Chapter 1.

General introduction
Introduction
During 1990-2000, the prevalence of stunted growth and underweight in preschool children in developing countries had declined from 38% to 30% for stunted growth and 30% to 23% for underweight. Despite the corresponding decrease of 42 and 27 millions in the number of stunted and underweight children respectively, around a third of preschool children in the developing world are still suffering from malnutrition.1,2

Interestingly, however, overweight in children has also become a growing concern because overweight in childhood is known to be tracked into adulthood and is related to an increased risk for, among others, cardiovascular morbidity and mortality.3-6 Several studies have shown that the prevalence of overweight and obesity are increasing both in developed and developing countries.7,8 Although in the least developed countries problems of malnutrition are still outweighing those of overweight and obesity, in the countries that undergo nutrition transition these problems seem to be gradually replacing those of malnutrition.9,10

This problem of under- or overnutrition becomes more complicated because recent epidemiological studies showed that a population can suffer from both problems at the same time, resulting in a double burden of under- and overnutrition. Stunted growth, a hallmark of chronic early childhood malnutrition, is associated with an increased risk of obesity and chronic degenerative diseases later in life.11,12

Malnutrition and Growth Impairment in Children in the Developing Countries
Malnutrition in infants and young children is a serious health problem because around 56% of all child deaths in the developing worlds are attributable to malnutrition’s potentiating effect on childhood infection. Children suffering from malnutrition have increased risk for severe diarrhoeal episodes and heightened susceptibility to certain infectious diseases, such as diarrhea, malaria, measles and pneumonia.13,14 Furthermore, although there is an association between increasing severity of anthropometric deficits and mortality, around 83% of malnutrition related mortality is associated with mild to moderate malnutrition instead of the severe one.14 Worse still, it has been shown that malnutrition early in childhood is associated with poor performance in intellectual functioning later in childhood, adolescence, and adulthood.15-17

Growth impairment is the result of a wide range of factors with complex interaction, namely socioeconomic, demographic and genetic factors.18 In underprivileged population, however, most of the growth deficits occurring in the community are related to inadequate food intake and/or severe and repeated infections. Perinatal factors related to maternal
undernutrition or disease, such as low birth weight, may also contribute.\textsuperscript{19-21} A common feature of the growth pattern in infants of the developing world is acceptable growth during the first 4 to 6 months followed by a progressive decline in growth rate relative to the affluent population. This decline in growth rate usually begins at the age of 6 months up to the age of 12 to 24 months or perhaps longer.\textsuperscript{22}

**Obesity and the Nutrition Transition in the Developing Countries**

Overweight or obesity has become a global epidemic and is still increasing both in developed and developing countries. Although the prevalence of overweight and obesity in less developed countries have not reached the level observed in many developed countries, an upward trend is clearly observed.\textsuperscript{9,10} This is a serious health problem because overweight in adolescents is associated with 30 to 80\% excess of mortality at middle age.\textsuperscript{4,14,23} The risk is especially high for mortality associated with cardiovascular diseases.\textsuperscript{23}

Overweight in childhood and adolescence is associated with increased risk of insulin resistance, dyslipidemia, and elevated blood pressure later in life.\textsuperscript{24,25} Increased left ventricular mass, a risk factor for cardiovascular disease in adulthood, is already present in obese children.\textsuperscript{26} These above arguments together with the knowledge that childhood obesity is indeed tracked into adulthood make the need for prevention of childhood obesity very important.\textsuperscript{6,27,28}

The emergence of obesity epidemic in the developing countries is associated with “the nutrition transition”. The world is moving toward higher fat and refined carbohydrate diet. Major dietary changes include a large increase in the consumption of animal food products and a fall in the intake of cereal and fiber. Asian countries, with a diet that is traditionally high in carbohydrate and low in fat, have shown a decline in the proportion of energy obtained from complex carbohydrate along with an increase in the proportion of fat.\textsuperscript{29-31}

A major change in economic structure associated with the nutrition transition is the result of the change from agrarian economy to industrialization. This change in economic structure together with the use of electricity, is associated with shifts toward occupation require less energy and alteration in the use of leisure time. In the past, leisure activities in children often mean active play, while today, it may mean a quite sedentary activity such as watching television or playing computer games.\textsuperscript{29}

In the developed countries, poor people have a higher risk for obesity. On the contrary, in the developing countries, wealthier segments of the population are at increased risk. However, recent studies of adult population in developing countries showed that obesity
could no longer be considered a disease of the rich. The burden of obesity in particular developing country tends to shift towards the lower socioeconomic status group of that country as its Gross National Product (GNP) increases.\textsuperscript{32,33}

One possible explanation for differences in the association between socioeconomic status and the prevalence of obesity in developed and developing countries is the influence of socioeconomic status on the people’s lifestyle, i.e. diet and physical activity. For example, with respect to the food consumption pattern, in the developing countries, richer people have better access to find and consume more meat and energy dense food which are much more expensive than other foods such as vegetables. On the other hand, in the developed countries, people with a higher socioeconomic status who are usually more educated and more aware of the cardiovascular risk, consume and prefer more vegetables and fruits.\textsuperscript{33}

**Early Origin of Adult Diseases**

The emergence of cardiovascular disease (CVD) epidemic in the developing countries has not yet attracted adequate public health awareness. In contrast to developed countries, it has not been widely realized that developing countries contribute a greater share to the global burden of CVD. The World Health Report 1999 noted, in 1998, 85\% of the global burden of CVD arose from the low and middle income countries.\textsuperscript{34}

It is estimated that, from 1990 to 2020, the CVD mortality in developing countries will increase 124\% for men and 107\% for women. This is much higher than the estimation in developed countries, which are 78\% for men and 56\% for women. Furthermore, CVD deaths in developing countries occur at earlier age. For example, in 1990, the proportion of CVD deaths at the age of less than 70 years was 26.5\% in developed and 46.7\% in developing countries.\textsuperscript{34}

The emergence of obesity and cardiovascular diseases epidemic in the developing countries seems not only to be associated with the change in lifestyle, i.e. “too much eating” and “too little exercises”. There is increasing evidence that malnutrition early in life is one of the additional risk factors for the development of obesity and other chronic diseases later in life.\textsuperscript{11,35}

Stunted growth, a marker of chronic early childhood malnutrition, is associated with increased risk for obesity.\textsuperscript{36} Compared to non-stunted adults, stunted individuals had higher fasting glucose, higher triglycerides, higher low-density lipoprotein cholesterol, higher total cholesterol and higher blood pressure.\textsuperscript{36-38} In a recent report on a 10-year follow-up of children who were stunted during the first 2 years of life, it was observed that children who
were stunted at young age had significantly higher systolic blood pressure at the age of 11-12 year.\textsuperscript{39}

It has been widely recognized that malnutrition during fetal life is a risk factor for the development of cardiovascular disease in adulthood.\textsuperscript{40} A systematic review of published papers reported a 2 mmHg decrease in systolic blood pressure per 1 kg increase in birth weight.\textsuperscript{41} Similar findings are observed in both developing and developed countries.\textsuperscript{42-45} Furthermore, birth weight is also inversely associated with later body mass index (BMI), abdominal fat, blood lipid concentration as well as the development of type 2 diabetes and coronary heart disease \textsuperscript{40;46-49}.

While the influence of prenatal growth on cardiovascular risk later in life is fairly clear, the effect of post-natal growth on risk of obesity and cardiovascular disease is still controversial. While most studies reported a direct association between rapid post-natal growth and increased risk of obesity and cardiovascular disease,\textsuperscript{41;44;50-53} some studies observed higher risk for developing hypertension, hyperlipidemia, insulin resistance and coronary heart diseases in children who gained less weight during the first two years of life.\textsuperscript{42;43;47;54}

If the latter were true, these long term effects of early life malnutrition, in addition to the already well-known immediate effects of malnutrition, will have enormous economic and social implications for the developing countries. This burden will be particularly heavy, not only due to most of the undernourished children reside in such countries, but also because there may not be enough resources to deal with the chronic degenerative diseases later on.

\textbf{Indonesia: a Country in Transition}

The Republic of Indonesia is the largest archipelago in the world, consisting of five main and approximately 17,500 smaller islands. The islands are spread between the Asian continent and Australia, and between the Pacific and the Indian oceans. The country has a total area of around 2.9 million km square with only around 50% land territory. Indonesia is a tropical country with annual mean temperatures of 21-33\degree C.\textsuperscript{55}

Indonesia has the world’s fourth largest population, i.e. after China, India and the United States. Based on the census of 2000, the total population of Indonesia was around 206 millions, and was estimated to be almost 220 million in 2003. The annual population growth rate declined from 2.1\% in 1970-1990 to 1.4\% in 1990-2003. In 2003, the proportion of children under-5-year-old was around 10\% of the total population, while that of under-15-year-old was 29\%.\textsuperscript{56;57}

The per capita GDP (Gross Domestic Product) was approximately 695 US Dollars in
2001, with an annual growth rate of around 3.2% in 1990-1999. However, this economic growth was not associated with a decline in the proportion of people living in income poverty. At the same period of time, the proportion of poor people increased from 15 to 18%.\textsuperscript{58} The World Bank data showed that, while in the average 80.4% of all households had electricity, only 35.0% of all households from the poorest quintile of the population had electricity compared to 99.9% of all households from the richest quintile. Similar to electricity, in the average 18.3% of all households had television, 10.8% in the poorest quintile vs. 95.8% in the richest quintile.\textsuperscript{59}

Similarly, the development of health and welfare in Indonesia did not reach everyone. Despite a decline in the average Infant Mortality Rate (IMR) from 128 per live births in 1960 to 41 in 2003, Indonesia was among the 13 countries in the world showing an increased gap of IMR between the wealthiest and the poorest segments of the population.\textsuperscript{56,58} It was observed that in 1997, the average IMR of the rural population was 58.0 while that of the urban was 35.7. A detailed look, however, showed that the IMR of the poorest quintile in urban area was 46.5 vs. 25.6 in the richest quintile. The corresponding number for the rural area was 78.9 vs. 18.5.\textsuperscript{59}

Documentation on the risk of cardiovascular diseases in children in Indonesia is not easily found. A recent study in an adult population reported increasing prevalence of elevated blood pressure from 22.1% (95% CI: 20.7-23.6) in 2001 to 26.7% (95% CI: 25.2-28.3) in 2005. In the same period, the prevalence of overweight and obesity also increased from 8.5% (95% CI: 7.3-9.8) to 14.2 (95% CI: 12.7-15.9). This study also observed that the prevalence of both elevated blood pressure and obesity were highest in the urban and the richest tertile of the rural population. Furthermore, in that area, non-communicable diseases (NCD) are responsible for 52% of total deaths, predominantly cerebrovascular and cardiovascular diseases.\textsuperscript{60}

**Aim of the Thesis**

The aim of this thesis is to assess problems of under- and overnutrition in children in Indonesia. The studies focus on the association of socioeconomic status and birth weight with malnutrition, stunted growth, overweight and obesity, as well as with the risk of elevated blood pressure in prepubertal children.

The questions asked in this thesis are:

1. How does socioeconomic status influence the prevalence of malnutrition, stunted growth, overweight and obesity?
2. How is the tracking for underweight, overweight, and obesity?
3. How is the interaction between birth-weight, stature, body mass index (BMI) and socioeconomic status on blood pressure of prepubertal children?

Outline of The Thesis
Chapter 2  Comparison on the prevalence of malnutrition in under-two-year-old children in two rural areas in Indonesia in association with socioeconomic status and other risk factors, e.g. diet and breastfeeding status.
Chapter 3  Assessment on the association of socioeconomic status, i.e. rural, poor urban and nonpoor urban, on the prevalence of stunted growth and obesity in prepubertal children.
Chapter 4  Assessment on the influence of rural or urban residences on the association between low weight at birth and stunted growth or overweight in prepubertal children.
Chapter 5  Assessment on the tracking for underweight, overweight and obesity as urban prepubertal children grew into adolescence. This chapter also presents the trend on the prevalence of underweight, overweight and obesity in prepubertal children in an urban area in Indonesia.
Chapter 6  Assessment on the association between socioeconomic status, i.e. rural, poor urban and nonpoor urban, and blood pressure in prepubertal children.
Chapter 7  An extended version of the project described in Chapter 6, which besides socioeconomic status, also describes the influence of stature, BMI and birth weight on blood pressure.
Chapter 8  General Discussion reviewing all data and presenting suggestions for future research.

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Chapter 2.

Role of breastfeeding in protecting children from malnutrition: a comparative study of nutritional status in children under-two-years of age in two districts in Indonesia

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ABSTRACT

Background. Prevalence of childhood malnutrition in Indonesia is quite high with uneven distribution throughout the country.

Methods. This study is a cross-sectional survey comparing pattern and determinants of nutritional status of children under-two-years-old between two districts in Indonesia, i.e. Belu, East Nusa Tenggara and Purworejo, Central Java. Children in each study area were randomly selected using a two-stage cluster and proportionate to estimate size sampling method. A total of 1427 children (711 of Belu and 716 of Purworejo) underwent interviews on households’ characteristics, feeding pattern, and anthropometric measurements.

Results. The mean weight-for-age z-scores of children in Belu were consistently below the NCHS-WHO reference curve and those of Purworejo. By the age of 6 to 23 months, children in both populations had significantly lower risk of being underweight if they were still breastfed, OR=0.43 (95%CI 0.29;0.62). Stratified data showed that the protective effect of breastfeeding was only statistically significant in Belu, OR=0.44 (95%CI 0.27;0.71), and not in Purworejo, OR=0.79 (95%CI 0.36;1.72).

Conclusions. The protective effect of breastmilk is significant in Belu where intake of animal source protein is significantly lower. We concluded that breastfed children have a lower risk of being undernourished.

Key words: breastfeeding, malnutrition, nutritional status, poorer area.
INTRODUCTION

Malnutrition, especially undernutrition, in infants and young children is one of the most serious health problems in the developing world. Apart from increasing risk of morbidity and mortality which contributes to more than half of the underfive children death in the developing countries, undernutrition in childhood is also related to concurrent and possibly later delayed intellectual and motor development, as well as deficit in growth and other adverse health effects.1-4

According to UNICEF, in the developing countries, 31% of under-five children suffer from being underweight.1 As in many developing countries, malnutrition is also a problem in Indonesia. The prevalence of underfive children suffering from underweight, i.e. a weight below -2 standard deviations of the mean of the most widely used reference population, the NCHS-WHO, was 42% in 1992, decreasing to 36% and 34% in 1995 and 1997, respectively. Although the overall prevalence has been decreasing, it is difficult to say whether this is true for all areas in Indonesia. Data shows that there are differences in the prevalence of malnutrition among different areas of Indonesia, which may be due to the difference in the development and prosperity among specific areas.5-8

Assuming that people in East Nusa Tenggara had a lower development and prosperity compared to almost the rest of Indonesia, this study intended to see the variability of nutritional status of children in Belu, East Nusa Tenggara, and compared it to those in Purworejo, Central Java, an area with presumably better development and prosperity. It also intended to see whether feeding pattern, especially breastfeeding status, had any influence on nutritional status of children in those areas.

METHODS

Study site and population

Belu is located on Timor island, one of the islands in East Nusa Tenggara, a province located in the southeastern part of Indonesia, while Purworejo is located in Central Java, one of the provinces in Java.

Study design and time frame

To assess the nutritional status of children under two years of age in both areas, we conducted cross-sectional studies on a random sample of each population. The study was part of a multicenter study on complementary feeding, carried out between September 1997 - February 1998. The study was approved by the ethical committee of Gadjah Mada University, Yogyakarta, Indonesia.
**Sampling strategy**

The study took advantage of a large surveillance program performed by Community Health and Nutrition Research Laboratory (CHN-RL). In the CHN-RL surveillance studies, a two-stage cluster sampling method was used to select a sample of households representative of each district. The first stage was to select clusters, which were the standard statistical enumeration areas or "wilcah" developed by the Central Bureau of Statistics for the population census in 1990. The sample was chosen with probability proportionate to estimated size (PPES) sampling methods, after the clusters were listed systematically into urban and rural areas. Twenty percent of "wilcah" in the district were selected. In the second stage, to get representative sample for the surveillance program, approximately 138 households from every "wilcah" were selected as the sampling units.

Children less than 2 years old listed in the CHN-RL households sample, were divided into 7 age groups, i.e. 0-1, 2-3, 4-5, 6-8, 9-11, 12-17 and 18-23 months. The reason for this division laid on the protocol for the complementary feeding study, i.e. based on types, composition and consistency of foods usually consumed by each age group. About 100 children from each age group were included. An equal number of children for every age group were randomly selected from each "wilcah".

**Data collection**

Information was collected using a pre-coded questionnaire form. Training of interviewers before the actual data collection began consisted of field practice, testing of questionnaires, cross data editing and discussions. All interviewers spoke the native languages. Data was collected from the children's caregivers, preferably mothers. Information on birth date was ascertained by the child's birth certificate or the child's growth chart at the local village health post.

Data on breastfeeding consisted of breastfeeding status, frequency of breastfeeding during the last 24 hours, and age of weaning if the child was no longer breastfed. Data on complementary feeding was collected by a 24-hour dietary recall. This dietary recall data was analyzed using International Minilist on WorldFood Assessment Program (University of California, Berkeley, Ca., USA).

Training for standardization of anthropometric measurements, followed by field practice and testing were performed prior to data collection. The children were twice weighted naked or in light underwear which was later subtracted from the total weight, to the nearest 0.1 kg, using a standard hanging Salter scale (UK) and a Seca digital scale.
(Germany). All equipment was standardized to WHO recommendations. The mother’s height was measured with a stadiometer to the nearest 0.1 cm, while her weight was measured with a Seca digital scale (Germany) to the nearest 0.1 kg. Due to the tropical weather, light clothing was the custom in the area.

**Data entry and analysis**

Field supervisors monitored the quality and completeness of the data collection. Ten percents of the households were randomly selected for spot-checking and rechecking of data contents. Field supervisors also edited and cross-edited questionnaire forms, and in case of incomplete data, further visit to the household were conducted. Data were entered using dSurvey (dSurvey, a survey research system, 1989). Double data entry was performed on 10% randomly selected data.

Anthropometric index (weight-for-age) was converted to z-scores of the WHO-NCHS reference population using EPINUT (Epi Info 6, Center for Disease Control, USA). Biologically implausible values, such as z-scores less than -6.00 or larger than +6.00 were directly excluded from the analysis. Values that were most likely to represent errors, i.e. 4 z-score units outside the observed mean z-score for every age in completed month, were also excluded from the analysis (flexible exclusion range). Descriptive, univariate and multivariate statistical analyses were performed with SPSS for Windows (version 7.5, SPSS Inc. Chicago, Ill, USA).

**RESULTS**

Of the 1434 eligible children from both districts, i.e. 717 from each district, only 1427 children (711 children from Belu and 716 children from Purworejo) underwent anthropometric measurements. The households’ characteristics of the study sample can be seen in Table 1.

The pattern of anthropometric indices and the prevalence of undernutrition (z-score less than -2 with respect to the NCHS-WHO reference population) to age can be seen in Figure 1. The weight-for-age faltering in both districts began in the second and third month of life up to the end of the second year. The mean weight-for-age-indices of children in Belu were consistently below the NCHS-WHO weight-for age reference curve. They were also always below those of Purworejo, with higher deviations after 4 to 5 months of life (Figure 1).

The prevalence of underweight (weight-for-age z-score less than -2) increased sharply in both districts after 6 to 8 months of life. The increase was much larger in Belu than in
Breastfeeding was very common in both population, the ever breastfed rates in Belu and Purworejo were 99.2% and 99.0% respectively, indicating that virtually all children in both samples received breast milk some time during their life. The proportion of the population ever breastfed was relatively constant in the different age groups.

Breastfeeding rate in the second year of life was significantly higher in Purworejo than in Belu ($p<0.001$), difference in proportion: 24.2% (95% CI:15.9;32.5%). Animal-product foods were rarely given. In children not breastfed, only 65% children in Belu and 87.2% children in Purworejo received animal source protein at the day of the 24-hour recall. Median amount in intake of animal source protein (in percentage of recommended dietary allowance, RDA) were higher in Purworejo than in Belu, i.e. 53.5% vs. 20.1% ($p=0.003$, Mann-Whitney test).

Table 2 showed that the difference in the prevalence of underweight (weight-for-age z-scores less than -2 NCHS-WHO growth reference curve) between Belu and Purworejo was significant in children between 6 to 23 months of age. In this age group, children still breastfed had a significantly lower risk of having z-score less than -2, OR:0.43(95%CI: 0.29;0.62). This association was still significant after adjusting for some other variables (Table 3).

Data stratified into each district showed that the protective effect of breastfeeding was only statistically significant in Belu, OR: 0.44 (95%CI: 0.27;0.71), and not in Purworejo, OR:0.79(95%CI: 0.36;1.72). Further analysis of feeding pattern showed that not breastfed children in Belu received significantly lower amount of animal source protein (Table 4).

**DISCUSSION**

The strength of our study is that it includes a presumably representative sample of the study population and collects a relatively good quality of data, especially the anthropometric measurement. As this is a cross sectional survey, it should be noted, however, that it is not appropriate to imply causal relationships.

Our findings suggest that the people living in Belu enjoy a lower standard of health and welfare compared to those living in Purworejo, i.e. parents of children in Belu attained lower levels of education than those in Purworejo; there are more children in Belu who have deceased sisters or brothers; there are more people live in a house and there are more under-five children in one house in Belu than that in Purworejo.

As about 99% of children in both studies had been breastfed, comparing their growth to NCHS-WHO reference curve may not be the most appropriate method. The growth curve
of breastfed infants living under favorable conditions (the breastfed pooled data set) has been shown to deviate from NCHS-WHO reference curve. During the first two or three months of life, breastfed infants tend to grow better relative to the NCHS-WHO reference curve, but then, their growth decline, and it will cross the WHO-NCHS curve at 5 or 6 months of age. At 12 months, the mean weight-for-age z-score of this data set is -0.6.9

The growth pattern of infants in Belu and Purworejo is comparable to the breastfed-pooled data set, but with different magnitudes. In Purworejo, in the first 2 months of life, the infants have higher growth rate compared to the NCHS-WHO reference population, then it declines. Weight-for-age z-scores cross the NCHS-WHO curve at the 4th - 5th month of life. The faltering continues, and in the last quarter of the first year, the mean(SD) weight-for-age z-score is -0.99(1.0). The mean weight-for age-indices of children in Belu were consistently below the NCHS-WHO weight-for age reference curve. They were also always below those of Purworejo, with higher deviations after 4 to 5 months of life.

Compared to the data set of breastfed infants, the growth faltering in children in both Belu and Purworejo occurs earlier and in larger magnitudes, although the problem is worse in Belu. Similar patterns have been reported from other studies in Indonesia, although in those studies the magnitude of faltering is not as grave as in this study.5,8,10

Using the classification of WHO on the prevalence of underweight for global monitoring, the prevalence of underweight in the first six months of life in both populations can be classified as low, i.e. less than 10%. In the third quarter of the first year (age 6 - 8 months), the prevalence of underweight in Belu increases to moderate, i.e. between 10 to 19%, while that in Purworejo remains low. Beginning from the last quarter of the first year and throughout the second year, the prevalence of underweight in Belu is very high, i.e. more, than 30%, while that in Purworejo raises gradually from moderate to high and very high within the same time frame.11

Our data showed that although in the first 6 months of life, the mean weight-for-age z-score of infants in Belu were always below the NCHS-WHO reference curve and always below those of Purworejo, the prevalence of underweight in both populations were low. We could not find the explanation for this in our data, but it might be due to the fact that most children in this age group were still breastfed.

Breastfeeding was very common in both populations. Since the proportion of children ever breastfed is high and have similar proportions across different age groups, giving breastmilk is the norm in both population. Breastfeeding rate in the second year of life was significantly higher in Purworejo than in Belu.

By the age of 6 to 23 months, children in both population had significantly lower risk of
being underweight if they were still breastfed, OR (95%CI); 0.43(0.29; 0.62). This association is still significant after adjusting for parental level of education (mothers' and fathers'), number of people in the house, number of under-five children in the house, number of respondent's siblings, number of died siblings, mothers' age and mothers' body mass index.

Data stratified into each district showed that the protective effect of breastfeeding was only statistically significant in Belu, and not in Purworejo. This association might be partly due to the fact that not breastfed children in Belu received significantly lower amount of animal source protein. The animal source protein from breastmilk which was withheld was not replaced by appropriate foods. Duration of not given breastmilk was negatively correlated with weight-for-age z-score (correlation coefficient (r)= -0.28, p=0.001).

Other variables significantly related to the risk of underweight in these children are level of mothers' education and mother's body mass index. A SUSENAS data (Indonesia National Household Survey) in 1987 also reported the close relationship between nutritional status and level of mothers' education. The effect of mothers' body mass index might be a little complicated, it might be related to infants' birth weight, to her breast milk energy density, and to mothers' or the families' socio-economic status.

The result of this study suggests that children under two years of age in Belu have a worse problem of undernourishment than those in Purworejo. Although the pattern of growth faltering relative to the NCHS-WHO reference population shows similarities to children in Purworejo and to the growth curve of breastfed infants living under favorable conditions (the breastfed pooled data set), its magnitude is considerably higher. Breastfed children have a lower risk of being undernourished. The protective effect of breastmilk is especially significant in Belu where intake of animal source protein is significantly lower.

References


Table 1. The households’ characteristics of the study sample.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Belu</th>
<th>Purworejo</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Households’ density:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean(SD) no. of people in the house</td>
<td>6.6 (2.4)</td>
<td>5.3 (1.4)</td>
<td>p&lt;0.001^a</td>
</tr>
<tr>
<td>Mean(SD) no. of under-five-year- old children in the house</td>
<td>1.6 (0.6)</td>
<td>1.3 (0.5)</td>
<td>p&lt;0.001^a</td>
</tr>
<tr>
<td><strong>Siblings:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median (Q1;Q3) no. of the respondent’s siblings</td>
<td>3.0 (2.0;4.0)</td>
<td>2.0 (1.0;3.0)</td>
<td>p&lt;0.001^c</td>
</tr>
<tr>
<td>Percentage of respondents who had died siblings (%)</td>
<td>21.6</td>
<td>12.8</td>
<td>p&lt;0.001^c</td>
</tr>
<tr>
<td>Sibling’s mortality rate^b (%)</td>
<td>9.4</td>
<td>6.1</td>
<td></td>
</tr>
<tr>
<td><strong>Parental level of education:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mothers’ (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No formal education</td>
<td>7.7</td>
<td>2.2</td>
<td>p&lt;0.001^c</td>
</tr>
<tr>
<td>Less than 6 years education</td>
<td>23.5</td>
<td>9.5</td>
<td></td>
</tr>
<tr>
<td>6 years education</td>
<td>43.9</td>
<td>51.5</td>
<td></td>
</tr>
<tr>
<td>9 years education</td>
<td>14.3</td>
<td>18.3</td>
<td></td>
</tr>
<tr>
<td>12 years education or more</td>
<td>10.6</td>
<td>18.5</td>
<td></td>
</tr>
<tr>
<td>Fathers’ (%)</td>
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<td></td>
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<tr>
<td>No formal education</td>
<td>11.0</td>
<td>0.7</td>
<td>p&lt;0.001^c</td>
</tr>
<tr>
<td>Less than 6 years education</td>
<td>18.2</td>
<td>6.8</td>
<td></td>
</tr>
<tr>
<td>6 years education</td>
<td>38.4</td>
<td>46.2</td>
<td></td>
</tr>
<tr>
<td>9 years education</td>
<td>13.2</td>
<td>19.2</td>
<td></td>
</tr>
<tr>
<td>12 years education or more</td>
<td>19.2</td>
<td>27.1</td>
<td></td>
</tr>
<tr>
<td><strong>Other characteristics of mothers:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean(SD) age of mother (years)</td>
<td>28.2(6.1)</td>
<td>29.2(5.7)</td>
<td>p&lt;0.001^a</td>
</tr>
<tr>
<td>Mean(SD) height of mother (cm)</td>
<td>152.0(32.3)</td>
<td>147.1(19.9)</td>
<td>p=0.001^a</td>
</tr>
<tr>
<td>Mean(SD) BMI^d of mother</td>
<td>19.7(3.0)</td>
<td>21.7(2.9)</td>
<td>p=0.001^a</td>
</tr>
</tbody>
</table>

^a:t-test, ^b:mortality rate among siblings ever born to the same mother: sum of (siblings ever born-siblings alive)/ sum of siblings ever born, ^c:Mann-Whitney test, ^d:BMl= body mass index
Figure 1. Pattern of mean weight-for-age z-scores of children in Belu and Purworejo related to WHO-NCHS growth reference curve.

Table 2. Comparison of proportions of children with low weight-for-age z-scores (less than -2 WHO-NCHS growth reference curve) in Belu and Purworejo.

<table>
<thead>
<tr>
<th>Age (months)</th>
<th>Belu (%)</th>
<th>Purworejo (%)</th>
<th>Odds Ratio</th>
<th>95% CI</th>
<th>p^a</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-3</td>
<td>2.5</td>
<td>0.5</td>
<td>5.0</td>
<td>0.6; 43.4</td>
<td>p=0.2</td>
</tr>
<tr>
<td>4-5</td>
<td>6.3</td>
<td>1.8</td>
<td>3.6</td>
<td>0.7; 18.3</td>
<td>p=0.15^b</td>
</tr>
<tr>
<td>6-8</td>
<td>18.3</td>
<td>1.0</td>
<td>22.8</td>
<td>3.0; 173.8</td>
<td>p&lt;0.001</td>
</tr>
<tr>
<td>9-11</td>
<td>43.9</td>
<td>11.1</td>
<td>6.3</td>
<td>3.0; 13.1</td>
<td>p&lt;0.001</td>
</tr>
<tr>
<td>12-17</td>
<td>58.7</td>
<td>24.5</td>
<td>4.4</td>
<td>2.4; 7.9</td>
<td>p&lt;0.001</td>
</tr>
<tr>
<td>18-23</td>
<td>59.4</td>
<td>32.4</td>
<td>3.1</td>
<td>1.7; 5.4</td>
<td>p&lt;0.001</td>
</tr>
</tbody>
</table>

^a: chi-square test, ^b: Fisher’s exact test
Table 3. Odds ratio of being underweight (weight-for-age z-scores less than −2 NCHS-WHO growth reference curve) in children 6 to 23 months of age.*

<table>
<thead>
<tr>
<th>Covariates</th>
<th>Multivariate OR</th>
<th>95% CI</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Breastfeeding status</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>not breastfed</td>
<td>1.0 (referent)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>still breastfed</td>
<td>0.39</td>
<td>0.26; 0.58</td>
<td>p&lt;0.001</td>
</tr>
<tr>
<td><strong>Mother’s education</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No formal education</td>
<td>1.0 (referent)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Less than 6 years education</td>
<td>1.31</td>
<td>0.66; 2.61</td>
<td>p=0.44</td>
</tr>
<tr>
<td>6 years education</td>
<td>0.52</td>
<td>0.31; 0.89</td>
<td>p=0.02</td>
</tr>
<tr>
<td>9 years education</td>
<td>0.33</td>
<td>0.18; 0.60</td>
<td>p&lt;0.001</td>
</tr>
<tr>
<td>12 years education or more</td>
<td>0.19</td>
<td>0.09; 0.42</td>
<td>p&lt;0.001</td>
</tr>
<tr>
<td><strong>Mother’s body mass index</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(continuous variable)</td>
<td>0.93</td>
<td>0.88; 0.98</td>
<td>p=0.006</td>
</tr>
</tbody>
</table>

*0 = underweight, 1 = not underweight; from a multiple logistic regression model adjusted for: no. of people in the house, no. of under-5-year old children in the house, no. of respondent’s siblings, no. of died siblings, mothers’ age (as continuous variables) and fathers’ education (five dummy variables).

Table 4. Comparison in intake of calorie, protein and animal source protein (in percentage of recommended dietary allowance, RDA) between not breastfed children in Belu and in Purworejo.

<table>
<thead>
<tr>
<th>Median intake of (% RDA)</th>
<th>Belu</th>
<th>Purworejo</th>
<th>p&lt;sup&gt;a&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>calorie</td>
<td>88.4</td>
<td>91.6</td>
<td>p=0.98</td>
</tr>
<tr>
<td>protein</td>
<td>116.8</td>
<td>145.3</td>
<td>p=0.09</td>
</tr>
<tr>
<td>animal source protein</td>
<td>20.1</td>
<td>53.5</td>
<td>p=0.003</td>
</tr>
</tbody>
</table>

<sup>a</sup> Mann-Whitney test
Chapter 3.

Influence of socioeconomic status on the prevalence of stunted growth and obesity in prepubertal Indonesian children

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ABSTRACT
This cross-sectional study assesses the prevalence of stunting, overweight, and obesity in prepubertal children from different socioeconomic groups in Indonesia. Children from rural, poor urban, and nonpoor urban communities were studied (n = 3,010). The prevalences of stunting, wasting, overweight, and obesity were 19.3%, 5.0%, 2.7%, and 0.8%, respectively. The odds ratios (OR) for stunting, as compared with nonpoor urban children, were higher among rural children (2.92; 95% confidence interval [CI], 2.37–3.59) than among poor urban children (1.58; 95% CI, 1.18–2.13). The prevalence of wasting was not influenced by socioeconomic status. Both rural and poor urban children were significantly less likely to be overweight than were nonpoor urban children: in comparison with nonpoor urban children, the OR values were 0.19 (95% CI, 0.10–0.36) for rural and 0.13 (95% CI, 0.04–0.43) for poor urban children. Boys were more likely to be stunted or obese than girls: OR for stunting, 1.75 (95% CI, 1.44–2.12); OR for obesity, 4.07 (95% CI, 1.40–11.8). Stunted children were less likely than non-stunted children to be overweight: OR, 0.10 (95% CI, 0.03–0.43). In Indonesia, undernutrition is still related to poverty, whereas obesity is more related to prosperity.

Key words: obesity, overweight, socioeconomic status, stunted growth
INTRODUCTION

Stunted growth affects 32% of children under five years of age in the developing world [1]. The process of becoming stunted due to chronic undernutrition begins at birth, or even before, and continues during the first three years of life. The stunting that occurs during these early years cannot be restored thereafter [2]. Unfortunately, when energy intake is adequate, these stunted children are at higher risk of overweight [3]. Besides being a risk factor for adult obesity, childhood obesity is associated with chronic diseases in later life, such as hyperinsulinemia, hypertension, hyperlipidemia, type 2 diabetes mellitus, and atherosclerotic cardiovascular disease [4, 5]. Increasing levels of these chronic diseases will pose a particular burden for developing countries. They will face the double burden of infectious and poverty-related diseases (e.g., malaria, chronic undernutrition), and the emerging concerns of chronic diseases related to early malnutrition, e.g., stunting and a related likelihood of being overweight.

Indonesia, a developing country with a total population of approximately 200 million, underwent a significant improvement in standard of living (defined by higher income and improved nutrition and health care) from 1960 to 1998. The infant mortality rate decreased from 128 to 40 per 1,000 live births, while the mortality rate among children under five years of age decreased from 216 to 58 per 1,000 live births [6]. However, this new prosperity was not evenly distributed. In 1999, 27% of the population was still living below the national poverty line [7]. With a documented increase in the prevalence of obesity among urban adults in Indonesia from 4.9% in 1988 to 7.6% in 1993 [8], it is important to know the prevalence of under- and overnutrition among children from different socioeconomic levels in Indonesia.

The aim of this study was to assess the prevalence of stunting, overweight, and obesity in school-aged prepubertal children from different socioeconomic levels (rural, poor urban, and nonpoor urban) in Indonesia. The study was also intended to investigate the association between stunting and overweight or obesity in relation to socioeconomic status.

SUBJECTS AND METHODS

Study population and design

The study was performed in two adjacent areas in Central Java. Yogyakarta, an urban area, was a city with approximately 487,115 inhabitants at the time of the study. Gunung Kidul, a rural area located about 20 to 40 km from Yogyakarta, had approximately 710,691 inhabitants [9]. We chose these two study areas because of the relatively homogeneous
ethnicity of their populations. Most of the people in both areas are of Javanese descent. The altitude of both study areas is less than 500 m above sea level [9].

A cross-sectional study was conducted in school-aged prepubertal children in both areas. We randomly selected 33 of 509 public primary schools in the rural area and 37 of 172 public primary schools in the urban area. In Indonesia, it is obligatory for children to enter primary school at the age of six or seven years. Prepubertal children (under eight years old for girls and under nine years old for boys) from the first- and second-year class of every school were included. The ages of the children finally studied ranged from 6 to 7.9 years for girls and from 6 to 8.9 years for boys. This study selected prepubertal children because puberty, which normally occurs after the age of eight in girls and nine in boys, may interfere with the interpretation of measurements due to changes in body composition and differences between children in the timing of the adolescent growth spurt.

We excluded children with prominent chronic diseases, such as congenital heart disease or major thalassemia. Also excluded were children who had physical handicaps that might interfere with the measurements.

The study was performed from February to May 1999 and was approved by the ethical committee of Gadjah Mada University, Yogyakarta, Indonesia.

**Data collection**

Anthropometric data were collected by health professionals. Heights and weights were measured using the standard techniques described by the World Health Organization (WHO) [10]. Training for standardization of the measurements, followed by field practice and testing, was performed prior to data collection. All measurements were performed between 8 and 10 a.m. The children, wearing light clothing, were weighed to the nearest 0.1 kg with a Seca digital scale (Germany). Height was measured to the nearest 0.1 cm with a portable stadiometer. Height was measured with the child standing facing the fieldworker, without shoes.

Birth dates were verified by a copy of the child’s birth certificate filed at the school. Definitions of rural and urban populations were based on agricultural activities and population densities. Yogyakarta had a population density of 14,988/km², while Gunung Kidul had a population density of 498/km² [9]. In the urban area, the socioeconomic status of each child was individually deduced from his or her living environment. Children living in the urban slum area were considered poor, and those not living in this area were considered to be not poor.
Data on height-for-age and weight-for-height were converted to z-scores of the WHO/National Center for Health Statistics (NCHS) reference population using the EPINUT component of the Epi Info 6.04 package (Centers for Disease Control and Prevention, Atlanta, Ga., USA). Biologically implausible values, such as z-scores below –6.00 or above +6.00, were excluded from the analysis. Values that were most likely to represent errors, i.e., those that were 4 z-score units (standard deviations) outside the observed mean for every age in one full year, were also excluded (flexible exclusion range) [11].

Children were classified as stunted if they had height-for-age z-scores (HAZ) below -2.00, and as not stunted if they had HAZ of –2.00 or more. Children were classified as overweight if they had weight-for-height z-scores (WHZ) based on WHO-NCHS references above +2.00. Wasting or thinness was defined by weight-for-height z-scores (WHZ) below –2.00 [11].

The body-mass index (BMI) was calculated by dividing the weight in kilograms by the square of the height in meters (kg/m²). The BMI reference proposed in 2000 by the International Obesity Task Force (IOTF) was used to classify children as overweight or not overweight and as obese or not obese. According to the IOTF cutoffs for persons 18 years of age, overweight is defined by a BMI above 25 kg/m² and obesity by a BMI above 30 kg/m². The cutoff points were tabulated at the exact half-year of ages. The cutoff points for overweight ranged from BMIs of 17.34 to 18.35 in 6- to 8-year-old girls and from 17.55 to 19.10 in 6- to 9-year-old boys. For obesity, the cutoff points ranged from 19.65 to 21.57 in 6- to 8-year-old girls and from 19.78 to 22.77 in 6- to 9-year-old boys [12]. Seven classes of age at midyear in our study population were defined as follows: 6 years old (6 to < 6.25 years), 6.5 years old (6.25 to < 6.75 years), 7 years old (6.75 to < 7.25 years), 7.5 years old (7.25 to < 7.75 years), 8 years old (7.75 to < 8.25 years), 8.5 years old (8.25 to < 8.75 years), and 9 years old (8.75 years or older). The maximum ages were 8 years for girls and 9 years for boys.

**Data entry and analysis**

Data were entered and analyzed using SPSS for Windows (Version 9, SPSS, Chicago, Illinois, USA). The odds ratios for stunting, wasting, overweight, and obesity within socioeconomic and sex groupings were compared by $\chi^2$ tests. Odds ratios are presented with 95% confidence intervals (95% CI). Quantitative data, i.e., data on height-for-age z-scores (HAZ) and weight-for-height z-scores (WHZ) and BMI, were analyzed by t-tests and Pearson’s correlation.
RESULTS
We visited 70 schools to examine a total of 3,689 prepubertal children who were listed as first- and second-year students. We excluded 674 children because they were not in the required age range. Of the remaining 3015 children, four missed the measurement session and one had a HAZ and a WHZ in the flexible exclusion range.

Of the 3,010 children for whom complete data were available, 1,218 were from the rural area and 1,792 were from the urban area. The children from the urban area were then subdivided into 440 poor (from the urban slum area) and 1,352 nonpoor children. The total population studied consisted of 1,738 boys (57.7%) and 1,272 girls (42.3%).

In our sample, the prevalence of stunting was 19.3% (HAZ < −2.00, 580 children). The prevalence of wasting was 5.0% (WHZ < −2.00, 152 children), while the prevalence of overweight was 2.3% (WHZ > +2.00, 70 children). However, when the BMI reference was taken, the prevalence of overweight was 2.7% (80 children). The level of agreement between these two cutoffs for overweight was good (κ = 0.89, p < .001, Fisher’s exact test). The prevalence of obesity based on the BMI reference proposed by IOTF was 0.8% (25 children). Details on the odds ratios for stunting, wasting, overweight, and obesity based on socioeconomic status are given in table 1.

Urban children, whether they were poor or nonpoor, were on average taller than rural children (p < .001), with mean (95% CI) differences in HAZ of 0.61 (0.54–0.68) and 0.20 (0.11–0.30), respectively. Nonpoor urban children were heavier than rural children (p < .001), with mean (95% CI) differences in weight-for-height z-scores (WHZ) and BMI of 0.24 (0.16–0.32) and 0.46 (0.32–0.59), respectively. There were no significant differences in WHZ and BMI between poor urban and rural children (p > .05).

The odds ratios for stunting (OR, 1.75; 95% CI, 1.44–2.12; p < .001) and obesity (OR, 4.07; 95% CI, 1.40–11.8; p = .005) were higher in boys than in girls. Details on the odds ratios and prevalences of stunting, wasting, overweight, and obesity in boys and girls are presented in table 2. The differences between boys and girls in the odds ratios for stunting were observed in both the rural and the urban areas (table 3).

Stunted children had lower odds ratios for overweight (OR, 0.10; 95% CI, 0.03–0.43; p < .001). Overall, the mean WHZ and BMI of stunted children were lower than those of nonstunted children. The differences were statistically significant for the rural, poor urban, and nonpoor urban children (table 4). There were significant positive correlations between HAZ and WHZ (ρ = 0.24, p < .001) and between HAZ and BMI (ρ = 0.30, p < .001).
DISCUSSION
The overall prevalence of stunting in rural children was almost three times higher than in nonpoor urban children. To a lesser degree, poor urban children also had significantly higher (p = 0.002, table 1) odds ratios for stunting than the nonpoor urban children. A similar study comparing the prevalence of stunting in slightly older (seven to nine years) urban and rural children in Malaysia found comparable results [13]. Two studies on urban school-children in Jakarta and Manila also found higher prevalences of stunting in children of lower socioeconomic status. These two studies used attendance at public or private schools as a proxy for socioeconomic status, whereas in our study we used the child’s area of residence as a proxy for socioeconomic status [14, 15]. Characteristics such as family income or parental education would be better indicators of socioeconomic status, but in these populations these were not always easy to assess. Many families had multiple irregular incomes, and extended families with multiple breadwinners and child caregivers were common. Our study and the other three studies mentioned above seem to suggest that, at least in these three Asian countries, stunted growth is still strongly associated with poverty.

Boys in our study had a higher odds ratio for stunting than girls. For reasons that are unclear, similar findings have been reported from Kuala Lumpur, Jakarta, Manila, and South Africa [13–16]. This difference in prevalence persisted after the data had been stratified into the three socioeconomic levels, suggesting the relative independence of the difference from socioeconomic status. Our previous study in children under two years of age in another area of Indonesia showed that beginning at the age of seven months, the difference in mean length-for-age between the Indonesian children and the NCHS/WHO reference population was larger in boys than in girls [17]. Another study from Indonesia showed that the size of this difference between boys and girls increased up to the age of 35 months [18]. It is still not known whether this greater difference persists until the final height has been attained, or whether there will still be a catch-up growth in adolescence.

The overall prevalence of wasting was almost one-quarter that of stunting. Unlike stunting, there was no significant difference in the prevalence of wasting between children of different socioeconomic status. Studies from other areas yielded variable findings [13–15]. Stunting, which occurs mostly in the first three years of life, reflects long-term undernutrition and poor health, whereas wasting is more a reflection of recent energy imbalance. Our study seems to suggest that the problems of undernutrition are important in the poorer segment of the population, especially in the early years of life [2, 3, 11].

The prevalence of overweight, based on either the IOTF or the WHO-NCHS references, was, on average, four to five times higher in nonpoor urban children than in rural
children or poor urban children, as was the prevalence of obesity. A higher prevalence of obesity in the higher socioeconomic portion of the population has been found in other studies in developing countries [13–15]. A study of school-children in Manila found that children from higher socioeconomic groups tended to consume more food, including animal food, fats and oils, and beverages, resulting in higher intake of calories. Moreover, children from high socioeconomic groups were apparently less physically active, were more likely to be driven to school instead of walking, and were more likely to prefer television and computer games over outdoor games [19].

In industrialized countries, and increasingly in developing countries, wealth has been associated with diets high in fat in combination with a sedentary lifestyle. But as global income rises and luxuries become affordable to most people in developed and middle-income countries, the pattern is changing [20]. In most developed countries, obesity is now associated with low income and social class [21–24]. Studies in developed countries have shown that children from lower-income families consume more energy-dense but inexpensive foods than those from higher-income families. These children also engage in less physical activity either because there is no safe place to play outdoors or because there is no money to pay for safe indoor facilities [25–27].

Nonpoor urban boys had significantly higher odds ratios for obesity than nonpoor urban girls. Similar findings were reported from the cities of Jakarta, Manila, and Kuala Lumpur [13–15]. Two studies in the United States found a higher prevalence of obesity in younger school-aged boys compared with girls, followed by an increasingly higher prevalence in girls compared with boys, as they approached adolescence [25, 28]. Other studies in similar age groups in South Africa, France, Russia, and China produced variable results in the prevalence of obesity between boys and girls in different age groups. [3, 16, 29, 30].

There is no clear explanation for this gender difference in susceptibility to obesity across race and age groups. The difference may be due to the variability of age range between studies, which, combined with the variability of the timing of maturation and body composition across race and gender, may lead to apparent differences in the prevalence of obesity. This assumption needs further study to be verified.

Unlike the study by Popkin et al. [3], our study did not indicate any association between stunting and overweight or obesity. Stunted children had a significantly lower mean BMI and WHZ, irrespective of socioeconomic status. It is possible that short children were at higher risk for later obesity during adolescence or adulthood, but further investigation is needed to determine whether this is the case.
In summary, our study indicates that poorer segments of the population in Indonesia still face the problem of undernutrition and will benefit from intervention to improve the quantity and quality of their food intake, especially in the early years of life. This study also shows that children from higher socioeconomic groups will benefit from programs to reduce the risk of obesity, such as public health campaigns on healthful food and encouragement to increase physical activity. However, one of the limitations of our study is that it did not assess some important predictors of height and obesity, such as parental height and obesity status, as well as lifestyle factors, such as diet and physical activity [22, 24, 31, 32].

References
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TABLE 1. Socioeconomic differences in the prevalence and odds ratio of stunting, wasting, overweight, and obesity

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Socioeconomic Status</th>
<th>n</th>
<th>Prevalence (%)</th>
<th>Odds Ratio (95% CI)</th>
<th>p^a</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stunted^b</td>
<td>Rural</td>
<td>1,218</td>
<td>28.2</td>
<td>2.92 (2.37-3.59)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td>Poor urban</td>
<td>440</td>
<td>17.5</td>
<td>1.58 (1.18-2.13)</td>
<td>.002</td>
</tr>
<tr>
<td></td>
<td>Nonpoor urban</td>
<td>1,352</td>
<td>11.8</td>
<td>1.00 (reference)</td>
<td>-</td>
</tr>
<tr>
<td>Wasted^c</td>
<td>Rural</td>
<td>1,218</td>
<td>4.8</td>
<td>0.97 (0.68-1.40)</td>
<td>0.89</td>
</tr>
<tr>
<td></td>
<td>Poor urban</td>
<td>440</td>
<td>6.4</td>
<td>1.32 (0.84-2.09)</td>
<td>0.23</td>
</tr>
<tr>
<td></td>
<td>Nonpoor urban</td>
<td>1,352</td>
<td>4.9</td>
<td>1.00 (reference)</td>
<td>-</td>
</tr>
<tr>
<td>Overweight based on IOTF^d</td>
<td>Rural</td>
<td>1,218</td>
<td>1.0</td>
<td>0.19 (0.10-0.36)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td>Poor urban</td>
<td>440</td>
<td>0.7</td>
<td>0.13 (0.04-0.43)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td>Nonpoor urban</td>
<td>1,352</td>
<td>4.9</td>
<td>1.00 (reference)</td>
<td>-</td>
</tr>
<tr>
<td>Overweight based on NCHS/WHO^e</td>
<td>Rural</td>
<td>1,218</td>
<td>1.0</td>
<td>0.23 (0.12-0.43)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td>Poor urban</td>
<td>440</td>
<td>0.5</td>
<td>0.11 (0.03-0.44)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td>Nonpoor urban</td>
<td>1,352</td>
<td>4.1</td>
<td>1.00 (reference)</td>
<td>-</td>
</tr>
<tr>
<td>Obesity based on IOTF^f</td>
<td>Rural</td>
<td>1,218</td>
<td>0.2</td>
<td>0.09 (0.02-0.39)</td>
<td>&lt;.001^g</td>
</tr>
<tr>
<td></td>
<td>Poor urban</td>
<td>440</td>
<td>0.0</td>
<td>Not calculated</td>
<td>.005^g</td>
</tr>
<tr>
<td></td>
<td>Nonpoor urban</td>
<td>1,352</td>
<td>1.8</td>
<td>1.00 (reference)</td>
<td>-</td>
</tr>
</tbody>
</table>

CI, Confidence interval; IOTF, International Obesity Task Force; NCHS, National Center for Health Statistics; WHO, World Health Organization; BMI, body-mass index.

^a. \( \chi^2 \).

^b. Height-for-age z-score (HAZ) < –2.00.

^c. Weight-for-height z-score (WHZ) < –2.00.

^d. Percentiles passing BMI of 25 kg/m\(^2\) (IOTF); see Subjects and Methods section for detailed information.

^e. Weight-for-height z-score (WHZ) > +2.00.

^f. Percentiles passing BMI of 30 kg/m\(^2\) (IOTF); see Subjects and Methods section for detailed information.

^g. Fisher exact test
TABLE 2. Sex differences in the prevalence and odds ratio of stunting, wasting, overweight, and obesity

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Sex</th>
<th>n</th>
<th>Prevalence (%)</th>
<th>Odds Ratio (95% CI)</th>
<th>p²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stuntedb</td>
<td>M</td>
<td>1,738</td>
<td>22.8</td>
<td>1.75 (1.44-2.12)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>1,272</td>
<td>14.5</td>
<td>1.00 (reference)</td>
<td>-</td>
</tr>
<tr>
<td>Wastedc</td>
<td>M</td>
<td>1,738</td>
<td>5.5</td>
<td>1.24 (0.76-2.03)</td>
<td>0.22</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>1,272</td>
<td>4.5</td>
<td>1.00 (reference)</td>
<td>-</td>
</tr>
<tr>
<td>Overweight based on IOTFd</td>
<td>M</td>
<td>1,738</td>
<td>3.0</td>
<td>1.40 (0.88-2.24)</td>
<td>0.15</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>1,272</td>
<td>2.0</td>
<td>1.00 (reference)</td>
<td>-</td>
</tr>
<tr>
<td>Overweight based on NCHS-WHOe</td>
<td>M</td>
<td>1,738</td>
<td>2.5</td>
<td>1.25 (0.76-2.03)</td>
<td>0.38</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>1,272</td>
<td>2.0</td>
<td>1.00 (reference)</td>
<td>-</td>
</tr>
<tr>
<td>Obesity based on IOTFf</td>
<td>M</td>
<td>1,738</td>
<td>1.3</td>
<td>4.07 (1.40-11.8)</td>
<td>.005</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>1,272</td>
<td>0.3</td>
<td>1.00 (reference)</td>
<td>-</td>
</tr>
</tbody>
</table>

CI, Confidence interval; IOTF, International Obesity Task Force; NCHS, National Center for Health Statistics; WHO, World Health Organization; BMI, body mass index.

a. \( \chi^2 \).
b. Height-for-age z-score (HAZ) < -2.00.
c. Weight-for-height z-score (WHZ) < -2.00.
d. Percentiles passing BMI of 25 kg/m\(^2\) (IOTF); see Subjects and Methods section for detailed information.
e. Weight-for-height z-score (WHZ) > +2.00.
f. Percentiles passing BMI of 30 kg/m\(^2\) (IOTF); see Subjects and Methods section for detailed information.
TABLE 3. Odds ratios for stunting and obesity among boys relative to girls in rural and urban areas

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Socioeconomic Status</th>
<th>Prevalence (%)</th>
<th>Odds Ratio (95% CI)</th>
<th>p&lt;sup&gt;a&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stunted&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Rural</td>
<td>31.8</td>
<td>22.9</td>
<td>1.57 (1.21-2.04)</td>
</tr>
<tr>
<td></td>
<td>Poor urban</td>
<td>22.3</td>
<td>10.9</td>
<td>2.35 (1.36-4.07)</td>
</tr>
<tr>
<td></td>
<td>Nonpoor urban</td>
<td>14.4</td>
<td>8.6</td>
<td>1.79 (1.26-2.54)</td>
</tr>
<tr>
<td>Obesity based on IOTF&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Rural</td>
<td>0.3</td>
<td>0.0</td>
<td>not calculated</td>
</tr>
<tr>
<td></td>
<td>Poor urban</td>
<td>0.0</td>
<td>0.0</td>
<td>not calculated</td>
</tr>
<tr>
<td></td>
<td>Nonpoor urban</td>
<td>2.6</td>
<td>0.7</td>
<td>4.00 (1.36-11.8)</td>
</tr>
</tbody>
</table>

CI, Confidence interval; IOTF, International Obesity Task Force; BMI, body mass index.

a. χ<sup>2</sup>

b. Height-for-age z-score (HAZ) < -2.00.

c. Percentiles passing BMI of 30 kg/m<sup>2</sup> (IOTF); see Subjects and Methods section for detailed information.

TABLE 4. Mean weight-for-height z-score (WHZ) and body mass index (BMI) of stunted compared with nonstunted children

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Socioeconomic Status</th>
<th>Stunted&lt;sup&gt;b&lt;/sup&gt;</th>
<th>Nonstunted&lt;sup&gt;b&lt;/sup&gt;</th>
<th>p&lt;sup&gt;c&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean weight for height z-score (WHZ)</td>
<td>Overall</td>
<td>-0.85 (0.77)</td>
<td>-0.56 (1.07)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td>Rural</td>
<td>-0.86 (0.66)</td>
<td>-0.66 (0.91)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td>Poor urban</td>
<td>-0.97 (0.77)</td>
<td>-0.72 (0.86)</td>
<td>.02</td>
</tr>
<tr>
<td></td>
<td>Nonpoor urban</td>
<td>-0.78 (0.96)</td>
<td>-0.44 (1.22)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Mean BMI (SD)</td>
<td>Overall</td>
<td>14.20 (1.06)</td>
<td>14.70 (1.73)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td>Rural</td>
<td>14.18 (0.90)</td>
<td>14.49 (1.37)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td>Poor urban</td>
<td>14.06 (1.03)</td>
<td>14.41 (1.25)</td>
<td>.02</td>
</tr>
<tr>
<td></td>
<td>Nonpoor urban</td>
<td>14.31 (1.35)</td>
<td>14.93 (2.03)</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

a. Height-for-age z-score (HAZ) < −2.00.

b. Height-for-age z-score (HAZ) ≥ −2.00.

c. t-test.
Chapter 4.

Association between low weight at birth
and stunted growth or overweight
in rural and urban Indonesian prepubertal children

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†† Passed away

Submitted
ABSTRACT

Low birth weight (LBW) is associated with both stunted growth and overweight later in life. A cross-sectional survey of 2833 school-aged prepubertal children was performed to assess relative contribution of LBW on the prevalence of stunted growth or overweight in rural and urban children in Indonesia. Each child had data on age, sex, stature, BMI and birth weight. Compared to the urban population, prevalence of stunted growth was higher in the rural, i.e. 16.3 vs. 32.7%. There was no significant difference in the prevalence of LBW. Stunted children were more likely to be born with LBW, OR (95% CI): 1.80 (1.31; 2.47), p<0.001. After stratifying data into rural and urban residences, the contribution of LBW to stunted growth appeared to be only significant in urban population, OR (95% CI): 2.42 (1.59; 3.68). In the rural areas, similar proportions of LBW were found in stunted and not stunted children. We observed no association between LBW and overweight. The observed variation in the prevalence of stunted growth among rural and urban children might have its origin in the socioeconomic status influencing pattern of post-natal growth.

Keywords: rural – urban - low birth weight – stunted growth – overweight – prepubertal children
INTRODUCTION

Around one third of children in the developing world are growth retarded before the age of 5 years. Stunted linear growth is an important public health problem because of its association with increased risk for morbidity and mortality, as well as its association with poor functional outcomes such as impaired cognitive development later in life.

Low birth weight (LBW) infants, especially those who were born small for gestational age (SGA), have been reported to have a tendency to remain shorter than infants born with a normal weight. Although there might be some biological potency for catch-up growth after birth, the opportunity might be low in children who lived in the poor environments not supporting for catch-up growth.

In addition to its association with prenatal growth deficits, stunted growth in the developing world might be also related to post-natal failure of growth. It had been widely recognized that children in the developing countries experienced retarded growth during the first years of life. A previous longitudinal study in a rural area in Indonesia observed a significant deficit in growth starting from the age of 6 to 7 months. This deficit increased the prevalence of stunted growth from only 7% in the neonatal period to 24% at the age of 12 months.

In some developing countries, especially in those experiencing nutrition transition, problems of overnutrition is gradually replacing or adding to the existing problem of undernutrition. Furthermore, several countries have documented the association between stunted growth and higher risk of obesity. Studies in affluent populations observed that rapid weight gain during infancy, particularly in those born with lower weight, was associated with an increased risk for obesity.

The contributions of pre- and postnatal growth deficits for subsequent stunted growth or overweight might be manipulated by the socioeconomic status of that population. Therefore, it is questioned in which way a poor or a relatively rich environment influences the association between a low weight at birth and stunted growth or overweight during childhood. The aim of this study was to assess the relative contribution of low weight at birth on the prevalence of stunted growth or overweight in school-aged prepubertal children from rural and urban communities in Indonesia.

SUBJECTS AND METHODS

Study Population and Design

The study was performed in two adjacent areas in Central Java. Yogyakarta, an urban area, had approximately 487,000 inhabitants, and Gunung Kidul, a rural area located about
20 to 40 km from Yogyakarta, had approximately 710,000 inhabitants at the time of the
study. Definition of rural and urban population was based on agricultural activities and
population densities. Yogyakarta had a population density of 14,988/km$^2$, while Gunung
Kidul had 498/km$^2$. These two study areas were chosen because of the relatively
homogenous ethnicity of their populations. Most of the people in both areas are of Javanese
descent. The altitude of both study areas is less than 500 meter above sea level.

A cross sectional study was conducted in school-aged prepubertal children in both
areas. Thirty-three out of 509 public primary schools in the rural area and 37 out of 172 in
the urban area were randomly selected. Prepubertal children (under 8 years of age for girls
and under 9 years of age for boys) from the first- and second year classes of each school
were included. As it is obligatory for children to enter primary school at the age of 6 to 7
years, the ages of the children finally studied ranged from 6 to 7.9 years for girls and from 6
to 8.9 years for boys. In this study only prepubertal children were selected since puberty,
which normally occurred after the age of 8 years in girls and 9 years in boys, may interfere
with the interpretation of the measurements due to the changes in body composition and to
the differences between children in the timing of the adolescent growth spurt.

Children with prominent chronic diseases, i.e. congenital heart diseases or major
thalassemia, and children who had physical handicaps that might interfere with the
determination of anthropometric measurements, were excluded. The study was conducted
from February to May 1999 and was approved by the ethical committee of Gadjah Mada
University, Yogyakarta, Indonesia.

**Data Collection**

Anthropometric data were collected by health professionals. Heights and weights were
measured using the standard techniques described by the World Health Organization (WHO)
18. Training for standardization of the measurements, followed by field practice and testing,
was performed prior to data collection. All measurements were performed at around 8 to 10
a.m. The children, wearing light clothing, were weighed to the nearest 0.1 kg using a Seca
digital scale (Germany). Height was measured to the nearest 0.1 cm using a portable
stadiometer with the child standing facing the fieldworker, without shoes. Information on
birth weight was collected using a questionnaire sent to the parents with the help of the
teachers. Birth dates were verified by the copy of the child's birth certificate filed at the school.
Statistical analyses

The body mass index (BMI) was calculated by dividing weight in kilograms by the square of height in meters (kg/m²). Data on BMI were converted to percentiles based on the year 2000 sex-specific Center for Disease Controls and Prevention (CDC) growth charts using the nutritional anthropometry module (NutStat) of the CDC’s Epi Info 2000 (Centers for Disease Control and Prevention, Atlanta, Georgia, USA). Children were classified as at risk for overweight if they had a BMI equal to or above the 85th percentile.19

Data on height were converted to standard deviation scores (height SDS) using the same references above and statistical software. Biologically implausible values, such as height SDS below -6.00 or above +6.00, were excluded from the analysis. Values that were most likely to represent errors, i.e., those with 4 SDS units outside the observed mean for every age in one full year, were also excluded (flexible exclusion range). Children were classified as stunted if they had height SDS below -2.00, and as not stunted if they had height SDS of -2.00 or more.20 Children were considered to have low birth weights (LBW) when their birth weights were less than 2500 g, and considered to have normal birth weights when they were 2500 g or above.1 Overweight, stunted growth and LBW were all defined using an international reference standard to allow for international comparison.

The prevalence of stunted growth, overweight and LBW in rural children was compared to urban children as the reference by using \( \chi^2 \)-test. Similar analyses were used to compare the prevalence of LBW in stunted vs. not stunted children and in overweight vs. not overweight children. Odds ratios were presented with 95% confidence intervals (95% CI). Test of interaction was performed to assess significance of the difference in odds ratios observed in subgroup analyses (rural vs. urban).21 All statistical analyses were performed using the CDC’s Epi Info 2000 (Centers for Disease Control and Prevention, Atlanta, Georgia, USA).

RESULTS

Seventy schools were visited to examine a total of 3689 prepubertal children listed as first- and second-year students. Six hundred and seventy-four (674) children were excluded because they were not in the required age range. Of the remaining 3015 children, four missed the measurement session and one had a height SDS in the flexible exclusion range.

Of the 3010 children for whom measurements were available and considered valid, 2833 (94%) children reported their birth weight, i.e. 1125 rural children (92.4% of the eligible rural children) and 1708 (95.3%) urban children. After stratifying the data into urban and rural residence, there was no significant difference in the prevalence of stunted growth.
and overweight between those who reported their birth weight and those who did not (data not shown).

Of all children studied, 1625 were boys (57.4%) and 1208 were girls. There was no significance difference in the ratio of males to females (p≥0.05), i.e. 58.8% and 56.4% boys in the rural and urban population, respectively. None of the children had either a prominent chronic disease or a physical handicap that might interfere with the measurements.

The overall prevalence of LBW was 7.0% (199 children). There was no significant difference in the odds to have been born small between the rural and the urban children. The overall prevalence of stunted growth, i.e. having a height SDS of less than −2.0, was 22.8%. Rural children, as compared to urban children, had significantly higher odds to be stunted. On the other hand, the prevalence of overweight was almost three times higher in the urban compared to rural children (Table 1).

The prevalence of LBW in stunted children (10.4%) was higher than those in not-stunted children (6.0%). Compared to not-stunted children, the odds ratio (95% CI) for being born small in stunted children was 1.80 (1.32; 2.45), p<0.001. In rural children, with higher prevalence of stunted growth (32.7%), there was no significant difference in the odds to be born small between the stunted and the not-stunted children. In urban children, with a lower prevalence of stunted growth (16.3%), the odds to be born small in stunted children was more than twice those of not-stunted children. Test of interaction showed a significant difference in the observed odds ratios from urban vs. rural population, the ratio of the odds ratios (95% CI) was 1.88 (1.11 – 3.17), p= 0.02 (Table 2A). This study did not reveal any association between LBW and the tendency to be overweight, i.e. OR (95% CI) of 0.84(0.38; 1.82), p=0.79.

DISCUSSION
This study addressed the question whether low weight at birth was associated with the prevalence of stunted growth or overweight in prepubertal children from rural and urban communities in Indonesia. We observed that, although only approximately 10% of stunted children had low weight at birth, the proportion of LBW in stunted children was significantly higher than in not-stunted children. However, when the data were stratified into rural and urban residences, a significantly higher proportion of LBW in stunted children was only observed in the urban population.

In the rural population, where the prevalence of stunted growth was highest, there was no significant association between LBW and stunted growth. As there was no significant difference in the odds for low weight at birth between the rural and the urban children, the
observed higher prevalence of stunted growth in rural children might be associated with an increased incidence of impaired growth post-natally occurring in this population.

It had been widely recognized that children in the developing countries experienced growth faltering during the first years of life. A common feature of growth pattern in infants from the developing world is acceptable growth during the first 4 to 6 months, followed by a progressive decline in growth rate compared to the affluent population. This decline in growth rate usually began at the age of 6 months up to the age of 12 to 24 months or perhaps longer.9

A previous follow-up study in a rural area in Indonesia documented a significant decline in growth rate starting from the age of 6 to 7 months. This decline was responsible for the increase in the prevalence of stunted growth from only 7% in the neonatal period to 24% at the age of 12 months.10 A study in the Philippines showed that the prevalence of stunted growth increased from around 35% at the age of 12 months to 65% at the age of 2 years, with the rural area worse off than the urban.6

This study and previous studies in Indonesia showed that most children in Indonesia were not born with low birth weight. The prevalence of a low birth weight is about 6 to 9%.1;10 However, most infants did exhibit growth deficits during the first years of life.10 Our previous study documented a worse growth in the poorer area.22

Since stunted growth reflects long-term cumulative inadequacies of health and nutrition, and had been strongly associated with poverty.6;20 we may conclude that the rural communities in this study seemed to be poorer than the urban area. The rural area had an increased prevalence of stunted growth, i.e. 32.7%, compared to 16.3% in the urban area.

Our study did not detect any association between LBW and overweight. A study in Sweden observed a positive association between rapid growth in infancy and early childhood with BMI in young adulthood. In that study, individuals with lower birth weights gained more weight in infancy.23 Similar studies in Britain documented the role of infant and childhood growth on the association between LBW and obesity.15;24 The association between LBW and overweight seemed to be mediated through rapid weight gain in infancy or childhood, which probably did not occur in our population. Since our study did not examine growth longitudinally, we cannot precisely assess the influence of postnatal growth.

In summary, the present study showed that, in an area where the prevalence of stunted growth was very high (the rural area), low weight at birth was not an important contributor for stunted growth. However, in an area where the prevalence of stunted growth was less (the urban area), low weight at birth became an important risk factor for subsequent stunted growth. Furthermore, as we did not observe a significant difference in
the prevalence of low birth weight among the rural and urban populations, we conclude that the observed difference in the prevalence of stunted growth among rural and urban prepubertal children might have its origin in the socioeconomic environment influencing the pattern of post-natal growth.

Reference

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Table 1. Prevalence and odds ratio of stunted growth\(^a\), overweight\(^b\) and low weight at birth (LBW)\(^c\) in rural children compared to urban children as the reference

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Population</th>
<th>Prevalence (%)</th>
<th>Odds Ratio (95% CI)</th>
<th>(\chi^2)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rural</td>
<td>Urban</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stunted Growth(^a)</td>
<td>All</td>
<td>32.7</td>
<td>16.3</td>
<td>2.49 (2.08 – 2.98)</td>
</tr>
<tr>
<td></td>
<td>Boys</td>
<td>31.7</td>
<td>16.2</td>
<td>2.40 (1.90 – 3.05)</td>
</tr>
<tr>
<td></td>
<td>Girls</td>
<td>34.1</td>
<td>16.5</td>
<td>2.62 (2.00 – 3.44)</td>
</tr>
<tr>
<td>Overweight(^b)</td>
<td>All</td>
<td>2.0</td>
<td>5.5</td>
<td>0.36 (0.23 – 0.57)</td>
</tr>
<tr>
<td></td>
<td>Boys</td>
<td>2.1</td>
<td>5.9</td>
<td>0.34 (0.19 – 0.62)</td>
</tr>
<tr>
<td></td>
<td>Girls</td>
<td>1.9</td>
<td>5.0</td>
<td>0.38 (0.18 – 0.79)</td>
</tr>
<tr>
<td>Low weight at birth (LBW)(^c)</td>
<td>All</td>
<td>7.5</td>
<td>6.7</td>
<td>1.12 (0.84 – 1.50)</td>
</tr>
<tr>
<td></td>
<td>Boys</td>
<td>6.3</td>
<td>5.4</td>
<td>1.19 (0.78 – 1.81)</td>
</tr>
<tr>
<td></td>
<td>Girls</td>
<td>9.1</td>
<td>8.5</td>
<td>1.08 (0.72 – 1.63)</td>
</tr>
</tbody>
</table>

\(^a\) having a height standard deviation scores (height SDS) below –2.00
\(^b\) having a BMI equal to or above the 85\(^{th}\) percentile of the CDC 2000 reference population
\(^c\) LBW= low birth weight, having a birth weight less than 2500 g
\(^d\) \(\chi^2\)
Table 2. Odds ratio for low weight at birth (LBW)\(^a\) among stunted\(^b\) or overweight\(^c\) children relative to those not-stunted nor overweight respectively, stratified by rural and urban residence

A. Stunted\(^b\) or not-stunted

<table>
<thead>
<tr>
<th>Population</th>
<th>Prevalence (%) of LBW(^a) in</th>
<th>OR (95%CI)</th>
<th>p(^d)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Stunted(^b)</td>
<td>Not Stunted</td>
<td></td>
</tr>
<tr>
<td>Rural</td>
<td>All 8.7</td>
<td>6.9</td>
<td>1.29 (0.82 – 2.04)</td>
</tr>
<tr>
<td></td>
<td>Boys 7.1</td>
<td>6.0</td>
<td>1.21 (0.63 – 2.33)</td>
</tr>
<tr>
<td></td>
<td>Girls 10.8</td>
<td>8.2</td>
<td>1.35 (0.71 – 2.58)</td>
</tr>
<tr>
<td>Urban</td>
<td>All 12.5</td>
<td>5.6</td>
<td>2.42 (1.59 – 3.68)</td>
</tr>
<tr>
<td></td>
<td>Boys 10.3</td>
<td>4.5</td>
<td>2.45 (1.32 – 4.53)</td>
</tr>
<tr>
<td></td>
<td>Girls 15.4</td>
<td>7.1</td>
<td>2.40 (1.35 – 4.27)</td>
</tr>
</tbody>
</table>

B. Overweight\(^c\) or not-overweight

<table>
<thead>
<tr>
<th>Population</th>
<th>Prevalence (%) of LBW(^a) in</th>
<th>OR (95%CI)</th>
<th>p(^d)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Overweight(^c)</td>
<td>Not Overweight</td>
<td></td>
</tr>
<tr>
<td>Rural</td>
<td>All 4.3</td>
<td>7.5</td>
<td>0.56 (0.07 – 4.19)</td>
</tr>
<tr>
<td></td>
<td>Boys 0</td>
<td>6.5</td>
<td>Not applicable</td>
</tr>
<tr>
<td></td>
<td>Girls 11.1</td>
<td>9.0</td>
<td>1.26 (0.15 – 10.32)</td>
</tr>
<tr>
<td>Urban</td>
<td>All 6.4</td>
<td>6.8</td>
<td>0.94 (0.36 – 2.29)</td>
</tr>
<tr>
<td></td>
<td>Boys 7.0</td>
<td>5.3</td>
<td>1.35 (0.47 – 3.88)</td>
</tr>
<tr>
<td></td>
<td>Girls 5.4</td>
<td>8.6</td>
<td>0.61 (0.14 – 2.58)</td>
</tr>
</tbody>
</table>

\(^a\) LBW= low birth weight, having a birth weight less than 2500  
\(^b\) having a height standard deviation scores (height SDS) below –2.00  
\(^c\) having a BMI equal to or above the 85\(^{th}\) percentile of the CDC 2000 reference population  
\(^d\) \(\chi^2\) or Fisher’s exact test  
\(^e\) Ratio for odds ratio observed in urban vs. rural population (95%CI): 1.88 (1.11 – 3.17), p=0.02 (test of interaction)  
\(^f\) Fisher’s exact test  
\(^g\) Yates corrected
Chapter 5.

Tracking for underweight, overweight and obesity from childhood to adolescence: a 5-year follow-up study in urban Indonesian children

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ABSTRACT

Aims: To assess tracking of body mass index (BMI) of urban Indonesian children from childhood to adolescence and to compare the prevalence of underweight, overweight and obesity in 6- to 8-year-old children from two surveys: years 1999 and 2004.

Methods: A longitudinal study assessing BMI tracking of 308 urban children followed from age 6–8 to 11–13 years and two cross-sectional surveys comparing the prevalence of underweight, overweight and obesity in 6- to 8-year-old children: year 1999 (n = 1,524) and 2004 (n = 510).

Results: Childhood BMI determined 52.3% variation of later BMI. After 5.1 (0.6) years the prevalence of overweight and obesity increased from 4.2 and 1.9% in childhood to 8.8 and 3.2% in adolescence. The prevalence of underweight decreased from 27.3 to 18.8%. All obese children remained obese, 84.6% overweight children stayed overweight, 56.0% underweight children remained underweight. In cross-sectional comparison the prevalence of overweight and obesity raised from 5.3 to 8.6% and from 2.7 to 3.7%, respectively. The prevalence of underweight remained constant.

Conclusions: The prevalence of overweight and obesity increases as children grow into adolescence. Overweight or obese children are more likely to remain overweight or obese. Cross-sectional comparison shows, while the prevalence of underweight stays constant, the prevalence of overweight and obesity increases.

Key Words: tracking of underweight, overweight and obesity – underweight – overweight – obesity - urban Indonesian children
INTRODUCTION

The worldwide prevalence of obesity is increasing rapidly [1, 2]. This epidemic is alarming because obesity is known to be associated with an increased risk for cardiovascular diseases and other chronic diseases related to metabolic syndrome, such as dyslipidemia and diabetes mellitus type 2 [3].

Childhood and adolescence are identified as the two critical periods for the development of obesity later in life. Studies from industrialized countries have shown that most obese children remain obese through adolescence and into adulthood [4–7]. However, this kind of tracking might be not observed in most developing countries. A study in China showed that only a very small proportion of overweight Chinese children continued to be overweight in adolescence, while, on the other hand, the probability for underweight children to remain underweight in adolescence was much higher [8]. It therefore seems that the pattern of tracking for underweight or overweight is different between the developed and developing countries.

Our previous report on the prevalence of overweight and obesity in Indonesia showed that the prevalence of obesity in urban children was >5 times higher than that of rural children. However, a considerable prevalence of undernutrition was also observed [9]. Whether these underweight, overweight and obese urban children remained in the same weight status when entering adolescence is not known.

The objective of the present study was to assess tracking for underweight, overweight and obesity in children in Indonesia after 5 years of follow-up, i.e. from age 6–8 to age 11–13 years. Furthermore, this study also compared the prevalence of underweight, overweight and obesity in 6- to 8-year-old children from two 5-year-apart cross-sectional surveys.

METHODS

The presented study is based on two cross-sectional surveys on the prevalence of obesity in primary school-children in Yogyakarta, a city in Indonesia that has approximately 487,000 inhabitants and a population density of 14,988/km² [10]. The first survey was performed in 1999 and included 3,010 prepubertal students of grades 1 and 2 (age between 6 and 8 years), from both urban and rural communities. The results of this study have been reported elsewhere [9]. The second survey was done in 2004 and enrolled 2,497 elementary school-children of grades 1–6 (age between 6 and 13 years), but only from the urban community.

The data obtained from these two surveys are divided into two analyses – longitudinal and cross-sectional (fig. 1).
**Longitudinal Analysis**

To demonstrate from childhood to adolescence, a cohort data obtained from 308 children who had their body weight and height measured in both surveys were used. In the first survey they were in late childhood (ages between 6 and 8 years), while in the second survey, which was around 5 years later, they were in early adolescence (ages between 11 and 13 years).

**Cross-Sectional Analysis**

In addition, to assess the trends in the prevalence of underweight, overweight and obesity in children of the same age over time, data of urban children aged 6–8 years from the first survey ($n = 1,524$) were compared to those with a similar age range from the second survey ($n = 510$).

**Data Collection**

Anthropometric data were collected by health professionals. Heights and weights were measured using the standard techniques described by the WHO [11]. Training for standardization of the measurements, followed by field practice and testing, was performed prior to data collection. All measurements were performed between 8 and 10 a.m. The children, wearing light clothing, were weighed to the nearest 0.1 kg with a Seca digital scale (Germany). Height was measured to the nearest 0.1 cm with a portable stadiometer. Height was measured with the child standing facing the fieldworker, without shoes.

The body mass index (BMI) was calculated. Data on BMI were converted to z-scores (BMI z-scores) and percentiles based on the year 2000 sex-specific Center for Disease Controls and Prevention (CDC 2000) growth charts using the nutritional anthropometry module (NutStat) of the CDC's Epi Info 2000 (CDC, Atlanta, Ga., USA). Children were classified as underweight if they had a BMI below the 5th percentiles of the year 2000 CDC growth charts. They were classified as overweight when the BMI was equal to or above the 85th percentile and as obese when the BMI was at the 95th percentile or above [12].

**Data Entry and Analyses**

Data were entered and analyzed using SPSS for Windows (Version 12, SPSS, Chicago, Ill., USA) and the CDCs Epi Info 2000 (CDC, Atlanta, Ga., USA).
**Longitudinal Analysis**

In this study, tracking was defined as the maintenance of ranking as underweight, overweight or obese between the two measurements: (1) prospective tracking was percentage of those underweight (overweight or obese) at the age of 6–8 years who were still underweight (overweight or obese) at the age of 11–13 years, and (2) retrospective tracking was percentage of those underweight (overweight or obese) at the age of 11–13 years who were already underweight (overweight or obese) at the age 6–8 years.

Other measures of tracking, such as relative risk (95% CI) of those underweight (overweight or obese) to remain underweight (overweight or obese) compared to those not underweight (overweight nor obese), correlation coefficient between BMI or BMI z-score, as well as agreement (k) in the classification of underweight (overweight or obese) between childhood and adolescence were also presented [13]. In addition, the mean increase (95% CI) of BMI relative to the CDC 2000 reference population (BMI z-score) between childhood and adolescence was also presented.

**Cross-Sectional Analysis**

Mean (SD) BMI and BMI z-scores, as well as the prevalence of underweight, overweight and obesity in 6- to 8-year-old children from the 1999 and 2004 surveys were analyzed.

**RESULTS**

**Longitudinal Analysis**

The cohort comprised 153 (49.7%) boys and 155 girls. At the beginning of the follow-up, when they were aged between 6 and 8 years, they had a mean (SD) age of 7.1 (0.4) years. In early adolescence (age 11–13 years), after a follow-up period of 5.1 (0.6) years, they were 12.1 (0.4) years old.

The absolute BMI increased from 14.5 (1.7) in childhood to 17.5 (3.0) in adolescence, i.e. mean difference (95% CI) of 3.0 (2.7; 3.2), p < 0.001. The BMI relative to the CDC 2000 reference population (BMI z-score) increased from –1.0 (1.2) in childhood to –0.5 (1.2) in adolescence, i.e. mean difference (95% CI) of 0.5 (0.4; 0.6), p < 0.001. Consequently, the prevalence of underweight declined from 27.3 to 18.8%, while the prevalence of overweight and obesity rose from 4.2 to 8.8% and from 1.9 to 3.2%, respectively. There was a higher prevalence of overweight and obesity in boys (table 1).

The correlation coefficient (r) between age 6–8 and age 11–13 measures was 0.73, p < 0.001 for BMI (0.78, p < 0.001 for boys and 0.65, p < 0.001 for girls) and 0.70, p < 0.001 for BMI z-score (0.71, p < 0.001 for boys and 0.67, p < 0.001 for girls). Overall, BMI in
childhood determined 52.3% variation (adjusted $R^2$, $p < 0.001$) of later BMI (61.1%, $p < 0.001$ for boys and 41.3%, $p < 0.001$ for girls). Similar to correlations, adjusted $R^2$ for BMI z-scores were slightly lower than for BMI, i.e. 48.2%, $p < 0.001$ (50.6%, $p < 0.001$ for boys and 44.4%, $p < 0.001$ for girls). Furthermore, a multiple regression analyses showed that adjusting for covariates such as age and sex did not significantly alter the association between the initial and the subsequent BMI or BMI z-scores.

As there was around 30% decrease in the prevalence of underweight, there were more children moved out from being classified as underweight than those moved in, i.e. 44.0 vs. 19.0%. On the other hand, since the prevalence of overweight was doubled, only 15% overweight children were not overweight adolescents compared to almost 60% overweight adolescents who were not overweight in childhood. However, the relative risk to be an overweight adolescent was still higher for overweight children (table 1).

All 6 obese children (5 boys and 1 girl) became obese adolescents. The relative risk for remaining obese, i.e. BMI z-score at the 95th percentile or above, was around 75 times than those not classified as obese (table 1). Furthermore, for those who were already classified as overweight (but not obese), the relative risk (95% CI) for becoming obese was 16.2 (2.0; 134.3), $p < 0.001$, i.e. 1 out of 6 overweight (but not obese) children becoming obese compared to 3 out of 292 not overweight children.

Cross-Sectional Analysis
Cross-sectional comparison between the 1999 and 2004 surveys showed no significance difference in BMI or BMI z-score. Mean (SD) BMI in 6- to 8-year-old children in 1999 and 2004 were 14.7 (1.8) and 14.9 (2.2), respectively. These BMIs had corresponding mean (SD) BMI z-scores of –0.9 (1.2) and –0.9 (1.4). However, from these two surveys it was observed that, while the prevalence of underweight was relatively constant, the prevalence of overweight and obesity tended to increase (table 2).

DISCUSSION
Prevalence and Tracking of BMI
This study describes the tracking of BMI as Indonesian urban children grew from childhood into adolescence. BMI z-score and percentiles based on the year 2000 sex-specific CDC 2000 growth chart were used throughout the analyses to facilitate international comparison and to allow combination of data across gender and age. This reference chart also enabled us to classify children and adolescence into underweight, overweight and obesity using one reference standard [12].
In this study an increase in BMI z-score as the children grew into adolescence was observed. Consequently, during the 5-year follow-up, the prevalence of underweight decreased, while the prevalence of overweight and obesity increased. However, BMI in childhood indeed tracked into adolescence, since BMI in childhood was highly correlated to BMI in adolescence. The correlation observed was similar to those in Australia (Caucasian children) and in Jamaica (Black children) [4, 14], and well above the correlation observed in China, which was only around 0.4 [8]. Overall, BMI in childhood determined around 50% of the variation in later BMI.

**Overweight and Obesity**

This study observed that the tracking pattern for overweight and obesity was similar to what had been reported earlier from developed countries [4, 6]. Most overweight children remained overweight and virtually all obese children stayed obese. The relative risk for overweight (obese) children to remain overweight (obese) was much higher than the risk for those not overweight (obese) to become overweight (obese). However, around half of the overweight (obese) adolescents were not overweight (obese) in childhood, because the prevalence of overweight and obesity were doubled as the children entering adolescence. Similar findings were observed in the USA (The Bogalusa Study), Australia and Iceland [4–7].

This study showed a tracking pattern which resembled more the pattern observed in developed countries rather than in developing countries, e.g. China. The explanation for this observation was not related to the fact that our sample comprised only urban children while those in China were both rural and urban, because the tracking for overweight and obesity in our sample was still significantly higher than that observed in urban Chinese children [8].

**Underweight**

There was approximately 30% decline in the prevalence of underweight as the children grew into adolescence. However, more than half of the underweight children remained underweight in adolescence. This prospective tracking was significantly higher than that observed in China, which was only around 33% [8]. The reason for high tracking for both overweight and underweight in our sample might be due to the fact that, unlike China, Indonesia was among the countries which underwent a widening gap in welfare [15].

Cross-sectional comparison on the prevalence of underweight, overweight and obesity between the 1999 and 2004 surveys also showed that, while the prevalence of overweight and obesity tended to increase, the prevalence of underweight seemed to be constant. Again, according to The United Nations Development Programme (UNDP), despite
an increase in gross domestic product of around 3.2% within a 10-year period (1990–1999),
the proportion of people with income poverty raised from 15 to 18% [15]. Our data might
indicate that those who were poor and had limited access to food remained underweight,
while those who were richer and exposed to unlimited energy intake tended to become
overweight. Unfortunately, we do not have data on tracking of energy intake nor
socioeconomic status to be able to explain this tracking of BMI.

CONCLUSION
In summary, the present results show that urban Indonesian children are increasing in
weight and BMI explained by the increase in the prevalence of overweight and obesity from
childhood into adolescence. Those who were overweight or obese during childhood were
most likely to remain overweight or obese in adolescence. On the other hand, despite a
decline in the prevalence of underweight from childhood into adolescence, the probability to
remain underweight in adolescence is more than 50%.

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Table 1. Tracking for underweight\textsuperscript{a}, overweight\textsuperscript{b} and obesity\textsuperscript{c} from childhood to adolescence (longitudinal analysis)

<table>
<thead>
<tr>
<th></th>
<th>Boys (n = 153)</th>
<th>Girls (n = 155)</th>
<th>All (n = 308)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Underweight\textsuperscript{a}</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prevalence of underweight\textsuperscript{a}</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>At the age of 6–8 years, n (%)</td>
<td>42 (27.5)</td>
<td>42 (27.1)</td>
<td>84 (27.3)</td>
</tr>
<tr>
<td>At the age of 11–13 years, n (%)</td>
<td>30 (19.8)</td>
<td>28 (18.1)</td>
<td>58 (18.8)</td>
</tr>
<tr>
<td>Agreement, $\kappa$\textsuperscript{d}</td>
<td>0.57*</td>
<td>0.64*</td>
<td>0.49*</td>
</tr>
<tr>
<td>Relative risk\textsuperscript{e} (95% CI)</td>
<td>17.2 (6.4; 46.3)*</td>
<td>8.1 (3.7; 17.6)*</td>
<td>11.4 (6.2; 20.9)*</td>
</tr>
<tr>
<td>% Prospective tracking\textsuperscript{f} (95% CI)</td>
<td>61.9 (45.7; 76.0)</td>
<td>50.0 (34.4; 65.6)</td>
<td>56.0 (44.7; 66.6)</td>
</tr>
<tr>
<td>% Retrospective tracking\textsuperscript{g} (95% CI)</td>
<td>86.7 (68.4; 95.6)</td>
<td>75.0 (54.8; 88.6)</td>
<td>81.0 (68.2; 89.7)</td>
</tr>
<tr>
<td><strong>Overweight\textsuperscript{b}</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prevalence of overweight\textsuperscript{b}</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>At the age of 6–8 years, n (%)</td>
<td>10 (6.5)</td>
<td>3 (1.9)</td>
<td>13 (4.2)</td>
</tr>
<tr>
<td>At the age of 11–13 years, n (%)</td>
<td>20 (13.1)</td>
<td>7 (4.5)</td>
<td>27 (8.8)</td>
</tr>
<tr>
<td>Agreement, $\kappa$\textsuperscript{d}</td>
<td>0.56**</td>
<td>0.38**</td>
<td>0.52**</td>
</tr>
<tr>
<td>Relative risk\textsuperscript{e} (95% CI)</td>
<td>11.7 (6.4; 21.4)**</td>
<td>20.3 (6.3; 65.7)**</td>
<td>15.6 (9.2; 26.5)**</td>
</tr>
<tr>
<td>% Prospective tracking\textsuperscript{f} (95% CI)</td>
<td>90.0 (54.1; 99.5)</td>
<td>66.7 (12.5; 98.2)</td>
<td>84.6 (53.7; 97.3)</td>
</tr>
<tr>
<td>% Retrospective tracking\textsuperscript{g} (95% CI)</td>
<td>45.0 (23.8; 68.0)</td>
<td>28.6 (5.1; 69.7)</td>
<td>40.7 (23.1; 61.0)</td>
</tr>
<tr>
<td><strong>Obesity\textsuperscript{c}</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prevalence of obesity\textsuperscript{c}</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age Group</td>
<td>Underweight</td>
<td>Overweight</td>
<td>Obesity</td>
</tr>
<tr>
<td>---------------------------</td>
<td>-------------</td>
<td>------------</td>
<td>---------</td>
</tr>
<tr>
<td>6–8 years</td>
<td>5 (3.3)</td>
<td>1 (0.6)</td>
<td>6 (1.9)</td>
</tr>
<tr>
<td>11–13 years</td>
<td>7 (4.6)</td>
<td>3 (1.9)</td>
<td>10 (3.2)</td>
</tr>
<tr>
<td>Agreement, κ&lt;sup&gt;d&lt;/sup&gt;</td>
<td>0.83**</td>
<td>0.50**</td>
<td>0.74**</td>
</tr>
<tr>
<td>Relative risk&lt;sup&gt;e&lt;/sup&gt; (95% CI)</td>
<td>74.0 (18.7; 293.1)**</td>
<td>77.0 (19.4; 305.1)**</td>
<td>75.5 (28.5; 199.9)**</td>
</tr>
<tr>
<td>% Prospective tracking&lt;sup&gt;f&lt;/sup&gt; (95% CI)</td>
<td>100.0 (46.4; 100.0)</td>
<td>100 (5.5; 100.0)</td>
<td>100.0 (51.7; 100.0)</td>
</tr>
<tr>
<td>% Retrospective tracking&lt;sup&gt;g&lt;/sup&gt; (95% CI)</td>
<td>71.4 (30.3; 94.9)</td>
<td>33.0 (1.8; 87.5)</td>
<td>71.4 (30.2; 94.9)</td>
</tr>
</tbody>
</table>

p < 0.001 (Pearson χ<sup>2</sup>); ** p < 0.001 (Fisher’s exact test).

<sup>a</sup> Underweight: BMI below the 5<sup>th</sup> percentiles.
<sup>b</sup> Overweight: BMI equal to or above the 85<sup>th</sup> percentiles.
<sup>c</sup> Obesity: BMI equal to or above the 95<sup>th</sup> percentiles of the CDC 2000 reference population.
<sup>d</sup> κ (kappa) level of agreement in classification of underweight, overweight or obesity between age 6–8 and 11–13 years.
<sup>e</sup> Relative risk for underweight (overweight or obese) children to remain underweight (overweight or obese) compared to the risk to be underweight (overweight or obese) in not underweight (not overweight or not obese children).
<sup>f</sup> Prospective tracking: percentage of those underweight (overweight or obese) at the age of 6–8 years that were still underweight (overweight or obese) at the age of 11–13 years.
<sup>g</sup> Retrospective tracking: percentage of those underweight (overweight or obese) at the age of 11–13 years that were already underweight (overweight or obese) at the age 6–8 years.
Table 2. Prevalence (%) of underweight\textsuperscript{a}, overweight\textsuperscript{b} and obesity\textsuperscript{c} in 6- to 8-year-old children from the 1999 and 2004 surveys (cross-sectional analysis)

<table>
<thead>
<tr>
<th></th>
<th>Prevalence of</th>
<th>overweight\textsuperscript{b}</th>
<th>obesity\textsuperscript{c}</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>underweight\textsuperscript{a}</td>
<td></td>
<td></td>
</tr>
<tr>
<td>All</td>
<td>1999 (n = 1,524)</td>
<td>25.0</td>
<td>5.3</td>
</tr>
<tr>
<td></td>
<td>2004 (n = 510)</td>
<td>25.9</td>
<td>8.6*</td>
</tr>
<tr>
<td>Boys</td>
<td>1999 (n = 754)</td>
<td>26.5</td>
<td>5.8</td>
</tr>
<tr>
<td></td>
<td>2004 (n = 238)</td>
<td>29.8</td>
<td>8.4</td>
</tr>
<tr>
<td>Girls</td>
<td>1999 (n = 770)</td>
<td>23.5</td>
<td>4.8</td>
</tr>
<tr>
<td></td>
<td>2004 (n = 272)</td>
<td>22.4</td>
<td>8.8**</td>
</tr>
</tbody>
</table>

\* Significantly different to the prevalence of the 1999 survey, \( p=0.007 \) (Pearson \( \chi^2 \)); \textbf{**} \( p = 0.02 \).

\textsuperscript{a} Underweight: BMI below the 5\textsuperscript{th} percentiles.

\textsuperscript{b} Overweight: BMI equal to or above the 85\textsuperscript{th} percentiles.

\textsuperscript{c} Obesity: BMI equal to or above the 95\textsuperscript{th} percentiles of the CDC 2000 reference population.
Fig. 1. Selection of samples for cross-sectional and longitudinal analyses. Cross-sectional analyses: 1,524 urban children 6–8 years old from a total of 3,010 (urban and rural) children in the 1999 survey and 510 children 6–8 years old from a total of 2,497 urban children 6–13 years old in the 2004 survey. Longitudinal analyses: 308 children who had their body weight and height measured in both surveys. In the 1999 survey they were between 6 and 8 years old, while in the second survey they were between 11 and 13 years old.
Chapter 6.

The influence of socioeconomic status on blood pressure of Indonesian prepubertal children

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†† Passed away

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http://www.nature.com/jhh/journal/v20/n7/pdf/1002028a.pdf
This cross-sectional study assesses the association between socioeconomic status and blood pressure of school-aged prepubertal children living in Indonesia. It has been shown that elevated blood pressure (BP) contributes to the development of coronary artery disease and to the pathogenesis of cerebrovascular accidents. There is also evidence of some correlations between childhood BP and BP in adulthood. In contrast to consistently negative association between socioeconomic status (SES) and BP in industrialized countries, findings in developing countries are heterogeneous. The aim of the present study was to evaluate the association between nutritional status and SES, defined as rural, poor urban and nonpoor urban, and BPs of school-aged prepubertal children living in Indonesia.

A cross-sectional study was conducted in school-aged prepubertal children in two adjacent areas in Central Java, Indonesia. Definitions of rural and urban populations were based on agricultural activities and population densities. Yogyakarta, the urban area, was a city with a population density of 14 988/km², whereas Gunung Kidul, a rural area located about 20–40 km from Yogyakarta, had a population density of 498/km². In the urban area, the SES of each child was individually deduced from his or her living environment. Children living in the urban slum area were considered poor, and those not living in this area were considered to be not poor.

We randomly selected 33 of 509 public primary schools in the rural area and 37 of 172 public primary schools in the urban area. Prepubertal children (under 8 years old for girls and under 9 years old for boys) from the first-and second-year class of every school were included. The ages of the children finally studied ranged from 6 to 7.9 years for girls and from 6 to 8.9 years for boys. The study was performed from February to May 1999.

Anthropometric data were collected by health professionals. Heights and weights were measured using the standard techniques described by the World Health Organization (WHO). Blood pressure was measured using the standard techniques described by the Task Force on Blood Pressure Control in Children, using one mercury-gravity manometer with two sets of cuff, a children cuff (for arm circumferences <18 cm) and a small adult cuff (for arm circumferences ≥18 cm) (Riester, Germany).

The body mass index (BMI) was calculated by dividing the weight in kilograms by the square of the height in metres (kg/m²). Data on BMI-for-age were converted to z-scores based on the year 2000 sex-specific Center for Disease Controls and Prevention (CDC) growth charts using the nutritional anthropometry module (NutStat) of the CDC's Epi Info 2000 (Centers for Disease Control and Prevention, Atlanta, GA, USA). Data on height-for-age were also converted to z-scores using the same above references and statistical
software. Children were classified as stunted if they had height-for-age z-scores (HAZ) below -2.00, and as not stunted if they had HAZ of -2.00 or more.\textsuperscript{4}

Of the 3010 children for whom anthropometric measurements were available and considered valid, we were only able to measure BP of 2922 children, that is, 1169 rural children (96% of the eligible rural children) and 1753 urban children (98%). Children from the urban area were then subdivided into poor (431, from the urban slum area) and nonpoor (1322).

Rural and poor urban children had significantly lower mean HAZ and BMI-for-age z-scores (BMIZ) than nonpoor urban children. The rural communities seemed to be the poorest among the three SES groups studied as they had the largest prevalence of stunted growth, that is, 28% as compared to 17% in poor urban children and 12% in nonpoor urban children. Stunted growth reflected long-term cumulative inadequacies of health and nutrition, and had been strongly associated to poverty.\textsuperscript{4,6}

The mean (s.d.) systolic and diastolic BP of our subjects was 99.6 (10.0) mm Hg for systolic BP and 59.6 (10.3) mm Hg for diastolic BP at the fourth Korotkoff sound. The poor urban children had significantly lower systolic and diastolic BP than the nonpoor urban children. On the other hand, there were no significant difference in both systolic and diastolic BP of rural children and nonpoor urban children.

Linear regression analyses, either separately or adjusted for every other variable in the models, showed that both systolic and diastolic BPs were independently and positively related to age, HAZ and BMIZ. In the model comparing rural and nonpoor urban children, nonsignificant lower systolic and diastolic BPs were observed in the rural children, that is, regression coefficients (95% confidence interval (CI)) of -0.66 (-1.45 to 0.13) mm Hg for systolic BP and -0.71 (-1.51 to 0.08) mm Hg for diastolic BP. After adjustment for sex, age, HAZ and BMIZ, the association of SES to systolic BP inversed; the systolic BP of rural children was significantly higher than those of nonpoor urban children, that is, regression coefficients (95% CI) of 1.19 (0.42–1.96) mm Hg. Although not statistically significant, the diastolic BP of rural children after adjustment for the above confounding factors was also higher than those of the nonpoor urban children.

On the other hand, in the models comparing poor urban and nonpoor urban children, the influence of SES on both systolic and diastolic BPs diminished after adjusting for age, stature and BMI, that is, showing that the influence of SES on BPs in these two groups of urban children was more related to the difference in the distribution of stature and BMI rather than the influence of the SES per se (Table 1).
A study of 3157 people born in 1946 in Britain showed that poor childhood growth provided some of the explanation for the influence of childhood social class gradient on later BP. However, a Finnish cohort study of people born in 1934–44 found an enhanced effect of negative association of poor childhood growth with adult BP among those grown up in poor social conditions. So far, it is not yet clear whether the influence of childhood SES on later BP is mediated, at least partly, through the growth in early life.

Albeit small, that is, 1.19 mm Hg (95% CI 0.42–1.96), the elevated systolic BP observed in these rural children was independent of other important predictors of BP in childhood, that is, sex, age, stature and BMI. Studies in adults in developing countries found a difference of around 2–5 mm Hg between SES groups studied. In summary, our study indicated that the poorer segment of the population, that is, the rural community, faced a higher risk for developing hypertension, which might be related to the observed higher prevalence of stunted growth in this community. As one-third of the children in developing countries become growth retarded before the age of 5 years, if linear growth retardation potentiates the effects of later weight gain on BP, then stunting in early childhood will be an important contributor to the rise of cardiovascular disease in developing countries. For disease prevention, we should therefore focus on optimization of fetal and infant growth.

Summary table
What is known about this topic:
- Obesity is associated with higher BP, both in children and in adults
- In developed countries, lower SES is associated with higher BP

What this study adds:
- The prevalence of obesity in prepubertal children in Indonesia is higher in the upper SES, so they seem to have higher BP
- However, for a given BMI, poor children had higher systolic BP than nonpoor children

References


**Tabel 1** Adjusted and unadjusted regression coefficients ($\beta$) for systolic and diastolic BP as the dependent variables and sex, age, HAZ, BMIZ and SES as the independent variables.

*(a) Systolic BP (mmHg)*

<table>
<thead>
<tr>
<th>Variables</th>
<th>Unadjusted $\beta^a$ (95%CI)</th>
<th>Adjusted*b $\beta^a$ (95%CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex (1=male, 0=female)</td>
<td>0.49 (-0.24 to 1.23)</td>
<td>0.42 (-0.35 to 1.18)</td>
</tr>
<tr>
<td>Age (months)</td>
<td>0.07 (0.03 to 0.12)**</td>
<td>0.12 (0.07 to 0.17)**</td>
</tr>
<tr>
<td>HAZ</td>
<td>2.57 (2.21 to 2.94)***</td>
<td>2.23 (1.81 to 2.65)***</td>
</tr>
<tr>
<td>BMIZ</td>
<td>2.83 (2.53 to 3.13)***</td>
<td>2.38 (2.05 to 2.71)***</td>
</tr>
<tr>
<td>SES (rural=1, nonpoor urban=0) (n=2491)</td>
<td>-0.66 (-1.45 to 0.13)</td>
<td>1.19 (0.42 to 1.96)**</td>
</tr>
<tr>
<td>SES (poor=1, nonpoor urban=0) (n=1753)</td>
<td>-1.79 (-2.90 to -0.67)**</td>
<td>...</td>
</tr>
<tr>
<td>Constant</td>
<td>--</td>
<td>93.3 (88.9 to 97.7)***</td>
</tr>
</tbody>
</table>

Adjusted R square: 0.15, 0.17

*(b) Diastolic BP at the fourth Korotkoff sound (mmHg)*

<table>
<thead>
<tr>
<th>Variables</th>
<th>Unadjusted $\beta^a$ (95%CI)</th>
<th>Adjusted*b $\beta^a$ (95%CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex (1=male, 0=female)</td>
<td>-0.42 (-1.18 to 0.34)</td>
<td>-0.41 (-1.22 to 0.40)</td>
</tr>
<tr>
<td>Age (months)</td>
<td>0.09 (0.04 to 0.14)**</td>
<td>0.13 (0.08 to 0.18)**</td>
</tr>
<tr>
<td>HAZ</td>
<td>1.89 (1.50 to 2.28)***</td>
<td>1.71 (1.27 to 2.15)***</td>
</tr>
<tr>
<td>BMIZ</td>
<td>1.84 (1.52 to 2.16)***</td>
<td>1.43 (1.08 to 1.77)***</td>
</tr>
<tr>
<td>Model Description</td>
<td>Coefficient</td>
<td>Lower CI</td>
</tr>
<tr>
<td>-------------------</td>
<td>-------------</td>
<td>----------</td>
</tr>
<tr>
<td>SES (rural=1, nonpoor urban=0) (n=2491)</td>
<td>-0.71</td>
<td>(-1.51 to 0.08)</td>
</tr>
<tr>
<td>SES (poor=1, nonpoor urban=0) (n=1753)</td>
<td>-1.68</td>
<td>(-2.82 to -0.53)**</td>
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<tr>
<td>Constant</td>
<td>51.6</td>
<td>(46.9 to 56.2)***</td>
</tr>
</tbody>
</table>

Adjusted R square: 0.07

Abbreviations: BMIZ, body mass index-for-age z-scores; BP, blood pressure; HAZ, height-for-age z-scores; SES, socioeconomic status.

Model 1 is comparing rural to nonpoor urban children, whereas model 2 is comparing poor to nonpoor urban children.

*P<0.05; **P<0.01; ***P<0.001.

\[ \beta \]: unstandardized coefficient

\[ ^{a} \]: With adjustment for every other variable in the model.
Chapter 7.

The Influence of socioeconomic status and birth weight on blood pressure of Indonesian prepubertal children

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Submitted
ABSTRACT

Objective. To assess the influence of socioeconomic status and birth weight on blood pressure of school-aged prepubertal children living in Indonesia.

Subjects and Methods. A cross-sectional survey in 2922 school-aged prepubertal children from the rural, poor urban and nonpoor urban communities was performed. Data on age, sex, stature, BMI, birth weight, systolic and diastolic BP were collected from all children.

Results. Overall and within every socioeconomic status group, blood pressures were positively associated with stature and body mass index (BMI). Children from poor-socioeconomic families, i.e. rural and poor urban, had significantly lower height and BMI, and hence, in the unadjusted analyses, poor socioeconomic status was associated with lower systolic and diastolic BP. However, after adjustment for age, sex, stature and BMI, rural children was found to have significantly higher systolic BP compared to nonpoor urban children, i.e. regression coefficient (95% CI) of 1.19(0.42 to 1.96). Birth weight was not associated with blood pressure in childhood.

Conclusion. This study indicated that for a given stature and BMI, poor children had a higher systolic BP.

Key words: socioeconomic status, blood pressure, prepubertal children, Indonesia
INTRODUCTION

It has been shown that elevated blood pressure (BP) contributes to the development of coronary artery disease and to the pathogenesis of cerebrovascular accidents, heart failure, and renal failure in adults [1]. There is also evidence of some correlations between childhood BP and BP in adulthood [2]. Moreover, elevated BP in childhood, along with obesity, is significantly associated with carotid vascular changes and increased left ventricular mass, both in childhood and adulthood [3,4,5].

Both obesity and a low birth weight are a risk factor for elevated BP, both in children and in adults [6,7,8,9]. Most epidemiological studies have also shown a relationship between high BP and socioeconomic status (SES) [10,11]. In industrialized countries, individuals of lower SES have been reported to have higher blood pressure both during childhood and adulthood, which may be related to obesity, since obesity is more prevalent among this population [12]. However, at present, in contrast to the Western populations, obesity is not a problem among people of a lower socioeconomic class in developing countries [13].

In contrast to consistently negative association between SES and BP in industrialized countries, findings in developing countries are heterogeneous [11,14,15,16]. The basis of these inconsistent associations of SES and BP in developing countries is not clear. One explanation might be due to the variation in the stages of modernization, economic development or adoption of westernized lifestyles between various communities [15]. One of the most marked societal and environmental changes in developing countries is urbanization [17,18]. With urbanization, a marked increase in consumption of energy rich as well as salted foods and a decrease in energy expenditure through less physical activity is observed [19,20].

Low childhood socioeconomic circumstances have been shown to have long lasting negative influences on adult health, i.e. poorer cardiorespiratory fitness, higher systolic BP, and higher prevalence of obesity, irrespective of where one ends-up in the socioeconomic hierarchy as an adult [21,22]. The effect of these adverse socioeconomic circumstances might or might be not associated with pre- and postnatal growth [22,23]. Therefore, the aim of the present study is to evaluate the association between nutritional status and SES, defined as rural, poor urban and nonpoor urban and blood pressures of school aged prepubertal children living in Indonesia. The contribution of birth weight on the actual blood pressure in the population studied was also taken into account.
SUBJECTS AND METHODS

Study population and design
The study was performed in two adjacent areas in Central Java. Yogyakarta, an urban area, was a city with approximately 487,115 inhabitants at the time of the study. Gunung Kidul, a rural area located about 20 to 40 km from Yogyakarta, had approximately 710,691 inhabitants [24]. We chose these two study areas because of the relatively homogeneous ethnicity of their populations. Most of the people in both areas are of Javanese descent. The altitude of both study areas is less than 500 m above sea level [24].

A cross-sectional study was conducted in school-aged prepubertal children in both areas. We randomly selected 33 of 509 public primary schools in the rural area and 37 of 172 public primary schools in the urban area. In Indonesia, it is obligatory for children to enter primary school at the age of six and seven years. Prepubertal children (under eight years old for girls and under nine years old for boys) from the first- and second-year class of every school were included. The ages of the children finally studied ranged from 6 to 7.9 years for girls and from 6 to 8.9 years for boys. This study selected prepubertal children because puberty, which normally occurs after the age of eight in girls and nine in boys, may interfere with the interpretation of anthropometric measurements due to changes in body composition and differences between children in the timing of the adolescent growth spurt. The results of the study on anthropometric status of these prepubertal children had been reported elsewhere [25].

We excluded children with prominent chronic diseases, such as congenital heart disease or major thalassemia as well as children with physical handicaps that might interfere with the measurements. The study was performed from February to May 1999 and was approved by the ethical committee of Gadjah Mada University, Yogyakarta, Indonesia.

Data collection
Anthropometric data were collected by health professionals. Heights and weights were measured using the standard techniques described by the World Health Organization (WHO) [26]. Training for standardization of the measurements, followed by field practice and testing, was performed prior to data collection. All measurements were performed between 8 and 10 a.m. The children, wearing light clothing, were weighed to the nearest 0.1 kg with a Seca digital scale (Germany). Height was measured to the nearest 0.1 cm with a portable stadiometer. Height was measured with the child standing facing the fieldworker, without shoes. Right mid-upper-arm circumference was measured at a point midway between the
olecranon and the acromion, using a non-stretchable measurement tape, to the nearest 0.1 cm.

Blood pressure (BP) was measured using the standard techniques described by the Task Force on Blood Pressure Control in Children [27]. To minimize the children’s apprehension, a physician, helped by the teacher in the class, first explained the procedure of blood pressure measurement to all children. All measurements were performed in the classrooms of the children by one physician using one mercury-gravity manometer with two sets of cuff, a children cuff (for arm circumferences of 13 to 20 cm) and a small adult cuff (for arm circumferences of 17 to 26 cm) (Riester, Germany). After at least 10 minutes rest from recent activity, children were asked to sit in a comfortable sitting position with their right arm fully exposed and resting on a supportive surface at the heart level. Blood pressures were measured on the right arm, using appropriate cuff size, i.e. the children cuff, when their arm circumferences were less than 18 cm, or the small adult cuff when their arm circumferences were 18 cm or above.

The cuff was then rapidly inflated to about 20 mm Hg above the point at which the radial pulse disappears. The pressure within the cuff is then released at a rate about 2 to 3 mm Hg per second while auscultation was performed over the brachial artery. The systolic BP was determined by the onset of the “tapping” Korotkoff sound. The diastolic BP were defined as both, the muffling of the Korotkoff sounds (the fourth Korotkoff sound) and the disappearance of the sound (the fifth Korotkoff sound)[1,27]. Both systolic and diastolic BP were recorded to the nearest 2 mm Hg.

Information on birth weight was collected using a questionnaire sent to the parents with the help of the teachers. Birth dates were verified by a copy of the child’s birth certificate filed at the school. Definitions of rural and urban populations were based on agricultural activities and population densities. Yogyakarta had a population density of 14 988/km², while Gunung Kidul had a population density of 498/km² [24]. In the urban area, the socioeconomic status of each child was individually deduced from his or her living environment. Children living in the urban slum area were considered poor, and those not living in this area were considered to be not poor.

**Statistical analyses**
The body mass index (BMI) was calculated by dividing the weight in kilograms by the square of the height in meters (kg/m²). Data on bmi-for-age were converted to z-scores and percentiles based on the year 2000 sex specific Center for Disease Controls and Prevention (CDC) growth charts using the nutritional anthropometry module (NutStat) of the CDC’s Epi
Info 2000 (Centers for Disease Control and Prevention, Atlanta, Georgia, USA). Children were classified as obese if they had a BMI-for-age at the 95th percentiles or above based on the year 2000 CDC growth charts [28].

Data on height-for-age were also converted to z-scores using the same above references and statistical software. Biologically implausible values, such as z-scores below -6.00 or above +6.00, were excluded from the analysis. Values that were most likely to represent errors, i.e., those that were 4 z-score units outside the observed mean for every age in one full year, were also excluded (flexible exclusion range). Children were classified as stunted if they had height-for-age z-scores (HAZ) below -2.00, and as not stunted if they had HAZ of −2.00 or more [29].

Children were considered to have low birth weights (LBW) when their birth weights were below the 10th percentile of our study subjects’ sex-specific birth weight distribution, and considered to have high birth weights (HBW) when they were above the 90th percentiles. Children who were born between the 10th and the 90th percentile were considered to have normal birth weight (NBW).

All statistical analyses were performed using SPSS for Windows (Version 9, SPSS, Chicago, Illinois, USA). The mean and distribution of continuous data, i.e. data on age, height-for-age z-scores (HAZ), BMI-for age z-scores (BMIZ), systolic and diastolic BP between the three socioeconomic status groups, between stunted and not stunted children, between obese and not obese children and between the three birth weight groups were compared using t-tests. Estimates differences were presented as mean difference and 95% confidence interval (95% CI).

Univariate regression analyses were used to assess the associations between systolic and diastolic BP as the dependent variables and socioeconomic status groups, sex, age, HAZ and BMIZ as the independent variables separately. Multiple regression analyses were then performed to estimates the independent contribution of socioeconomic status to blood pressures adjusted for sex, age, HAZ and BMIZ with or without birth weight status. Two sets of multiple regression models were constructed, one set of models was to compare rural to nonpoor urban children while the other set was to compare poor urban to nonpoor urban children.

To assess predictors of systolic and diastolic BP within the rural, poor urban and nonpoor urban children or within the LBW, NBW and HBW children separately, multiple regression models with sex, age, HAZ and BMIZ as the independent variables and systolic and diastolic BP as the dependent variables were constructed for each socioeconomic status.
and birth weight group. Regression coefficients were presented as unstandardized estimates (95% CI). Statistical significances (α) were set to p<0.05.

RESULTS
We visited 70 schools to examine a total of 3689 prepubertal children who were listed as first- and second-year students. We excluded 674 children because they were not in the required age range. Of the remaining 3015 children, four missed the anthropometric measurement session and one had a HAZ in the flexible exclusion range.

Of the 3010 children for whom anthropometric measurements were available and considered valid, we were only able to measure blood pressure of 2922 children, i.e. 1169 rural children (96% of the eligible rural children) and 1753 urban children (98%). The characteristics of the 88 children without the blood pressure measurements were comparable to the rest of the children except that they were significantly older.

Children from the urban area were then subdivided into 431 poor (from the urban slum area) and 1322 nonpoor. The total population studied consists of 1690 (57.8%) boys and 1232 girls. The three groups did not differ with respect to the ratio of males to females (p≥0.05), i.e. 59%, 58% and 57% boys in the rural, poor urban and nonpoor urban, respectively.

Rural and poor urban children had significantly lower mean height-for-age z-scores (HAZ) and BMI-for-age Z-scores (BMIZ), compared to nonpoor urban children (Table 2). The overall prevalence of stunted growth, i.e. HAZ less than −2.00, was 19%. The prevalence of stunted growth was 28% in the rural, 17% in the poor urban and 12% in the nonpoor urban children. On the other hand, the prevalence of obesity, i.e. having a BMI centile of 95th or above, being 2.0% overall, was highest among the nonpoor urban children, i.e. 3.5%, compared to among the poor urban (0.7%) and among the rural children (0.7%).

The mean (SD) systolic and diastolic blood pressures (BPs) of our subjects was 99.6 (10.0) mmHg for systolic BP, 59.6 (10.3) mmHg for diastolic BP at the 4th Korotkoff (K4) sound and 49.4 (15.4) for diastolic BP at the 5th Korotkoff (K5) sound. The poor urban children had significantly lower systolic and diastolic BP than the nonpoor urban children. On the other hand, there were no significant difference in both systolic and diastolic BP of rural children and nonpoor urban children (Table 1).

Stunted children had significantly lower mean of systolic and diastolic BPs, mean(SD) systolic BP of 96.5 (8.8) mmHg in stunted children compared to 100.3 (10.1) in not stunted children, mean(SD) K4 of 57.3 (10.0) mmHg in stunted children compared to 60.2 (10.3) in not stunted children, and mean(SD) K5 of 57.3 (10.0) mmHg in stunted children compared
to 60.2 (10.3) in not stunted children. Similar findings were observed when the data were stratified into each socioeconomic status groups. Obese children had significantly higher mean of systolic and diastolic BPs compared to not obese children (data not shown).

Linear regression analyses, either separately or adjusted for every other variable in the models, showed that both systolic and diastolic BPs were independently and positively related to age, HAZ and BMIZ. In the model comparing rural and nonpoor urban children, a non-significant lower systolic and diastolic BPs were observed in the rural children, i.e. regression coefficients (95% CI) of –0.66 (-1.45 to 0.13) mmHg for systolic BP and –0.71 (-1.51 to 0.08) mmHg for diastolic BP. After adjustment for sex, age, HAZ and BMIZ, the association of socioeconomic status with systolic BP inversed, the systolic BP of rural children was significantly higher than those of nonpoor urban children, i.e. regression coefficients (95% CI) of 1.19 (0.42 to 1.96) mmHg. Although not statistically significant, the diastolic BP of rural children after adjustment for the above confounding factors was also higher than those of the nonpoor urban children (Table 2).

On the other hand, comparing poor urban children to nonpoor urban children showed that in the unadjusted model, socioeconomic status was significantly associated with both systolic and diastolic BPs, i.e. poor urban children had significantly lower systolic and diastolic BPs. But, in the model adjusted for sex, age, HAZ and BMIZ, the association between socio-economic status and BPs diminished (Table 2). Comparing predictors of systolic and diastolic BPs showed that, within each socioeconomic status group, age, HAZ and BMIZ were independently and positively associated with both systolic and diastolic BPs (Table 3).

Of the 2922 children, 2750 (94.1%) reported their birth weight; i.e. 1079 out of 1169 (92.3%) rural children, 348 out of 431 (92.3%) poor urban children and 1273 out of 1322 (96.3%) nonpoor urban children. There were no significance difference in the prevalence of having low birth weight (LBW) between the rural, i.e. prevalence of 7.9%, the poor urban (9.4%) and the nonpoor urban children (6.9%) (p≥0.05). However, the nonpoor urban children had significantly higher odds to have high birth weight (HBW), i.e. the prevalence of HBW of 12.4%, compared to 6.8% in the rural children and 7.2% in the poor urban children.

There were no difference in the mean systolic and diastolic BP of LBW children compared to those of NBW children but there were significantly higher systolic and diastolic BP at the 4th Korotkoff sound of HBW children compared to NBW children (Table 4). Since the mean HAZ and BMIZ of HBW children were also significantly higher than those of the NBW children, adjusting the regression of birth weight to BPs with BMIZ and HAZ showed that birth weight was not, anymore, a significant predictor of either systolic or diastolic BPs.
Putting the variable birth weights into the models in Table 2 and 3 did not significantly altered the models. Within each birth weight group, systolic and diastolic BPs were significantly associated with BMIZ (data not shown).

**DISCUSSION**

Our study, similar to other previous studies in both developing and developed countries, showed that systolic and diastolic blood pressures (BPs) in children were positively associated with age, stature and BMI [6,7,8,9]. Similar associations existed within every socioeconomic status groups studied, i.e. age, stature and BMI were significant predictors of blood pressures both in low and high socioeconomic status children.

We observed that, in average, the systolic and diastolic BPs of poor urban children were lower than those of non-poor urban children, while the BPs of rural children were not significantly lower than those of the non-poor urban children. Rural children had average BPs comparable to non-poor urban children despite the fact that they had, in average, lower BMI-for-age z-scores (BMIZ) and height-for-age z-scores (HAZ).

In the models comparing poor urban and non-poor urban children, the influence of socioeconomic status on both systolic and diastolic BPs diminished after adjusting for age, stature and BMI, i.e. showing that the influence of socioeconomic status on BPs in these two groups of urban children was more related to the difference in the distribution of stature and BMI, rather than from the influence of the socioeconomic status per se.

Comparing rural children to non-poor urban children showed different things. In the unadjusted models, systolic and diastolic BPs of rural children were not different. When the models were adjusted for sex, age, stature and BMI, the rural children had significantly higher systolic BP compared to non-poor urban children of a given stature and BMI. Although not statistically significant, similar associations were also observed for diastolic BP.

In association with birth weight, our study observed that higher birth weight children had higher systolic BP. The association was explained by higher current BMIZ and HAZ in HBW children.

The rural communities in this study seemed to be the poorest among the three SES groups studied since they had the largest prevalence of stunted growth, i.e. 28%, compared to 17% in poor urban children and 12% in non-poor urban children. Stunted growth reflected long-term cumulative inadequacies of health and nutrition, and had been strongly associated with poverty [29,30]. However, stunted growth was not the only risk factor for raised BP in these rural children, as what had been suggested by a study in Brazil [31],
because even in this poor population, BPs were positively associated with both height and BMI.

Poor rural communities in developing countries are at higher risk for suffering from impaired prenatal and early postnatal growth, since these are the times when growth is most vulnerable to the adverse effect of poverty, poor nutrition and high prevalence of infectious diseases [30,32,33,34]. However, studies showed that some of these growth retarded infants did exhibit catch up growth at later ages [35,36]

The associations between impaired fetal growth and raised blood pressure in later life, either in developing or developed countries, have been widely reported [37,38]. Some studies have also observed the relation of rapid growth in late childhood or adolescent period to higher BP later in life [6,36,39]. Nevertheless, the influence of infant growth to later BP has been variable.

Walker et al. 2001, in a study in Jamaica found no difference in systolic or diastolic BPs between stunted and non stunted children. However, he reported larger effects of increasing weight on systolic BP of 11 to 12 year-old Jamaican children who were stunted between the age of 9 to 24 months [36]. Cheung et al. observed that after controlling for birth length and ponderal index, the change in ponderal index from 6 to 18 months was inversely associated with systolic BP in young Hongkong adults [40]. Similarly, Adair and Cole, in a longitudinal study in The Philippines showed that growth rate in infancy was negatively associated with later BP[39]. On the other hand, several studies in developed countries had failed to observe any association between growth in infancy and later BP once birth weight had been taken into account [41,42].

A study of 3157 people born in 1946 in Britain showed that poor childhood growth provided some of the explanation for the influence of childhood social class gradient on later blood pressure [23]. However, A Finnish cohort study of people born in 1934-44 found an enhanced effect of negative association of poor childhood growth with adult BP among those grown up in poor social conditions [22]. So far, it is not yet clear whether the influence of childhood socioeconomic status on later blood pressure is mediated, at least partly, through the growth in early life.

The much higher prevalence of stunted growth observed in the rural children in our study indicated that the rural community had more probability to suffer from pre- and postnatal growth restriction. If linear growth retardation in early life did indeed potentiate the effects of later weight gain on blood pressure, this and the poor socioeconomic circumstances might be the explanation for the observed higher systolic BP in the rural
children in our study. But of course, due to the cross sectional nature of our study, we could not verify this hypothesis.

Albeit small, i.e. 1.19 mmHg (95%CI 0.42 to 1.96), the elevated systolic BP observed in these rural children was independent of other important predictors of BP in childhood, i.e. sex, age, stature and BMI. Studies in adults in developing countries found a difference of around 2 to 5 mmHg between SES groups studied [14,16]. Previous studies in Indonesian adults found no consistent difference in the prevalence of hypertension between rural and urban population [43]. In 1986, a study in some subdistricts in Yogyakarta observed a slightly higher prevalence of hypertension in rural children (aged 6 to 14 years old) compared to urban children [44].

In summary, our study indicated that the poorer segment of the population, i.e. the rural community, faced a higher risk for developing hypertension, which might be related to the observed higher prevalence of stunted growth in this community. Since one third of the children in developing countries become growth retarded before the age of 5 years [45], if linear growth retardation potentiates the effects of later weight gain on blood pressure, then stunting in early childhood will be an important contributor to the rise of cardiovascular disease in developing countries. For disease prevention, we should therefore focus on optimization of fetal and infant growth.

References
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Table 1. Comparison of height-for-age (HAZ), body mass index-for-age z-scores (BMIZ), systolic and diastolic blood pressure (BP) of children from different socioeconomic status (SES) groups

<table>
<thead>
<tr>
<th>Variables</th>
<th>Mean (SD)</th>
<th>Mean difference (95% CI)</th>
<th>p</th>
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<td>Height-for-age z-scores (HAZ)</td>
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<tr>
<td>Rural (n=1169)</td>
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<td>&lt;0.001</td>
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<td>Poor urban (n=431)</td>
<td>-1.27 (0.85)</td>
<td>-0.40 (-0.50 to -0.30)</td>
<td>&lt;0.001</td>
</tr>
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<td>Nonpoor urban (n=1322)</td>
<td>-0.87 (0.94)</td>
<td>0 (reference)</td>
<td>-</td>
</tr>
<tr>
<td>Body mass index-for-age z-scores (BMIZ)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rural (n=1169)</td>
<td>-1.08 (1.01)</td>
<td>-0.24 (-0.33 to -0.15)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Poor urban (n=431)</td>
<td>-1.12 (1.08)</td>
<td>-0.28 (-0.40 to -0.16)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Nonpoor urban (n=1322)</td>
<td>-0.84 (1.26)</td>
<td>0 (reference)</td>
<td>-</td>
</tr>
<tr>
<td>Systolic BP (mmHg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rural (n=1169)</td>
<td>99.5 (9.5)</td>
<td>-0.66 (-1.45 to 0.13)</td>
<td>0.10</td>
</tr>
<tr>
<td>Poor urban (n=431)</td>
<td>98.4 (9.6)</td>
<td>-1.79 (-2.90 to -0.67)</td>
<td>0.002</td>
</tr>
<tr>
<td>Nonpoor urban (n=1322)</td>
<td>100.1 (10.5)</td>
<td>0 (reference)</td>
<td>-</td>
</tr>
<tr>
<td>Diastolic BP at the 4th Korotkoff sound (mmHg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rural (n=1169)</td>
<td>59.5 (10.0)</td>
<td>-0.71 (-1.51 to 0.08)</td>
<td>0.08</td>
</tr>
<tr>
<td>Poor urban (n=431)</td>
<td>58.5 (11.3)</td>
<td>-1.68 (-2.82 to -0.53)</td>
<td>0.004</td>
</tr>
<tr>
<td>Nonpoor urban (n=1322)</td>
<td>60.2 (10.2)</td>
<td>0 (reference)</td>
<td>-</td>
</tr>
<tr>
<td>Diastolic BP at the 5th Korotkoff sound (mmHg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rural (n=1169)</td>
<td>49.3 (15.3)</td>
<td>-0.74 (-1.93 to 0.45)</td>
<td>0.22</td>
</tr>
<tr>
<td>Poor urban (n=431)</td>
<td>47.9 (16.3)</td>
<td>-2.11 (-3.78 to -0.43)</td>
<td>0.01</td>
</tr>
<tr>
<td>Nonpoor urban (n=1322)</td>
<td>50.0 (15.2)</td>
<td>0 (reference)</td>
<td>-</td>
</tr>
</tbody>
</table>
Tabel 2. Adjusted and unadjusted regression coefficients ($\beta$) for systolic and diastolic blood pressure as the dependent variables and sex, age, height-for-age z-scores (HAZ), body mass index-for-age z-scores (BMIZ) and socioeconomic status (SES) as the independent variables. Model 1 is comparing rural to nonpoor urban children while model 2 is comparing poor to nonpoor urban children.

(a) Systolic blood pressure (mmHg)

<table>
<thead>
<tr>
<th>Variables</th>
<th>Unadjusted $\beta^1$ (95%CI)</th>
<th>Adjusted$^2$ $\beta^1$ (95%CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Model 1 (n=2491)</td>
<td>Model 2 (n=1753)</td>
</tr>
<tr>
<td>Sex (1=male, 0=female)</td>
<td>0.49 (-0.24 to 1.23)</td>
<td>0.42 (-0.35 to 1.18)</td>
</tr>
<tr>
<td>Age (months)</td>
<td>0.07 (0.03 to 0.12)**</td>
<td>0.12 (0.07 to 0.17)**</td>
</tr>
<tr>
<td>HAZ</td>
<td>2.57 (2.21 to 2.94)*****</td>
<td>2.23 (1.81 to 2.65)*****</td>
</tr>
<tr>
<td>BMIZ</td>
<td>2.83 (2.53 to 3.13)*****</td>
<td>2.38 (2.05 to 2.71)*****</td>
</tr>
<tr>
<td>SES (rural=1, nonpoor urban=0) (n=2491)</td>
<td>-0.66 (-1.45 to 0.13)</td>
<td>1.19 (0.42 to 1.96)**</td>
</tr>
<tr>
<td>SES (poor=1, nonpoor urban=0) (n=1753)</td>
<td>-1.79 (-2.90 to -0.67)**</td>
<td>...</td>
</tr>
<tr>
<td>Constant</td>
<td>...</td>
<td>93.3 (88.9 to 97.7)*****</td>
</tr>
<tr>
<td>Adjusted R square</td>
<td>0.15</td>
<td>0.17</td>
</tr>
</tbody>
</table>
### (b) Diastolic blood pressure at the fourth Korotkoff sound (mmHg)

<table>
<thead>
<tr>
<th>Variables</th>
<th>Unadjusted</th>
<th>Adjusted $^*$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$\beta^1$ (95%CI)</td>
<td>$\beta^2$ (95%CI)</td>
</tr>
<tr>
<td>Model 1 (n=2491)</td>
<td>Model 2 (n=1753)</td>
<td></td>
</tr>
<tr>
<td>Sex (1=male, 0=female)</td>
<td>-0.42 (-1.18 to 0.34)</td>
<td>-0.41 (-1.22 to 0.40)</td>
</tr>
<tr>
<td>Age (months)</td>
<td>0.09 (0.04 to 0.14)**</td>
<td>0.13 (0.08 to 0.18)**</td>
</tr>
<tr>
<td>HAZ</td>
<td>1.89 (1.50 to 2.28)***</td>
<td>1.71 (1.27 to 2.15)***</td>
</tr>
<tr>
<td>BMIZ</td>
<td>1.84 (1.52 to 2.16)***</td>
<td>1.43 (1.08 to 1.77)***</td>
</tr>
<tr>
<td>SES (rural=1, nonpoor urban=0) (n=2491)</td>
<td>-0.71 (-1.51 to 0.08)</td>
<td>0.60 (-0.21 to 1.41)</td>
</tr>
<tr>
<td>SES (poor=1, nonpoor urban=0) (n=1753)</td>
<td>-1.68 (-2.82 to -0.53)**</td>
<td>...</td>
</tr>
<tr>
<td>Constant</td>
<td>...</td>
<td>51.6 (46.9 to 56.2)***</td>
</tr>
</tbody>
</table>

### Adjusted R square

<table>
<thead>
<tr>
<th></th>
<th>Model 1</th>
<th>Model 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.07</td>
<td>0.08</td>
</tr>
</tbody>
</table>
(c) Diastolic blood pressure at the fifth Korotkoff sound (mmHg)

<table>
<thead>
<tr>
<th>Variables</th>
<th>Unadjusted β¹ (95%CI)</th>
<th>Adjusted² β¹ (95%CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Model 1 (n=2491)</td>
</tr>
<tr>
<td>Sex (1=male, 0=female)</td>
<td>-1.11 (-2.23 to 0.02)</td>
<td>-1.47 (-2.70 to -0.25)*</td>
</tr>
<tr>
<td>Age (months)</td>
<td>0.15 (0.08 to 0.22)***</td>
<td>0.22 (0.14 to 0.30)***</td>
</tr>
<tr>
<td>HAZ</td>
<td>2.53 (1.95 to 3.10)***</td>
<td>2.51 (1.84 to 3.19)***</td>
</tr>
<tr>
<td>BMIZ</td>
<td>1.61 (1.12 to 2.09)***</td>
<td>1.02 (0.49 to 1.54)***</td>
</tr>
<tr>
<td>SES (rural=1, nonpoor urban=0)</td>
<td>-0.74 (-1.93 to 0.45)</td>
<td>0.93 (-0.30 to 2.17)</td>
</tr>
<tr>
<td>SES (poor=1, nonpoor urban=0)</td>
<td>-2.11 (-3.78 to -0.43)*</td>
<td>...</td>
</tr>
<tr>
<td>Constant</td>
<td>...</td>
<td>34.3 (27.2 to 41.3)***</td>
</tr>
</tbody>
</table>

Adjusted R square

<table>
<thead>
<tr>
<th></th>
<th>Model 1</th>
<th>Model 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.04</td>
<td>0.06</td>
</tr>
</tbody>
</table>

¹ β: unstandardized coefficient
² With adjustment for every other variable in the model
* p<0.05; ** p<0.01; ***p<0.001
Table 3. Adjusted regression coefficients ($\beta$) for systolic and diastolic blood pressure as the dependent variables and sex, age, height-for-age z-scores (HAZ), and body mass index-for-age z-scores (BMIZ) as the independent variables in children within different socioeconomic status (SES) groups

A. Systolic blood pressure (mmHg)

<table>
<thead>
<tr>
<th>Variables</th>
<th>Rural</th>
<th>Poor urban</th>
<th>Nonpoor urban</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n = 1169</td>
<td>n = 431</td>
<td>n = 1322</td>
</tr>
<tr>
<td>Sex (1=male, 0=female)</td>
<td>-0.42 (-1.52 to 0.68)</td>
<td>0.72 (-1.09 to 2.52)</td>
<td>1.12 (0.06 to 2.18)*</td>
</tr>
<tr>
<td>Age (mos)</td>
<td>0.09 (0.02 to 0.16)*</td>
<td>0.13 (0.01 to 0.25)*</td>
<td>0.15 (0.08 to 0.23)***</td>
</tr>
<tr>
<td>HAZ</td>
<td>1.80 (1.19 to 2.42)***</td>
<td>1.46 (0.40 to 2.52)**</td>
<td>2.54 (1.97 to 3.12)***</td>
</tr>
<tr>
<td>BMIZ</td>
<td>2.36 (1.84 to 2.89)***</td>
<td>2.39 (1.57 to 3.21)***</td>
<td>2.36 (1.93 to 2.78)***</td>
</tr>
<tr>
<td>Constant</td>
<td>97.3 (91.4 to 103.3)***</td>
<td>90.9 (80.5 to 101.3)***</td>
<td>90.1 (83.6 to 96.6)***</td>
</tr>
<tr>
<td>Adjusted R square</td>
<td>0.10</td>
<td>0.10</td>
<td>0.18</td>
</tr>
</tbody>
</table>

B. Diastolic blood pressure at the fourth Korotkoff sound (mmHg)

<table>
<thead>
<tr>
<th>Variables</th>
<th>Rural</th>
<th>Poor urban</th>
<th>Nonpoor urban</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n = 1169</td>
<td>n = 431</td>
<td>n = 1322</td>
</tr>
<tr>
<td>Sex (1=male, 0=female)</td>
<td>-0.98 (-2.18 to 0.21)</td>
<td>-1.95 (-4.16 to 0.25)</td>
<td>0.09 (-1.00 to 1.18)</td>
</tr>
<tr>
<td>Age (mos)</td>
<td>0.08 (0.005 to 0.16)*</td>
<td>0.20 (0.05 to 0.34)**</td>
<td>0.19 (0.11 to 0.27)***</td>
</tr>
<tr>
<td>HAZ</td>
<td>1.77 (1.10 to 2.43)***</td>
<td>1.14 (-0.15 to 2.44)</td>
<td>1.60 (1.01 to 2.20)***</td>
</tr>
</tbody>
</table>
### BMIZ

<table>
<thead>
<tr>
<th></th>
<th>Rural</th>
<th>Poor urban</th>
<th>Nonpoor urban</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMIZ</td>
<td>1.11 (0.54 to 1.67)***</td>
<td>1.78 (0.77 to 2.78)**</td>
<td>1.62 (1.18 to 2.06)***</td>
</tr>
<tr>
<td>Constant</td>
<td>56.7 (50.2 to 63.2)***</td>
<td>45.3 (32.6 to 58.0)***</td>
<td>46.3 (39.6 to 52.9)***</td>
</tr>
</tbody>
</table>

**Adjusted R square**: 0.04

---

### C. Diastolic blood pressure at the fifth Korotkoff sound (mmHg)

<table>
<thead>
<tr>
<th>Variables</th>
<th>Rural</th>
<th>Poor urban</th>
<th>Nonpoor urban</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n = 1169</td>
<td>n = 431</td>
<td>n = 1322</td>
</tr>
<tr>
<td></td>
<td>( \beta^1 ) (95%CI)</td>
<td>( \beta^1 ) (95%CI)</td>
<td>( \beta^1 ) (95%CI)</td>
</tr>
<tr>
<td>Sex (1=male, 0=female)</td>
<td>-1.91 (-3.76 to -0.07)*</td>
<td>-1.99 (-5.18 to 1.20)</td>
<td>-1.08 (-2.72 to 0.55)</td>
</tr>
<tr>
<td>Age (mos)</td>
<td>0.17 (0.05 to 0.29)**</td>
<td>0.33 (0.12 to 0.54)**</td>
<td>0.28 (0.17 to 0.40)***</td>
</tr>
<tr>
<td>HAZ</td>
<td>2.52 (1.49 to 3.55)***</td>
<td>2.98 (1.13 to 4.85)**</td>
<td>2.43 (1.54 to 3.31)***</td>
</tr>
<tr>
<td>BMIZ</td>
<td>0.35 (-0.53 to 1.23)</td>
<td>1.19 (-0.26 to 2.63)**</td>
<td>1.40 (0.75 to 2.06)***</td>
</tr>
<tr>
<td>Constant</td>
<td>39.6 (29.6 to 49.6)***</td>
<td>25.2 (6.8 to 43.5)***</td>
<td>29.1 (19.1 to 39.2)***</td>
</tr>
</tbody>
</table>

**Adjusted R square**: 0.02

---

1. \( \beta \): unstandardized coefficient with adjustment for every other variable in the model.
2. * p<0.05; ** p<0.01; ***p<0.001
Table 4. Comparison of height-for-age (HAZ), body mass index-for-age z-scores (BMIZ), systolic and diastolic blood pressure (BP) of children from different birth weight groups.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Mean (SD)</th>
<th>Mean difference (95% CI)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Height-for-age z-scores (HAZ)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LBW(^1) (n=193)</td>
<td>-1.45 (0.87)</td>
<td>0.26 (0.12 – 0.40)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HBW(^2) (n=242)</td>
<td>-0.77 (0.95)</td>
<td>-0.42 (-0.54 – -0.29)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>NBW(^3) (n=2315)</td>
<td>-1.19 (0.94)</td>
<td>0 (reference)</td>
<td>-</td>
</tr>
<tr>
<td><strong>Body mass index-for-age z-scores (BMIZ)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LBW(^1) (n=193)</td>
<td>-1.25 (1.13)</td>
<td>0.24 (0.07 – 0.41)</td>
<td>0.005</td>
</tr>
<tr>
<td>HBW(^2) (n=242)</td>
<td>-0.40 (1.03)</td>
<td>-0.61 (-0.76 – (-0.46)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>NBW(^3) (n=2315)</td>
<td>-1.01 (1.14)</td>
<td>0 (reference)</td>
<td>-</td>
</tr>
<tr>
<td><strong>Systolic BP (mmHg)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LBW(^1) (n=193)</td>
<td>99.54 (9.43)</td>
<td>0.061 (-1.38 – 1.50)</td>
<td>0.93</td>
</tr>
<tr>
<td>HBW(^2) (n=242)</td>
<td>102.34 (11.30)</td>
<td>2.86 (1.37 – 4.34)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>NBW(^3) (n=2315)</td>
<td>99.48 (9.83)</td>
<td>0 (reference)</td>
<td>-</td>
</tr>
<tr>
<td><strong>Diastolic BP at the 4(^{th}) Korotkoff sound (mmHg)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LBW(^1) (n=193)</td>
<td>58.87 (9.56)</td>
<td>-0.71 (-2.23 – 0.81)</td>
<td>0.36</td>
</tr>
<tr>
<td>HBW(^2) (n=242)</td>
<td>61.09 (10.57)</td>
<td>1.51 (0.13 – 2.89)</td>
<td>0.03</td>
</tr>
<tr>
<td>NBW(^3) (n=2315)</td>
<td>59.58 (10.40)</td>
<td>0 (reference)</td>
<td>-</td>
</tr>
<tr>
<td><strong>Diastolic BP at the 5(^{th}) Korotkoff sound (mmHg)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LBW(^1) (n=193)</td>
<td>48.42 (14.84)</td>
<td>-0.99 (-3.25 – 1.26)</td>
<td>0.39</td>
</tr>
<tr>
<td>HBW(^2) (n=242)</td>
<td>50.12 (15.77)</td>
<td>0.71 (-1.33 – 2.75)</td>
<td>0.50</td>
</tr>
<tr>
<td>Group</td>
<td>n</td>
<td>Mean (SD)</td>
<td>Reference</td>
</tr>
<tr>
<td>-------</td>
<td>----</td>
<td>-----------</td>
<td>-----------</td>
</tr>
<tr>
<td>NBW$^3$ (n=2315)</td>
<td>2315</td>
<td>49.41 (15.39)</td>
<td>0 (reference)</td>
</tr>
</tbody>
</table>

1. LBW = low birth weight, having a birth weight below the 10th percentile of our study subjects’ sex-specific birth weight distribution, i.e. below 2500 g for both boys and girls
2. HBW = high birth weight, having a birth weight above the 90th percentile of our study subjects’ sex-specific birth weight distribution, i.e. above 3600 g for boys and above 3500 g for girls
3. NBW = normal birth weight, having a birth weight between the 10th and the 90th percentile of our study subjects’ sex-specific birth weight distribution
Chapter 8.

General Discussion
INTRODUCTION

The aim of this thesis is to assess problems of under- and overnutrition in children in Indonesia. The studies focus on the association of socioeconomic status and birth weight with malnutrition, stunted growth, overweight and obesity, as well as with the risk of elevated blood pressure in prepubertal children.

The questions asked in this thesis are:

4. How does socioeconomic status influence the prevalence of malnutrition, stunted growth, overweight and obesity?
5. How is the tracking for underweight, overweight, and obesity?
6. How is the interaction between birth-weight, stature, body mass index (BMI) and socioeconomic status on blood pressure of prepubertal children?

1. How does socioeconomic status influence the prevalence of malnutrition, stunted growth, overweight and obesity?

Influence of socioeconomic status on the prevalence of malnutrition in infants and younger children

Similar to what had been observed in many developing countries and previous studies in Indonesia,\(^1\)\(^2\) pattern of weight increments in infants and younger children during the first 2 to 3 months of life were comparable to the reference population (Chapter 2). However, it was then followed by a decline up to the age of two years. It was also observed in the study that children in the poorer area exhibited an earlier and a sharper decline, resulting in a larger downward deviation from the NCHS-WHO reference standard.

Consequently, beginning from the age of 6 months, the study in Chapter 2 observed a significantly higher prevalence of malnourished children in the poorer area. At the age of 6 to 8 months, the prevalence of malnutrition was 18.3% in the poorer area vs. 1.0% in the more affluent area (p<0.001). The prevalence continued to rise in both areas studied, but the prevalence in the poorer area was always significantly higher, i.e. 43.9% vs. 11.1% (p<0.001) at the age of 9 to 11 months, 58.7% vs. 24.5% (p<0.001) at the age of 12 to 18 months and 59.4% vs. 32.4% (p<0.001) at the age of 18 to 24 months.

This observed high prevalence of malnutrition in the two areas studied was attributable to, among others, the quality of the weaning foods. The median consumption of animal food protein, in percentage of recommended daily allowance (RDA), was well below the recommendation. It was, however, significantly lower in the poorer area, i.e. 20.1% vs.
53.5%. This was the reason why we also observed that, especially in the poorer area, breastfeeding was an important contributor to nutritional status. It seemed that, in those not breastfed, the essential nutrients derived from breast milk, which was withheld, was not adequately replaced.

The study in Chapter 2 also observed that other proxies of socioeconomic status, i.e. maternal education and maternal body mass index (BMI), were independently associated with nutritional status. Several other studies had also reported a close relationship between nutritional status and the level of maternal education.\(^3\) The effect of maternal body mass index (BMI) on the infants’ nutritional status might be related to the infants' birth weight, to the mothers’ breast milk energy density,\(^4\) and to the mothers' or the families' socio-economic status.\(^5\)

**Influence of socioeconomic status on the prevalence of stunted growth and obesity in prepubertal children**

The study in Chapter 3 showed that, in older children, the prevalence of stunted growth, but not the prevalence of underweight or wasting, was significantly higher in the poorer segment of the population. Stunted growth, which occurs mostly in the first three years of life, reflects long-term malnutrition and poor health, whereas wasting is more a reflection of recent energy imbalance.\(^6\) In association with the result of the study in Chapter 2, it seemed that most of the effects of malnutrition occurring in the first years of life had never fully recovered, resulting in a higher prevalence of short children in the poorer segment of the population. A longitudinal study in the Philippines showed that 70% of children who were stunted at the age of 2 years were still stunted at the age of 8.5 years.\(^7\)

However, in contrast to what had been observed in China, Russia, South Africa and Brazil,\(^8\)-\(^10\) our study did not observe any association between stunted growth and obesity. Stunted children had lower odds to be obese. This study found that the prevalence of overweight and obesity were five times higher in the wealthier part of the population (Chapter 3).

It seemed that in Indonesia, similar to what was observed in other developing countries,\(^11\);\(^12\) so far malnutrition and stunted growth were still the problems of the poor while overweight and obesity were the problems of the rich. The poorer segment of the population will benefit from interventions to improve the quantity and quality of food intake, especially in the early years of life, while the wealthier segment needs programs to reduce the risk of obesity.
Association between low weight at birth and stunted growth or overweight in prepubertal children

The contribution of low weight at birth to the prevalence of stunted growth in older children differed between the rural and the urban population (Chapter 4). In rural children, with a higher prevalence of stunted growth, low weight at birth was not a significant contributor to the prevalence of subsequent stunted growth. On the other hand, in the urban population, with a lower prevalence of stunted growth, the odds to be born small in stunted children were more than twice higher than in those not stunted. Since the study did not detect a significant difference in the prevalence of low weight at birth, the observed difference in the prevalence of stunted growth might be due to, as shown by the study in Chapter 2, the occurrence of considerable postnatal growth deficit in infants in the rural area.

This observation is important because the question whether infants born with a low birth weight will catch up in growth may not be the most appropriate issue to be asked in a poor area. It is better, perhaps, to ask whether the infants will suffer from declining growth rate. It seems that, in a poor area, postnatal growth deficit has a larger contribution to subsequent stunted growth. A longitudinal study in a rural West Java-Indonesia observed an increase in the prevalence of stunted growth from only 7% in the neonatal period to 24% at the age of 12 months.\(^2\) A study in the Philippines showed that, in the second year of life, birth weight was no longer significantly associated with the likelihood for stunted growth.\(^13\)

The study in Chapter 4 did not detect any association between low weight at birth and overweight. The association between low weight at birth and overweight seemed to be mediated through rapid weight gain in infancy or childhood which probably did not occur in our population.

2. **How is the tracking for underweight, overweight, and obesity?**

The study in Chapter 5 observed an increase in the mean BMI z-score as Indonesian urban children grew into adolescence. Consequently, during those five-years of follow-up, the prevalence of underweight decreased, while the prevalence of overweight and obesity increased. However, body mass index (BMI) in childhood did indeed track into adolescence, since BMI in childhood was highly correlated with BMI in adolescence.

The study observed that most overweight children remained overweight and virtually all obese children stayed obese. The relative risk of overweight (obese) children to remain overweight (obese) was much higher than the risk of those not overweight (obese) to
become overweight (obese). However, around half of the overweight (obese) adolescents were not overweight (obese) in childhood, because the prevalence of overweight and obesity were doubled as the children entering adolescence (Chapter 5).

In the mean time, the study also found approximately 30% decline in the prevalence of underweight as the children grew into adolescence. However, more than a half of the underweight children remained underweight in adolescence.

The observed tracking pattern for overweight and obesity was similar to what had been reported earlier in some developed countries and was well above that observed in the developing countries, e.g. China. On the other hand, with a similar follow-up period, the observed prospective tracking for underweight was also significantly higher than that observed in China, which was only around 33%. This information suggested that children who were on the overweight or obese side tended to remain overweight or obese while those in the underweight side tended to remain underweight.

In association with the overall development of welfare in Indonesia, The United Nations Development Program (UNDP) reported that, despite an increase in Gross Domestic Product (GDP) of around 3.2% per year within 10 years time (1990-1999), the proportion of people lived in income poverty raised from 15% to 18%. It seemed that, in Indonesia, those who were poor and had limited access to food remained underweight, while those who were wealthier and exposed to unlimited energy intake tended to become overweight. Consequently, unless measures for preventing obesity is instituted, increase in welfare may lead to the emergence of obesity epidemics.

3. **How is the interaction between birth-weight, stature, body mass index (BMI) and socioeconomic status on blood pressure of prepubertal children?**

Our study, similar to other previous studies in both developed and developing countries, observed that systolic and diastolic blood pressures in children were positively associated with age, stature and body mass index (BMI). Similar associations existed within every socioeconomic status group studied, i.e. age, stature and BMI were significant predictors of blood pressures both in low and high socioeconomic status children (Chapter 7).

Classification of socioeconomic status used in the study of blood pressure (Chapter 6 and 7) was the same as the one used in Chapter 3, i.e. rural, poor urban and nonpoor urban. In association with the result of the study in Chapter 3, it was observed that the rural population was the poorest among the three population groups studied since it had the highest prevalence of stunted growth. The nonpoor urban population was the richest since it
had the lowest prevalence, while the poor urban population stood between the two. This was because it had been recognized that stunted growth reflected long-term cumulative inadequacies of health and nutrition, and had been strongly associated with poverty.\textsuperscript{6,13}

The systolic and diastolic blood pressures of children from the richest part of the population, i.e. the nonpoor urban children, were significantly higher than those of the poor urban children. However, surprisingly, blood pressures of the rural children were not significantly lower than those of the non-poor urban children. Rural children had average blood pressures comparable to non-poor urban children despite the fact that they had, in average, lower BMI and stature.

Linear regression analyses, either separately or adjusted for every other variable in the model showed that both systolic and diastolic blood pressures were independently and positively related to age, stature and BMI. In the model comparing rural and nonpoor urban children, non-significant lower systolic and diastolic blood pressures were observed in the rural children. After adjustment for sex, age, stature and BMI, the association between socioeconomic status and systolic blood pressures inverted; the systolic blood pressures of the rural children were significantly higher than those of the nonpoor urban children. Although not statistically significant, the diastolic blood pressures of the rural children, after adjustment for the above confounding factors, were also higher than those of the nonpoor urban children.

On the other hand, in the models comparing poor urban and nonpoor urban children, the influence of socioeconomic status on both systolic and diastolic blood pressures diminished after adjusting for age, stature and BMI, that is, showing that the influence of socioeconomic status on blood pressures in these two groups of urban children was more related to the difference in the distribution of stature and BMI rather than the influence of the socioeconomic status per se.

Poor rural communities in the developing countries are at higher risk for suffering from impaired prenatal and early postnatal growth, since these are the times when growth is most vulnerable to the adverse effect of poverty, poor nutrition and high prevalence of infectious diseases.\textsuperscript{2,13} However, stunted growth was not the only risk factor for raised blood pressures in these rural children, because even in this poor population, blood pressures were positively associated with both height and BMI.

Adair and Cole, in a longitudinal study in The Philippines showed that the highest odds for increased blood pressures occurred among boys who were relatively thin at birth but relatively heavy as adolescents. Larger weight increment from birth to the age of 2 years
decreased the odds for high blood pressures whereas larger increment from the age of 8 to 11 years increased the odds for high blood pressures.\textsuperscript{21}

Walker et al. 2001, in a study in Jamaica observed that nutritional status in infancy did not influence systolic and diastolic blood pressures of 11 to 12 year-old Jamaican adolescents. However, the effect of increasing weight on systolic blood pressures was higher in adolescents who were stunted at the age of 9 to 24 months.\textsuperscript{22}

Cheung et al. found that birth length, ponderal index at birth and postnatal change in ponderal index between the age of 6 to 18 months were independently and inversely associated with systolic blood pressures in young Hongkong adults.\textsuperscript{23} Similarly, studies in Finland showed that low weight gain during infancy, irrespective of birth weight, followed by rapid gain in weight and height later in childhood was associated with higher risk for coronary heart diseases.\textsuperscript{24;25}

In association with birth weight, our study observed that higher birth weight children had higher systolic blood pressures. The association was explained by higher current stature and BMI in high birth weight children (Chapter 7).

In summary, it seemed that, beside BMI and stature, living in a poor condition was also associated with increased risk for having high blood pressures. This association might be related to the higher prevalence of malnutrition in the first years of life.

**RECOMMENDATIONS AND FUTURE RESEARCH**

Associations between small body size at birth and later cardiovascular disease and its biological risk factors have been almost consistently found. Small size at birth had been associated with increased risk for adult abdominal obesity and lower fat free mass,\textsuperscript{26-28} elevated plasma cortisol,\textsuperscript{29} hypertension,\textsuperscript{30;31} dyslipidemia,\textsuperscript{32} and coronary heart disease,\textsuperscript{25} as well as type-2 diabetes mellitus and insulin resistance.\textsuperscript{24;33-35}

The association between low birth weight and cardiovascular diseases in later life is thought to be the result of persistence of changes in morphology and physiology that accompany slow fetal growth. The fetal origin hypothesis proposes that adult chronic diseases originate through plasticity during development whereby malnutrition, hypoxia, or stresses in utero initiate pathological changes, such as reduced number of nephrons and altered function of beta cells.\textsuperscript{33;36-39}

However, most studies also showed that the associations between birth weights and later indicators of chronic diseases were stronger after controlling for current weight, indicating the role of postnatal growth in modifying the influence of birth weight on
subsequent cardiovascular risks. It was postulated that it was the combination of slow intrauterine growth followed by rapid compensatory growth that led to the disease.

A closer look on postnatal growth showed that the association between compensatory growth and later risk of cardiovascular disease was not uniform across gender and age groups. A study from Finland reported that, on average, higher weight or body mass index during the first two years of life reduced the risk for coronary events, while higher weight or increase in weight after this period was associated with higher risk for coronary events. Similarly, a study in The Philippines observed that higher weight gain after the age of 8 years increased the odds for high blood pressure in boys, whereas larger infant weight and length gains reduced the odds. A study in India showed that growth of children in whom impaired glucose tolerance or diabetes later developed was characterized by a low BMI between birth and two years of age followed by a young age at adiposity rebound (defined as the age after infancy at which BMI starts to rise), and a sustained accelerated gain in BMI until adulthood. The last two studies, however, reported that despite a higher increase in weight or BMI, these individuals were not obese in childhood.

Although rapid gain in weight during infancy has not been associated with increased risk for cardiovascular disease, it has been identified as a risk factor for later obesity. However, most studies only focused on gain in weight or gain in BMI while it seems that gain in height may have a different influence. A study in Sweden reported that linear catch-up growth did not increase the risk for elevated blood pressure in young adults who had been small for gestational age. Studies in Jamaica and Brazil observed an increase risk for elevated blood pressure and obesity in short individuals.

Our study and previous studies in Indonesia showed that most children in Indonesia were not born with low weight at birth, that is, prevalence of low birth weights of only around 6 to 9% (paper in Chapter 4). However, most infants, especially those coming from the poorer society exhibited growth deficits during the first years of life (paper in Chapter 2). In association with the emergence of cardiovascular disease epidemic in the developing countries as a whole and a reported increase in the prevalence of elevated blood pressure and obesity in Indonesian adults, the influence of postnatal growth deficit on cardiovascular risk in the absence of intrauterine growth restriction should be investigated further.

Given the functional consequences of small body size among adults, compensatory growth has typically been viewed as desirable for children with poor nutritional histories. However, in association with the risk of cardiovascular diseases, compensatory growth after postnatal growth deficits should be regarded with caution.
Another interesting aspect of the growth of Indonesian children was observed in the recently published growth chart. During childhood, the average height standard deviation scores (SDS) of Indonesian children were constantly at around 1.0 to 1.5 SDS below those of the CDC (Center for Disease Controls and Prevention) 2000 growth chart. In adolescence, however, an abrupt downward deviation occurred, resulted in a much lower adult height, i.e. around 2.0 and 2.5 SDS below the CDC 2000 growth chart for women and men respectively.55

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Chapter 9.

Summary and conclusions
Samenvatting en conclusies
Ringkasan dan Kesimpulan
Summary and conclusions:

**The impact of socio-economic status on the risk of malnutrition and overweight in Indonesian children: an epidemiological study**

Chapter 1 provides information on the existence of problems of “under and over nutrition” in the developing countries and their possible associations with risk of cardiovascular disease later in life. Furthermore, this chapter also describes background information on the situation of Indonesia as a country in both nutritional and epidemiological transition. By the end of this chapter, the aim and the study questions of the thesis are described.

The study in Chapter 2 assesses risk factors for malnutrition in children under two years old from two rural areas in Indonesia. One area was shown to be poorer than the other. This study shows that most Indonesian children have acceptable weight during the first 6 months of life. However, as they grew, the prevalence of malnutrition increases. The decline in nutritional status is worse in the poorer area, resulting in a significantly higher prevalence of malnutrition from the age of 6 months onwards.

The malnutrition might be associated with the poor quality of the weaning food since the consumption of animal food protein, especially in the poorer area, is quite low. It is concluded that being still breastfed helps protecting the children from malnutrition.

The study in Chapter 3 compares the prevalence of stunted growth, underweight, overweight and obesity in school-aged prepubertal children from different socioeconomic levels, i.e. rural, poor urban, and nonpoor urban. The study observes that poor children, i.e. rural and poor urban children have significantly higher prevalence of stunted growth compared to nonpoor urban children. The prevalence of stunted growth in the rural children is almost three times higher than in the nonpoor urban children. On the other hand, while there is no significant difference in the prevalence of underweight, these poor children have a lower prevalence of overweight and obesity. The prevalence of overweight and obesity is around four to five times higher in the nonpoor urban children.

Chapter 4 discusses the relative contribution of low weight at birth on the prevalence of stunted growth or obesity in prepubertal children from the rural and the urban population. The study shows that, in an area where the prevalence of stunted growth is very high (the rural area), low weight at birth is not an important contributor for stunted growth. However, in an area where the prevalence of stunted growth is less (the urban area), low weight at
birth is an important risk factor for subsequent stunted growth. Furthermore, as there is no significant difference in the prevalence of low birth weight among the rural and the urban populations, it is concluded that the observed difference in the prevalence of stunted growth among rural and urban prepubertal children might have its origin in the socioeconomic environment influencing the pattern of post-natal growth.

The study in Chapter 5 describes the tracking of body mass index (BMI) as Indonesian urban children grew from childhood into adolescence. The study shows that urban Indonesian children are increasing in weight and BMI explained by the increase in the prevalence of overweight and obesity from childhood into adolescence. Those who are overweight or obese during childhood are most likely to remain overweight or obese in adolescence. On the other hand, despite a decline in the prevalence of underweight from childhood into adolescence, the probability to remain underweight in adolescence is more than 50%.

The study in Chapter 6 and 7 assesses the influence of socioeconomic status and birth weight on blood pressure of school-aged prepubertal children. The study observed that systolic and diastolic blood pressures in children in Indonesia are positively associated with age, stature and BMI. However, this study indicates that, for a given age, stature and BMI, the poorer segment of the population, that is, the rural community, faced a higher risk for developing hypertension, which might be related to the observed higher prevalence of stunted growth in this community.

In association with birth weight, our study observed that higher birth weight children had higher systolic blood pressure. The association is explained by the fact that high birth weight children are in average taller and heavier than the normal birth weight children.

In Chapter 8, the results of the studies described in this thesis are discussed. It is concluded that problems of "under and over nutrition" exist in Indonesia. Children from the rural area, especially those coming from the poorer area have a higher risk to be malnourished. On the other hand, those coming from the urban population, especially those from the nonpoor community have a higher risk to be overweight or obese. However, irrespective of their present height or BMI, being from the poorer area is an independent risk for higher blood pressure.
Samenvatting en conclusies:

**Socioeconomische status en het risico op onder- en overgewicht bij kinderen in Indonesië: een epidemiologisch onderzoek**

Hoofdstuk 1 geeft een overzicht van de bestaande problemen van “onder- en overvoeding” op de kinderleeftijd in ontwikkelingslanden en de mogelijke verbanden met hart- en vaatziekten op oudere leeftijd. Daarnaast geeft dit hoofdstuk achtergrondinformatie over Indonesië als land met betrekking tot de transitie in voeding en epidemiologie. Tenslotte worden het doel en de vraagstellingen van het onderzoek die geleid hebben tot dit proefschrift beschreven.

Hoofdstuk 2 beschrijft een onderzoek waarin risicofactoren voor het ontwikkelen van ondervoeding in kinderen jonger dan 2 jaar, afkomstig uit twee plattelandsgebieden, worden vastgesteld. Het ene gebied heeft een lagere socio-economische status dan het andere gebied. Het onderzoek toont aan dat de meeste kinderen een acceptabel gewicht hebben gedurende de eerste 6 maanden na de geboorte. Echter, naarmate de kinderen ouder worden neemt het aantal kinderen met ondervoeding toe. In het gebied met de laagste socio-economische status is de voedingsstatus het meest aangedaan, hetgeen resulteert in het vaker voorkomen van ondervoeding vanaf de leeftijd van 6 maanden. Verondersteld wordt dat ondervoeding geassocieerd kan zijn met de slechte kwaliteit van vaste voeding aangezien de consumptie van dierlijk voedings eiwit met name in het armste plattelandsgebied, vrij laag is. In ieder geval blijkt dat het langdurig geven van borstvoeding kinderen beschermt voor ondervoeding.

In Hoofdstuk 4 wordt de invloed van een laag geboortegewicht op het voorkomen van lengtegroeivertraging en obesitas in prepuberale kinderen, afkomstig van het platteland en de stad, beschreven. De studie toont aan dat, in een gebied waar latere lengtegroeivertraging vaak voorkomt (het platteland), een laag geboortegewicht geen belangrijke factor is die hiertoe bijdraagt. Echter, in een gebied waar lengtegroeivertraging minder vaak voorkomt (de stad), blijkt een laag geboortegewicht wel een belangrijke risicofactor te zijn voor latere lengtegroeivertraging. Een verschil in het voorkomen van een laag geboortegewicht tussen de populaties van het platteland en de stad is er niet. Men zou hieruit kunnen concluderen dat het verschil in voorkomen van latere lengtegroeivertraging tussen de prepuberale plattelands- en stadskinderen veroorzaakt wordt door de socio-economische omgeving die het postnatale groeipatroon beïnvloedt.

Het onderzoek in hoofdstuk 5 beschrijft de body mass index (BMI) in Indonesische stadskinderen vanaf de kinderleeftijd tot aan de adolescentie. De studie toont aan dat stadskinderen zodanig in gewicht en BMI toenemen dat er in de loop van de tijd een toename optreedt van het aantal kinderen met overgewicht en obesitas. De kinderen, die op de kinderleeftijd overgewicht hebben of obees zijn, hebben over het algemeen ook op adolescentie leeftijd overgewicht of zijn zelfs obees. Aan de andere kant van het spectrum blijkt dat, ondanks een afname in het voorkomen van ondergewicht vanaf de kinderleeftijd tot aan de adolescentie, de kans op ondergewicht in de adolescentie meer dan 50% is.

Het onderzoek beschreven in de hoofdstukken 6 en 7 laat de invloed zien van de socio-economische status en het geboortegewicht op de bloeddruk van schoolgaande prepuberale kinderen. Dit onderzoek toont aan dat zowel de systolische als de diastolische bloeddruk in Indonesische kinderen positief gerelateerd zijn aan de leeftijd, de lichaamslengte en de BMI. De studie geeft tevens aan dat bij een bepaalde leeftijd, lengte en BMI, de armsten van de bevolking, dat is de plattelandsgemeenschap, een hoger risico hebben op het ontwikkelen van hoge bloeddruk. Dit zou gerelateerd kunnen zijn met het vaker voorkomen van latere lengtegroeivertraging in deze gemeenschap. Tevens wordt aangetoond dat kinderen met een hoog geboortegewicht een hogere systolische bloeddruk hebben. Deze kinderen bleken gemiddeld langer en zwaarder zijn dan de kinderen met een normaal geboortegewicht.

In hoofdstuk 8 worden de resultaten van dit proefschrift bediscussieerd in het kader van reeds bekende gegevens uit de literatuur.
Geconcludeerd wordt dat in Indonesië problemen van zowel ondervoeding als overvoeding bestaan. Kinderen van het platteland, met name zij die wonen in gebieden met een lagere socio-economische status, een verhoogd risico hebben op ondervoeding. Hiertegenover staan de kinderen uit de stads-gemeenschap, met name zij die afkomstig zijn uit het niet-arme deel van de bevolking, die een verhoogd risico hebben op overgewicht en obesitas. Verrassend is dat, ongeacht de lengte of BMI van het kind, de afkomst uit een gebied met een lage socio-economische status een onafhankelijke risicofactor blijkt te zijn voor een hogere bloeddruk.
Ringkasan dan kesimpulan:

**Pengaruh Status Sosial-ekonomi terhadap Risiko Terjadinya Malnutrisi dan Obesitas pada Anak Indonesia: Suatu Studi Epidemiologis**

Bab 1 membahas masalah “kelebihan dan kekurangan gizi” di negara-negara berkembang dan kemungkinan hubungannya dengan peningkatan risiko penyakit kardiovaskular di kemudian hari. Bab ini juga membahas situasi Indonesia sebagai negara yang sedang melalui transisi epidemiologi dan nutrisi. Sebagai penutup, pada akhir bab disampaikan tujuan dan pertanyaan penelitian tesis ini.


Malnutrisi ini mungkin berkaitan dengan rendahnya kualitas makanan penyapih karena asupan protein hewani, terutama di daerah yang lebih miskin, sangat rendah. Karena itulah penelitian ini juga menemukan bahwa status masih menyusui membantu melindungi anak dari malnutrisi.

Bab 4 membahas kontribusi relatif berat badan lahir rendah terhadap prevalensi perawakan pendek (stunted) dan obesitas pada anak usia sekolah prapubertas dari daerah pedesaan dan perkotaan. Penelitian ini menunjukkan bahwa di daerah dengan prevalensi perawakan pendek (stunted) yang tinggi (daerah pedesaan), berat badan lahir rendah bukan merupakan kontributor perawakan pendek (stunted) yang signifikan. Namun, di daerah dengan prevalensi perawakan pendek (stunted) yang lebih rendah (daerah perkotaan), berat badan lahir rendah menjadi faktor risiko perawakan pendek yang penting. Selanjutnya, karena tidak ditemukannya perbedaan signifikan prevalensi berat badan lahir rendah antara populasi pedesaan dan perkotaan, perbedaan prevalensi perawakan pendek (stunted) antara anak prapubertas pedesaan dan perkotaan mungkin disebabkan oleh perbedaan lingkungan sosial-ekonomi yang mempengaruhi pertumbuhan pasca-natal.

Penelitian pada Bab 5 menunjukkan adanya penelusuran (tracking) indeks massa tubuh (IMT) pada saat anak perkotaan Indonesia tumbuh dari masa anak ke masa remaja. Penelitian ini juga menunjukkan bahwa seiring dengan pertambahan usia dari masa anak ke masa remaja, prevalensi berat badan lebih (overweight) dan obesitas juga meningkat. Anak yang sudah mempunyai berat badan lebih (overweight) atau obes cenderung akan tetap mempunyai berat badan lebih (overweight) atau obes pada masa remaja. Sebaliknya, meskipun prevalensi berat badan kurang (underweight) berkurang, kemungkinan anak yang mempunyai berat badan kurang (underweight) untuk tetap underweight pada masa remaja lebih dari 50%.

Penelitian pada Bab 6 dan 7 menilai pengaruh status sosial-ekonomi dan berat badan lahir terhadap tekanan darah anak usia sekolah prapubertas. Penelitian ini menunjukkan bahwa tekanan darah sistolik dan diastolik anak Indonesia berbanding lurus dengan umur, tinggi badan dan indeks massa tubuh (IMT). Namun, penelitian ini juga menunjukkan bahwa untuk usia, tinggi badan dan indeks massa tubuh (IMT) yang setara, segmen masyarakat yang termiskin, dalam hal ini penduduk pedesaan, mempunyai risiko lebih besar untuk menderita hipertensi. Hal ini mungkin berkaitan dengan tingginya prevalensi perawakan pendek (stunted) pada komunitas ini.

Anak yang lahir dengan berat badan lahir yang lebih berat mempunyai tekanan darah sistolik yang lebih tinggi. Hal ini karena anak dengan berat badan lahir yang lebih berat juga cenderung mempunyai perawakan yang lebih tinggi dan mempunyai IMT yang lebih besar daripada anak yang lahir dengan berat badan lahir yang normal.
Bab 8 membahas keseluruhan hasil penelitian yang disampaikan pada tesis ini. Tesis ini menyimpulkan adanya masalah “kelebihan dan kekurangan gizi” di Indonesia. Anak dari daerah pedesaan, terutama yang berasal dari daerah yang lebih miskin, mempunyai risiko yang lebih tinggi untuk mengalami malnutrisi. Sebaliknya, anak dari daerah perkotaan, terutama yang bukan berasal dari keluarga miskin, mempunyai risiko lebih tinggi untuk menderita berat badan lebih (overweight) atau obesitas. Namun, tanpa memperhatikan tinggi badan atau berat badan saat ini, berasal dari daerah yang lebih miskin merupakan faktor risiko independen untuk tekanan darah yang lebih tinggi.