Body weight, weight gain and hyperglycaemia are associated with hypertensive disorders of pregnancy in women with gestational diabetes

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Abstract

Aim. – The aim of this study was to measure the capacity of glucose- and weight-related parameters to predict pregnancy-induced hypertensive disorders in women with gestational diabetes.

Methods. – An observational study was conducted involving 2037 women with gestational diabetes. The associations of glycaemic and weight-related parameters with pregnancy-induced hypertensive disorders were obtained by univariate and adjusted multivariate analyses. Also, model predictability and attributable predictor risk percentages were calculated, and collinearity and factor interactions examined.

Results. – Multivariate analyses revealed that hypertensive disorders were mainly predicted by average third-trimester glycated haemoglobin (HbA1c) levels ≥ 5.9%, by being overweight or obese before pregnancy and by excess gestational weight gain after adjusting for age, tobacco use, chronic hypertension, parity, urinary tract infections and gestational age at delivery. Prepregnancy body weight (overweight and obesity) had the strongest impact on pregnancy-related hypertensive disorders (attributable risk percentages were 51.5% and 88.8%, respectively). The effect of being overweight or obese on hypertensive disorders was enhanced by HbA1c levels and gestational weight gain, with elevated HbA1c levels multiplying the effect of being overweight before pregnancy.

Conclusion. – The average third-trimester HbA1c level is a novel risk factor for pregnancy-induced hypertensive disorders in women with gestational diabetes. HbA1c levels ≥ 5.9%, prepregnancy overweight or obesity and excess gestational weight gain are all independent risk factors of pregnancy-related hypertensive disorders in such women. In treated gestational diabetes patients, the strongest influence on hypertensive disorders is prepregnancy obesity.

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1. Introduction

Maternal diabetes and obesity are major causes of pregnancy-induced hypertensive disorders (PIHD) such as gestational hypertension and/or preeclampsia [1,2]. Now, gestational weight gain is gaining attention as a predisposing condition [3–7] possibly because of the renewed interest in determining optimal weight gain during pregnancy [8]. However, in women with gestational diabetes (GDM), the combined effect of glucose- and weight-related factors on PIHD development is still unknown.

Rather than any degree of glucose intolerance, PIHD may only affect women with severe and sustained hyperglycaemia [9] in the setting of GDM. Consistently, fasting glucose levels ≥ 5.8 mmol/L have been associated with gestational hypertension or preeclampsia [10–12]. It is possible that, because obese women have fasting glucose values above those of normal-weight women, maternal obesity may mitigate the effect of fasting glucose levels on hypertensive disorders [10,12].

Other than the link between PIHD and factors related to metabolic status during pregnancy at baseline (before treatment), such as hyperglycaemia severity at GDM diagnosis [12–14] and prepregnancy obesity [12,14,15], little is known about the effects of glycaemia and weight control on PIHD in GDM pregnancies up to delivery. Higher average glycated haemoglobin A1c (HbA1c) values have been described in women with GDM and...
PIHD [16] and, clearly, intensive GDM treatment reduces the risk of gestational hypertension [17] and preeclampsia [6]. However, appropriate glycaemic control may be insufficient to avoid PIHD in obese pregnant women with GDM [12].

The effect of gestational weight gain on PIHD has been explored in both epidemiological [5,7] and pregestational diabetes [4] studies. In cases of GDM, one hypothesis is that the boundaries of glycaemic control are mediated, at least in part, by lower gestational weight gain in women who undergo dietary interventions [6]. In the present study, a cohort of Spanish women with GDM were assessed for the combined effects of GDM severity, third-trimester glycaemic control, prepregnancy body mass index (BMI) and gestational weight gain on pregnancy-induced hypertensive disorders.

2. Methods

2.1. Setting and patient population

The present observational study reviewed a prospective database that included women with singleton pregnancies, diagnosed with GDM between 1987 and 2008, who were followed at a diabetes and pregnancy unit in a third-level hospital in Spain (n = 2568). National Diabetes Data Group (NDDG) criteria—specifically, a 100-g oral glucose tolerance test (OGTT) with fasting glycaemia values of 5.8 mmol/L that at 1 h were 10.6 mmol/L, at 2 h were 9.2 mmol/L and at 3 h were 8.1 mmol/L [18]—were applied to diagnose GDM after universal screening. Of these women, eight with suspected undiagnosed preexisting diabetes, one with glomerulopathy, 13 with liver disease, five with positive antiphospholipid autoantibodies and three with beta-thalassaemia were excluded from the analyses. Women taking particular medications (such as corticosteroids) were referred to a specialized clinic and were also not included in our GDM cohort. Thus, complete data were available in 2037 (80.0%) of the followed women.

The present study was approved by the Hospital Ethics Committee, and adhered to the principles for medical research involving human subjects as established by the World Medical Association (Declaration of Helsinki).

2.2. Outcome and explanatory variables

In this study, a number of glycaemic- and weight-related parameters were evaluated. GDM severity was assessed by a 100-g 3-h OGTT at diagnosis, with fasting, 1-h, 2-h and 3-h glucose levels being measured in venous plasma. The OGTT fasting glucose and area under the glucose curve were used to define baseline glucose intolerance. In addition, the mean monthly HbA1c level was used as a glycaemic control parameter from the time of GDM diagnosis to delivery. HbA1c levels were measured by high-performance liquid chromatography, using Diamat and Variant automated analyzers (Bio-Rad Laboratories, Richmond, CA, USA), with exchangeable results that were calibrated according to the Diabetes Control and Complications Trial (DCCT) normality range. BMI before pregnancy was calculated on the basis of self-reported prepregnancy weight and measured height. Gestational weight gain was quantified as the difference between weight at the last prenatal visit and the prepregnancy self-reported weight. Gestational weight gain was defined, as suggested by Institute of Medicine (IOM) guidelines [8], for each prepregnancy BMI category. In addition, serum fasting insulin levels, measured by two-sided immunoenzymometric assay (ST AIA-PACK IRI, Tosoh Bioscience, South San Francisco, CA, USA), were obtained in 899 women during the first prenatal visit before starting GDM treatment to assess baseline insulin resistance.

Fasting glucose categories were defined as ≤ or > 5.8 mmol/L (NDDG criteria), and the 75th percentile was used as the 100-g OGTT area under the curve (AUC) threshold value. As an HbA1c threshold value has not been defined in GDM, associations between PIHD and HbA1c increments of 0.1% of the mean value were used. Also evaluated were the univariate associations between PIHD and each HbA1c value. Overweight (25–29.9 kg/m²) and obese (≥ 30 kg/m²) prepregnancy BMI categories were defined, and any weight gain over the upper limit of the recommended range (> 16 kg for normal weight, > 11.5 kg for overweight, > 9 kg for obesity) was considered excess gestational weight gain.

Chronic hypertension was defined as either confirmed hypertension or a prescription for antihypertensive treatment before 20 weeks of gestation. Pregnancy-related hypertensive disorders included gestational hypertension and/or preeclampsia. Gestational hypertension was defined as new-onset hypertension (≥ 140 and/or 90 mmHg, confirmed by at least two determinations) after 20 weeks of pregnancy. Preeclampsia was diagnosed if new proteinuria (≥ 300 mg/24 h) was identified [19].

2.3. Statistical analysis

Our study looked for associations between various parameters and PIHD by first obtaining pairwise correlation coefficients between variables that could predict PIHD. Second, the relative effects of glycaemic and weight-related variables on PIHD development were assessed using parameters that were routinely measured in clinical practice and were not strongly correlated (pairwise r Pearson coefficient < 0.7), but were significantly (P < 0.05) associated with the outcome, which were then fed into a stepwise multiple logistic regression model along with selected covariates that might plausibly be related to outcome. The effects of these factors were measured by adjusted odds ratios (AOR) with 95% confidence intervals (95% CI). The overall multivariate model predictability was estimated by the area under the receiver operating characteristic (AUROC) curve (with 95% CI).

Additional multicollinearity analyses were performed by sequentially subtracting the factors that remained in the regression model to identify the AUROC curve variations that would be expected from any independent influence of these factors. The attributable risk percentages (ARE%) for the risk factors obtained were calculated, using the formula ARE% \( (AOR - 1) / AOR \) to estimate the PIHD excess that could be predicted by one risk factor in women exposed to that factor. Also examined was the effect of interactions among risk...
3. Results

A total of 144 women (7.1%) were diagnosed with gestational hypertension (6.5%) and/or preeclampsia (0.6%). In addition to the data distribution displayed in Table 1, insulin treatment was used by 1052 women (51.7%). Of all the women studied, 1968 (96.6%) were Caucasian.

Regarding the characteristics of PIHD vs non-PIHD women, mean age was 34 ± 4 years vs 33 ± 5 years, respectively (P = 0.037). Hypertension identified during pregnancy persisted in 32 (71.1%) women with pregestational chronic hypertension compared with 5.7% of the non-PIHD women (P = 0.000). Superimposed preeclampsia developed in two (4.4%) of the 45 women with chronic hypertension. Any smoking status was less frequent in the PIHD vs non-PIHD women (4.7% vs 7.5%, respectively; P = 0.048), whereas parity (range; 0–1; P = 0.144) and urinary tract infection rates (8.5% vs 8.2%, respectively; P = 0.968) were similar in both PIHD and non-PIHD groups. Insulin treatment was more frequent in PIHD than in non-PIHD women (9.1% vs 4.8%, respectively; P = 0.000). Mean gestational age at delivery was 38 ± 1 years in PIHD women vs 39 ± 1 years in non-PIHD women (P = 0.000). As for baseline insulin resistance, higher median (interquartile range) fasting insulin levels were detected before GDM treatment in 64 women who developed PIHD [13.0 (10.0–18.0) μU/mL] vs [10.0 (7.0–13.0) μU/mL] in 835 non-PIHD women (P = 0.000).

In addition, a persistent association was observed between PIHD and average HbA1c levels in univariate and multiple regression analyses. Average third-trimester HbA1c values ≥ 5.9% were significantly associated with PIHD, whereas lower HbA1c values showed no statistically significant association with PIHD on categorical analyses (Fig. 1). Other univariate analyses demonstrated that all glycaemic and weight-related parameters were associated with PIHD except for the 100-g OGTT AUC (Table 2).

Multivariate analyses included the 100-g OGTT fasting glycaemia values, average third-trimester HbA1c values, prepregnancy BMI and excess gestational weight gain, and were adjusted for the following selected covariates: maternal age; tobacco use; parity; chronic arterial hypertension; urinary tract infections; and gestational age at delivery. The adjusted analysis demonstrated that average third-trimester HbA1c levels ≥ 5.9% had a significant effect on PIHD. Being overweight or obese were also strong predictors of pregnancy-related hypertensive disease, and excess weight gain had an independent effect (Table 3). Previous pairwise correlation analyses showed no strong correlations between predictive parameters (r Pearson coefficients range: 0.09–0.17). Introducing interactions between parameters did not improve multivariate model predictability, while inclusion of insulin use failed to modify effects of the selected glycaemic and weight-related parameters. Prepregnancy obesity and excess gestational weight gain were also associated with PIHD in the subgroup with chronic hypertension. As expected, tobacco use and early pregnancy termination were associated with a reduced PIHD incidence.

The PIHD predicted variability by the model with no selected covariates was 0.77 (0.72–0.82) according to the AUROC curve, whereas the AUROC of the model including covariates was 0.84 (0.79–0.88). The combination of average third-trimester HbA1c levels ≥ 5.9% and being overweight/obese before pregnancy increased PIHD risk for individual factors on a multiplicative scale compared with average third-trimester HbA1c levels ≥ 5.9% only (Figs. 2 and 3).

4. Discussion

Prepregnancy BMI, average third-trimester HbA1c levels and excess gestational weight gain are independent PIHD risk factors, according to the latest IOM recommendations [8]. In the present study, these main predictors of pregnancy-induced hypertensive disorders were examined in a cohort of Spanish women with GDM. In our followed women, glycaemic variables were more weekly associated with PIHD than being obese before pregnancy.

Our present study has a few limitations. First, the evidence obtained is limited by the retrospective nature of the study, although the data were prospectively collected. Second, prepregnancy BMI was calculated on the basis of self-reported weight. However, regarding the reliability of these data, a validation study of reproductive-aged women found that 84% were classified into appropriate BMI categories on the basis of self-reported weight and height [20]. On the other hand, one strength of our data was that major covariate information was available thanks to the close collaboration of the obstetricians and endocrinologists at our institution’s diabetes and pregnancy unit. All women were diagnosed with GDM according to NDDG criteria and received GDM treatment. As such, our cohort was representative of the Spanish population with GDM [14]; nevertheless, differences

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Table 1
Characteristics of a cohort of women with gestational diabetes.

<table>
<thead>
<tr>
<th>Maternal characteristics</th>
<th>Mean ± SD, median (IQR) or n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>n</strong></td>
<td>2037</td>
</tr>
<tr>
<td>Age (years)</td>
<td>33 ± 4</td>
</tr>
<tr>
<td>Chronic arterial hypertension</td>
<td>45 (2.3)</td>
</tr>
<tr>
<td>Smoking status (any)</td>
<td>430 (21.1)</td>
</tr>
<tr>
<td>Parity</td>
<td>1.0 (0.0–1.0)</td>
</tr>
<tr>
<td>Urinary tract infection</td>
<td>153 (8.2)</td>
</tr>
<tr>
<td>Gestational age at diagnosis (weeks)</td>
<td>27 ± 5</td>
</tr>
<tr>
<td>Gestational age at delivery (weeks)</td>
<td>39 ± 1</td>
</tr>
<tr>
<td>Fasting glycaemia by 100-g OGTT (mmol/L)</td>
<td>5.0 ± 0.8</td>
</tr>
<tr>
<td>Area under the 100-g OGTT curve (mmol/l/min)</td>
<td>28.5 ± 2.8</td>
</tr>
<tr>
<td>Average third-trimester HbA1c (%)</td>
<td>5.2 ± 0.4</td>
</tr>
<tr>
<td>Prepregnancy BMI (kg/m²)</td>
<td>24.7 ± 4.7</td>
</tr>
<tr>
<td>Excess gestational weight gain°</td>
<td>299 (14.7)</td>
</tr>
<tr>
<td>Pregnancy-induced hypertensive disorder</td>
<td>144 (7.1)</td>
</tr>
<tr>
<td>Gestational arterial hypertension</td>
<td>132 (6.5)</td>
</tr>
<tr>
<td>Preeclampsia</td>
<td>12 (0.6)</td>
</tr>
</tbody>
</table>

IQR: interquartile range; OGTT: oral glucose tolerance test for diagnosis of gestational diabetes; BMI: body mass index.

° Above the upper limit (kg) per each prepregnancy BMI category recommended by Institute of Medicine guidelines (2009).
Fig. 1. Univariate associations between average third-trimester HbA1c levels (%) and pregnancy-induced hypertensive disorders in 2037 women with gestational diabetes.

In screening practices, GDM diagnostic criteria, metabolic features and treatment modalities [21] limit our ability to arrive at general conclusions.

Our data nonetheless extend the previous research on GDM-associated hypertensive disorders by evaluating a number of glycaemic parameters. Average third-trimester HbA1c values proved to have greater PIHD predictive effects than either OGTT or AUC. However, HbA1c levels are not routinely measured in GDM women. One reason for this may be because HbA1c levels are a retrospective measurement of glycaemic control during the past four weeks prior to its measurement. Thus, they cannot entirely be a substitute for self-measurement of capillary glucose levels. On the other hand, HbA1c levels can be accurately measured and have good correlation with average glycaemia [1,17]. Another argument against HbA1c measurement may be the lack of any established normal HbA1c ranges during pregnancy. As background, however, there is an average 0.4% HbA1c decrease during pregnancy because of accelerated erythrocyte exchange and turnover [22]. In addition, the red blood spread phenomenon leads to changes in HbA1c that are faster than the 6- to 8-week interval needed outside of gestation [23].

HbA1c baseline values were not associated with pregnancy-induced hypertensive disorders in the Hyperglycaemia and Adverse Pregnancy Outcome (HAPO) Study [24]. However, in

Table 2
Univariate associations between glucose/weight-related factors and pregnancy-induced hypertensive disorders in 2037 women with gestational diabetes.

<table>
<thead>
<tr>
<th>Factor</th>
<th>Pregnancy-induced hypertensive disorders, n (%)</th>
<th>OR (95% CI)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>100-g OGTT fasting glycaemia ≥ 5.8 mmol/L.</td>
<td>34 (15.3)</td>
<td>110 (6.1)</td>
<td>2.78 (1.84–4.20)</td>
</tr>
<tr>
<td>100-g OGTT AUC ≥ 75th percentile</td>
<td>42 (8.8)</td>
<td>94 (6.2)</td>
<td>1.45 (0.99–2.12)</td>
</tr>
<tr>
<td>HbA1c ≥ 5.9%</td>
<td>16 (18.8)</td>
<td>119 (6.7)</td>
<td>3.21 (1.81–5.70)</td>
</tr>
<tr>
<td>Prepregnancy overweightb</td>
<td>103 (14.0)</td>
<td>40 (3.1)</td>
<td>3.11 (1.98–4.89)</td>
</tr>
<tr>
<td>Prepregnancy obesityc</td>
<td>63 (27.4)</td>
<td>80 (4.5)</td>
<td>12.08 (7.79–18.74)</td>
</tr>
<tr>
<td>Excess gestational weight gaind</td>
<td>41 (13.7)</td>
<td>103 (5.9)</td>
<td>2.52 (1.72–3.51)</td>
</tr>
</tbody>
</table>

Statistical significance if P < 0.05; OGTT: oral glucose tolerance test at diagnosis of gestational diabetes; AUC: area under the curve.

a Average third-trimester HbA1c.
b Prepregnancy body mass index (BMI) 25–29.9 kg/m².
c Prepregnancy BMI ≥ 30 kg/m².
d Above the upper interval (kg) per prepregnancy BMI categories recommended by Institute of Medicine guidelines (2009).
that study, HbA1c was only measured in those with a diagnosis of relatively mild gestational glucose intolerance. An earlier study [16] described higher HbA1c values during the third trimester in women with GDM and hypertensive disorders. Despite the fact that HbA1c changes are faster during rather than outside of pregnancy, there is still a delay in PIHD risk evaluation using HbA1c measurements compared with OGTT. However, this HbA1c relationship favours intensive glycaemic control to hypothetically reduce PIHD occurrence in women at risk. Our adjusted analysis found that average third-trimester HbA1c levels ≥ 5.9% had a significant influence on PIHD, but HbA1c levels remained below risk values in the majority (95.8%) of our cohort.

A result in line with previous research conclusions was that pregnancy-related hypertensive disease was more strongly related to prepregnancy BMI than to glucose-related variables. In previous studies, prepregnancy BMI was linked to PIHD in both non-GDM [1,3,5,14,25,26] and GDM [10,12,14,27,28] pregnancies whereas, in our cohort, it accounted for 51.5% and 88.8% of hypertensive disorders in those who were

Fig. 2. Prevalence (%) of pregnancy-induced hypertensive disorders (PIHD) in 2037 women with gestational diabetes grouped according to prepregnancy body mass index scores. The rate of PIHD in women with average third-trimester HbA1c levels ≥ 5.9% and excess gestational weight gain are shown within categories of normal weight, overweight and obesity before pregnancy.

Fig. 3. Prevalence (%) of pregnancy-induced hypertensive disorders (PIHD) in 2037 women with gestational diabetes (from left to right): with average third-trimester HbA1c ≥ 5.9% only; who were overweight or obese prepregnancy only; with excess gestational weight gain only; and with these three risk factors combined.

Table 3
Stepwise multiple logistic regression: glucose and/or weight-related risk factors for pregnancy-induced hypertensive disorders in 2037 women with gestational diabetes.

<table>
<thead>
<tr>
<th>Factor</th>
<th>Factor present</th>
<th>Factor absent</th>
<th>P value</th>
<th>ARE% (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>HbA1c ≥ 5.9%</td>
<td>2.52 (1.12–5.69)</td>
<td>1.00 (reference)</td>
<td>0.026</td>
<td>60.3 (10.7–82.4)</td>
</tr>
<tr>
<td>Prepregnancy overweight</td>
<td>2.06 (1.11–3.83)</td>
<td>1.00 (reference)</td>
<td>0.022</td>
<td>51.5 (9.9–73.9)</td>
</tr>
<tr>
<td>Prepregnancy obesity</td>
<td>8.94 (4.98–16.04)</td>
<td>1.00 (reference)</td>
<td>0.000</td>
<td>88.8 (79.9–93.8)</td>
</tr>
<tr>
<td>Excess gestational weight gain</td>
<td>1.91 (1.08–3.37)</td>
<td>1.00 (reference)</td>
<td>0.025</td>
<td>47.6 (7.4–70.3)</td>
</tr>
</tbody>
</table>

Statistical significance if P < 0.05.
a OR (odds ratio) adjusted for maternal age, tobacco use, parity, chronic arterial hypertension, urinary tract infection and gestational age at delivery.
b Attributable risk percentage in women exposed to factor.
c Averaged third-trimester HbA1c.
d Prepregnancy body mass index (BMI) 25–29.9 kg/m².
e Prepregnancy BMI ≥ 30 kg/m².
f Above the upper interval (kg) per prepregnancy BMI categories recommended by Institute of Medicine guidelines (2009).
overweight and obese, respectively, with 60.3% attributable to high HbA1c levels. This considerable effect of prepregnancy BMI was observed regardless of GDM severity. In fact, similar conclusions were also noted in two previous approaches [11,12]. Based on our data, the effect of being overweight or obese before pregnancy was also multiplied in the GDM group with average third-trimester HbA1c levels ≥ 5.9%.

Gestational weight gain is still a controversial issue [3,5–7,14,25,27], although growing evidence suggests that gestational weight gain can affect desirable GDM pregnancy outcome [6,17]. Adequate weight gain may be one positive effect of treating GDM. Indeed, parallel reductions in gestational weight gain and preeclampsia were observed by Landon et al. [6] in obese GDM women who received intensive GDM treatment. Gestational weight gain was reduced by around 2.2 kg in a meta-analysis of antenatal dietary and lifestyle interventions in obese pregnant women [29]. However, reversal of GDM-associated complications by reducing gestational weight gain was not demonstrated. In our present cohort, the association between excess gestational weight gain and PIHD was evident throughout all prepregnancy BMI categories. Thus, it may be hypothesized that achieving desirable weight gain should be a goal of GDM treatment. Medical counselling also strongly correlates with actual gestational weight gain [30]. This suggests that safe and effective weight-management strategies for pregnant women need to be developed.

Our present results establish a new association between average HbA1c levels and excess weight gain and pregnancy-induced hypertensive disorders in women with GDM. Insulin resistance appears to be a key pathogenetic factor for GDM-associated PIHD [3]. It may therefore be speculated that prepregnancy BMI acts as a surrogate of insulin resistance, which can lead to early placental dysfunction. In contrast, disturbed glucose metabolism and marked weight gain appear to be delayed, presenting at a more advanced gestational stage. This would explain the prolonged and appreciable effect of prepregnancy BMI on PIHD. During the third trimester, synergy between factors may be due to enhanced endothelial dysfunction, which itself may be a major pathogenetic factor of PIHD. Hyperglycaemia, circulating triglycerides, free fatty acids and adipose tissue cytokines such as tumour necrosis factor (TNF)-α might also be mediators of this placental and systemic endothelial hypertensive vasculopathy [1].

In conclusion, average third-trimester HbA1c levels are more effective indicators of pregnancy-related hypertensive disease in women with GDM than OGTT values. Those HbA1c levels along with prepregnancy BMI and excess gestational weight gain are all independent risk factors of pregnancy-induced hypertensive disorders. However, the usual HbA1c values in women treated for GDM are unlikely to have relevant effects on the hypertensive condition. In fact, the strongest influence is exerted by prepregnancy BMI, with an added effect of excess gestational weight gain, although one positive note is that careful attention to the latter may reduce PIHD in GDM pregnancies. Clearly, this possibility suggests that GDM should be treated for reasons beyond glycaemia.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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Appendix A. Supplementary data

Supplementary data (French abstract) associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.diabet.2013.12.011.

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