Response to the correspondence referring to our article “Stunting is not a synonym of malnutrition” (2018EJCN0997RR) by Conny Tanjung, Titis Prawitasari, Damayanti Rusli Sjarif

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

Thank you for the opportunity to answer the response [1] referring to our article, "Stunting is not a synonym of malnutrition" (2018EJCN0997RR) [2].

Stunting defined as height for age <-2SD of the WHO child growth standards [1], is not a synonym of malnutrition. The lack of association between nutrition and stature has explicitly been described already 100 years ago. In view of thousands of severely starving children shortly after World War I, Schlesinger [2] stated: *"The child's longitudinal growth is largely independent of the extent and nature of the diet... Even during severe dietary restrictions, impairments of infant growth are markedly small, and occur slowly and delayed."*

In view of the rich literature on nutrition and growth of the early 20th century, and the still amazingly diverse professional opinions on this issue, we are pleased to reply to the comments of Tanjung et al.[1] They precisely reflect the current state of debate, and we will thus try to provide some more insights into this fascinating topic.

Tanjung et al. [1] refer to the widespread opinion that "stunting may begin very early in life, typically in utero". This may be right for some populations, but it does not apply for Indonesians. Pulungan et al. published Indonesian national growth charts [3]. The average lengths of newborn boys with 50.12 cm, and newborn girls with 49.81 cm are even greater than WHO standards (boys 49.88, girls 49.15 [1]). The same applies for birth weights (average Indonesian boys' birth weight is 3.46 (WHO 3.35) kg, girls' weight is 3.39 [WHO 3.232 kg). The data clearly show that though stunted at later age [4], Indonesian children do not suffer from prenatal growth impairment. This is in line with historic European observations. Mid-19th century European children and adults were significantly shorter than WHO standards and references, but their birth weights and lengths were close to or even above WHO values [5, pp 442-443].

The authors cite "many important factors that can cause stunting such as household and family factors, inadequate infant feeding practices and infection". This is true. Yet, we feel it would be insolent to assume that 50% of the parents in rural Soe, among them many civil servants, government employees, teachers, dentists, and architects, with diplomas, magister, and university degrees, would had offered adverse housing conditions or inadequate feeding practices to induce stunted children.

We agree that stunting may be caused by familial short stature, constitutional delay of growth and development, genetic disorders and endocrine disorders. We did not measure parental height. But we documented timely eruption of the deciduous teeth in children of the first to third year of primary school, and there was no delay in age of menarche in older girls. We failed to find evidence supporting constitutional delay of growth and development. And we feel that it may be quite preposterous to assume that half of the rural children of Soe may have suffered from genetic or endocrine disorders.

It is true and it has never been questioned that children who suffer from starvation do not grow. It is also true that when a period of early starvation has been overcome, one might no longer find the characteristic losses in subcutaneous tissue as these children quickly recover from leanness. But, re-

feeding causes significant catch-up in height [6]. Already half a century ago, ample evidence had been collected in thousands of children from post-war Germany who were sent to Switzerland for re-feeding [7]. These children gained 3-5 cm in height within 6-8 weeks. Being aware of this and similar arguments, we did inquire about the food situation in Soe, but people did not remember any relevant food shortage during the last decade. In addition, there was no time trend in the extent of shortness: the younger children – being closer to some putative adverse early life event – were not shorter in height z-scores than the older children.

Tanjung et al. cite the work of Vonaesch et al. [8]. We are well aware of this work, but we deliberately decided not to discuss it in our manuscript. These authors present several putative explanations in order to understand the association of being too short and too fat. They mention food that is low in animal proteins, low in micronutrients and fat, and rich in carbohydrates. This is not new. We refrained from quantifying food uptake in our sample, however we closely observed the children during school meals, and except for the very obese school children of Ubud, failed to notice any exceptional consumption of food of poor quality. Indonesian children particularly those of rural West Timor, tend to consume traditional food for lunch. We also failed to observe "opportunistic overeating" leading to excessive food intake and hence overweight, as mentioned by Vonaesch et al., except for the obese children of Ubud. It may be mentioned that also the obese school children showed on average negative height z-scores.

We gathered evidence to question the validity of WHO growth charts for the Indonesian population. The WHO child growth standards for infants and children [1] are based on data obtained in Ghana, India, Norway, Brazil, Oman, and North America. These standards lack data from Indonesia or from any other Southeast Asian population. The same applies for the WHO Reference 2007. This reference was a reconstruction of the 1977 US National Center for Health Statistics (NCHS)/WHO reference [9], and we fail to find any convincing reason to understand why an almost half a century old national US based reference should be valid for modern Southeast Asian populations.

Tanjung et al. write about the "remarkable similarity in linear growth of the six Multicentre Growth Reference Study Group populations, demonstrating that, when health, environmental and care needs are met, human growth potential is universal to at least 5 years of age". We are sorry. It may be laudable to believe in such a statement, but it simply lacks truth. Ample historic evidence was collected in the seminal book *"Origin of the study of human growth"* [5] showing growth in healthy upper, and lower social strata of the last centuries. Already at birth, populations may differ in length and weight. Average Indian birth weights ranges far below WHO standards with average values close to or even below 3000 g regardless of caste or wealth as shown by Subramanyam et al. [10]. In contrast to the Multicentre Growth Reference Study Group [11] that referred to a highly selected group of 301 Indian infants for the longitudinal, and only 1490 children for the cross-sectional sample, Subramanyam et al. sampled children aged 0 to 59 months with birth weight data obtained from health cards (n = 3227) and maternal recall (n = 16787). Thus, we do not follow in the belief that "this growth standard [Multicentre Growth Reference Study] is a symbol of children's right to achieve their genetic growth potential". In addition, we rather consider the term "genetic growth potential" a beautiful dream. There is no evidence that such a potential in fact exists and can be achieved under favorable circumstances [12, 13, pp 333-4].

Quite in contrast to the notion of a predetermined potential, the NCD Risk Factor Collaboration [14] published trends in adult human height showing that average height of the same populations may change by up to 20 cm within a single century. This is particularly obvious in the Dutch. Dutch men of the mid-19th century reached on average 163 cm, with a stunting rate of over 50%. Less than one percent were taller than 180 cm [15]. Also the healthy, wealthy Dutch merchants were short when compared with their modern descendants, and they were neither malnourished, nor chronically ill, nor did half of them suffer from endocrine or genetic disorders. The empirically measurable evidence from living populations confirms that healthy, well-nourished and genetically similar people (e.g., same-sex siblings) have a 'growth potential' for a wide range of sizes and shapes. Something other than nutrition and infection underlies this growth variation.

Tall individuals often dominate others. Stature is a signal among social mammals and contests for social dominance stimulate growth, even before the animals eat more food [16]. Dominance stimulates growth also in the human [17, 18]. 19th century Europeans living in the colonies, were tall. Even their children were tall. The children of the Dutch masters raised under the tropical conditions of Indonesia

were not only taller than the Indonesians. Anticipating their future dominance, they were even taller than their European cousins raised in Amsterdam [19].

Being tall is a matter of education and politics. Europeans, particularly the Dutch and the Scandinavian people who since more than a century, are known for their advanced educational system and their democratic history, are tall. Upward social change stimulates height of the following generation [20]. Better educated Indonesian mothers give birth to taller children as we also mentioned in our present paper [21 instead [2]]. Migrants who integrate best into their host population are taller in height than those who do not [22, 23]. Short stature populations, who regularly contact tall people and start to assimilate in habits and living condition, tend to become taller. The tallest Indonesians live on the tourist island Bali [3], and the tallest Balinese live near the beaches of Kuta [Balinese pediatricians, pers. communication 2018].

The false equalization of stunting and malnutrition is an American invention. Let us terminate this reply by adding some notes on this matter.

In 1971, Seoane and Latham [24] and in 1972 and 1973 Waterlow [25, 26] invented a medical classification of malnutrition based on height-for-age. This purely anthropometric definition of nutritional status was also discussed in detail in a World Health Organization 1971 report [27] and later, became quite broadly accepted after publication of a Nestle nutrition workshop in 1988 [28]. This invention published in the Lancet in 1973 [26], was made acceptable by introducing “two hypothetical children with the same deficit in weight for age”, and broadly fantasizing about nutritional growth failure. The author recommended “for the sake of brevity” to call this condition “stunting". Waterlow and others based their invention on the work of Gomez [29] who had reported on 584 severely malnourished children, age 6 months to 5 years, from the Hospital Infantil, Mexico City. Gomez found that weight was “*from 40 to 60 percent of the average normal theoretic weight in 509 children, and under 40 percent in 75 children*”; and that “*the difference in height from the normal theoretic height increases with the age of the child; at 5 years, the difference was 15.8 percent”.* This corresponds to some -3.6 height SDS when referred to modern WHO standards, and does not surprise: starving children do not grow. But being short is not an indicator for starvation. None of these authors was aware of the older European literature, nor did they mention the work of Keys and colleagues who had meticulously studied and reviewed historic literature before starting their fundamental experiments on human starvation [30].

These are the reasons why we are pleased to comment on the comments of Tanjung et al. They are an ideal opportunity for some additional annotations that ***stunting is not a synonym of malnutrition***.

Literature (more literature is available at the authors‘)

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