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Comparison of Two Mathematical Models for the Screening of Preeclampsia Using Free Cloud Computing Resources

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Abstract
Aim: To develop and validate a free computational tool for preeclampsia (PE) risk assessment and compare the performance of two widely used mathematical screening models: the Fetal Medicine Foundation (FMF) competing risks model and the Fetal Medicine Barcelona (FMB) multivariate Gaussian distribution model. *Methods*: A Python-based computational engine was developed using Google Colab, which integrated maternal characteristics, mean arterial pressure (MAP), uterine artery pulsatility index (UtA-PI), and placental growth factor (PlGF). Simulated cohorts of 100,000 pregnancies (97,000 non-PE and 3,000 with PE) were assessed. Concentrations of PlGF were simulated across three analytical platforms (Roche, Thermo Fisher, and Perkin Elmer) at 11–13 weeks of gestation (WG) and with a Roche platform at 10 WG. The model performance was evaluated using Receiver Operating Characteristic (ROC) curve analysis, detection rates (DRs), and screen-positive rates (SPRs). *Results:* The computational tool showed excellent agreement with validated online calculators (proportional differences of 2.1% for FMF and 5.7% for FMB). The FMF model consistently outperformed the FMB model across all platforms (AUC, 0.885–0.888 vs. 0.845–0.846). Platform-specific PlGF differences significantly affected the risk thresholds but not the overall diagnostic accuracy. Both models maintained comparable performance at 10 WG. *Conclusion*: The FMF model outperformed the FMB model owing to the broader integration of maternal risk factors and platform-specific medians. This free, open-access tool supports informed PE screening decisions and is particularly relevant given the widespread commercial use of both models.

Keywords: Competing Risks; Gaussian Distribution; Preeclampsia; Python; Screening

Introduction

Preeclampsia (PE) is a multisystem syndrome of pregnancy characterized by systolic blood pressure (SBP) ≥ 140 mmHg and/or diastolic blood pressure (DBP) ≥ 90 mmHg, measured at least twice within a 4-hour interval, in women who were previously normotensive. This condition must be accompanied by at least one of the following criteria emerging after 20 weeks of gestation (WG): proteinuria, evidence of maternal organ dysfunction, and/or uteroplacental dysfunction [1,2]. PE is one of the most severe complications of pregnancy and a leading cause of maternal and perinatal morbidity and mortality worldwide, accounting for approximately 14% of maternal deaths and contributing to approximately 500,000 fetal and neonatal deaths annually [2–5].

Preeclampsia is classified into two primary subtypes: Type I PE occurs earlier in pregnancy and is characterized by placental dysfunction, superficial trophoblastic invasion and elevated peripheral vascular resistance. Type II PE manifests later and is associated with poor maternal cardiovascular adaptation [6,7]. The global prevalence of PE is estimated to range between 2% and 5%. In Spain, reported data indicate a prevalence of 0.7% to 1.1% for preterm PE and up to 1.6% for term PE [8,9]. Preventive treatment with low-dose aspirin reduces the incidence of preterm PE by more than 60% [10,11], making effective first-trimester screening essential.

The FMF competing risk model, integrating mean arterial pressure (MAP), uterine artery pulsatility index (UtA-PI), and placental growth factor (PlGF) measured between 11+0 and 13+6 WG with maternal characteristics, detects 81% of preterm PE cases at a 10% screen-positive rate (SPR), compared to 41% with maternal risk factors alone [5]. More recently, the Fetal Medicine Barcelona (FMB) proposed an alternative multivariate Gaussian distribution model using the same markers, with PlGF measurable from 8+0 to 13+6 WG, reporting a detection of 94% of early onset PE at a 10% SPR [12]. Both screening strategies combined with prophylactic aspirin have been shown to be cost-effective [9,13–15].

This study aimed to develop and validate a free, open-access computational tool for PE risk assessment and compare the performance of two widely used mathematical screening models: the FMF competing risks model and the FMB multivariate Gaussian distribution model, using Google Colab and comparing their diagnostic performance across different analytical platforms and gestational ages (GAs).

Materials and Methods

Development of the Computational Tool

A computational engine was developed in Python (version 3.10.12) using the open-source Google Colab environment [16], incorporating NumPy, Pandas, Openpyxl, SciPy, and Datetime libraries. Input data were provided in an Excel file (1_Data_Screening_PE.xlsx), and risk estimates were exported to Google Drive as XLSX output. The full mathematical details of both algorithms are provided in Supplementary Material S1, and the Excel file and tool are accessible via the links in Supplementary Material S2.

Validation of the Computational Tool

The tool was validated by comparing its risk estimates with those of freely available online calculators implementing the same models [17,18]. In total, 195 simulated patients were assessed using the FMF model and 133 using the FMB model, covering all relevant variable combinations (see Supplementary Material S3).

Simulation of Pregnancies

Using Python's random module, 100,000 virtual patients were computationally generated based on published parameters from large population-based PE screening studies [8,19–26], with no real patient data used. The simulated cohort comprised 97,000 patients without PE and 3,000 with PE (prevalence 3%), within a GA range of 11+0 to 13+6 WG. Based on these studies, the following risk factor frequencies were defined a priori to differ between PE and non-PE groups: Afro-Caribbean ethnicity, IVF conception, and family history of PE were set at twice the frequency in the PE group; type I/II diabetes at three times; SLE, APS, and previous PE at five times; and chronic hypertension (CH) at twelve times. Conversely, smoking was modelled as 20% less prevalent in the PE group. All continuous variables followed a Gaussian distribution. Additionally, a separate cohort of 30,000 patients was simulated using PlGF at 10 WG (Roche platform).

Simulation of Screening Markers

Patients with PE had, on average, 8% higher MAP, 14% higher UtA-PI, and 33% lower serum PlGF. Concentrations of PlGF were simulated for three platforms (Roche Diagnostics, Thermo Fisher, and Perkin Elmer), with Thermo Fisher averaging 23% lower and Perkin Elmer 38% lower than Roche [27–29]. Afro-Caribbean patients have 50% higher PlGF concentrations [30]. Population medians were derived from 2,500 non-PE simulated patients using polynomial regression (order 2 after logarithmic transformation).

Statistical Analysis

Statistical analyses were performed using MedCalc (v20.218). Passing-Bablok regression and Bland-Altman analyses were used to assess the agreement between the computational engine and web calculators. Medians and proportions were compared using the Mann-Whitney U and chi-squared tests, respectively. Receiver operating characteristic (ROC) curve analysis and area under the curve (AUC) were used to evaluate and compare the model performance.

Results

Validation of the Computational Tool

Both models showed excellent agreement with web-based calculators, with proportional differences of 2.1% higher for FMF and 5.7% lower for FMB when using Google Colab (Figure 1). Using predefined risk thresholds (1:150 for FMF and 1:226 for FMB), each model misclassified only one patient. A programming error was identified in the FMF web calculator: the a priori risk was not increased when APS or SLE was present individually, but only when both conditions coexisted.

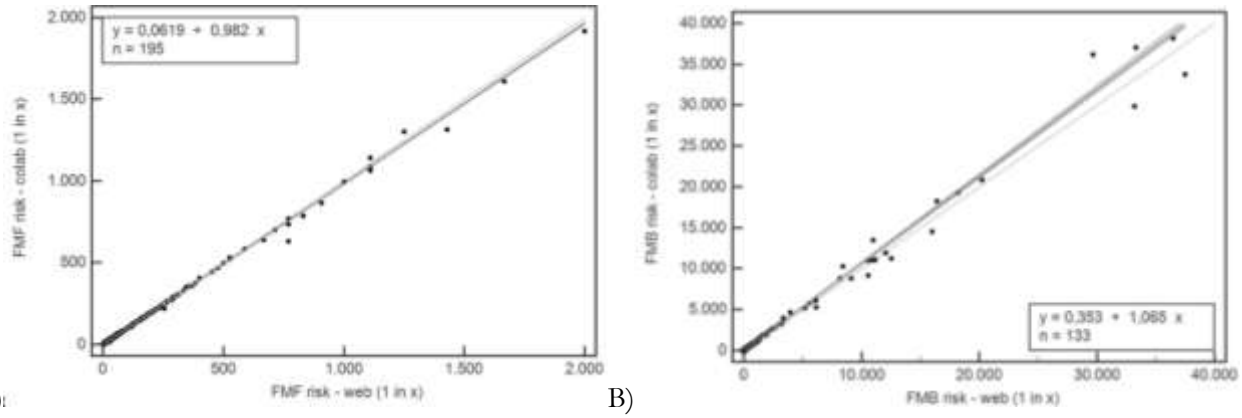

Figure 1. (A) Passing–Bablok regression analysis comparing the risk of PE obtained using the FMF, and **(B)** FMB models, with values calculated in Google Colab versus those from the web-based calculators.

Study Population

The maternal and pregnancy characteristics are presented in Table 1. All variables showed significant differences between the PE and non-PE groups, except for GA at examination (p = ns).

Distribution of PlGF Results across Platforms

The simulated PlGF concentrations were significantly different between the platforms (Figure 2). The results from Thermo Fisher were, on average, 26.4% lower than those from Roche, and the results from Perkin Elmer were 47.4% lower than those from Roche and 21.7% lower than those from Thermo Fisher.

Screening Performance at 11–13 Weeks of Gestation

The FMF model consistently outperformed the FMB model across all three platforms. Using Roche Diagnostics as the reference platform, AUC values were 0.888 (95% CI: 0.886–0.890) for FMF versus 0.845 (95% CI: 0.843–0.847) for FMB (Table 2; $p<0.0001$). Similar results were obtained with Thermo Fisher (AUC: 0.887 vs. 0.846) and Perkin Elmer (AUC: 0.885 vs. 0.846) platforms (Supplementary Material S4, Tables S-I and S-II). When using platform-specific medians derived from the simulated population, AUC values improved slightly for both models (FMF: 0.891–0.894; FMB: 0.846–0.848), with consistent cutoff values across platforms (Supplementary Material S4, Table S-III).

Table 1. Maternal and pregnancy characteristics in the screening population.

Characteristic	Non-PE (n=97,000)	PE (n=3,000)	p value
Maternal age (years)	32.5 (28.5–36.6)	33.7 (29.6–37.8)	<0.0001
> 35 years	32,567 (33.6)	1,232 (41.1)	
> 40 years	10,668 (11.0)	421 (14.0)	
Maternal weight (kg)*	66 (59–73)	72 (65–79)	<0.0001
Maternal height (cm)	165 (158–171)	164 (157–171)	<0.0001
BMI (kg/m²)	24.2 (21.2–27.6)	26.9 (23.5–30.4)	<0.0001
GA at examination (weeks)	12.6 (11.9–13.3)	12.6 (11.9–13.3)	ns
Racial origin			**<0.0001**
Caucasian	89,046 (91.8)	2,584 (86.1)	
Afro-Caribbean	4,903 (5.1)	319 (10.6)	
South Asian	987 (1.0)	32 (1.1)	
East Asian	1,023 (1.0)	36 (1.2)	
Mixed	1,041 (1.1)	29 (1.0)	
Mode of conception			**<0.0001**
Spontaneous	89,173 (91.9)	2,529 (84.3)	
Ovulation induction	1,004 (1.0)	34 (1.1)	
IVF	6,823 (7.1)	437 (14.6)	
Medical history			
Chronic hypertension	1,006 (1.0)	351 (11.7)	<0.0001
Diabetes mellitus, type I	458 (0.5)	44 (1.5)	<0.0001
Diabetes mellitus, type II	473 (0.5)	37 (1.2)	<0.0001
SLE	90 (0.1)	18 (0.6)	<0.0001
APS	50 (0.05)	8 (0.27)	<0.0001
Cigarette smoker	9,733 (10.0)	246 (8.2)	0.001
Family history of PE	2,957 (3.0)	153 (5.1)	<0.0001
Obstetric history			**<0.0001**
Nulliparous	48,652 (50.2)	1,498 (49.9)	
Parous with no previous PE	46,494 (47.9)	1,208 (40.3)	
Parous with previous PE	1,854 (1.9)	294 (9.8)	
Interpregnancy interval (years)	3.5 (2.4–4.6)	4.5 (3.0–5.9)	<0.0001
GA in previous delivery (weeks)	40 (38–41)	35 (34–37)	<0.0001
Screening markers			
MAP (mmHg)	82.4 (78.1–86.7)	89.8 (84.4–95.0)	<0.0001
UtA-PI	1.63 (1.39–1.88)	1.86 (1.58–2.14)	<0.0001
PlGF (pg/mL) – Roche (11–13 WG)	53.4 (41.0–69.5)	35.5 (27.4–46.4)	<0.0001
PlGF (pg/mL) – Thermo (11–13 WG)	41.0 (31.3–53.8)	27.3 (20.9–35.8)	<0.0001
PlGF (pg/mL) – PerkinElmer (11–13 WG)	32.9 (25.0–43.4)	22.0 (16.7–29.0)	<0.0001
PlGF (pg/mL) – Roche (10 WG)	33.4 (28.1–41.6)	22.1 (18.7–29.0)	<0.0001

Data are given as median (interquartile range) or n (%). APS: antiphospholipid syndrome; BMI: body mass index; GA: gestational age; IVF: in vitro fertilization; MAP: mean arterial pressure; ns: non-significant; PE: preeclampsia; PlGF: placental growth factor; SLE: systemic lupus erythematosus; UtA-PI: uterine artery pulsatility index; WG: weeks of gestation.*at the time of first-trimester PE screening

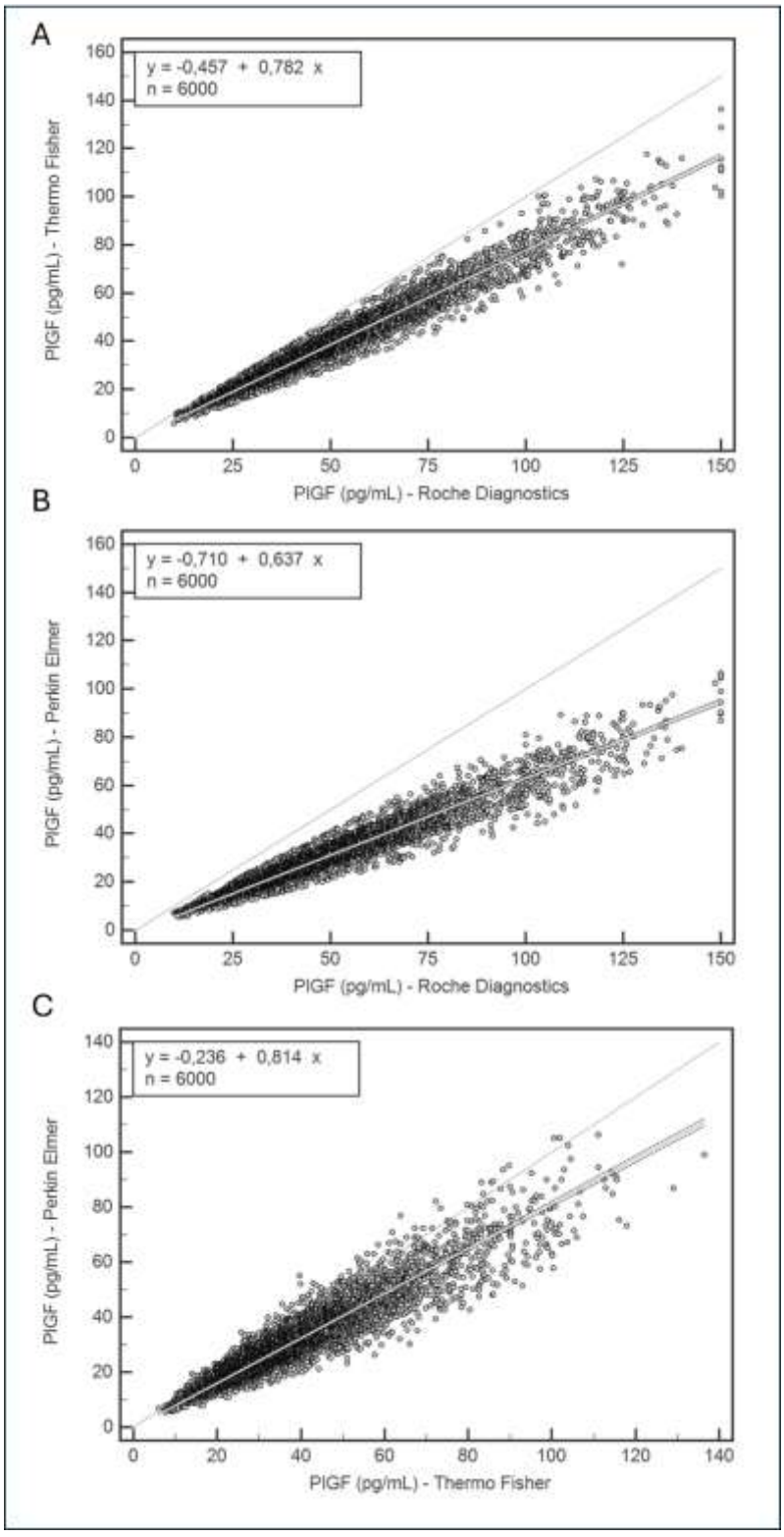

Figure 2. (A) Passing–Bablok regression analysis comparing PlGF results from Thermo Fisher and Roche assays, **(B)** Perkin Elmer and Roche assays, and **(C)** Perkin Elmer and Thermo Fisher assays, using simulated concentrations from 3,000 non-PE and 3,000 PE pregnancies.

Table 2. Performance of PE screening at fixed screen-positive rates (SPR) using the FMF and FMB mathematical models with Roche Diagnostics PlGF platform (11–13 WG).

SPR	FMF model			FMB model			p value
	Cutoff	DR (%)	AUC	Cutoff	DR (%)	AUC	
10%	127	67.4 (65.7–69.1)	**0.888** (0.886–0.890)	380	60.0 (58.3–61.8)	**0.845** (0.843–0.847)	<0.0001
15%	182	75.8 (74.3–77.4)		780	68.4 (66.7–70.1)		
20%	244	81.2 (79.7–82.6)		1368	73.9 (72.3–75.4)		
25%	313	84.8 (83.5–86.1)		2261	78.1 (76.5–79.5)		

Values in parentheses are 95% confidence intervals. AUC: area under receiver-operating-characteristics curve; DR: detection rate; SPR: screen-positive rate; WG: weeks of gestation.

Detection rates stratified by maternal risk factors showed similar sensitivities for both models when no risk factors were present (63.8% vs. 62.4%). However, when risk factors were present, the FMF model showed significantly higher detection rates (DRs) across all subgroups (Supplementary Material S4, Table S-IV).

Screening Performance at 10 Weeks of Gestation

When PlGF at 10 WG was incorporated into the simulation, the FMF model continued to outperform the FMB model in terms of AUC. The AUC values were similar to those at 11–13 WG: 0.890 for FMF (vs. 0.888 at 11–13 WG) and 0.850 for FMB (vs. 0.845). An 8% reduction in the PlGF difference between the PE and non-PE groups (from −33% to −25%) resulted in a 4–5% decrease in the DR at the same SPR (Supplementary Material S4, Table S-V).

Discussion

The FMF model consistently outperformed the FMB model in terms of diagnostic accuracy. This advantage is primarily attributable to the FMF's more comprehensive a priori risk calculation, which incorporates a broader range of maternal risk factors, including autoimmune diseases, diabetes, and obstetric history, and applies platform-specific PlGF medians. In contrast, the FMB model relies solely on Roche Diagnostics medians, which creates cutoff variability when other platforms are used, although the overall AUC remains unaffected. This distinction has practical implications: laboratories using Thermo Fisher or Perkin Elmer platforms must recalibrate risk thresholds when applying the FMB model to avoid suboptimal detection or excess screen-positive rates.

Among all published PE risk models, the FMF model remains the only one with extensive internal and external validation, whereas the FMB strategy has not yet been externally validated [5]. However, both models have been reported to show comparable predictive performance in direct comparisons [31]. Recent evidence suggests that PlGF measured before 11 WG can be integrated into both algorithms with no significant loss of performance in early onset or preterm PE detection [31–33]. However, a Danish national study found that reliable PlGF sampling may extend from 10 to 14 WG but not earlier [34], a finding supported by our simulation, where an 8% reduction in PlGF discriminatory power led to a notable 4–5% drop in DR.

A programming error was identified in the FMF web calculator: the a priori risk was not increased when APS or SLE was present individually, but only when both conditions coexisted simultaneously, contrary to the model's specification. This discrepancy highlights the value of independent computational validation tools, such as the one presented here.

The present findings have important clinical implications for first-trimester PE screening. The superior performance of the FMF model suggests that incorporating a broader range of maternal risk factors may improve identification of women who could benefit from preventive aspirin therapy, with a detection rate of 67.4% versus 60.0% at a 10% SPR, a difference that, extrapolated to a cohort of 100,000 pregnancies, could translate into approximately 200 additional PE cases identified and potentially treated with prophylactic aspirin.

Several limitations of this study should be acknowledged. First, its simulation-based design, although grounded in published population data, may not fully reflect the complexity and variability of real-world clinical populations. Model performance was evaluated using simulated datasets and comparison with online calculators rather than prospective clinical cohorts; therefore, the reported DRs represent model-predicted estimates rather than empirically observed outcomes. Additional limitations include the use of a fixed PE prevalence of 3%, the absence of stratification by PE subtype (preterm versus term), and the derivation of population medians from the same simulated dataset, which may introduce circularity. Finally, the study assessed predictive performance rather than clinical outcomes, such as reduction in PE incidence or improvement in maternal and neonatal health. Accordingly, the tool should be regarded as a research and educational instrument rather than a standalone clinical decision support system, and prospective clinical validation remains essential.

Regarding generalizability, the simulation parameters were derived primarily from published European populations, with a predominantly Caucasian composition (~91%), which may limit applicability in more ethnically diverse clinical settings. Although the FMF model has undergone extensive external validation, prospective multicenter studies in diverse populations are required to confirm the applicability of both the computational tool and the observed differences between screening models in routine clinical practice.

Conclusions

The FMF model outperformed the FMB model owing to its more comprehensive integration of maternal risk factors and greater robustness across analytical platforms. While platform differences and non-local medians can shift risk cutoff values, the overall screening accuracy, as measured by the AUC, remains unaffected. PE risk algorithms retain validity before 11 WG, provided that PlGF has sufficient discriminatory power. This free, open-access computational tool enables clinically relevant simulations and supports informed decisions regarding PE screening strategies and provides an independent means of validating the accuracy of existing online calculators.

List of Abbreviations: APS: antiphospholipid syndrome; AUC: area under the curve; BMI: body mass index; CH: chronic hypertension; DR: detection rate; FMB: Fetal Medicine Barcelona; FMF: Fetal Medicine Foundation; GA: gestational age; IVF: in vitro fertilization; MAP: mean arterial pressure; PE: preeclampsia; PlGF: placental growth factor; ROC: receiver operating characteristic; SLE: systemic lupus erythematosus; SPR: screen-positive rate; UtA-PI: uterine artery pulsatility index; WG: weeks of gestation.

Author Contributions: Conceptualization: EMM, ZCA; Formal analysis: EMM, SAR, SMP, LJM; Methodology: EMM, SAR, SMP, MCSB, LJM, Project Administration: ZCA, RVO; Supervision: MCSB, ZCA, RVO, Writing: EMM, Writing – review and editing: SAR, SMP, MCSB, LJM, ZCA, RVO. All authors read and approved the final manuscript.

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Data Availability Statement: The authors declare that the data supporting the findings of this study are available within the paper as tables and figures, as well as in the Supplementary Material, linked here: https://drive.google.com/file/d/1GmbT1qxfV7owngTLECqcPQnDzAtZSxYq/view?usp=sharing

Conflict of Interest: The authors declare no conflict of interest.

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