Development of a Prediction Equation for Insulin Sensitivity From Anthropometry and Fasting Insulin in Prepubertal and Early Pubertal Children

Terry T.-K. Huang, PhD, MPH
Maria S. Johnson, PhD
Michael I. Goran, PhD

OBJECTIVE — To test the utility of homeostasis model assessment (HOMA) in predicting insulin sensitivity [\(10^{-4} \text{ mmol}^{-1} \cdot \text{L}^{-1} \cdot \text{min}^{-1}\)] in children and to develop and compare two new prediction equations for insulin sensitivity in children using demographic and anthropometric measures in the presence or absence of fasting insulin.

RESEARCH DESIGN AND METHODS — We studied 156 white and African-American children with complete data (mean age 9.7 ± 1.8 years, 87.8% Tanner Stage 1 or 2). For development of new equations, two-thirds of the children were randomly assigned to a development group, whereas the remaining children were assigned to a cross-validation group.

RESULTS — A modified HOMA equation accurately predicted insulin sensitivity, but its utility is similar to fasting insulin alone. Demographic and anthropometric measures alone did not predict insulin sensitivity accurately, even when precise measures of body composition were included in the prediction model. Ethnicity, calf skinfold, and fasting insulin together explained 73% of the variance in insulin sensitivity and accurately predicted insulin sensitivity. The regression of measured versus predicted insulin sensitivity in the cross-validation group was not significantly different from the line of identity (\(P > 0.05\)). Mean difference between measured and predicted insulin sensitivity was also not significant (\(P > 0.05\)). Some bias was apparent, particularly in white boys.

CONCLUSIONS — Ethnicity, calf skinfold, and fasting insulin can accurately predict insulin sensitivity with greater precision than HOMA or fasting insulin alone (\(R^2 = 0.73\)). Future studies, however, are needed to examine whether a universal equation is possible. A cross-validated prediction equation may be useful in population-based studies when complex measures of insulin sensitivity are not available.

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RESEARCH DESIGN AND METHODS

Subjects
A total of 156 children were recruited by newspaper and radio advertisements and by word of mouth. Subjects were screened by medical history and were ineligible if they were 1) <4 years of age; 2) taking medications known to affect body composition or physical activity (e.g., prednisone, Ritalin, or growth hormone); 3) previously diagnosed with syndromes known to affect body composition or fat distribution (e.g., Cushing’s syndrome, Down’s syndrome, type 1 diabetes, or hypothyroidism); or 4) diagnosed previously with any major illness. Because the intent was to recruit a heterogeneous group of children, there were no criteria for other characteristics such as obesity. This study was approved by the Institutional Review Board at the University of Alabama at Birmingham. Parents provided informed consent before testing began.

Protocol and measurements of anthropometry
Children (n = 156) were admitted to the General Clinical Research Center late in the afternoon for an overnight visit. On the following morning after an overnight fast, blood was collected and a tolbutamide-modified frequently sampled intravenous glucose tolerance test (FSIGTT) was performed.

Tolbutamide-modified FSIGTT
At 0600 on the morning after admission to the General Clinical Research Center a topical anesthetic (Emla cream) was applied to the antecubital space of both arms, and at 0700 flexible intravenous catheters were inserted. Three blood samples (2 ml) were collected for determination of basal glucose and insulin. At time 0, glucose (25% dextrose, 11.4 g/m²) was administered intravenously. Blood samples (2 ml) were then collected at the following times relative to glucose administration at 0 min: 2, 3, 4, 5, 6, 8, 10, 14, 19, 22, 25, 30, 40, 50, 70, 100, 140, and 180 min. Tolbutamide (125 mg/m²) was injected intravenously at 20 min. Sera were analyzed for glucose and insulin, and values were entered into the MINMOD computer program (Version 3.0) for determination of insulin sensitivity (10–12).

Assay of glucose and insulin
Glucose was measured in 10 μl serum using an Ektachem DT II System (Johnson and Johnson Clinical Diagnostics, Rochester, NY). In our laboratory, this analysis has a mean intra-assay coefficient of variation (CV) of 0.61% and a mean interassay CV of 1.45%.

Insulin was assayed in duplicate 200-μl aliquots with Coat-A-Count kits (Diagnostic Products, Los Angeles, CA). According to the supplier, cross-reactivity of this assay with proinsulin is 40% at midcurve; C-peptide was not detected. In our laboratory, this assay had a sensitivity of 11.4 pmol/l (1.9 μU/ml), a mean intra-assay CV of 5%, and a mean interassay CV of 6%. Commercial quality control sera of low, medium, and high insulin concentration (Lyphochek; Bio-Rad, Anaheim, CA) were included in every assay to monitor variation over time.

Assessment of insulin sensitivity by HOMA
The HOMA yields an equation (5) where insulin resistance = [fasting insulin (μU/ml) * fasting glucose (mmol/l)] / 22.5.
RESULTS — The descriptive characteristics of the sample with regard to ethnicity, gender, age, Tanner Stage, weight, height, waist circumference, calf skinfold thickness, suprailiac skinfold thickness, fasting glucose, fasting insulin, and insulin sensitivity are shown in Table 2. The development group and the cross-validation group did not differ on any of these variables (P > 0.05). Because the current sample is part of a larger, heterogeneous observational study on childhood obesity, the range of insulin sensitivity reflects that heterogeneity. The range of insulin sensitivity in the current study is consistent with what we have published previously. Although insulin sensitivity obtained from the minimal model cannot be compared directly with that obtained from the clamp technique, the two methods have been shown to correlate as highly as 0.89 (13). Therefore, they both give valid assessments of overall insulin sensitivity (13).

Fitting HOMA in children
Regression of FSIGTT-measured insulin sensitivity on HOMA-estimated insulin resistance in the development group yielded an equation where log, insulin sensitivity = 2.393 – (0.306 \times \text{HOMA insulin resistance}). Insulin sensitivity predicted by HOMA accounted for 63.4% of the variance in observed insulin sensitivity. Although insulin sensitivity obtained from the minimal model cannot be compared directly with that obtained from the clamp technique, the two methods have been shown to correlate as highly as 0.89 (13). Therefore, they both give valid assessments of overall insulin sensitivity (13).

Figure 1 — Measured versus modified HOMA-predicted loge insulin sensitivity in the cross-validation sample (n = 52). Unit of insulin sensitivity = 10^{-4} \text{ min}^{-1}/(\mu \text{IU/ml}). Conversion between loge insulin sensitivity and insulin sensitivity on its natural scale is shown in Table 1. Insulin sensitivity measured by FSIGTT, regressed against insulin sensitivity predicted from equation using HOMA in the development group. Regression trend (dashed line) not significantly different from line of identity.

Table 2 — Descriptive characteristics of the subjects

<table>
<thead>
<tr>
<th>Variable</th>
<th>Statistics</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
</tr>
<tr>
<td>Age (years)</td>
<td>9.7 ± 1.8</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>40.5 ± 14.0</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>139.7 ± 12.5</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>67.9 ± 12.0</td>
</tr>
<tr>
<td>Suprailiac skinfold (mm)</td>
<td>16.2 ± 10.3</td>
</tr>
<tr>
<td>Calf skinfold (mm)</td>
<td>17.0 ± 9.1</td>
</tr>
<tr>
<td>Fasting glucose (mg/dl)</td>
<td>93.5 ± 5.9</td>
</tr>
<tr>
<td>Fasting insulin (\mu\text{IU/ml})</td>
<td>13.7 ± 7.8</td>
</tr>
<tr>
<td>Insulin sensitivity [10^{-4} \text{ min}^{-1}/(\mu\text{IU/ml})]</td>
<td>5.4 ± 3.6</td>
</tr>
</tbody>
</table>

Gender 63.5% girls 36.5% boys
Ethnicity 43.0% white 57.0% African-American
Tanner stage 68.6% Stage 1 19.2% Stage 2 9.0% Stage 3 3.2% Stage 4
In addition, bias was detected by regressing the difference between FSIGTT-measured insulin sensitivity and insulin sensitivity predicted by the equation containing HOMA on FSIGTT-measured insulin sensitivity (intercept $\pm$ SEM = 0.70 ± 0.09, $P < 0.001$; slope $\pm$ SEM = 0.50 ± 0.06, $P < 0.001$; $r = 0.77$, $P < 0.001$; see Fig. 2).

**Development of prediction equation with demographic and anthropometric measures**

For children randomly assigned to the development group, the potential demographic and anthropometric predictors of insulin sensitivity were entered into a stepwise prediction model, and an equation including calf skinfold thickness ($P < 0.001$), ethnicity ($P < 0.001$), weight ($P < 0.05$), and gender ($P < 0.05$) was defined (Table 3). In this equation, calf skinfold thickness accounted for 42.5% of the variance in insulin sensitivity and ethnicity accounted for 13.5% of the variance, whereas weight and gender accounted for 2.5 and 1.8% of the variance, respectively (total $R^2 = 0.60$). Cross-validation of the equation was performed in the randomly assigned validation group. Through regression analysis of the measured versus predicted insulin sensitivity, the equation with only demographic and anthropometric measures was significantly different from the line of identity (intercept $\pm$ SEM = 0.64 ± 0.15, $P < 0.001$; slope $\pm$ SEM = 0.83 ± 0.14, $P > 0.05$; see Fig. 3). By paired Student’s $t$ tests, there was a significant mean difference between measured and predicted insulin sensitivity, using the equation with only demographic and anthropometric measures (untransformed mean $\pm$ SD = 5.4 ± 3.9 vs. 3.0 ± 1.5 × 10^{-4} min^{-1}/(μU/ml), $P < 0.001$).

**Development of prediction equation with demographic, anthropometric, and fasting blood measures**

A second stepwise regression model was performed using potential demographic, anthropometric, as well as fasting insulin and glucose measures. This analysis

<table>
<thead>
<tr>
<th>Step variable</th>
<th>Regression equation for insulin sensitivity</th>
<th>Model $R^2$</th>
<th>$P$ value variable entered</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Calf skinfold (SF)</td>
<td>$-0.059 * \text{Calf SF} + 2.350$</td>
<td>0.42</td>
<td>$&lt;0.001$</td>
</tr>
<tr>
<td>2. Ethnicity</td>
<td>$-0.067 * \text{Calf SF} + 0.601 * \text{Ethnicity} + 2.153$</td>
<td>0.56</td>
<td>$&lt;0.001$</td>
</tr>
<tr>
<td>3. Weight</td>
<td>$-0.044 * \text{Calf SF} + 0.581 * \text{Ethnicity} - 0.017 * \text{Weight} + 2.469$</td>
<td>0.58</td>
<td>$&lt;0.02$</td>
</tr>
<tr>
<td>4. Gender</td>
<td>$-0.041 * \text{Calf SF} + 0.616 * \text{Ethnicity} - 0.018 * \text{Weight} + 0.228 * \text{Gender} + 2.345$</td>
<td>0.60</td>
<td>$&lt;0.05$</td>
</tr>
</tbody>
</table>
yielded an equation with fasting insulin (P < 0.001), ethnicity (P < 0.001), and calf skinfold thickness (P < 0.001) as the significant predictors (Table 4). In this equation, fasting insulin accounted for 63.8% of the variance in insulin sensitivity, ethnicity accounted for 5.1% of the variance, and calf skinfold thickness accounted for 3.7% of the variance (total \( R^2 = 0.73 \)).

Cross-validation of this equation showed that the regression of measured versus predicted insulin sensitivity was not significantly different from the line of identity (intercept \( \pm \text{SEM} = 0.07 \pm 0.15 \); slope \( \pm \text{SEM} = 0.97 \pm 0.09, P > 0.05 \); see Fig. 4). In addition, paired Student’s \( t \) tests showed no significant difference between measured and predicted insulin sensitivity (untransformed mean \( \pm \text{SD} = 5.4 \pm 3.9 \) vs. \( 4.8 \pm 2.4 \times 10^{-4} \text{ min}^{-1}/(\text{mU/ml}), P > 0.05 \)). The equation including fasting insulin was therefore successfully cross-validated. Table 5 shows the means of measured and predicted insulin sensitivity by gender and ethnicity.

An examination of the discrepancy between measured and predicted insulin sensitivity as a function of measured insulin sensitivity in the cross-validation group is shown in Fig. 5 (intercept \( \pm \text{SEM} = -0.40 \pm 0.11, P < 0.001 \); slope \( \pm \text{SEM} = 0.30 \pm 0.07, P < 0.001 \); \( r = 0.53, P < 0.001 \)). In this case, an intercept of 0 and a slope of 0 would indicate absence of bias. Because some bias was apparent in the group as a whole (i.e., significant slope), we conducted a stratified analysis by gender and ethnicity. We found that the bias occurred largely in white boys (intercept \( \pm \text{SEM} = -1.24 \pm 0.18, P < 0.001 \); slope \( \pm \text{SEM} = 0.73 \pm 0.09, P < 0.001 \)) and, to a lesser extent, in African-American girls (intercept \( \pm \text{SEM} = -0.48 \pm 0.20, P < 0.05 \); slope \( \pm \text{SEM} = 0.49 \pm 0.16, P < 0.01 \). Bias was not detected in either white girls (intercept \( \pm \text{SEM} = -0.26 \pm 0.17, P > 0.05 \); slope \( \pm \text{SEM} = 0.13 \pm 0.10, P > 0.05 \)) or African-American boys (intercept \( \pm \text{SEM} = -0.04 \pm 0.24, P > 0.05 \); slope \( \pm \text{SEM} = 0.19 \pm 0.18, P > 0.05 \)).

**CONCLUSIONS** — The current study is the first in children to examine the possibility of predicting insulin sensi-

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Table 4 — Regression of insulin sensitivity on demographic, anthropometric, and fasting blood measures

<table>
<thead>
<tr>
<th>Step variable</th>
<th>Regression equation for insulin sensitivity</th>
<th>Model ( R^2 ) entered</th>
<th>( P ) value variable entered</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Fasting insulin</td>
<td>(-0.077 * \text{Fasting Insulin} + 2.467)</td>
<td>0.64</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>2. Ethnicity</td>
<td>(-0.078 * \text{Fasting Insulin} + 0.357 * \text{Ethnicity} + 2.280)</td>
<td>0.69</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>3. Calf skinfold (SF)</td>
<td>(-0.058 * \text{Fasting Insulin} + 0.459 * \text{Ethnicity} - 0.026 * \text{Calf SF} + 2.370)</td>
<td>0.73</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>
tivity (as based on an FSIGTT and the minimal model) by using simple measures of demography and anthropometry, in the presence or absence of fasting insulin. The main findings of this paper are: 1) the modified HOMA method accurately predicted insulin sensitivity in this sample of children but did not account for more of the variance in observed insulin sensitivity than fasting insulin alone; 2) insulin sensitivity in children could not be accurately predicted by demographic and anthropometric measures alone; 3) insulin sensitivity could be accurately predicted in children by an equation with demographic, anthropometric, and fasting insulin measures, and this equation was better than HOMA or fasting insulin alone; and 4) although the combination of demographic, anthropometric, and fasting insulin measures accurately predicted insulin sensitivity in the group as a whole, the equation lacked individual precision in white boys and, to a lesser extent, in African-American girls.

In the first part of the study, we attempted to cross-validate an equation containing the HOMA index in a cohort of children. We found that, in children, insulin sensitivity could be validly predicted using this method. However, similar to findings from previous studies in adults, the modified HOMA equation may not be very precise and stable in children, in whom predicted values of insulin sensitivity from the equation containing HOMA accounted for only ~63% of the variance in observed insulin sensitivity. Therefore, the equation containing HOMA was not significantly better than an equation containing fasting insulin alone (Step 1, Table 4). These findings suggest that in children, a prediction equation for insulin sensitivity warrants the inclusion of variables other than fasting insulin and glucose. Therefore, in the second part of the study, we attempted to develop new equations that included demographic, anthropometric, and fasting blood measures.

The current study was not able to cross-validate a prediction equation of insulin sensitivity, using basic demographic and anthropometric measures. We decided to use only conventional measures of adiposity because our goal was to develop a prediction equation of insulin sensitivity that was easily accessible. Use of BMI as a potential predictor variable, rather than simple weight and height

![Graph](https://via.placeholder.com/150)

**Figure 4**—Measured versus predicted \( \log_e \) insulin sensitivity in the validation group (n = 52). Unit of insulin sensitivity = \( 10^{-4} \) min\(^{-1}/(\mu U/ml) \). Conversion between \( \log_e \) insulin sensitivity and insulin sensitivity on its natural scale is shown in Table 1. Insulin sensitivity measured by FSIGTT in the cross-validation group and regressed on insulin sensitivity predicted from the equation developed in the development group (Step 3, Table 4). Regression trend (dashed line) not significantly different from line of identity (solid line) (\( P > 0.05 \)).

<table>
<thead>
<tr>
<th>Subgroup</th>
<th>Measured insulin sensitivity</th>
<th>Predicted insulin sensitivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>African-American girls</td>
<td>3.5 ± 1.7</td>
<td>3.1 ± 1.4</td>
</tr>
<tr>
<td>White girls (n = 17)</td>
<td>5.7 ± 2.6</td>
<td>5.9 ± 2.3</td>
</tr>
<tr>
<td>African-American boys</td>
<td>3.8 ± 3.3</td>
<td>3.0 ± 2.4</td>
</tr>
<tr>
<td>White boys (n = 14)</td>
<td>7.7 ± 5.5</td>
<td>5.9 ± 1.7</td>
</tr>
</tbody>
</table>

Data are means ± SD.

Table 5—Measured insulin sensitivity versus insulin sensitivity predicted by demographic, anthropometric, and fasting insulin measures by gender and ethnicity
measurements, did not yield different results. However, it is possible that more precise and complex measures of adiposity, such as dual energy X-ray absorptiometry (DEXA), would better correlate with insulin sensitivity. Nevertheless, in a separate stepwise regression analysis in a subset of children, we found that the inclusion of total body fat and lean tissue mass measured by DEXA did not improve the prediction equation significantly ($R^2 = 0.01$). Therefore, it seems that insulin sensitivity cannot be sufficiently predicted by demographic and anthropometric measures alone.

An equation with demographic, anthropometric, and fasting insulin measures validly predicted insulin sensitivity in our sample. Comparing the equation with demographic and anthropometric measures alone and the one with demographic, anthropometric, as well as fasting insulin, the latter accounted for 13% more of the variance in insulin sensitivity. Fasting insulin was shown to be a major predictor of insulin sensitivity, accounting for ~64% of insulin sensitivity alone. Similar findings were shown in adults (2,4). This may explain why an equation without fasting insulin may not be adequate for the estimation of insulin sensitivity. However, because fasting insulin is not normally measured in most clinical settings, it may not always be readily available. Nevertheless, compared with direct measures of insulin sensitivity, fasting insulin is still a much less complex measure to obtain.

It is also noteworthy that because fasting insulin alone accounted for most of the variance in observed insulin sensitivity, it did yield a univariate equation (Step 1, Table 4) that was successfully cross-validated in the validation group (regression results not shown). However, fasting insulin alone was not as accurate as the combination of fasting insulin with demographic and anthropometric measures (Step 3, Table 4). In addition, to rely on fasting insulin alone, the correlation between (measured-predicted) and measured insulin sensitivity was 0.75, suggesting that there was substantially more bias than the new equation developed in this study (i.e., recall that a correlation of 0 represents no bias in this instance; in other words, at any given level of measured insulin sensitivity, the difference between measured and predicted insulin sensitivity is 0). The additional measures provided more accuracy and slightly more precision in the prediction by accounting for an additional 9% of the variance in observed insulin sensitivity. These measures are simple and inexpensive, therefore we believe they should be included.

We also note that fasting glucose was not a significant predictor of insulin sensitivity. This is not too surprising because fasting glucose does not vary greatly in healthy children. This may partly explain why HOMA is only as useful as fasting insulin alone in children, because it estimates insulin resistance by relying solely on the combination of fasting insulin and fasting glucose.

Although our equation with demographic, anthropometric, and fasting insulin measures was successfully cross-validated in the current sample, there seemed to be some bias in the prediction. In a separate stratified analysis by gender

Figure 5—Measured minus predicted versus measured $\log_2$ insulin sensitivity in the cross-validation group ($n = 52$). Unit of insulin sensitivity = $10^{-4}$ min$^{-1}$ (µU/ml). Conversion between $\log_2$ insulin sensitivity and insulin sensitivity on its natural scale is shown in Table 1. $Y =$ deviation between insulin sensitivity measured by FSIGTT in the cross-validation group and insulin sensitivity predicted by equation developed in the development group (Step 3, Table 4). Dashed line represents regression trend ($r = 0.53$, $P < 0.001$). Bias in prediction equation is present because slope of regression is significantly different from 0 ($P < 0.001$).
and ethnicity, where the deviation of measured and predicted insulin sensitivity was regressed against measured insulin sensitivity, we found that the bias occurred largely in white boys and, to a lesser extent, in African-American girls. Bias was not detected in either white girls or African-American boys. It is not clear why this may be the case, particularly given the fact that the current sample was limited by its size for us to draw any conclusions per any gender by racial subgroup. However, compared with white girls and African-American boys combined, white boys in our sample had significantly higher insulin sensitivity and African-American girls had significantly lower insulin sensitivity (mean 5.4, 7.8, 3.5 × 10^{-4} \text{ min}^{-1} / (\mu \text{IU/ml}) respectively, P < 0.001). Therefore, it may be that at higher and lower ranges of insulin sensitivity, the current prediction equation loses some degree of precision. Future studies are needed, however, to examine whether this remains a problem in larger samples.

There are some limitations of the study that should be considered. First, because only African-American and white children were included in the current study, generalization to other ethnic groups is not possible. Second, our sample size did not allow us to more carefully examine whether it would be useful to develop a separate equation for each gender by ethnicity subgroup or for different ranges of insulin sensitivity. This warrants further investigation in the future, when larger samples of children are available. Third, the fact that Tanner Stage was not selected as a significant predictor may be due to the fact that only 12% of our sample was far enough into puberty. Therefore, in pubertal and early pubertal children, fat composition and ethnic identity (other than fasting insulin) may be most important in predicting insulin sensitivity. Given that we have previously shown, in a longitudinal study, that even in the most obese children insulin sensitivity falls by 33% from Tanner I to Tanner III (14), we recognize that our equations may not be generalizable to all stages of maturation, and additional analysis will be required to incorporate Tanner stage into any future equations.

Finally, readings of insulin concentrations may be different, depending on the type of assay used. In the current study, polyclonal antibodies were used in the insulin assay. However, as long as the discrepancy from different antibodies is consistent across individuals, we believe that our equation would still be valid using other insulin assays. Previously, we had compared the proinsulin levels between African-American and Caucasian children and found that there was no difference (Gower BA, Goran MI, unpublished data). Our equation could make a difference if African-Americans had more proinsulin than Caucasians (and therefore had higher nonspecific insulin levels but not higher specific insulin levels). However, this was not the case; in fact, the data were identical regardless of the use of a specific insulin assay or a nonspecific one. Future studies in different laboratories should determine whether our equations are equally valid using other forms of assay.

In conclusion, insulin sensitivity in children cannot be validly predicted by the combination of merely demographic and anthropometric measures. An equation containing HOMA can validly predict insulin sensitivity, but its precision is not better than an equation containing fasting insulin alone. A combination of demographic (i.e., ethnicity, gender), anthropometric (i.e., calf skinfold, suprailiac skinfold, weight), and fasting insulin measures predicted insulin sensitivity in white and African-American children better than the equation with HOMA or fasting insulin alone. Our validated equation of insulin sensitivity may be useful in population-based studies when complex techniques of measuring insulin sensitivity are not available. Because some bias exists in the current equation, its use in diagnosing insulin sensitivity on an individual basis is not encouraged. Future studies need to further examine whether a universal equation is feasible. Nevertheless, the current study is the first of such in children and suggests that a simple and easily accessible prediction equation of insulin sensitivity in children is possible.

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References

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