Is age at menarche a good predictor of future body fat? The case of a developing country

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ABSTRACT

Age at menarche has been proposed to serve as a predictor of future body fat for the developed world. Our aim in this study is to determine whether this is also the case for a developing country—Indonesia. We analyze nationally representative data, concerning 9,543 women aged 15–62 in 2007–2008, and find that the relationship between age at menarche and body mass index is negative and statistically significant. The size of the relationship, however, is negligible. It thus appears that age at menarche is not a good predictor of future body fat in Indonesia and possibly other developing countries.

In 1997, the World Health Organization (WHO, 2000b) declared that obesity was a global epidemic. This global scale of obesity implies that the developing world is no longer safe from it. This is particularly concerning for women because, on average, obesity rates are higher for women than for men in the developing world (Prentice, 2006). Obesity is concerning because it contributes to type 2 diabetes, stroke, coronary artery disease, gallbladder disease, hyperlipidaemia, hypertension, and several cancers, to name only a few (Withrow & Alter, 2011). Obesity, therefore, is related to greater medical expenditures particularly for women. For example, obese (defined as body mass index [BMI] ≥ 30 kg/m²) women aged 18–64 in the United States in 2003 spent $1,457 more per year than did nonobese women; on the other hand, the analogous difference for men was only $405. Partly related to this (because it is more costly to hire obese women than nonobese women), among full-time female workers with health insurance in the United States, obese women earned $2.64 per hour less than nonobese women. In contrast, the corresponding figure for men was $0.58 and was statistically nonsignificant (Bhattacharya & Bundorf, 2009). Because obesity is related to medical expenditures and wages, obesity has implications for socioeconomic aspects such as income and health inequalities, health insurance, disability benefits, taxation, and negative externalities; therefore, obesity is also a public issue.
Kelly, Yang, Chen, Reynolds, and He (2008) estimated that the prevalence of overweight ($25 \leq \text{BMI} < 30$) and obesity among adults aged 20+ in 2005 was higher in the developed than developing world: 35.2% versus 19.6% for overweight and 20.3% versus 6.7% for obesity. The number of overweight and obese people, however, is greater in the developing than developed world: 611 million versus 326 million for overweight and 209 million versus 188 million for obesity. In addition to the number, the growth rate of this group is also particularly alarming for the developing world. Popkin, Adair, and Ng (2012) estimated that the proportion of overweight and obese women aged 18–49 grew about 0.7 percentage points per year in 42 developing countries in the 1990s and 2000s. The past has been bleak, but the future is even more bleak for the developing world. Kelly and colleagues (2008) projected that if recent trends continue, the numbers of overweight and obese people are expected to increase most rapidly in the developing world, ranging 62%–205% for overweight people and 71%–263% for obese people from 2005 estimates. This concern extends to children, as growing proportions of children suffer from obesity in some developing countries. For example, the prevalence of overweight and obesity among school-age children increased more than 0.5 percentage points per year in urban China and urban Brazil (Wang & Lobstein, 2006).

Considering the current situation and future prospect of overweight and obesity in the developing world, more attention needs to be paid to body fat in the developing world. Given that prevention of obesity is less expensive and painful than treatment, it is important to find a good predictor of future body fat. If found, such a predictor will provide potential patients with time and knowledge to prevent the accumulation of excess body fat. It is better if such a predictor is early and easily detected in one’s lifetime to allow more time for the prevention of obesity with fewer mistakes. Particularly for women, the medical literature has proposed a predictor that meets these criteria: age at menarche. Girls experience menarche before adulthood (early), and menarche is clearly visible (easily). Much evidence of this has been provided for the developed world, but relatively little is available for the developing world (see references in the discussion section).

It is instructive to note that as nutrition has improved and disease has decreased, age at menarche has decreased worldwide. There is evidence of leveling off in the developed world, but no such sign is in sight in the developing world (Graham, Larsen, & Xi, 1999; Onland-Moret et al., 2005). For example, Graham and colleagues (1999) observed that the mean age at menarche decreased from 15.8 years for women born between 1949 and 1953 to 13.7 years for women born from 1974 to 1978 (a reduction of 0.72 years per decade), in two rural (meaning poor) counties in Anhui, China. It is surprising that this fast decrease occurred despite the severe retrogression in material conditions due to the Great Leap Forward (1958–1961) and the Cultural Revolution (1966–1976). Regarding Indonesia, Sohn (2014a, 2015e) recorded that age at menarche decreased from 14.39 years for the birth year 1944 to 13.18 years for the birth year 1988, and the reduction accelerated for the later-born women.
When the growing prevalence of excess body fat—indicated by overweight and obesity—is considered together with the decreasing age at menarche in the developing world, it appears plausible that age at menarche is a good predictor of future body fat. This may not be true, however, especially when malnutrition coexists with obesity in some developing countries, that is, the dual burden of disease (Doak, Adair, Bentley, Monteiro, & Popkin, 2005). Moreover, a good predictor needs to satisfy an additional condition: a large relationship with what it predicts. This is related to the criterion of fewer mistakes. If the size is small, the predictor is more likely to make a mistake. This concern is particularly relevant to age at menarche because it is affected by many factors. Thus, although the relationship between age at menarche and future body fat is negative, if the magnitude of the relationship is small, age at menarche cannot be regarded as a good predictor of future body fat. Unfortunately, the literature typically highlights the sign of the relationship but neglects its size. A further criterion for a good predictor is a dose–response relationship. This criterion can be considered, however, if the predictor passes the criterion of a large relationship. As explained below, age at menarche does not pass this immediate criterion, and, therefore, we do not consider the criterion of a dose–response relationship.

The purpose in writing this study, therefore, is to determine whether age at menarche is a good predictor of future body fat in a developing country—Indonesia. The sheer number of the population alone (about 250 million; the fourth most populous country in the world) makes Indonesia an interesting case. In addition, its economy has rapidly grown since independence: its real GDP grew more than 22 times during 1949–2012 (van der Eng, 2010, and his extended estimates). Thus, the Indonesian population has been exposed to an obesogenic environment for a material length of time. In fact, the proportion of Indonesian women with a BMI ≥ 27 (obese for the Indonesian population, more about this cut-off point later) increased from 9.7% in 1993 to 19.6% in 2007 (Roemling & Qaim, 2012). Indonesian women have suffered from this more than men; as of 2007, while 8.9% of men were obese, 19.6% of women were obese (Roemling & Qaim, 2012). Moreover, the Islamic culture of Indonesia is of importance for cross-country comparisons because body fat is influenced by cultural as well as socioeconomic factors (De Garine & Pollock, 1995).

Methods

The main dataset is the Indonesian Family Life Survey (IFLS), an ongoing longitudinal survey. The baseline survey was initiated with more than 22,000 individuals in 7,224 households in 13 provinces in 1993 (IFLS1). Four follow-up surveys were performed in 1997 (IFLS2), 2000 (IFLS3), and 2007 (IFLS4); an ad hoc follow-up was conducted in 1998, but it involves only 25% of the original sample and is not publically available. Although not all regions were covered, great care was exercised to ensure that the sample was as nationally representative as possible. In addition,
IFLS4 contains the largest sample size among the IFLS surveys and relevant variables that are unavailable in the other IFLS surveys.

Ever-married women aged 15–49 and women in the previous survey (ever-married, aged 15–49 at that time) reported their age at menarche in whole years (Sohn, 2014a; 2015e, for more about age at menarche in Indonesia). One immediate concern with recalled age at menarche is measurement error and bias. For example, if respondents inaccurately recall their age at menarche, measurement error occurs. If younger respondents recall their age at menarche more accurately than older ones, bias occurs. Ample evidence from Livson and McNeill (1962) to Dorn, Sontag-Padilla, Pabst, Tissot, and Susman (2013), however, suggests that any measurement error or bias is small. The high reliability of recall is probably due to the salience of menarche in a woman’s life.

We proxy body fat with BMI. It is acknowledged that BMI cannot accurately distinguish between fat and lean body mass. For example, BMI may incorrectly classify a muscular man as obese. In addition, BMI cannot discern between peripheral fat and central fat, when the latter poses a more serious health concern than the former. For these reasons, Kragelund and Omland (2005) went so far as to bid “farewell” to BMI. As it is too expensive in large-scale surveys to measure visceral fat using laboratory methods such as dual-energy X-ray absorptiometry, computed axial tomography, and functional magnetic resonance imaging, medical researchers have proposed alternative anthropometrics such as waist circumference, waist-to-height ratio, waist-to-hip ratio, sagittal diameter, and abdominal skinfold thickness. Sohn (2014c) demonstrated that compared with other anthropometrics, however, BMI was the best predictor of chronic diseases, at least in Indonesia (Sohn, 2014b, for more information about BMI in Indonesia). Specially trained nurses measured weight and height (Sohn, 2015a, 2015b, 2015c, 2015d, 2015f; in press, for more about height in Indonesia); weight was measured to the nearest 0.1 kg and height to 0.1 cm. For analysis, weight is restricted to 30–150 kg, height to 120–200 cm, BMI to 10–40 kg/m², and age at menarche to 10–18 years. Numbers beyond these ranges are likely due to (probably random) recording errors. In addition, pregnant women are excluded because they could be mistaken as exhibiting high BMIs, even if they did not have high BMIs prior to pregnancy.

We estimate the relationship between age at menarche and BMI using the following ordinary least squares (OLS) specification:

$$\text{BMI}_i = \beta_1 \text{Menarche}_i + X_i \beta_2 + e_i$$ (1)

where $\text{BMI}_i$ refers to the BMI of woman $i$, $\text{Menarche}$ to age at menarche, $X$ to covariates of a constant, demographics, health-related behaviors, and socioeconomic status (SES), $e$ to an error term, and $\beta_1$ and $\beta_2$ to a coefficient and coefficient vector, respectively, to estimate. $\beta_1$ is the coefficient of interest and measures the size of the relationship between age at menarche and BMI. Cross-sectional sampling weights are applied, and standard errors are clustered at the county level.
Elements of $X$ are as follows: age; whether the woman was Javanese (the majority ethnic group); education level (no or elementary education, junior high school, senior high school, or college and above; Sohn, 2013a); residential location (urban or rural residence); whether the woman had had a general check-up performed during the past 5 years; whether the woman had visited a public hospital, puskesmas (government-mandated community health clinics), private hospital, clinic, health worker, or doctor’s practice or been visited by a health worker or doctor; number of days performing vigorous activities for at least 10 minutes continuously during the past 7 days; whether the respondent was the policy holder or primary beneficiary of health benefits or health insurance; number of health facilities for which the woman knew the locations; number of days eating meat during the past week; the natural logarithm of the sum of the values of 13 household assets; and province fixed effects. The 13 household assets are as follows: houses or land occupied by the household; other houses or buildings (including land); land not used for farm or nonfarm business; poultry; livestock or fishponds; hard stem plants not used for farm or nonfarm business; vehicles (cars, boats, bicycles, and motorbikes); household appliance (radio tape recorder, TV, fridge, sewing or washing machine, VCD player, HP, and etc.); savings, certificate of deposit, or stocks; receivables; jewelry; household furniture or utensils; and other assets. When observations with missing values are excluded, the sample size is 9,543.

Smoking status is not included in $X$ because female smoking in Indonesia is highly discouraged, and consequently, almost no Indonesian women smoke (Sohn, 2013b). In addition, as is typically done in social science surveys, all covariates were self-reported, which could cause concerns similar to those for recalled age at menarche. As shown below, however, once age is controlled for, other covariates do not influence $\beta_1$ much, which suggests that these concerns are not serious as far as estimation of $\beta_1$ is concerned. Another concern is selection bias because the sample consists only of ever-married women. For example, if compared with never-married women, ever-married women experienced menarche early (which would improve their marriageability especially for young women under age 20) and subsequently became heavier, $\beta_1$ is biased upward. This potential bias is probably small, however, because controlling for covariates beyond age does not influence $\beta_1$ much, implying that selection bias may be small. Another way of checking the size of this bias is to consider only women aged 30+ because almost all women in this age range are ever married (not shown). Below, we perform this exercise and show that the potential selection bias is small. Even if selection biases $\beta_1$ upward, this only reinforces our primary finding that $\beta_1$ is small. That is, when potentially upward biased $\beta_1$ is small, unbiased $\beta_1$ must be small.

In addition to OLS for the pooled sample, we also estimate specification (1) for a variety of subsamples to investigate whether the results for the pooled sample generally apply to most women, or whether some small groups exhibit particularly large $\beta_1$ while others do not. We also estimate quantile regressions, using specification (1) to examine whether $\beta_1$ for the mean BMI represents $\beta_1$ for the entire distribution of
BMI. Moreover, we shed further light on the upper ends of BMI (i.e., obesity), which is of emerging concern, by replacing BMI on the left-hand side of specification (1) with a dichotomous variable of obesity (i.e., a linear probability model). In addition to elucidating the emerging concern, this exercise is of interest because it allows us to examine whether the relationship is stronger in a continuous or discrete manner.

It is important to note that we do not evaluate causality between menarche, future body fat, and any other factors. This question is difficult to address using social science surveys. In fact, medical research has determined several obesity-related genes to be associated with age at menarche (Elks et al., 2010). Although genes are involved in driving both menarche and body fat (before and after menarche), environmental conditions mediate the relationship between age at menarche and BMI, which is supported by varying degrees of the relationship across countries (see the discussion section). The recent pandemic scale of obesity also proves that more than genes are involved. Thus, it is still meaningful to estimate the magnitude of the relationship to determine whether age at menarche is a good predictor of future body fat. Relatedly, our exercise is valid only for girls who are not obese before menarche. For girls who are obese in childhood, their obese status in childhood can predict their obese status in adulthood earlier and more accurately than age at menarche.

**Results**

**Descriptive statistics**

In Table 1, we present descriptive statistics. We employ many variables in the analysis, and it is time-consuming to discuss all the variables. We thus briefly discuss only the main anthropometrics. The mean height is 151.0 cm, which is short compared with those for other countries. For example, according to Deaton (2007), the mean height for women comparable with our sample is 155.0 cm for Latin America and the Caribbean, 156.9 cm for five Central Asian countries, and 157.8 cm for Africa. In fact, the mean height is so short that it is similar to that of hunter-gatherers and foragers living in subsistence-based economies (Walker et al., 2006). Moreover, this shortness is an extension of a historical trend, as Indonesia belongs to the region where people were the shortest in the world during 1810–1989 (Baten & Blum, 2012). To the extent that height reflects the nutritional and disease environment in childhood (Silventoinen, 2003), the short height implies that Indonesian women greatly suffered from an adverse environment.

Although the mean height of the sample is short, their mean weight (54.0 kg) is not light. As a result, the mean BMI (23.6 kg/m²) is not as small as the mean height suggests. One can appreciate the seriousness of this figure by comparing the prevalence of women with a BMI ≥ 25 across developing countries. In our sample, this figure is 34.1%, but the corresponding figure for comparable women in 42 developing countries is 25.3% (Popkin et al., 2012). Moreover, considering the lower cut-off points of obesity for Asians than for other populations (WHO, 2000a), implications for obesity-related diseases for Indonesian women are greater than the raw difference conveys.
The mean age at menarche is 13.9 years. According to Thomas, Renaud, Benefice, De Meeuws, and Guegan (2001), the mean age of menarche in 67 developed and developing countries is 13.5 years. Considering that their data are rather old and many developing countries have recently experienced economic growth, however, the Indonesian mean age at menarche is probably late. Just like height, to the extent that age at menarche reflects the nutritional and disease environment in childhood (Tanner, 1978), the late age at menarche is consistent with the argument suggested by their short height; that is, the Indonesian women suffered from an adverse environment.

**Baseline results**

Correlation coefficients between age at menarche, BMI, and current age provide an intuitive understanding of the sign and size of the relationship (Table 2). The correlation coefficient between age at menarche and BMI (−0.04) is negative but small, which anticipates a weak relationship between them in multiple regression models. Age at menarche and age are positively correlated, with a correlation coefficient of 0.15, implying that as the nutritional and disease environment improved
over time, younger women experienced menarche earlier. The positive correlation between age and BMI is consistent with the general trend that individuals gain weight as they age (Rokholm, Baker, & Sørensen, 2010); the size of the relationship (0.18) is greater than that between age at menarche and BMI. Overall, the results suggest that age is an important confounding factor between age at menarche and BMI. Thus, it is important to control for age when one investigates the relationship between age at menarche and BMI.

To illustrate this point more formally, we estimate specification (1), first without age, and then with age. In Column 1 of Table 3, we document that $\beta_1$ is $-0.056$ and weakly significant. This figure indicates that the relationship is indeed negative, as consistently demonstrated in the literature. Its magnitude, however, is negligible. For example, a reduction of 1 year in age at menarche, which is equivalent to the reduction experienced by birth cohorts 1944–1988, is related to an increase of only 0.056 in BMI, or 0.24% of the mean BMI.

According to the directions of the three correlation coefficients in Table 2, $\beta_1$ is probably biased downward (toward 0) if age is not taken into account. We thus add

Table 2. Correlation coefficients.

<table>
<thead>
<tr>
<th></th>
<th>BMI</th>
<th>Age at menarche</th>
<th>Current age</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI</td>
<td>1.000</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age at menarche</td>
<td>-0.037</td>
<td>1.000</td>
<td></td>
</tr>
<tr>
<td>Current age</td>
<td>0.182</td>
<td>0.147</td>
<td>1.000</td>
</tr>
</tbody>
</table>

Note: All correlation coefficients are statistically significant with $p$ values $<.01$.

Table 3. Relationship between age at menarche and BMI: OLS.

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at menarche</td>
<td>-0.056 (0.028)**</td>
<td>-0.123 (0.030)**</td>
<td>-0.104 (0.027)**</td>
<td>-0.122 (0.025)**</td>
</tr>
<tr>
<td>Current age</td>
<td>0.061 (0.006)**</td>
<td>0.062 (0.006)**</td>
<td>0.048 (0.005)**</td>
<td></td>
</tr>
<tr>
<td>Javanese</td>
<td>0.259 (0.098)**</td>
<td>0.559 (0.188)**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Junior high school</td>
<td>0.050 (0.157)</td>
<td>-0.400 (0.153)**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Senior high school</td>
<td>0.194 (0.131)</td>
<td>-0.608 (0.120)**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>College or above</td>
<td>0.677 (0.224)**</td>
<td>-0.510 (0.226)**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urban residence</td>
<td>0.953 (0.147)**</td>
<td>0.510 (0.150)**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paid work</td>
<td></td>
<td>-0.034 (0.106)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>General check-up last 5 yrs</td>
<td></td>
<td>0.248 (0.130)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hospital visit last 4 wks</td>
<td></td>
<td>-0.108 (0.101)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vigorous activities last wk</td>
<td></td>
<td>-0.082 (0.022)**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Health insurance</td>
<td></td>
<td>0.402 (0.147)**</td>
<td></td>
<td></td>
</tr>
<tr>
<td># Of known health facilities</td>
<td></td>
<td>0.435 (0.048)**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Meat consumption last wk</td>
<td></td>
<td>0.083 (0.040)**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ln(household assets)</td>
<td></td>
<td>0.156 (0.027)**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>24.44 (0.54)****</td>
<td>23.03 (0.38)****</td>
<td>22.10 (0.44)****</td>
<td>19.16 (0.44)****</td>
</tr>
<tr>
<td>Province fixed effects</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>N</td>
<td>9543</td>
<td>9543</td>
<td>9543</td>
<td>9543</td>
</tr>
<tr>
<td>Adjusted $R^2$</td>
<td>&lt;0.000</td>
<td>0.024</td>
<td>0.042</td>
<td>0.072</td>
</tr>
</tbody>
</table>

Note: Cross-sectional sampling weights are applied. Standard errors clustered at the county level are in parentheses.

* $p$ value $<.10$;
** $p$ value $<.05$;
*** $p$ value $<.01$. 
age to the specification (Column 2). The coefficient on age has the expected positive sign and implies that an increase of 10 years in age is related to an increase of 0.61 in BMI. More importantly, \( \beta_1 \) increases more than twice, but its magnitude remains negligible. Because it is possible that other omitted variables bias \( \beta_1 \) downward, we add basic demographic variables (Column 3). The size of \( \beta_1 \) decreases, but only slightly; when we control for more variables (Column 4), the size of \( \beta_1 \) increases again.

The coefficients for some variables are statistically significant and logical. For example, more days of vigorous activities during the previous week are negatively related to BMI. Variables related to higher SES—urban residence, possession of health insurance, number of known health facilities, meat consumption, and household assets—are positively related to BMI. These findings are in contrast with the negative relationship between SES and BMI in developed countries, but they are consistent with the positive relationship in developing countries (Hruschka & Brewis, 2013). Also note that as we control for more SES variables, the relationship between education and BMI turns from positive to negative. This suggests that once the SES part of education is separated from the rest of human capital (e.g., health consciousness, cognitive skills for health information, and forward-looking perspective), higher human capital is related to better health (implied by a low BMI), which is consistent with the original model in health economics (Grossman, 2000).

All of these supplementary results suggest that the covariates have considerable substance in explaining BMI. Nevertheless, the inclusion of them hardly influences \( \beta_1 : \beta_1 \) in Column 2 is almost identical to that in Column 4. The results in Column 4 strengthen the argument that potential downward bias in \( \beta_1 \) stemming from omitted variables is probably minimal. If such covariates exist, they need to be strongly correlated with age at menarche but weakly correlated with the rest of the covariates. Given the large number of covariates included in the specification, this possibility seems small. The changes in \( \beta_1 \) in Columns 2–4 also support this. Thus, even the largest \( \beta_1 \) in the table indicates that a reduction of 1 year in age at menarche is related to an increase of only 0.12 in BMI, or much smaller than 1% of the mean BMI.

To check a possible nonlinear relationship between age at menarche and BMI, we add the square of age at menarche to specification (1). The coefficients for both terms are not statistically significant, regardless of the presence and various combinations of the covariates (not shown). This suggests that the relationship is primarily linear.

We can further support this linear relationship by a graphical summary, presented in Figure 1. For this figure, we first estimate the following OLS specification.

\[
BMI_i = \alpha_1 + \alpha_2 Age_i + u_i
\]

(2)

where \( Age \) refers to age, \( u \) to an error term, and \( \alpha_1 \) and \( \alpha_2 \) to coefficients to estimate. We apply cross-sectional sampling weights and cluster standard errors at the county level. Second, we derive estimated \( u (\hat{u}) \), which allows us to adjust age for BMI. Third, we regress \( \hat{u} \) on age at menarche using locally weighted regressions (lowess), depicting the relationship between age at menarche and age-adjusted
BMI. Lowess is attractive in that it provides a nonparametric, hence, flexible way of examining the relationship. At the same time, lowess also checks whether the results in Table 3 are robust to methodological changes.

In Figure 1, the relationship is more complicated than suggested in Table 3. The ages at menarche 12–16 account for 90% of the sample, however, and the relationship is generally linear in this range. This strong linearity is notable because we nonparametrically estimate it. Using Figure 1, we support the plausibility of specification (1) and the results in Table 3. Nevertheless, the range of the y-axis indicates that the magnitude of the relationship is small even for this age range.

**Additional results**

Using the pooled estimations, we provide concise information on the relationship between age at menarche and BMI. The information may be too concise, however, if some subgroups exhibit a large relationship between the two, while other subgroups exhibit no relationship. To check this possibility, we divide the pooled sample into subgroups according to seven categories: non-Javanese versus Javanese, age <30 versus age ≥30, under senior high school versus senior high school and above, below versus above median household assets, no versus any meat consumption during the past week, no versus any vigorous activities during the past week, and rural versus urban residence. We control for only age to make \( \beta_1 \) large, which is against our argument that \( \beta_1 \) is small.

In Table 4, \( \beta_1 \) varies depending on the subgroups; however, irrespective of the subgroups, \( \beta_1 \) is consistently small. The smallest \( \beta_1 \) is estimated for women with any vigorous activities (-0.03, not statistically significant), while the largest \( \beta_1 \) is for women with a senior high school education or above (-0.25). In addition, when we separately analyze women under age 30 and 30+ (Rows 3 and 4), \( \beta_1 \) is...
greater for the younger group (−0.24) than for the older group (−0.09), which is consistent with the upward sample selection, as argued above. The difference in $b_1$ between the two groups, however, is not only due to the selection bias; some omitted cohort effects exist. That said, $b_1$ for women aged 30, for whom the selection bias should be negligible, remains small. Furthermore, when we compare $b_1$ for women aged 30 and $b_1$ for the pooled sample (Column 2 of Table 3), the upward selection bias in the latter does not seem serious. In any event, the challenge that the upward selection bias is serious only strengthens our argument that $b_1$ is small.

Another possibility against our argument is that although $b_1$ is small, it could be particularly high for obese women. This is possible because women who experience early menarche tend to have a higher BMI before and after menarche than women who experience late menarche. To check this possibility, we estimate quantile regressions for specification (1), controlling for only age (Table 5). The relationship between age at menarche and BMI monotonically increases with BMI. Thus, at quantile 0.95, a reduction of 1 year in age at menarche is related to an increase of 0.28 in BMI. Even this, however, equates to only 1.2% of the mean BMI.

Table 4. Relationship between age at menarche and BMI by subsample: OLS.

<table>
<thead>
<tr>
<th>Category</th>
<th>Age at menarche</th>
<th>Mean BMI</th>
<th>$N$</th>
<th>Adjusted $R^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>−0.064 (0.057)</td>
<td>23.5</td>
<td>5478</td>
<td>0.020</td>
</tr>
<tr>
<td>2</td>
<td>−0.185 (0.036)**</td>
<td>23.8</td>
<td>4065</td>
<td>0.029</td>
</tr>
<tr>
<td>3</td>
<td>−0.243 (0.052)**</td>
<td>22.3</td>
<td>3137</td>
<td>0.049</td>
</tr>
<tr>
<td>4</td>
<td>−0.090 (0.038)**</td>
<td>24.1</td>
<td>6406</td>
<td>0.001</td>
</tr>
<tr>
<td>5</td>
<td>−0.071 (0.037)**</td>
<td>23.6</td>
<td>6512</td>
<td>0.014</td>
</tr>
<tr>
<td>6</td>
<td>−0.247 (0.056)**</td>
<td>23.9</td>
<td>3031</td>
<td>0.095</td>
</tr>
<tr>
<td>7</td>
<td>−0.063 (0.036)**</td>
<td>23.1</td>
<td>4772</td>
<td>0.008</td>
</tr>
<tr>
<td>8</td>
<td>−0.172 (0.057)**</td>
<td>24.2</td>
<td>4771</td>
<td>0.034</td>
</tr>
<tr>
<td>9</td>
<td>−0.107 (0.056)**</td>
<td>23.1</td>
<td>3670</td>
<td>0.012</td>
</tr>
<tr>
<td>10</td>
<td>−0.132 (0.029)**</td>
<td>24.0</td>
<td>5873</td>
<td>0.034</td>
</tr>
<tr>
<td>11</td>
<td>−0.145 (0.024)**</td>
<td>23.8</td>
<td>7805</td>
<td>0.035</td>
</tr>
<tr>
<td>12</td>
<td>−0.034 (0.088)</td>
<td>23.1</td>
<td>1738</td>
<td>0.001</td>
</tr>
<tr>
<td>13</td>
<td>−0.059 (0.050)</td>
<td>23.2</td>
<td>4569</td>
<td>0.007</td>
</tr>
<tr>
<td>14</td>
<td>−0.174 (0.031)**</td>
<td>24.3</td>
<td>4974</td>
<td>0.052</td>
</tr>
</tbody>
</table>

Note: Age is controlled for but not listed. Cross-sectional sampling weights are applied. Standard errors clustered at the county level are in parentheses.

Table 5. Relationship between age at menarche and BMI: Quantile regression.

<table>
<thead>
<tr>
<th>Quantile</th>
<th>0.05</th>
<th>0.25</th>
<th>0.50</th>
<th>0.75</th>
<th>0.95</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at menarche</td>
<td>−0.035 (0.042)</td>
<td>−0.078 (0.032)**</td>
<td>−0.216 (0.034)**</td>
<td>−0.271 (0.045)**</td>
<td>−0.277 (0.072)**</td>
</tr>
<tr>
<td>Pseud $R^2$</td>
<td>0.001</td>
<td>0.015</td>
<td>0.029</td>
<td>0.031</td>
<td>0.023</td>
</tr>
</tbody>
</table>

Note: Age is controlled for but not listed. The mean BMI is 23.6. Cross-sectional sampling weights are applied. Standard errors clustered at the county level are in parentheses.
After knowing that important dynamics between age at menarche and BMI occur at the high ends of BMI, one could contend that the relationship between age at menarche and obesity may be larger than that between age at menarche and BMI. This exercise is of interest because it allows us to examine whether $\beta_1$ is greater in a continuous or discrete manner at the upper ends of BMI. We thus check this possibility by using specification (1). The dependent variable is a dummy variable of obesity, and age is the only covariate. We employed three cut-off points for obesity to define obesity: 25, 27, and 30. While the WHO (2000a) suggested a cut-off point of 25 for Asian adults, the WHO Expert Consultation (2004) suggested a cut-off point of 27 for Indonesians. Sohn (2014c) argued that this point is the best predictor of chronic diseases for Indonesians compared with other anthropometrics, including a cut-off point of 25. A cut-off point of 30 is conventional when BMI is used to define obesity.

In Table 6, the magnitude of $\beta_1$ is larger than the baseline case. Specifically, when we consider cut-off points of 25, 27, and 30, a 1-year reduction in age at menarche is related to an increase of 1.32 percentage points, 1.43 percentage points, and 0.71 percentage points, respectively, in obesity. These figures correspond to 3.9%, 7.0%, and 8.8% of the mean obesity rate for each. Thus, $\beta_1$ is greater in a discrete than continuous manner. More importantly, the practically meaningful relationship between age at menarche and BMI occurs at the high ends of BMI. It is, however, worth recalling that these numbers are upper bounds, given that the upward selection bias potentially exists and age is the only covariate. These caveats weaken the power of age at menarche as a predictor of future body fat in Indonesia.

### Discussion

We estimate the degree to which age at menarche can serve as a predictor of future body fat (proxied by BMI) in Indonesia. The relationship between age at menarche and BMI is statistically significant, but its size is small. This is the case whether we pool or divide the sample. As BMI becomes larger, the size monotonically increases, but the size remains small even at the high ends of BMI.

One can appreciate the small size of $\beta_1$ for Indonesia by comparing it with those for other countries. Recall that an upper bound of $\beta_1$ for the pooled sample is

### Table 6. Relationship between age at menarche and obesity: OLS.

<table>
<thead>
<tr>
<th></th>
<th>BMI ≥ 25</th>
<th>BMI ≥ 27</th>
<th>BMI ≥ 30</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age at menarche (/10)</strong></td>
<td>-0.132 (0.027)**</td>
<td>-0.143 (0.026)**</td>
<td>-0.071 (0.022)**</td>
</tr>
<tr>
<td><strong>N</strong></td>
<td>9543</td>
<td>9543</td>
<td>9543</td>
</tr>
<tr>
<td><strong>Adjusted R^2</strong></td>
<td>0.022</td>
<td>0.017</td>
<td>0.009</td>
</tr>
</tbody>
</table>

**Note:** Age is controlled for but not listed. Cross-sectional sampling weights are applied. Standard errors clustered at the county level are in parentheses.

* $p$ value < .10;
** $p$ value < .05;
*** $p$ value < .01.
—0.12. Although specifications and samples vary, comparisons suggest that, in general, $\beta_1$ is greater for developed than developing countries; moreover, $\beta_1$ for Indonesia is smaller even compared with that for India.

There are relatively many U.S. studies, and therefore, we first discuss $\beta_1$ for the United States. For a sample of White women aged 20–34 recruited in 1959–1974, Garn, LaVelle, Rosenberg, and Hawthorne (1986) calculated that the mean BMI was 23.58 for the earliest maturing women (age at menarche $\leq$ 11 years) and 21.66 for the latest maturing group (age at menarche $\geq$ 14 years). If we liberally assume the difference in the median ages at menarche between the two groups to be 6 years, $\beta_1$ is $-0.32$. Burke and colleagues (1992) provided mean current BMIs for women aged 18–30 who participated in a cardiovascular disease risk factor examination in 1985–1986. The mean BMI was 27.6 for the earliest maturing Black women (ages at menarche 6–11 years) and 24.1 for the latest maturing Black women (ages at menarche 14–19 years). If we again assume the difference in the median ages at menarche between the two groups to be 6 years, $\beta_1$ is $-0.58$. The corresponding figure for White women is $-0.43$. Wellens and colleagues (1992) provided the ages at menarche and BMIs of two groups of White students at the University of Texas at Austin (measured in 1970 and 1987) and of White participants in the Fels Longitudinal Study (mean age 18.3 years). They defined the earliest maturing group as age at menarche $< 12$ years and the latest maturing group as age at menarche $> 14$ years, and SDs of the ages at menarche of the three groups ranged 1.1–1.4. Thus, when we assume a difference of 5 years in the median ages between the two groups, $\beta_1$ is $-0.10$ for the 1970 sample, $-0.28$ for the 1987 sample, and $-0.70$ for the Fels sample. For a sample of women aged 18–37 recruited from Bogalusa, Louisiana, United States, in 1982–1996, Freedman and colleagues (2003) estimated that the age-adjusted difference in BMI between the earliest (age at menarche $< 12$) and latest (age at menarche $\geq 13.5$ years) maturing groups was 3.6 for White women and 3.2 for Black women. Given that the SD of age at menarche for both races is 1 year, it would be too liberal to use a difference of 5 years in the median ages at menarche between the two groups. Nevertheless, when we take this difference, $\beta_1$ is $-0.72$ for White women and $-0.64$ for Black women.

Regarding the United Kingdom, Power, Lake, and Cole (1997) evaluated British women aged 33 born in March 1958 and provided mean BMIs by age at menarche. The BMI difference between the earliest (age at menarche $\leq 11$ years) and latest (age at menarche $\geq 15$ years) maturing groups was 4.1. If we assume a liberal difference of 6 years in the median ages at menarche between the two groups, $\beta_1$ is $-0.68$. Pierce and Leon (2005) reported similar results for women aged 45–52, born in Aberdeen, Scotland, in 1950–1955. When they regressed age at menarche on BMI, controlling for age, $\beta_1$ was $-0.64$. For a sample of British women aged 53, born in one week in March 1946, Hardy, Kuh, Whincup, and Wadsworth (2006) calculated that the mean BMI was 28.7 for the earliest (age at menarche $\leq 12$ years 3 months) and 25.8 for the latest (age at menarche $\geq 14$ years 7 months) maturing women. When we assume a difference of 5 years in the median ages between the
two groups, $\beta_1$ is $-0.58$. Using the same sample, Pierce, Kuh, and Hardy (2010) regressed age at menarche on BMI, allowing us to check whether the rough estimate of $-0.58$ is plausible. They found that $\beta_1$ was $-0.75$. These two related studies deserve attention because they demonstrated that a difference of 5 years in age at menarche between the earliest and latest maturing groups for Hardy and colleagues (2006) is indeed liberal.

Going beyond the U.S. and the UK, Jacobson (1954) listed height and weight by age at menarche for Norwegian hospital nurses aged 20–34; the earliest ages at menarche were 11–13, and the latest 15–18. If we assume the difference in the median ages at menarche between the two groups to be 5 years, $\beta_1$ is $-0.17$. Laitinen, Power, and Järvelin (2001) analyzed a sample of northern Finnish women aged 31 born in 1966 and estimated that $\beta_1$ was $-0.56$ (when BMI at 14 years of age was not controlled for). Hulanicka, Lipowicz, Koziel, and Kowalisko (2007) examined a representative sample of women aged 50 who were born in the city of Wrocław, Poland, in 1953. They did not report $\beta_1$ but estimated a correlation coefficient of $-0.20$ between age at menarche and BMI at age 50, which is much greater than ours ($-0.04$).

Note that $\beta_1$ (or the correlation coefficient) in all these studies is greater than that in our study. In the previous studies, however, $\beta_1$ is likely to be biased downward for at least one of the following reasons: age was not adjusted, the study period was old, the sample was young, and a liberally assumed difference in the median or mean ages between the earliest and latest maturing groups. As in Table 3, after age adjustment, $\beta_1$ increases more than twice. Even if age adjustment did not increase $\beta_1$ in some previous studies as much as in ours, age adjustment would increase it to a nonnegligible extent. In addition, obesity emerged as a serious public health issue in the United States only in the 1980s and spread to other developed countries thereafter. Specifically, obesity showed little change in the United States in 1960–1980. The prevalence of obesity, however, increased by 8 percentage points between the 1976–1980 survey and the 1988–1994 survey, followed by a similar increase between the 1988–1994 survey and the 1999–2000 survey. Subsequently, the growth rate declined but continued to be positive, with an ambiguous sign of leveling-off (Flegal, Carroll, Kit, & Ogden, 2012). This pattern also occurred in other developed countries (Rokholm et al., 2010). Thus, if the study period were the 1990s or later, $\beta_1$ in some previous studies would be greater. Relatedly, as BMI increases with age up to some old ages (Rokholm et al., 2010), $\beta_1$ for old women would be greater than that for young women. This suggests that $\beta_1$ in some previous studies with young women is likely to be biased downward. In addition, by comparing $\beta_1$ from Hardy and colleagues (2006) and that of Pierce and colleagues (2010), one can see that the assumed differences (e.g., 5 or 6 years) in the median or mean ages between the earliest and latest maturing groups for rough estimations of $\beta_1$ appear too great. A difference of 4 years is more plausible, but we adopt liberal differences above to make the estimates against our argument. If all these
factors are considered, the differences between $\beta_1$ for the developed countries and that in this study would be much greater than the raw differences imply.

Only a couple of studies exist for developing countries. For example, Sharma, Talwar, and Sharma (1988) provided anthropometrics of 147 healthy unmarried women aged 18–21 recruited from an urban Punjabi population of Chandigarh and Ludhiana, India; all of them belonged to middle socioeconomic group families. According to this information, the BMIs of the earliest (age at menarche 11 years) and the latest (age at menarche 16 years) maturing women were 19.68 and 18.23. When we divide this difference by 5 years, $\beta_1$ is $-0.29$. This figure is smaller than that of the developed countries but greater than ours. This is the case especially when some of the factors potentially biasing $\beta_1$ downward are taken into account. $\beta_1$ similar to ours is reported only for one province of China. Feng and colleagues (2008) estimated that for women aged 25–64 recruited from Anhui in 2004–2005, the age-adjusted $\beta_1$ was $-0.13$.

The socioeconomic circumstances of Indonesia and Anhui, China, help explain the small size of $\beta_1$ for both. Despite Indonesia’s fast economic growth since the late 1960s, the standard of living continues to be low. For example, Indonesia ranked 121 in the human development index in 2012. Malnutrition remains of important concern in Indonesia (Roemling & Qaim, 2013). People in Anhui suffered severe drought and famine in the late 1970s, in addition to the Great Leap Forward and the Cultural Revolution, which devastated the entire country in the late 1950s–mid-1970s. It appears, therefore, that the relationship between age at menarche and BMI is strong when malnutrition poses little concern. Otherwise, it is weak, and, consequently, age at menarche cannot serve as a good predictor of future body fat. It is revealing that in India, $\beta_1$ was large for girls from affluent families (Sharma et al., 1988).

**Conclusions**

When age at menarche fails to be a good predictor of future body fat, other predictors—if causal factors are difficult to obtain—need to be sought. In the meantime, national programs aimed at preventing obesity need to be launched in the developing world. This is particularly the case because more people in the developing than developed world are afflicted by obesity at present and are projected to be in the future. This is also beneficial in the developing world since prevention is more cost effective than treatment. Furthermore, such national programs should not be limited to the two most commonly advanced reasons for the increase in the prevalence of obesity—food marketing practices and institutionally driven reductions in physical activity. McAllister and colleagues (2009) challenged that these might not be the main driving force behind the obesity epidemic. Instead, they provided 10 putative contributors to obesity that have been supported by strong evidence, but neglected by the public. It is worth keeping in mind that there are many levers to prevent obesity.

A simple copy of programs implemented in the developed world is not recommended for the developing world, however, because malnutrition remains a serious
public health concern (Doak et al., 2005). A naïve attempt to reduce weight may cause the unintended consequence of an increase in malnutrition. Approaches different from those for the developed world need to be envisioned for the developing world. For this vision, one of the 10 contributors to obesity proposed by McAllister and colleague (2009)—intrauterine and intergenerational effects—is worth highlighting. David Barker (1998) is credited with finding a link between early fetal growth patterns and risk of several chronic diseases in adulthood. One of the many findings is that intrauterine malnutrition is related to obesity in adulthood, which has also been supported by experiments on rats (Jones & Friedman, 1982). The dual burden of disease, partly driven by the nutrition transition, persists in the developing world; children are typically underweight and adults obese (Doak et al., 2005). This contributor is especially relevant in Indonesia. This is because, as shown in Table 1, Indonesian women are very short, and the small uterine environment constrains fetal growth. Consequently, intrauterine growth restriction is related to obesity in adulthood, and their offspring also become obese. This is aggravated by the nutrition transition, and nutritional strategies that promote catch-up growth may backfire in the form of adult and intergenerational obesity. Thus, efforts unique to the developing world are desirable to end this vicious circle.

Future research can improve on this study. One way would be to replicate our strategy for other developing countries. We mention one study each for India and China, but both studies relied on isolated samples, especially for the former. Although the large populations in these countries make it difficult to collect nationally representative samples, it is worthwhile, considering the rapidly growing prevalence of obesity. At the same time, one can perform replications for other less populous developing countries. In particular, since extremely high rates of obesity have been observed in several of the Pacific islands—for example, 79% of adults were recorded as obese (BMI ≥ 30) in Nauru in 1994 (Prentice, 2006), it is urgent to call attention to these populations. It would be also interesting to employ proxies for body fat other than BMI and check whether the relationship between age at menarche and these proxies remains weak. In this pursuit, however, one should bear in mind that such proxies need to be related to morbidity and mortality. Otherwise, even if the relationship is strong, it is not useful to reduce morbidity and mortality.

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References


