Original Article

Maternal and environmental risk for faltered growth in the first 5 years for Tanjungsari children in West Java, Indonesia

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Background and Objectives: Low birth weight leads to growth faltering, attributable inter alia to malnutrition and maternal health and literacy. Risk for growth faltering in rural children under five is studied. Study Design: The Risk Approach Strategy in Tanjungsari, West Java has been analysed for all pregnancies during 1988-1989 and 4,698 singleton infants born between 1 January 1988 and 31 April 1990. Weight and body length/height measurements were repeated over 60 months, and plotted against WHO standards. Weight-for-age and height-forage z-scores were calculated using 2006 WHO growth as reference. The correlation between shortness (so-called stunting) and its presumptive risk factors was determined. A subset underwent DNA analysis for insulin-like growth factor-1 (IGF-1), and insulin receptor substrate-1 (IRS-1) polymorphism. Results: Weight and body length/height follow-ups were followed-up for 3795 infants; 14.2% of the cohort had low birth weight (<2500 g) (LBW) and 85.8% normal birth weight (NBW). LBW infants showed a similar velocity but tended to catch up more slowly (GEE; p<0.001). Relative to WHO references, the differential for stature increased with age, largely offset by reduced weight-for-age so that weight-for-height tracked close to the WHO reference; this contrasts with more divergence internationally. Birth length and weight, along with potable water access were correlated with stunting for children under 2 years. Neither the observed IGF-1, IRS-1 or combined gene polymorphisms were associated with LBW. Conclusions: The prediction by factors operative during pregnancy for early life stature ,with some adaptation for LBW infants, endures to 60 months.

Key Words: birth weight, postnatal growth, stunting, shortness, birth length, water quality, Tanjungsari cohort

INTRODUCTION

Postnatal growth of low birth weight (LBW) infants is of interest in longer term health outcome projection, for which growth curves are employed to monitor this population.¹ For the early diagnosis of an abnormal trajectory, velocity standards are necessary. These standards can only be obtained from longitudinal studies. Community-based recording of postnatal growth is critical, particularly to contrast rural and urban communities in regard to factors affecting growth (e.g. maternal nutrition and infection or environmental factors such as sanitation) Figure 1 shows how disparate these can be by location and age in Indonesia).^{2,3}

The first year of life is critical because it is during this time that a substantial amount of energy is needed for growth; this risk is evident in many settings.^{4,5} Such growth may not be supported by enough food or its quality.³⁻⁵ Growth provides an objective indicator of children's well-being and support, and it is also an accurate marker of disparities in human development.^{4,6}

Weight and length/height are the main measures of growth and plotting these two indicators on a growth

chart is a simple and reliable method to identify faltering among infants and children. Growth faltering is not a disease or symptom of a disease, but it might belie or foreshadow a poorer quality of life. Growth faltering is marked by a loss of weight or failure to gain the expected weight between consecutive measurements.⁷ Plotting length/height and weight measurements against a standardised growth chart can be used to diagnose undernutrition and over-nutrition at any given age and to monitor and observe growth retardation or growth failure in real time before intervention.^{8,9}

Three *types of undernutrition* are recognised: underweight (decreased weight for age), *stunting (pathologically and nutritionally decreased height for age)*, and wasting (decreased weight for height)^{10,11} In public health,

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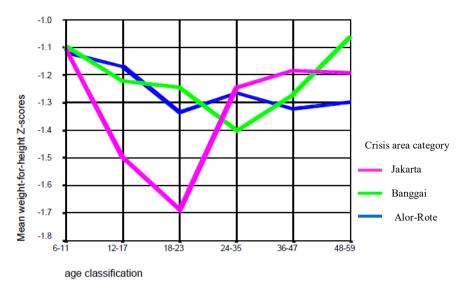


Figure 1. Mean weight-for-height z score (WHZ) of children 6-59 months of age in urban (Jakarta) and rural area (Banggai & Alor-Rote). Source: Bardosono 2007³.

stunting and wasting are considered significant indices of child health due to the compounded risk they provide of morbidity and mortality.^{5,9,12} As over-fatness or obesity may accompany the body compositional features of lean mass in undernutrition, stunting has gained a more prominent place in growth monitoring. The problem this presents is that 'shortness' is not necessarily a nutritional problem and may represent nutritional adaptation.

Stunting as a form of shortness attributable to chronic malnutrition can be caused by many factors, such as maternal nutrition and infections; teenage motherhood and short birth intervals; fetal growth restriction and pre-term delivery; recurrent childhood infections; a range of environmental factors including pollutants; and genetic polymorphisms some of which involve insulin resistance identifiable for insulin-like growth factor-1 (-1245C>T IGF-1) and its receptor, insulin receptor substrate-1 (2914G>C IRS-1).2,13,14 The insulin-like growth factor-1 polymorphism is a single nucleotide polymorphism $C \rightarrow T$ at the -1245 promoter region, while insulin receptor substrate-1 polymorphism is a single nucleotide polymorphism $G \rightarrow C$ at the 2914 structural region. However, shortness is most often found among well individuals and may, as indicated, be adaptive to past adverse health exposures while not itself a health problem. Whether shortness can be said to have a nutritional basis will depend on a contextual analysis of food intake patterns or altered needs with underlying disease. It may also represent adaptation to past or anticipated nutritional disadvantage through epigenetic pathways.

The Tanjungsari cohort is a longitudinal study that started with the RAS (Risk Approach Strategy by Traditional Birth Attendants) research project in October 1987 until December 1989. A birth cohort was established in 1988–1990 in the Tanjungsari subdistrict (West Java, Indonesia), enrolling 4556 children followed for anthropometric measurements from birth to 60 months of age. It was intended that this study would help to design and implement evidence-based policy to reduce pathological shortness (stunting) in children under 5 years in Indonesia.¹⁵

MATERIALS AND METHODS

Study population and sampling

A longitudinal study, referred to as RAS, began in October 1987 and was in place by December 1989.15 The cohort included singleton infants born between 1 January 1988 and 16 April 1990.^{16,17} The inclusion criteria were singleton, availability of both birth weight and birth length measured within 24 hours of birth, and weight and length measured at least 3 times during 0-60 months follow up. Outliers were excluded where birthweight was <2000 g and >3500 g, or birth length <40 cm and >54cm. A subset of 175 adults (89 and 86 adults with LBW and NBW, respectively) was randomly selected for the study of fetal origin of adult disease (FOAD) and underwent DNA analysis. The study and genetic analysis protocol were approved by the Ethics Committee for Medical and Health Research, Faculty of Medicine, Universi-Padjadjaran (No.: 139/FKUP-RSHS/KEPK/Kep/ tas EC/2010). All subjects provided written consent to participation in the studies.

Baseline data collection

Birth weights were those obtained from infants within 24 hours of birth or within the subsequent 24 hours. Interviewers were female village health volunteers, recruited and trained to take the measurements at respondents' homes. The principle investigator (AA) and the research team conducted several weighing cross-checks. Special attention was paid to the way the weighing scales were held and read.¹⁵

Longitudinal data collection and analysis

All infants were revisited for anthropometric measurements on day 7, 28, 42 and at month 3, 6, 9, and 12. Thereafter, regular visits were conducted every 6 months until the child was 60 months (5 years) old.^{15,16} Recumbent measurements were taken from infants and children aged 0–24 months using a UK made somatometer, whereas older children were measured in the standing position (height) using a wall mounted microtoise. Babies were weighed without clothes to the nearest 100 g by

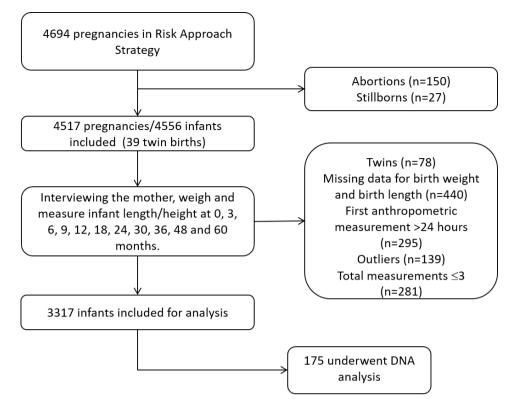


Figure 2. Flowchart for the enrolment of study participants.

using a spring scale (Kern and Sohn GmBH, Germany) with a maximum load of 7 kg; the scales were calibrated before each measurement with weights provided by the company. Salter scales with a maximum load of 25 kg or 50 kg, from the community integrated health post program were used to weigh older children. Available infants decreased by approximately 1%–2% at each examination through death or were lost to follow-up.

Maternal mid upper arm circumference was measured by a colored tape with a cut-off at 22 cm (the rule at that time). Maternal education was classified into less or at least 6 years formal education. Maternal age was classified into high (<16 or >40-year-old) or low risk.

Source of drinking water was classified into improved and unimproved, depending on the openness of the water source.

Exclusive breast feeding was defined as breastfed-only up to 3 months of age, following the recommendation at the time. Protein-rich food intakes were estimated by food recall during each visit.

DNA Polymorphism

Full medical check-ups were conducted for 175 randomly selected participants, with attention paid to any cardiovascular or metabolic problems.¹⁸ DNA isolation¹⁹ and PCR-RFLP for IRS-1 polymorphism²⁰ were conducted by the Health Research Unit, Faculty of Medicine, Universitas Padjadjaran, Indonesia. The sequencing for IGF-1 polymorphism¹⁹ was done at the Genetic Science Lab in Singapore.

Statistical analyses

Live-born singleton infants with known birth weight divided into two categories: LBW (low birth weight) for infants born with birth weight below 2500 g, and NBW (normal birth weight) for infants born weighing 2500 g or more. Birth length categorized into short and normal length with the cut-off point of 48 cm. Eleven measurement points (at age 0, 3, 6, 9, 12, 18, 24, 30, 36, 48, and 60 months) were available for analysis. We calculated the mean and standard deviation (SD) of weight and length (kg and cm) and plotted it against the WHO growth standards.²¹ The serial anthropometric measurements were plotted using Microsoft Excel. SPSS 20 was used for GEE analyses.

Weight-for-height, weight-for-age, and height-for-age were expressed as z-scores using WHO Anthro 2007 version 2.20, which uses the 2006 WHO growth standards as reference. The prevalence of being underweight (low weight-for-age z-score, WAZ), stunting (low height-forage z-score, HAZ), and wasting (low weight-for-height zscore, WHZ) was assessed relative to the 2006 WHO growth standards. Moderate stunting, wasting, and underweight were defined, respectively, as HAZ, WHZ, and WAZ between -2 and -3 SDs (inclusive) below the WHO growth standards. Severe stunting, wasting, and underweight were defined, respectively, as HAZ, WHZ, and WAZ below -3 SD of the WHO growth standards. Prevalences of wasting, stunting, and underweight were applied to the total population. The correlations of 'stunting' as the response variable with other variables (gender, mother's age, mother's education, last birth interval, midupper arm circumference, infant's birth weight, breastfeeding practise, source of drinking water, protein containing complementary feeding practice) as contributory variables were evaluated by Spearman's rho test. Generalized estimating equations (GEE) were used to generate binary logistic regression of predictors with stunting as the dependent variable. The level of significance for statistical tests was p < 0.05.

Table 1. Baseline characteristics of offspring and their mothers

	G	irls	Boys		
Variables	LBW	NBW	LBW	NBW	
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	
n (%)	199 (12.2)	1,437 (87.8)	149 (8.9)	1,532 (91.1)	
Birth weight (kg)	2.28 (0.12)	2.98 (0.31)	2.28 (0.12)	2.98 (0.31)	
Birth length (cm)	44.9 (1.92)	47.2 (2.01)	44.8 (1.65)	47.6 (2.11)	
Mother's age (y)	24.6 (5.83)	25.4 (5.85)	24.0 (5.74)	25.4 (5.78)	
Source of drinking water, n (%)					
Improved	75 (37.7)	657 (45.7)	60 (40.3)	683 (44.6)	
Unimproved	124 (62.3)	779 (54.2)	89 (59.7)	849 (55.4)	
Maternal MUÂC, n (%)	. ,		. ,		
<22 cm	10 (5)	34 (2.4)	15 (10.1)	41 (2.7)	
≥22 cm	51 (25.6)	457 (31.8)	46 (30.9)	463 (30.2)	
missing	138 (69.3)	945 (65.8)	88 (59.1)	1028 (67.1)	
Birth spacing, n (%)	. ,	. ,			
<24 mos	7 (3.5)	108 (7.5)	11 (7.4)	142 (9.3)	
>24 mos	104 (52.3)	893 (62.1)	68 (45.6)	913 (59.6)	
1st pregnancy	88 (44.2)	436 (30.3)	70 (47)	477 (31.1)	
Maternal age group, n (%)					
High risk age	75 (37.7)	489 (34)	69 (46.3)	512 (33.5)	
Normal age	124 (62.3)	947 (65.9)	80 (53.7)	1020 (66.6)	
Maternal formal education, n (%)					
<6 years	56 (28.1)	423 (29.4)	37 (24.8)	459 (30)	
≥6 years	143 (71.9)	1013 (70.6)	112 (75.2)	1072 (70)	

LBW: low birth weight; NBW: normal birth weight; MUAC: mid-upper arm circumference.

RESULTS

For 4505 singleton infants, birth weight and length data were available for 4059 (90.1%) infants, but the data for only 3764 (83.6%) were measured within 24 hours of birth, 139 birth data were out of range, and 308 infants were measured less than 4 times (including stillbirth), thus leaving 3317 (73.6%) infants eligible for inclusion (Figure 2). During follow-up, the number of children measured decreased due to changes of residency; the difference between the number of children at birth and at 60 months was 17%.

The mean birth weights were 2836 g (SD=348.4 g) and 2914 g (SD=361.9 g) in girls and boys, respectively. The mean heights were 46.89 cm (SD=2.19 cm) and 47.36 cm (SD=2.22 cm) in girls and boys, respectively. It was found that 199 (12.2%) girls and 149 (8.9%) boys weighed <2500 g, categorised as LBW.

Weight and length follow-up were analysed for the 3317 infants with complete birth weight and birth length measurements. Means of birth weight and birth length for LBW and NBW children, grouped by gender, are presented in Table 1. The birth weights for male and female infants with LBW children did not differ, while for NBW children they did (p=0.001). On the other hand, birth lengths did not differ.

The mean weight and height series from 0 to 60 months of age are plotted by gender against WHO growth standard in Figure 3. The pattern of weight and height growth for both LBW and NBW of both genders are similar. Mean weights and heights for LBW male and female children remain lower than in NBW children; the differences for weight and length growth with histories of NBW and LBW are significant (GEE p<0.001). In contrast with the WHO standards, both weight and, more especially, length growths are well below the standard.

We then calculated the degrees of wasting, underweight, and 'stunting' based on WHZ, WAZ, and HAZ. The prevalence of wasting (WHZ <-2 SD of the WHO growth standard) was less than 5% at each measurement. The prevalence was highest at 3 months (5.3%) and declined until 9 months, with noticeable peaks at 12 months, but it otherwise decreased throughout the measurement period. The prevalence of stunting was high at all points of measurement, ranging between 26.3% at 3 months and 75.8% at 24 months. The highest prevalence of severe stunting occurred at 18 months (44.1%) and then started to decline until 60 months (25.1%). The prevalence of underweight was not as high as that of stunting. This prevalence increased from 3 to 30 months and peaked at age 60 months (20.8%). The highest prevalence of severe underweight was that at 24 months (3.2%) (Table 2).

The mean WHZ, WAZ and HAZ for each age are plotted in Figure 4. The prominent low means of HAZ leads us to analyse its risk factors. Binary logistic regression for the association between stunting at age 0, 3, 6, 9, 12, 18, 24, 30, 36, 48, and 60 months as the response variable and its explanatory variables such as gender, mother's age, mother's education level, mother's MUAC, infant's birth weight, source of drinking water, breastfeeding practice, and complimentary feeding practise had been conducted. The mother's MUAC was highly correlated with the variable mother's height; this might be lead to collinearity problems. However, we assume that the variable mother's MUAC was more accurate as an independent variable for stunting than the variable mother's height. Mother's age, birth interval and exclusive breastfeeding at age 6 months did not come out as significant risk factors of stunting for children under 5 years old. The analysis of univariable binary logistic regression shown that the age of measurements, birth weight and length, sex, mother's education level, source of drinking water, mid-upper arm

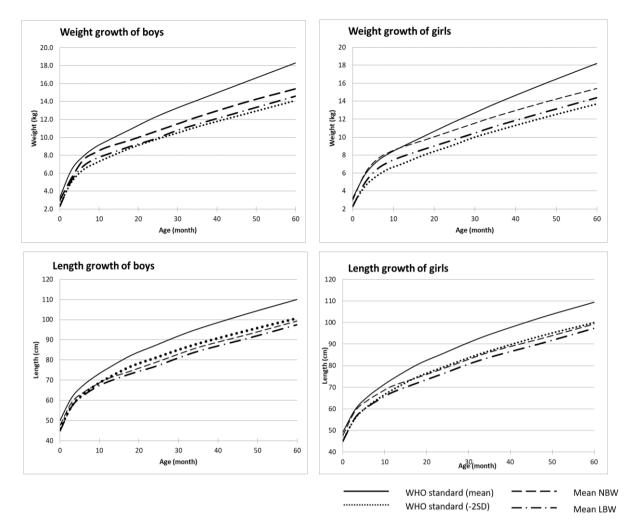


Figure 3. Mean weight and height of low birth weight and normal birth weight children compared to WHO child standard growth. The differences in weight and length growth with history of NBW and LBW (GEE; sig at p < 0.001) stratified by gender.

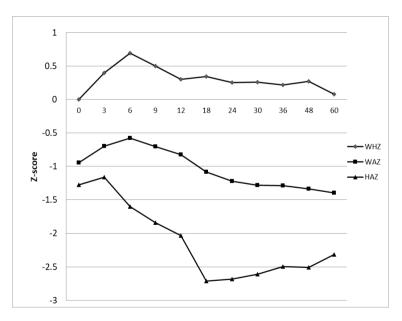


Figure 4. Mean z-score (Weight for Height Z-score (WHZ), Height for Age Z-score (HAZ), and Weight for Age Z-score (WAZ)) by age for total population, relative to 2006 WHO growth standards (0–60 months).

circumference of mother, exclusive breast feeding at age 3 and complimentary feeding practise (protein) at age 0-6 and 6-24 months significantly proven as the risk factors of stunting for children under 5 years.

The multivariable evaluation of predictors by applied the GEE show that only age, birth weight and length, sex, and source of drinking water were risk factors for shortness regarded as 'stunting'. With univariable analysis, it appears that non-breastfeeding was protective against

A		Wasting (WHZ) (%)		Shortness (HAZ) (%)			Underweight (WAZ) (%)		
Age (months)	Ν	<-2	<-3	Ν	<-2	<-3	Ν	<-2	<-3
(monuis)		z-score	z-score		z-score	z-score		z-score	z-score
0	2947	5.1	1	3317	29	7.9	3317	8.6	0.6
3	3261	5.3	1.5	3286	26.3	9.3	3295	11.5	2
6	3215	2.1	0.5	3235	37.7	14.6	3287	8.9	1.7
9	3234	1.8	0.3	3239	44.6	17	3295	10.2	1.5
12	3189	3.1	0.4	3188	44.9	20.8	3265	12.3	2
18	3161	2.1	0.3	3148	68.8	44.1	3201	16.3	3
24	3141	2.2	0.3	3132	75.8	38.4	3179	19.6	3.2
30	3072	1.5	0.1	3064	72.1	30.5	3119	20.1	2.2
36	3058	1.4	0.1	3059	68.9	25.5	3105	18.1	2.5
48	3059	1.1	1.1	3057	70.5	27.5	3073	18.3	1.9
60	2989	1.1	0.1	2986	60.6	25.1	3006	20.8	1.7

Table 2. Prevalence of wasting, shortness, and underweight in children by degree and age distribution (0-60 months)

[†]Degree of wasting is determined by Weight for Height Z-score (WHZ).

[‡]Degree of shortness is determined by Height for Age Z-score (HAZ).

[§]Degree of underweight is determined by Weight for Age Z-score (WAZ)

stunting, but this is not evident on multivariable analysis (Table 3).

Table 4 shows that no association was found between birth weight and either the G2914C IRS-1 or C1245T IGF-1 gene polymorphisms.

DISCUSSION

This longitudinal study of physical growth during the first five years of postnatal life in a rural area in West Java is of a kind not commonly available in Indonesia, although nutritional surveys which could be the basis of prospective studies often are. In the current study, a difference in growth (weight and length) has been observed between LBW and NBW infants. LBW infants grow with a similar velocity, and are even like the velocities targeted in WHO standard growth charts. Despite the similar velocities, the mean weight and length both in boys and girls in this population are very low. The weight growth of LBW children is just above the -2SD line. The heights are even more discrepant; the length growth of NBW girls coincides with the -2SD line, while the boys' is below it. Insofar as stature is genetically inherited or epigenetically determined, whether intergenerationally or during gestation, parental anthropometrics are risk factors for SGA (small for gestational age) offspring.²² Despite all of those factors, the failure to catch up leads to stunting,²³ Early hormonal intervention, at age 2, as proposed by Lee at al may be misplaced unless the functional relevance and pathogenesis are understood will save their future.²⁴ Another future implication is that proper catch-up growth yields taller young adults with better cognition and less prevalence of disability,25 although the influence of childhood and adolescent lifestyles should not be discounted.²⁶ Urbanisation and socioeconomic transition may increase the risk of metabolic and cardiovascular diseases among adults who were SGA and preterm infants who underwent catch-up growth.27

Among studies that have measured infant growth velocity, comparison is difficult due to lack of standardisation regarding calculation method,²⁸ but interpretations of such studies are nonetheless important to make. In a cohort study in Brazil, LBW was associated with a reduction in BMI and waist circumference at 8 years old, whereas rapid weight gain at 6 months resulted in increased BMI and waist circumference at the same age.²⁹

Stunting is expected to be high in this LBW population. Stunting at 48 and 60 months in LBW infants in this study is in line with that in other cohorts, including a pooled estimate from 19 cohort studies.^{30,31} However, comparisons should be made with caution because there seems to be variability in the infant growth chart with respect to ethnicity.³² The comparison with WHO growth standards showed clearly how small Tanjungsari's children were, especially since the WHO growth standard used in this study was 2006 and different from the 1990's standard.³³

The highest prevalence of wasting in our study was at 3 months (5.3%). It was lower than the prevalence reported by Helen Keller International using the NCHS (National Centre for Health Statistics) reference population among 'under-fives' (children under 5 years) in rural West Java in 1999-2003 (7%-10%) and much lower than in the Indonesia from 1997 (12.9%). The differences cannot be explained using different reference populations, which suggests that wasting might not be a major public health problem in Indonesia. The results of a study in India using WHO growth reference standards were in line with those of the present study.¹² The highest prevalence of being underweight in our study was at age 60 months, it was also lower (for underweight and severely underweight children, 20.8% and 1.7%, respectively) compared with national data from 1995 (31.6%) and rural West Java data from 1999-2003 (from 39% to 32% respectively).34 Compared with a retrospective birth cohort in Lambrene, Gabon, where underweight was more prevalent.³⁵

More than one-third of infants were moderately 'stunted' at birth, which deserves special attention because stunting reflects chronic malnutrition-and, if associated with LBW, a risk factor for health in later life. Equally, it represents sensing during early intrauterine life of a vulnerable environment in which to be born. 'Stunting' prevalence in our study ranged from 26.3% at 3 months to 75.8% at 24 months. The prevalence reported by Helen Keller International in rural West Java in 1999–2003 and

Table 3. Regression model for shortness and its predictors

	OR (95	
	Univariable	Multivariable
Age of measurements		
0 month	0.27 (0.24–0.29)**	0.25 (0.21–0.30)**
3 months	$0.23 \left(0.21 {-} 0.26\right)^{**}$	$0.18 (0.15 - 0.22)^{**}$
6 months	$0.39 (0.36 - 0.43)^{**}$	$0.36(0.30-0.43)^{**}$
9 months	$0.52(0.48-0.57)^{**}$	0.47 (0.40–0.56)**
12 months	0.53 (0.49–0.58)**	0.47 (0.40–0.56)**
18 months	1.43 (1.34–1.57)**	1.55 (1.31–1.85)**
24 months	2.04 (1.86–2.23)**	2.28 (1.90-2.72)**
30 months	1.68 (1.54–1.82)**	1.79 (1.51–2.11)**
36 months	1.44 (1.33–1.56)**	1.35 (1.16–1.57)**
48 months	1.55 (1.45–1.66)**	1.51 (1.32–1.73)**
60 months	1	1
Child factors		
Gender		
Girls	$0.77 \left(0.71 {-} 0.84\right)^{**}$	0.69 (0.58–0.82)**
Boys	1	1
Birth weight	±	1
Low birth weight ^{\dagger}	2.38 (2.05–2.75)**	1.85 (1.36–2.53)**
Normal birth weight	1	1.85 (1.50–2.55)
Birth length ^{††}	1	1
Short	2.42 (2.23-2.62)**	2.44 (2.03-2.92)**
Normal		
Birth interval	1	1
	0.00 (0.76, 1.05)	
≤ 24 months	0.90 (0.76–1.05)	
>24 months	1	
Environmental factors		
Source of drinking water		
Unimproved	1.42 (1.31–1.54) **	1.29 (1.08–1.54)**
Improved	1	1
Maternal factors		
Mother's formal education		
<6 years	1.25 (1.14–1.36) **	1.19 (0.95–1.45)
≥6 years	1	1
Mother's MUAC		
<22 cm	1.43 (1.12–1.83)**	1.03(0.75-1.39)
≥22 cm	1	1
Mothers' age		
High risk [‡]	0.99 (0.91–1.08)	
Normal risk	1	
Post-delivery factors		
Exclusive breastfeeding 0-3 months		
Liquid food	$0.80(0.65{-}0.98)$ *	1.15 (0.37-3.60)
Solid food	0.91 (0.77–1.09)	0.86 (0.60–1.26)
Exclusive breastfeeding	1	1
Exclusive breastfeeding 0-6 months	1	1
	0.07 (0.50, 1.80)	
Liquid food Solid food	0.97 (0.50 - 1.89) 0.95 (0.50 - 1.78)	
Solid food Evolutive breastfooding	0.95 (0.50–1.78)	
Exclusive breastfeeding	1	
Additional protein intake 0- 6 months		1.00 (1.1.47)
No	1.30 (1.19–1.42)**	1.20 (1-1.45)
Yes	1	1
Additional protein intake 6-24 months		
No	$1.45 (1.11 - 1.91)^{**}$	1.34 (0.77–2.32)
Yes	1	1

[†]Low birth weight <2500 gr.

^{††}Short: birth length <38 cm.

[‡]Age ≤ 20 years; age ≥ 35 years.

p*<0.05; *p*<0.01.

national data from 1997 and 2002 for children under 5 years was 42.2%.^{34,36} National data obtained from the World Bank in 1995 indicated a 47% prevalence of stunting in preschool children.³⁷ These numbers should be compared with caution because they were obtained using different reference populations; the previous studies used

NCHS data as their reference. Stunting prevalence was approximately 10% higher when using the WHO reference instead of NCHS.³⁶ Later studies in Guatemala and India used WHO reference standards, and gross comparison at ages 0–5 months (20% in India and 22% in Guatemala) and 18–23 months (58% in India and 54% in

	LBW n=89	NBW n=86	χ^2	р	Odds ratio (95% CI)
IRS-1gene					
Allele 2914 C mutant	3	3	0.002	0.966	0.97 (0.15-6.20)
Allele 2914 G wildtype	86	83			
IGF-1gene					
Allele -1245T mutant	61	54	0.642	0.423	1.29 (0.66-2.53)
Allele -1245C wildtype	28	32			

Table 4. Risk analysis for LBW by G2914C IRS-1 gene and -C1245T IGF-1 gene polymorphisms

Guatemala) revealed higher stunting prevalence than in ours.¹² Another study in Gabon yielded similar results to our study.³⁸ 'Stunting' in our cohort did not reduce with age. A later study in Indonesia has shown that 'stunting' remains a major finding in Indonesia.³⁹ The questions are why and how does it matter? The Tanjungsari study may provide some insight from its longitudinal design.

In our study WHZ decreased at age 9 months, later than reported elsewhere.⁴⁰ This correlated with earlier stunting at 3 months. A study conducted in the Philippines reported even earlier stunting onset: 2 months of age.² A multicentre study demonstrated that the HAZ graph decreases at a later age: at age 8 months in Ethiopia and Vietnam, and at age 7 months in India and Peru.³⁸ A bigger study conducted in 54 countries supported our results, finding a rapid decline in HAZ from birth until 24 months, with a similar trend in South Asian countries (Bangladesh, Cambodia, India, and Nepal).40 Infant length deviated from the reference from the first months of life, and marked deterioration continued during the first year and reached a nadir HAZ around 18-24 months of age; from there, it plateaued at a lower level without any major improvement in subsequent months.36,40 The age at which infants began to be underweight in the present study (6 months) was 3 months later than that in a previous study, but rapid decline did not occur.⁴⁰ The results of the two studies were therefore consistent. In another study conducted in Indonesia, growth faltering was observed to have begun at age 3-4 months.^{41,42} Growth deceleration before a child reaches 2 years of age has also been observed in a study performed in Egypt, Kenya, and Mexico.⁴² Infants born with low birth weight consistently have lower WHZ, WAZ, and HAZ compared with their normal birth weight peers from birth to 60 months old, suggesting that nutritional status at birth can predict children's well-being in the first 5 years of life.

'Stunting' is a chronic consequence of complex interactions between household, socioeconomic, and cultural influences on a child's nutritional intake.⁷ The prevalence of presumably pathological shortness or stunting in the present study was at a critical point (\geq 40%) for child development.⁴⁴ The findings are similar to previous studies in some 137 developing countries, in Metro Cebu Philippines, and in Nigeria, namely that limited maternal education, low birth weight and adverse environments are the major contributors.^{2,,43,45} In Bangladesh, too, children born of poorly educated mothers, exposed to unsafe water, and SGA had a higher risk of faltered growth during their first 5 years.⁴⁶ In the Philippines and the present study, did not influence birth anthropometry.⁴³ Inadequate of breastfeeding practice and the using of unimproved source of drinking water also highly contributed in the increasing stunting prevalence in this study. Low birth weight is a risk factor for stunting, but it can be fixed by appropriate breastfeeding practise.

Adding complementary liquid food during the first 3 months prevented stunting. Most of the liquid foods given to the children were formula milk. It was common in the rural area to put women behind during meals. The best of the meal was usually, and still, given to the father and then children. The low quality of food leads to anaemia and then low quality of breast milk. Considering that the habit of prioritizing men during meal is still practiced, then the promotion of exclusive breast feeding should be accompanied by educating the family on the importance of maternal nutrition. The next fact was supporting this condition.

In this study we did not consider the vulnerabilities of children such as diarrhoea, respiratory tract infections, prolonged illness, and loss of appetite as potential risk factors for stunting, but these are well-known to be involved in linear growth. This was a limitation of the present study which might have allowed a more reliable differentiation of the different forms of shortness.

Arends et al reported that polymorphisms of the IGF-1 gene had clinical relevance for short stature.⁴⁷ Although the allele type they investigated was not the same as that in the present study, Arends et al considered that there was a transmission disequilibrium of the 191-allele IGF-1 mutant allele in their subject population, which resulted in lower IGF-1 levels in the blood, lower birth weight, shorter birth length, and smaller head circumference; similar results were found in a study by Vaessen et al.^{47,48} Our study of Tanjungsari residents discovered transmission disequilibrium of the wild-type allele, but no relevance to birth weight, growth restriction, or nutritional state disturbance at age 5 years was discovered.

A 2002 study by Bezerra et al of IRS-1 gene polymorphisms reported that birth weight was lower in new-borns with the Gly972Arg polymorphism in IRS-1 as compared with control subjects (3141±31.8 vs 3373±80.3 g, p=0.008).⁴² In the Tanjungsari cohort study, it was found that this polymorphism was more prevalent in new-borns with a birth weight <3000 g (p=0.041). The small sample size could be the reason for this inconsistent result.

The present study provides data that may be of importance to clinicians and public health providers, principally because such information contributes to developing infant and child growth charts for Indonesian settings that might be more accurate in the local context than general global charts.³⁶ This study also involved and empowered

female community health volunteers, which is key to strengthening the healthcare system in rural settings.^{49,50}

Conclusions and recommendations

A community-based longitudinal study that has followed child growth from birth to 60 months has given insight into the later outcomes of LBW. There is a critical period in child malnutrition between 0-18 months. Birth weight is a determinant of postnatal growth in the first years of life. Among the three traditional malnutrition indices, 'stunting', presumed to be nutritionally related shortness' is a major and prevalent finding. However, it appears to be partly an adaptive phenomenon and not in itself necessarily pathogenetic for associated health outcomes. Interventions which are encouraged by so-called 'stunting' should be informed by its complexity and not reductionist or single factor in their approach. The Tanjungsari study identifies several determinants of shortness including ones which are sociodemographic (location, age, gender), birth weight and length, environmental (the water supply), and maternal literacy. Thus, further support is given to the education of women and the role of women in community development as it pertains to child growth and development invulnerable populations.⁵¹ Further studies in the same or similar settings should investigate the quality of life of children who survive to adulthood.⁵²⁻⁵⁵

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AUTHOR DISCLOSURES

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