the tall children. Quite in contrast, even the obese populations were shorter in height than WHO references. In Guatemala it has been known since the 1990s that the high prevalence of stunting is caused by factors other than nutrient intake [48]. A survey of eight rural, Maya villages, and an urban medical clinic in Quetzaltenango, Guatemala with a high percentage of Maya patients found that for 306 newborns, with a median age of 19 days postpartum, 38% were 'stunted' at birth and that maternal height explained only 3% of the variability in hSDS of the newborns [49]. The nutrient intake of the mothers may have been inadequate but as we discussed above, systematic reviews of numerous nutrient supplementation interventions in Guatemala and elsewhere find little or no positive impact on newborn length.

The same research team published a more recent report based on their participatory action research conducted within a socioecological framework [50]. The study enrolled a longitudinal cohort of 155 women, followed during pregnancy (6–9 months), early (0–6 weeks), and later (4–6 months) postpartum, and two cross-sectional cohorts (60 early and 56 later postpartum). The authors report that diet diversity and adult food security (38%) were low. Urinary and gastrointestinal infections were rare (<5%) but reports of distress in meaningful Maya social categories was frequent (20–50%). "Participants reported low maternal autonomy (81%), high paternal support (70%), small social support networks (2.7 ± 1.3 individuals), and high trust in family (88%) and community-based institutions (61–65%) but low trust in government services (6%). Domestic violence was commonly reported (22%). Infant stunting was common (36% early postpartum and 43% later postpartum) despite frequent antenatal care visits (7.5 + 3.8). Participant engagement with the research team did not influence study outcomes based on comparisons between longitudinal and cross-sectional cohorts" (p. 415). These findings confirm the earlier study of high stunting prevalence at birth and associate this with maternal psychosocial distress and insecurity more than diet or infection.

Education matters [22, 23]. Social strata and education are associated with child growth. The differences in height between the Indonesian groups of lower SES and higher SES support this view. But again, the within-group correlations were weak. Within each group, parental education failed to suggest major impact of education on height. We found no satisfying educational explanation for the one standard deviation height difference between rural Soe and urban Kupang.

Heredity has been an argument for short stature. West-Timor, a far-off region with little migration from outside and almost no physical contact with modern Europeans, might serve as an example of genetic isolation, but it fails to explain the difference of one SD between height of children raised only 110 km apart from each other. Similar considerations apply for Maya children from Guatemala born in the USA. They are, on average, 11 cm taller than their siblings born and raised in Guatemala [51]. The genetic argument is further weakened when considering recent Genome-Wide Association Studies stating that genetic loci associated with height only explain some 12.3% of the total variance in adult human height [52].

Why are these children so short?

This question is difficult, as it might imply a negative connotation of being short. We rather suggest asking why are these children not as tall as modern European children? Modern Indonesians are slightly taller than wealthy Europeans some 150 years ago. Kotelmann [53] studied upper class adolescent boys from an elite humanistic school in Hamburg. These boys were delayed in pubertal development by almost two years and reached an average near final height around 165 cm. Similar patterns of growth delay and short height were reported from aristocratic and working-class boys educated at Carlsschule, Stuttgart, an 18th century boarding school [54].

Recent evidence suggests community effects on height [55]. Among social mammals, strategic growth adjustments have been described [56], mechanisms that may also apply for the regulation of human growth [57].

We propose that living in poverty with food and housing insecurity, emotional trauma, and other stress insults are some, but not all of the factors that explain the comparably short stature of those many modern nations listed by the NCD Risk Factor Collaboration [58].

In addition, we propose that children can be short because they lack those social, economic, political, and psychological infrastructures that in the modern western world have stimulated growth beyond all previously recorded limits for height [58]. Good nutrition, health, general living conditions, and care giving are essential prerequisites for growth, but they do not in themselves maximize stature. We emphasize the bilateral link between height and social position [59]. Taller stature is associated with higher socioeconomic status [60], but also social position and political environment modulate growth [55, 61], a vision that opens new fields of biosocial research. Here, the classical verdict of Liberté, Égalité, Fraternité, becomes more than just a revolutionary

statement. Groups who feel liberty, who practice social equality and peaceful coexistence—the Northern European countries may serve as political examples—are known to be tall.

There are limits to this study. The underlying mechanisms of social–emotional growth adjustments are still unclear. The present investigation lacks detailed information on daily food intake and the composition of the diet, and confirmative laboratory data on serum iron, zinc, thyroid hormones, vitamin D levels, etc.

This work does not question the obvious causal association of energy insufficiency, essential nutrient, and chronic infection with reduced linear growth. We are not advocating the discredited idea that the short stature of children and adults living in poverty may be a genetic adaptation or beneficial homeostatic response acquired from generations of malnutrition and is without any pathological consequence.

But we do question the inappropriate use of global growth standards to conclude that stunting is *prima facie* evidence of malnutrition and chronic infection. We question that government ministries and nongovernmental organizations base their *raison d'être* on the term "stunting" as a synonym for "chronic malnutrition". We intend to stimulate the debate about the inappropriate misapplication of a global growth reference derived from high SES and mostly westernized populations when applied to apparently healthy and well-nourished low SES children raised in remote areas of former European colonies. We propose a more precise identification of growth inhibition caused by malnutrition based on a combination of measurements, including height, triceps and subscapular skinfolds, and other indicators of energy balance and nutrient adequacy.

We protest against the misinterpretation of short stature as a proxy indicator for malnutrition. *Malnutrition leads to stunting, but stunting by itself does not indicate malnutrition*.

Acknowledgements We gratefully acknowledge significant support by the local authorities, and in particular, by Octavianus NE, Jelalu ES, Lubis M, Arto KS, Siregar C, Rahayu S, Nasution A, Andreas ST, Harahap MA, Sihotang RD, Winny, Suryawan WB, Prayoga AW, Satria E, Paramita ADP, Febriyanti KD, Mahaputri AS, Dhyna AD, Pratama GA, Trisnia A, Purnama IA, and Wisudarma GA, from the Medical Faculty Nusa Cendana University, Kupang-East Nusa Tenggara, Indonesia; from the School of Medicine, University of Sumatera Utara, Medan, Sumatera Utara, Indonesia; and from Regional General Hospital Wangaya Denpasar.

Funding There was no funding for data collection, analysis, or interpretation of the data; there was no funding for trial design; subject recruitment; there was partial funding of travel expenses by the Indonesian Pediatric Society, managed by Pulungan A. The corresponding author MH has full access to all the data in the study and had final responsibility for the decision to submit for publication.

Author contributions Study design: CS, MH, BB, literature search: CS, MH, BB, measurements: Indonesia—CS, MH, and the Indonesian co-authors: DSL, FT; CPMVP, MI, LFI, NKM, MKEP, AVH, AT, SA, MGM, FRD, RRKR, SYT, PVK, BJM, RN, RP, IKR, ASL, PA, and AAIP equally contributed to the measurements of the children. Local organization of the study: MJ, JB, and AP; data analysis: CS, MH, and BB; data interpretation: CS, MH, and BB; writing: CS, MH, and BB; figures: BB, CS.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Publisher's note: Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

References

1. Lartey A. What would it take to prevent stunted growth in children in sub-Saharan Africa? Proc Nutr Soc. 2015;74:449–53.
2. Black RE, Victora CG, Walker SP, Bhutta ZA, Christian P, de Onis M et al. Maternal and child undernutrition and overweight in low-income and middle-income countries. Lancet 2013;382:427–51.
3. https://www.who.int/growthref/en/
4. Hermanussen M, Bogin B, Scheffler C. Stunting, starvation and refeeding: a review of forgotten 19th and early 20th century literature. Acta Paediatr. 2018;107:1166–76.
5. Hermanussen M, Bilogub M, Lindl AC, Harper D, Mansukoski L, Scheffler C. Weight and height growth of malnourished school-age children during re-feeding. Three historic studies published shortly after World War I. Eur J Clin Nutr. 2018;72:1603–19.
6. Goudet S, Griffiths P, Bogin B, Madise N. Interventions to tackle malnutrition and its risk factors in children living in slums: a scoping review. Ann Hum Biol. 2017;44:1–10.
7. Dangour AD, Watson L, Cumming O, Boisson S, Che Y, Velleman Y, et al. Interventions to improve water quality and supply, sanitation and hygiene practices, and their effects on the nutritional status of children. Cochrane Database Syst Rev. 2013: CD009382. https://doi.org/10.1002/14651858.CD009382.pub2.
8. Seoane N, Latham MC. Nutritional anthropometry in the identification of malnutrition in childhood. J Trop Pediatr Environ Child Health. 1971;17:98–104.
9. Waterlow JC. Classification and definition of protein-calorie malnutrition. Br Med J. 1972;2:566–9.
10. Waterlow JC. Note on the assessment and classification of protein-energy malnutrition in children. Lancet 1973;2:87–9.
11. Gomez F, Galvan RR, Frenk S, Munoz JC, Chavez R, Vazquez J. Mortality in second and third degree malnutrition. J Trop Pediatr. 1956;2:77–83.
12. Gueri M, Gurney JM, Jutsum P. The Gomez classification. Time for a change? Bull World Health Organ. 1980;58:773–7.
13. Joint FAO/WHO. Expert Committee on Nutrition. World Health Organization Technical Report Series. No. 477. Geneva: WHO; 1971.
14. Waterlow JC, editor. Linear growth retardation in less developed countries. Nestle Nutrition Workshop Series, 1988, Vol. 14. New York: Nestec Ltd; 1988.
15. Prendergast AJ, Humphrey JH. The stunting syndrome in developing countries. Paediatr Int Child Health. 2014;34:250–65.

16. Keys A, Brozek J, Henschel A, Mickelsen O, Longstreet Taylor H. The biology of human starvation. Minneapolis: The University of Minnesota Press; 1950.
17. Hermanussen M, Wit JM. How much nutrition for how much growth? Horm Res Paediatr. 2017;88:38–45.
18. Scheffler C, Krützfeldt LM, Dasgupta P, Hermanussen M. No association between fat tissue and height in 5019 children and adolescents, measured between 1982 and in 2011 in Kolkata/India. Anthropol Anz. 2018;74:403–11.
19. Mumm R, Scheffler C. Lack of evidence of nutritional influence on height in four low and middle-income countries. Anthropol Anz. 2019. https://doi.org/10.1127/anthranz/2019/0988 in press
20. Kristjansson E, Francis DK, Liberato S, Benkhalti Jandu M, Welch V, Batal M et al. Food supplementation for improving the physical and psychosocial health of socio-economically disadvantaged children aged three months to five years. Cochrane Database Syst Rev. 2015:CD009924. https://doi.org/10.1002/14651858.CD009924.pub2.
21. Grantham-McGregor SM, Fernald LC, Kagawa RM, Walker S. Effects of integrated child development and nutrition interventions on child development and nutritional status. Ann N Y Acad Sci. 2014;1308:11–32.
22. Pulungan AB, Julia M, Batubara JRL, Hermanussen M. Indonesian national synthetic growth charts. Acta Sci Paediatr. 2018;1:20–34.
23. Rietsch K, Eccard J, Scheffler C. Decreased external skeletal robustness due to reduced physical activity? Am J Hum Biol. 2013;25:404–10.
24. Vollmer S, Bommer C, Krishna A, Harttgen K, Subramanian SV. The association of parental education with childhood undernutrition in low- and middle-income countries: comparing the role of paternal and maternal education. Int J Epidemiol. 2017;46:312–23.
25. Imdad A, Yakoob MY, Bhutta ZA. Impact of maternal education about complementary feeding and provision of complementary foods on child growth in developing countries. BMC Public Health. 2011;11(Suppl 3):S25.
26. Behrman RE, Kliegman RM, Jenson HB. Nelson, textbook of pediatrics. 16th ed. Philadelphia, London, Toronto: Saunders; 1999.
27. https://en.wikipedia.org/wiki/List_of_countries_by_GDP_(PPP).
28. Badan Penelitian dan Pengembangan Kesehatan Kementerian Kesehatan RI. Riset Kesehatan Dasar. Jakarta: Indonesia; 2013. p.4.
29. http://www.globalhungerindex.org/pdf/en/2017/posters.pdf.
30. https://en.wikipedia.org/wiki/List_of_Indonesian_provinces_by_GRP_per_capita.
31. Schrade L, Scheffler C. Assessing the applicability of the digital laser rangefinder GLM Professional Bosch 250 VF for anthropometric field studies. Anthropol Anz. 2013;70:137–45.
32. Knussmann R. Anthropologie, Handbuch der vergleichenden Biologie des Menschen. Stuttgart: Fischer; 1988. P.
33. Maynard LM, Wisemandle W, Roche AF, Chumlea WC, Guo SS, Siervogel RM. Childhood body composition in relation to body mass index. Pediatrics 2001;107:344–50.
34. Wells JC. Sexual dimorphism of body composition. Best Pract Res Clin Endocrinol Metab. 2007;21:415–30.
35. Scheffler C, Obermüller J. Development of fat distribution patterns in children and its association with the type of body shape assessed by the Metric-Index. Anthropol Anz. 2012;69:45–55.
36. Frisancho AR, Garn SM. Skin-fold thickness and muscle size: implications for developmental status and nutritional evaluation of children from Honduras. Am J Clin Nutr. 1971;24:541–6.
37. Friedman JF, Phillips-Howard PA, Mirel LB, Terlouw DJ, Okello N, Vulule JM, et al. Progression of stunting and its predictors among school-aged children in western Kenya. Eur J Clin Nutr. 2005;59:914.
38. Frisancho AR. Triceps skin fold and upper arm muscle size norms for assessment of nutritional status. Am J Clin Nutr. 1974;27:1052–8.
39. Cleveland WS. Robust locally weighted regression and smoothing scatterplots. J Am Stat Assoc. 1979;74:829–36.
40. Schilitz A. Körperliche Entwicklung und Körperzusammensetzung von Brandenburger Schulkindern im Geschlechter- und Altersgruppenvergleich (Berichte aus der Biologie). Maastricht und Herzogenrath: Shaker; 2001.
41. Frisancho R. Anthropometric standards for the assessment of growth and nutritional status. University of Michigan Press; Michigan, USA, 1990.
42. Li LW, Wong HM, Peng SM, McGrath CP. Anthropometric measurements and dental caries in children: a systematic review of longitudinal studies. Adv Nutr. 2015;6:52–63.
43. Boersma B. Catch-up growth in children. Leiden: Thesis; 1998.
44. Brundtland GH. Height, weight and menarcheal age of Oslo schoolchildren during the last 60 years. Ann Hum Biol. 1980;7:307–22.
45. Hagen W. Zum Akzelerationsproblem. Dtsch Med Wochenschr. 1961;86:220–3.
46. Hermanussen M, Scheffler C, Groth D, Bogin B. Editorial Perceiving stunting - student research and the "Lieschen Müller effect" in nutrition science. Anthropol Anz. 2018;74:355–8.
47. de Onis M, Branca F. Childhood stunting: a global perspective. Matern Child Nutr. 2016;12:12–26.
48. Solomons NW, Mazariegos M, Brown KH, Klasing K. The underprivileged, developing country child: environmental contamination and growth failure revisited. Nutr Rev. 1993;51:327–32.
49. Solomons NW, Vossenaar M, Chomat AM, Doak CM, Koski KG, Scott ME. Stunting at birth: recognition of early-life linear growth failure in the western highlands of Guatemala. Public Health Nutr. 2015;18:1737–45.
50. Chomat AM, Solomons NW, Koski KG, Wren HM, Vossenaar M, Scott ME. Quantitative methodologies reveal a diversity of nutrition, infection/illness, and psychosocial stressors during pregnancy and lactation in rural Mam-Mayan mother–infant dyads from the Western Highlands of Guatemala. Food Nutr Bull. 2015;36:415–40.
51. Bogin B, Smith P, Orden AB, Varela Silva MI, Loucky J. Rapid change in height and body proportions of Maya American children. Am J Hum Biol. 2002;14:753–61.
52. Tyrrell J, Jones SE, Beaumont R, Astley CM, Lovell R, Yaghootkar H, et al. Height, body mass index, and socioeconomic status: mendelian randomisation study in UK Biobank. Br Med J. 2016;352:i582.
53. Kotelmann, L. (1879) Die Körperverhältnisse der Gelehrtenschüler des Johanneums in Hamburg: Ein statistischer Beitrag zur Schulhygiene. Z Königlichen Preuss Statist Bureau's. Nachdruck;1879.
54. Hartmann W. Beobachtungen zur Akzeleration des Längenwachstums in der zweiten Hälfte des 18. Jahrhunderts. "Carlsschule". Dissertation. Frankfurt; 1970.
55. Hermanussen M, Alt C, Staub K, Aßmann C, Groth D. The impact of physical connectedness on body height in Swiss conscripts. Anthropol Anz. 2014;71:313–27.
56. Huchard E, English S, Bell MB, Thavarajah N, Clutton-Brock T. Competitive growth in a cooperative mammal. Nature 2016;533:532–4.
57. Hermanussen M, Scheffler C. Stature signals status: the association of stature, status and perceived dominance—a thought experiment. Anthropol Anz. 2016;73:265–74.

58. NCD Risk Factor Collaboration (NCD-RisC). A century of trends in adult human height. eLife. 2016;5:e13410.
59. Hermanussen M. Stunted growth. Eur J Clin Nutr. 2016; 70:647–9.
60. Bogin B, Scheffler C, Hermanussen M. Global effects of income and income inequality on adult height and sexual dimorphism in height. Am J Hum Biol. 2017;e22980. https://doi.org/10.1002/ajhb.22980.
61. Bogin B, Hermanussen M, Scheffler C. As tall as my peers—similarity in body height between migrants and hosts. Anthropol Anz. 2018;74/5:363–74.