32.4 million small for gestational age (SGA) and 12.1 million preterm births occur in low- and middle-income countries (LMIC) annually. These newborns experience increased risk of neonatal and infant mortality and of long-term health consequences. Understanding causal mechanisms that lead to SGA and preterm birth is critical to reducing this burden. As part of the Child Health Epidemiology Reference Group, we explored nutritional and reproductive maternal risk factors associated with SGA and preterm outcomes in LMICs, including short maternal stature, young/advanced maternal age, low/high parity, and short/long birth interval. We highlight our findings and also summarize existing literature on how low/high BMI and low weight gain during pregnancy, respectively, are associated with SGA and/or preterm outcomes.

**Height**

Short maternal stature is associated with both SGA and preterm birth. Short stature may limit the uterine volume for fetal growth [1]. Main exposures that determine final attained height are considered to occur during the fetal period and early childhood, but there is increasing interest in exploring interventions in later childhood or adolescence to promote catch-up growth.

**BMI and Weight Gain during Pregnancy**

Systematic reviews have shown statistically significant associations between low BMI/low weight gain and fetal growth restriction [2, 3]. In addressing acute malnutrition during pregnancy through protein-energy supplementation, it is important to be cautious of potential
consequences; larger fetal size in areas where maternal stunting rates are high may potentially increase rates of cephalopelvic disproportion and obstructed labor.

### Maternal Age and Parity

We examined the association of maternal age and parity with SGA/preterm outcomes [4]. We found that women who were both age <18 years and nulliparous had the highest odds of SGA and preterm when compared to women in the reference category (age 18 to <35 years and parity 1–2; tables 1 and 2). Those who were parity ≥3 had heightened risk of preterm. We saw no impact of advanced age or high parity on SGA.

The association between young age and adverse neonatal outcomes may be operating through multiple biological mechanisms. The mother may have experienced incomplete physical growth prior to pregnancy, leading to lower stature and smaller pelvic dimensions, thus constraining fetal growth. Young age may also be a proxy for poor socioeconomic status and malnutrition. We hypothesize that the association between high parity and preterm birth may largely be due to residual confounding.

### Birth Interval

Several meta-analyses have reported increased risk of SGA and preterm among those born after a short birth interval. Understanding birth intervals as an exposure is difficult, particularly in low-resource settings.

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**Table 1. Summary of meta-analyses examining association between maternal short stature and SGA/preterm**

<table>
<thead>
<tr>
<th>Publication</th>
<th>Exposure/reference</th>
<th>SGA</th>
<th>Preterm</th>
</tr>
</thead>
<tbody>
<tr>
<td>WHO Collaborative Study [3]</td>
<td>lowest vs. highest quartile of height</td>
<td>crude OR 1.9 (1.8–2.0)</td>
<td>crude OR 1.2 (1.1–1.2)</td>
</tr>
<tr>
<td>Knowledge Synthesis Group [6]</td>
<td>varied by study</td>
<td>aOR 1.39 (1.15–1.68)</td>
<td>crude OR 1.23 (1.11–1.37); adjusted data mostly not significant</td>
</tr>
<tr>
<td>CHERG [under review]</td>
<td>&lt;145 cm</td>
<td>aRR 1.77 (1.61–1.95)</td>
<td>aRR 1.44 (1.20–1.75)</td>
</tr>
<tr>
<td></td>
<td>145 to &lt;150 cm</td>
<td>aRR 1.50 (1.41–1.60)</td>
<td>aRR 1.13 (1.04–1.22)</td>
</tr>
<tr>
<td></td>
<td>150 to &lt;155 cm</td>
<td>aRR 1.31 (1.26–1.36)</td>
<td>aRR 1.10 (1.02–1.18)</td>
</tr>
<tr>
<td></td>
<td>≥155 cm (ref.)</td>
<td>ref.</td>
<td>ref.</td>
</tr>
</tbody>
</table>
Interpregnancy interval (birth to next conception) is the preferred measure of the exposure, compared to birth intervals (birth to next birth). However, the former cannot be properly measured without ultrasound dating. It is also unclear how best to account for miscarriages and early stillbirths in these intervals, events that may not have as large a burden on the mother as a full pregnancy, but more than not having conceived at all.

Existing literature highlights various nutritional and reproductive health-related exposures associated with SGA and preterm birth. Some interventions have demonstrated efficacy in reducing these outcomes, but there is generally less evidence of effectiveness. Chronic malnutrition may require intergenerational intervention, and in addressing acute malnutrition, potential consequences of increasing fetal size need to be taken into account. While family planning can reduce the adverse effects of early pregnancy, increased access to contraceptives affects young age at first birth the least out of all reproductive health-related risk factors.
More research is necessary on how to maximize the effectiveness of known interventions, but also on uncovering new, efficacious interventions, taking into account the independent and shared causal mechanisms operating on SGA and preterm outcomes.

References


