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Original Article

Prognostic factors at birth for stunting at 24 months of age in rural Indonesia

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Abstract

Background The problems of stunting are its high prevalence as well as the complexity of its risk factors. Identifying the modifiable prognostic factors at birth may reduce the shortterm as well as longterm effects of stunting in later life.

Objective To estimate the influence of prognostic factors detected at birth for stunting at 24 months of age and the occurence of reversal of stunting at 24 months of age among children in a rural area of Indonesia.

Methods Subjects (n=343) were born to mothers participating in a randomized controlled, double-blind, community-based study of vitamin A and/or zinc supplementation during pregnancy and followed from June 1998 to October 2000. The children were followed prospectively from birth until 2 years of age with monthly measurements of length from birth to 12 months, and again at 18 and 24 months. Data on potential prognostic factors detected at birth, i.e., maternal, child, and household facilities, were collected by trained field workers at home visits. The incidence and risk ratio were calculated to assess the influence of the possible prognostic factors detected at birth on stunting at 24 months of age among these children.

Results Boys who were born prematurely had significantly higher risk of stunting at 24 months of age compared to girls born maturely. The incidences of stunting at 24 months of age according to gender, and gestational age were 33.9% boys *vs.* 22.5% girls (RR 1.80; 95%CI 1.06 to 3.09), and 33.3% premature *vs.* 27.6% mature (RR 7.11; 95%CI 2.07 to 24.48), respectively.

Conclusion Boys who were born prematurely have significantly higher risk to become stunted at 24 months of age. The occurrence of reversal of stunting at 24 months of age is low. **[Paediatr Indones. 2016;56:48-56.]**

Keywords: *stunting, 24 months, prognostic factors*

Stunting, defined anthropometrically as height-for-age Z-score (HAZ) <-2 SD,[1] is prevalent in children in developing countries. The prevalence of stunting ranges from 30 to 69% in Asian countries, including Indonesia.[2-7] In European countries, by comparison, only 3.3 to 6.2% children are stunted.[8] Early stunting, particularly during the first two years of life, decreases developmental test scores[9] and increases the risk of psychological problems[10] as well as risk of infection due to lower immune function.[11]

Stunting reflects the cumulative effects of several factors that occur through the life cycle. Both genetics and the environment are hypothesized to influence stunting. Stool regenerating gene protein (REG 1ß) has been observed to be higher at 3 months of age and predictive of a shortfall in linear growth later in life.[12] In addition, genetic studies among stunted children in developing countries revealed that male gender,[6,13-15] short mothers,[6,15] paternal education,[6,15] paternal occupation,[6,16] low birth weight,[6,14] early supplemental feeding,[6] poor preventive health care,[6] maternal zinc deficiency,[17] low weight gain during pregnancy,[18]

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short length at birth,[19] history of malnutrition during early life,[19] unhygienic environments,[19] tap water and toilet facilities,[14,15] family income,[14,16] poor breastfeeding practice,[20] lack of supplementation with vitamin A,[16] anemia,[16] infections such as diarrhea and respiratory infections, food-insecure households, maternal tobacco use,[22] preterm, and small-for-gestational age[23] were all potential risk factors for stunting.[21] However, other results have been inconclusive, as gender as well as maternal education and occupation were not found to be predictors of stunting in another study.[16]

Stunting may be reversed, especially if it occurs during the first two years of life. Of children stunted at 2 years of age, only 30% were no longer stunted at 8.5 years, and 32.5% were no longer stunted at 12 years.[24] Preventive actions can be included in the health program if modifiable factors related to stunting in early life can be identified. Nutritional support should be included in the rehabilitation program, since a diet rich in vitamin A, for instance, is related to a greater incidence of reversal of stunting.[25] The controversy about predictors of stunting among previous studies suggests that demographic factors are likely to play an important role. Therefore, we aimed to evaluate possible prognostic factors identified at birth for stunting at two years of age, in a rural area of Indonesia. We also aimed to evaluate the occurrence of reversal of stunting at two years of age.

Methods

We analyzed secondary data from the Zibuvita and the Pronak Studies done from September 1995 to December 1999 in the Purworejo District, Central Java, Indonesia. The Zibuvita Study was a community-based, double-blind, randomized controlled trial to evaluate the effects of prenatal vitamin A and zinc on maternal morbidity and pregnancy outcomes. Infants born to Zibuvita mothers (without congenital anomalies) whose mothers consented to participate were included in the Pronak Study, a longitudinal study monitoring children from birth onwards with respect to growth, development, feeding practices, and morbidity. The results of these studies were published elsewhere.[26,27]

We had an opportunity to analyze data from 343 infants who had complete or at least minimally missing data on body length from birth up to 12 months of age that could be imputed. As such, subjects who died before two years of age were excluded. This subset of data was selected from 1,956 children born to 2,173 women who were recruited by the Zibuvita Study.

Stunting (in this study we combined stunting and severe stunting into one category) was defined as height-for-age Z-score (HAZ) <-2SD according to the WHO 2006 child growth standard.[1] We evaluated the stunting status at 24 months of age and the reversal of stunting between a particular age and 24 months of age. Reversal of stunting is not as simple as change, for instance, from HAZ –2.1 SD to HAZ -1.9 SD, because a minor change of HAZ 0.2 SD is meaningless. Therefore, we defined reversal of stunting between particular ages (t_1) and 24 months of age to be "a child who was stunted at t_1 but not stunted at 24 months and for whom $HAZ_{24} > r \times HAZ_{t1}$, where HAZ_{24} was the child's HAZ at 24 months, HAZ_{t1} was the child's HAZ at t_1 month, and r was the correlation coefficient between HAZ_{24} and HAZ_{t1} calculated by statistic software (r = 0.148), for the subset of children who were stunted at t_1 months and not stunted at 24 months. Hence, we did not include children for whom $HAZ_{24} \leq r \times HAZ_{t1}$ in the category of those who recovered".[21]

We analyzed several possible prognostic factors for stunting at 24 months of age, which could be identified at birth. These factors included maternal, child, and household variables. Maternal variables analyzed were height (normal vs. short) and education (illiterate/low educatational level vs. high educational level). Normal maternal height was defined as ≥145 cm, while short was defined as <145 cm. Maternal education was classified as high educatational level if she passed high school. Illiterate/low educational level was defined as never attended school, graduated from primary school, or something in between. Child variables were weeks of gestation (premature vs. normal), birth weight (low vs. normal), birth length (stunted *vs.* not stunted at birth), size for gestational age (small-for-gestational age vs. appropriate-for-gestational age), and birth order (first born *vs.* non-first born). The definition of normal weeks of gestation was ≥37 weeks, while prematurity was defined as gestation <37 weeks. Normal birth weight was defined as ≥2,500 g and low birth weight as <2,500 g. Appropriate-for-gestational age was defined as birth weight ≥10th per-

centile, and small-for-gestational age as birth weight $<10^{th}$ percentile. Household variables analyzed consisted of source of drinking water (unimproved *vs.* improved), and sanitation facilities (unimproved *vs.* improved). We defined source of drinking water and sanitation facilities using WHO criteria.[29] Improved sources of drinking water included household connections, public standpipes, boreholes, protected drilled wells, protected springs, and rain water collections, while unimproved sources included unprotected wells, unprotected springs, vendor-provided water, and tanker truck-provided water. Improved sanitation facilities included connection to public sewers or septic systems, pour-flush latrines, and simple or ventilated improved pit latrines, while unimproved facilities included service or bucket latrines from which sewage was manually removed, public latrines, and open latrines.

Trained field workers collected sociodemographic and anthropometric data from the participating subjects at home visits. At birth, anthropometric data were collected within 48 hours of birth. Birth length was measured using a calibrated, locally-produced, wooden measuring board. Afterwards, subjects' lengths were measured monthly from birth to 12 months of age, and additional data were collected at 18 and 24 months of age. Body lengths were measured in triplicate and recorded as a mean value.

We transformed body length data to Z-score using *Epi Info version 3.3* (Centers for Disease Control and Prevention, Atlanta, GA, USA), then imported the data *to SPSS version 15.0* (SPSS Inc., Chicago, IL, USA) for statistical analysis. Our monthly data of body lengths from birth to 12 months of age was missing in 16% of the subjects' measurements, therefore, we performed imputations by assuming a linear growth pattern. For example, we imputed a missing length at 9 months of age as [(length at 8 mo + length at 10 mo)/2].

We calculated relative risk and 95% confidence intervals for each prognostic factor detected at birth on stunting status at 24 months of age using *SPSS version 15.0* (SPSS Inc., Chicago, IL, USA). All prognostic factors with $P<0.2$ in univariate analysis (unadjusted) then were included in a multivariate analysis (adjusted). Statistical significance was determined based on 95% confidence intervals. This study was approved by the Health Research Ethics Committee of the Gadjah Mada University Medical School, Yogyakarta.

Results

Out of 1,956 infants born live to Zibuvita mothers, 1,613 infants were excluded due to lack of maternal consent, being in another study involving micronutrient supplementation (namely, the Zinak Study), or who had missing body length data which could not be imputed.

Of 343 children, the prevalence of stunting at birth was 17/343 (5%). Later in life, 121/343 (35.3%) children became stunted between the age of 0 and 24 months. At 24 months of age, 96/343 (28%) children were still stunted. However, of the 121 children who were stunted between 0-24 months of age, 25 (20.7%) children were no longer stunted at 24 months of age (**Figure 1**).

Table 1 shows that the majority of mothers in our study had a low educational level (76%). Prematurity and small-for-gestational age occurred in 7.0% and 5.2% of the subjects, respectively. Only 12.6% of household facilities had unimproved water sources. We compared children who were not stunted at 24 months of age to children who were stunted at 24 months of age, to evaluate the potential prognostic factors related to stunting at 24 months of age. **Table 2** shows that gender and gestational age correlated to stunting at 24 months of age. The incidences of stunting at 24 months of age were higher in boys compared to girls (33.9% *vs.* 22.5%) and prematurity compared to maturity (33.3% *vs.* 27.6%). Hence, infants of male gender (RR 1.80; 95%CI 1.06 to 3.09) and prematurity (RR 7.11; 95%CI 2.07 to 24.48) had higher risk of stunting at 24 months of age compared to infants of female gender and normal gestational age.

Although 20.7% of children who were stunted sometime between 0-24 months of age were no longer stunted at 24 months, we could not determine that the proportion of reversal of stunting in our study was 20.7%. Based on our definition, only 3/121 (2.5%) experienced reversal of stunting at 24 months of age. Birth characteristics of 3 children who experienced reversal of stunting at 24 months of age were as follows: mature, not stunted at birth, appropriate-

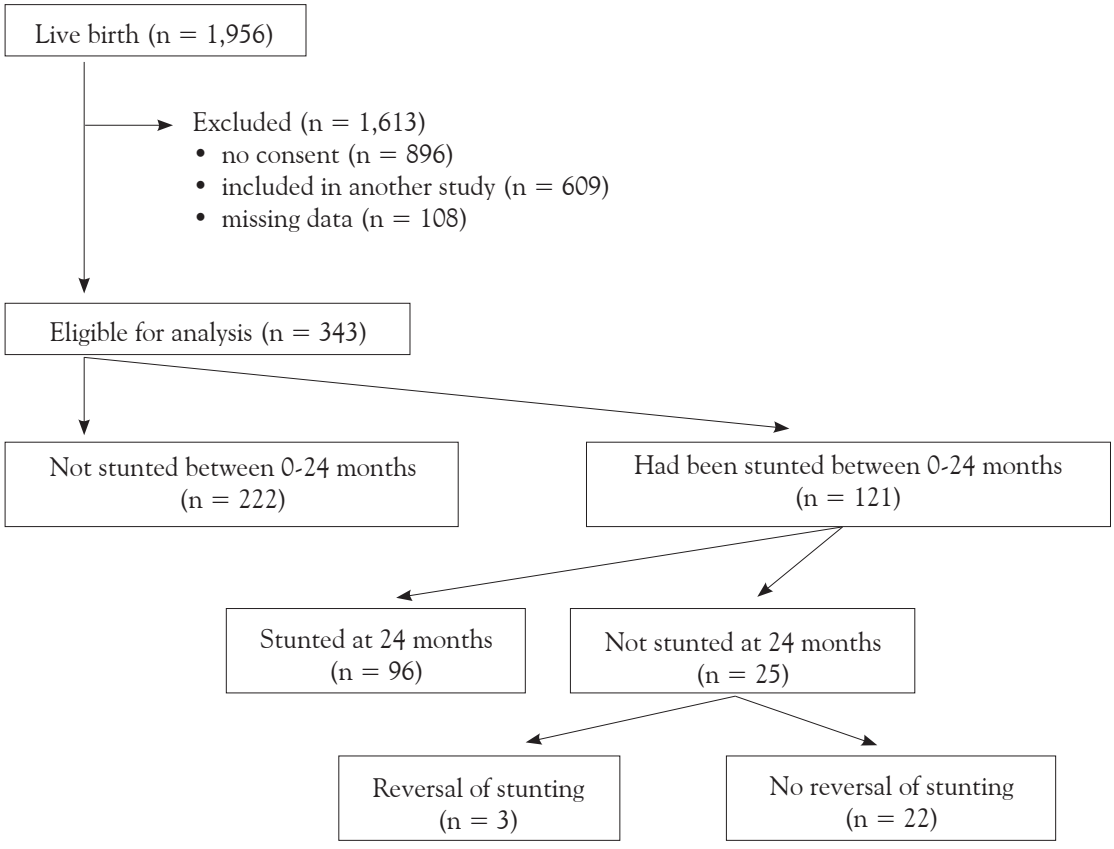

Figure 1. Study profile

for-gestational age, and born to mothers of normal height.

Discussion

Our multivariable analysis of factors identifiable at birth revealed that male gender and premature birth were significant prognostic factors of stunting at 24 months of age. Few children who had ever been stunted in the period of 24 months of life experienced reversal of stunting at 24 months of age.

Previous studies from African countries, Cambodia, India, and Brazil also reported male gender to be a predictor of stunting.[13-16,29] A meta-analysis of 16 demographic and health surveys from Sub-Saharan Africa observed that boys were more vulnerable to inequalities in health, especially in families with low socioeconomic status.[13] Moreover, the conclusion from 131 demographic health surveys and 48 multiple indicator cluster surveys from 80 countries done from 1990 to 2011 was that countries with higher prevalence of stunting tended to have larger socioeconomic inequalities.[30] Unfortunately, we were not able to analyze differences of stunting status by socioeconomic situation of families due to lack of data. However, gender as a risk factor of stunting at 24 months of age was reported to be more likely in boys the first year, and more likely in girls the second year, in a study of Filipino children. The researchers argued that differences in parental caregiving behavior led to the discrepancy. Boys were given solid food earlier, resulting in higher incidence of diarrhea compared to girls.[6]

Table 1. Basic characteristics of mothers and children according to stunting status from a longitudinal study

Characteristics	Proportion of total (%)	Stunting status of the children during the first 24 mo. of life*	
		Stunting (n=121)	No stunting (n=222)
Mother			
Height <145cm, n (%)	36/286 (12.6)	17 (14.0)	19 (8.6)
Illiterate/low educational level,‡ n (%)	259/341 (76.0)	97 (80.2)	162 (73.0)
Child			
Female gender, n (%)	178/343 (51.9)	57 (47.1)	121 (54.5)
First born, n (%)	72/299 (24.1)	29 (24.0)	43 (19.4)
Premature,§ n (%)	24/343 (7.0)	17 (14.0)	7 (3.2)
Small-for-gestational age,¶ n (%)	18/343 (5.2)	12 (9.9)	6 (2.7)
Stunting at birth, n (%)	17/343 (5.0)	17 (14.0)	0 (0)
Household facilities**			
Unimproved water sources, n (%)	43/340 (12.6)	16 (13.2)	27 (12.2)
Unimproved sanitation, n (%)	161/340 (47.4)	63 (52.1)	98 (44.1)

* stunting = HAZ <-2 SD of WHO Growth Standard 2006,
‡ never attended school or up to primary school graduate
§ gestational weeks <37
¶ birth weight <10th percentile
** according to WHO criteria

Small-for-gestational age was a risk factor for stunting in a meta-analysis,[23] which grouped children into four combinations of small-for-gestational age status and gestational age. Relative to the reference group, i.e., appropriate-for-gestational age (AGA) + full term, preterm + AGA, term + small-for-gestational age (SGA) and preterm + SGA children had increased risk of being stunted at 12-60 months of age with OR 1.93 (95%CI 1.71 to 2.18), OR 2.43 (95%CI 2.22 to 2.66), and OR 4.51 (95%CI 3.42 to 5.93), respectively. Therefore, according to the RR value of 3.3 (95%CI 0.93 to 11.8), small-for-gestational age in our study was an "almost" significant prognostic factor, supporting the meta-analysis results that the origin of stunting is during the fetal stage of life.

Low birth weight was reported to be a risk factor for stunting.[6,14] Children born with low birth weight had a risk of neurological abnormalities that led to poor growth.[14] However, a Filipino study showed that the effect of low birth weight on stunting was strongest during the first 6 months of age, then declined. This phenomenon occurred for several reasons. The length velocity of children born with low birth weight is higher during early life compared to those with normal birth weight. Thus, children who are stunted at birth may no longer be stunted several months later. Furthermore, feeding patterns influence the growth of low birth weight infants. Such children are less likely to initiate breastfeeding and are weaned earlier. Therefore, the consumption of infant formula in excessive quantities may influence growth.[6] We observed that although low birth weight may increase the risk of stunting, however our finding was not statistically significant (RR 3.55; 95%CI 0.97 to 12.56). Our results contrasted with those of the Filipino study, but we did not have sufficient data in our subjects' records to identify other factors that may have affected stunting after birth. Previous studies demonstrated that breastfeeding practice, feeding patterns, vitamin supplementation, infectious diseases, as well as family income affected stunting in children.[6,14,16,20,21]

Maternal variables were not significant prognostic factors for stunting at 24 months of age in our study. Low maternal educational level was not a prognostic factor for stunting, in contrast to a Cambodian study that demonstrated a lower prevalence of stunted children from educated parents. Ikeda *et al.* hypothesized that educated parents were better equipped to provide good care to their children than parents with low educational levels.[15] Again, we were unable to assess the influence of various family socioeconomic conditions on stunting status. However, an Indian study concluded that

Table 2. Incidences of stunting and risk for stunting at 24 months of age by prognostic factors identified at birth (maternal, child, and household factors)

Predictive factors	Incidence (%)	Unadjusted risk			Adjusted risk§	
		RR	95% CI	P value	RR	95% CI
Maternal factors						
Maternal height						
<145 cm	33.9	1.68	0.83 to 3.39		2.04	0.95 to 4.38
Maternal education						
High educational level	22.0	1		0.12	1	
Illiterate/low educational level *	30.1	1.54	0.89 to 2.65		1.15	0.58 to 2.47
Child factors						
Gender						
Female	22.5	1		0.19	1	
Male	33.9	1.35	0.86 to 2.10		1.80	1.06 to 3.09¶
Gestation at birth						
Mature	27.6	1		0.001	1	
Premature	33.3	5.02	2.02 to 12.48		7.11	2.07 to 24.48¶
Size-for-gestational age						
Appropriate-for-gestational age	26.8	1		0.007	1	
Small-for-gestational age†	50.0	3.96	1.45 to 10.85		3.32	0.93 to 11.80
Birth weight						
Normal	26.0	1		<0.001	1	
Low	54.2	8.08	2.94 to 22.26		3.55	0.97 to 12.56
Birth order						
Not first born	26.9	1		0.40		
First born	31.9	1.26	0.73 to 2.18		-	-
Household factors						
Water sources						
Improved	27.9	1		0.78		
Unimproved‡	30.2	1.10	0.57 to 2.13		-	-
Sanitation facilities						
Improved	26.8	1		0.16	1	
Unimproved‡	29.8	1.38	0.88 to 2.15		1.17	0.67 to 2.05

* never attended school up to primary school graduate

† birth weight <10th percentile

‡ according to WHO criteria

§ prognostic factors with P<0.2 were included in multivariate analysis

¶ significant

maternal education did not significantly contribute to stunting.[16] Maternal height was also not a prognostic factor for stunting in our study. In contrast, a Cambodian study showed that maternal height was a significant risk factor for stunting status of the child. Short maternal height correlated with intrauterine growth retardation and low birth weight, which are potential predictors of infant mortality and failure to thrive.[15]

A report from Brazil showed that economic development had not changed the effect of lack of toilet availability at home as an indicator of socioeconomic status on stunting. Two surveys done in 1987 and 2007[14] indicated that unavailability of toilets combined with low birth weight were still significant risk factors of stunting. Other risk factors identified in the 1987 survey were no longer seen to be potential risk factors for stunting in 2007, i.e., BCG vaccination and severe pneumonia requiring hospitalization. Socio-economic development along with health intervention improved child nutritional status during the period of time.[14]

Our study revealed a decline in stunting from birth to 24 months of age by 20.7%. However, we could not conclude that the prevalence of reversal of stunting in our community was 20.7%. According to our definition of reversal of stunting, only 2.5% children actually experienced reversal of stunting at 24 months of age. In other words, 18.2% of children who were no longer stunted at 24 months of age, increased only slightly in length, although they were above the HAZ cut-off of –2 SD. A previous study reported a higher prevalence of reversal of stunting. Of children who were stunted at 6 to 72 months of age, 26.3% of them improved their HAZ (>- 2SD) after 18 months of follow up.[25] Due to different definitions of reversal of stunting between studies, 26.3% improvement of stunting reported in that study can be deemed similar to the 20.7% in our study. We used the reversal of stunting definition from a publication based on nine prospective studies from developing countries.[21] Checkly *et al*. reported that 6% of children who were stunted at six months of age experienced reversal of stunting at 24 months of age. The percentages of reversal of stunting at 12 months and 18 months were 4% and 9%, respectively.[21]

Identification of prognostic factors of stunting at birth has a two-fold benefit. Intervention can be started early. A health promotion program should be prioritized for boys born small-for-gestational age or low birth weight. Moreover, health workers should be mindful of the health and nutritional status of pregnant women, since the results of our study indicate that intrauterine life has a role in the risk of future stunting.

A strength of our study was that trained field workers collected data from birth up to two years age. This longitudinal observation permits us to observe cause and effect relationships. However, body length measurements at only 18 and 24 months after one year of age was a limitation of our study. The impact of missing body length data from 12 months to 24 months was that the occurrence of stunting between 12 and 18 months, as well as between 18 and 24 months of age, could not be precisely identified. However, we hypothesized that as stunting is a chronic process, stunting between 12 and 24 months might be detected at 18 months of age.

Premature infants and stunted children have a higher risk of obesity.[33] Children born small-for-gestational age is "low-high-human" means that "low birth weight will be a high risk of high fat in the future," whereas obese children tend to be obese in adult life with high risk of cardiovascular disease and insulin resistance.[34,35] As such, we suggest evaluating the occurrence of obesity among surviving small-for-gestational age infants born to Zibuvita and Pronak women, as well as those who experienced stunting, since they are currently 14-19 years of age.

In conclusion, boys who were born prematurely with low birth weight and small-for-gestational age have significantly higher risk to become stunted at 24 months of age. Prevalence of the reversal of stunting is low, in which these children were likely born by mothers with normal height, mature gestational age, not stunted at birth, and size appropriate-for-gestational age.

Conflict of interest

None declared.

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