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Inflammatory indices beyond PSA density for prostate cancer detection in men with PSA 4–10 ng/mL

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Inflammatory indices beyond PSA density for prostate cancer detection in men with PSA 4–10 ng/mL

Article

Running title: Inflammatory markers in prostate cancer

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ABSTRACT

BACKGROUND: Whether routinely available systemic inflammatory indices provide incremental diagnostic information beyond prostate-specific antigen density (PSAD) in men with prostate-specific antigen (PSA) concentrations of 4–10 ng/mL remains uncertain.

METHODS: We retrospectively pooled 237 men undergoing initial prostate biopsy at two hospitals, including 110 with biopsy-detected prostate cancer and 127 with benign prostatic hyperplasia. Logistic regression models included PSAD alone (Model 0), PSAD plus ln(PIV) (Model 1), or PSAD plus ln(SIRI) (Model 2). We compared discrimination, model fit, prediction error, calibration, exploratory operating points, and internal validation using 1000 bootstrap resamples.

RESULTS: AUROCs were 0.835, 0.848, and 0.846 for Models 0, 1, and 2, respectively. The increases of 0.013 and 0.011 were not statistically significant in paired DeLong tests (P = 0.244 and P = 0.357). Both extended models improved fit and reduced apparent Brier scores. At 90.0% sensitivity, specificity was 43.3%, 54.3%, and 52.8%; the corresponding specificity increases were 11.0 percentage points (95% bootstrap interval −10.9 to 27.7) and 9.4 percentage points (−13.7 to 25.6). Optimism-corrected AUROCs were 0.834, 0.843, and 0.842.

CONCLUSIONS: PIV and SIRI improved model fit in this selected cohort but did not establish reliable incremental discrimination or clinical utility beyond PSAD. External evaluation in representative biopsy populations is required before either index is used to inform biopsy decisions.

INTRODUCTION

Biopsy decisions remain challenging for men with total prostate-specific antigen (tPSA) concentrations of 4–10 ng/mL. Contemporary guidelines recommend risk assessment beyond the serum tPSA concentration alone.[1–3] Magnetic resonance imaging (MRI) has changed the diagnostic pathway, as illustrated by PROMIS and PRECISION.[4,5] Standardized MRI assessment and prostate-specific antigen density (PSAD) further inform risk stratification.[6,7] Nevertheless, the potential contribution of inexpensive blood-based markers alongside PSAD warrants evaluation.

The pan-immune-inflammation value (PIV) and systemic inflammation response index (SIRI) combine routinely measured peripheral blood-cell counts.[8,9] Previous studies have evaluated predictive models and inflammatory indices for prostate cancer (PCa), including patients within the PSA gray zone.[10–13] However, an association with PCa or better performance than tPSA alone does not establish added value beyond PSAD. We therefore compared continuous PSAD alone with PSAD plus either log-transformed PIV or SIRI in a pooled two-center cohort. We examined whether improved model fit was accompanied by better discrimination or performance at exploratory high-sensitivity and high-specificity operating points.

MATERIALS AND METHODS

Study population and outcome

We retrospectively analyzed men undergoing their first transperineal prostate biopsy at two hospitals in Shenyang, China, between January 2020 and May 2026. The participating institutions were the Fourth Affiliated Hospital of China Medical University and Liaoning Cancer Hospital. Eligibility required tPSA of 4–10 ng/mL, complete values for the final model variables, and a definitive biopsy diagnosis. Exclusions included active infection or inflammatory disease, hematological disease, another malignancy, recent treatment likely to alter blood counts or PSA,

previous prostate surgery, and invalid data. Biopsy comprised 12 systematic cores, with three additional cognitively targeted cores when MRI identified a suspicious lesion.

The endpoint was any biopsy-detected PCa, compared with pathologically confirmed benign prostatic hyperplasia (BPH). The analysis therefore did not include the full spectrum of biopsy-negative diagnoses. The final dataset contained 237 complete observations, and no imputation was performed. No a priori sample-size calculation was undertaken. Center identifiers and the original screening denominator were unavailable in the analytic records. Supplementary Figure S5 shows the final sample; it cannot reconstruct all screened patients or reason-specific exclusions.

Ethics approval and informed consent

The Ethics Committee of the Fourth Affiliated Hospital of China Medical University approved the study (EC-2026-KS-190; August 13, 2026). Approval was also obtained from the Ethics Committee of Liaoning Cancer Hospital (2023GZR0314). The requirement for informed consent was waived by the Ethics Committee of the Fourth Affiliated Hospital of China Medical University and the Ethics Committee of Liaoning Cancer Hospital. The study followed the approved protocol and the Declaration of Helsinki and its subsequent amendments.

Predictors and model specification

PSAD was calculated as tPSA divided by prostate volume. PIV was calculated as neutrophils × monocytes × platelets / lymphocytes, and SIRI as neutrophils × monocytes / lymphocytes. Calculations used numerical blood-cell counts expressed in 10^9/L, tPSA in ng/mL, and prostate volume in mL. PSAD was expressed in ng/mL per mL. PIV and SIRI were transformed using natural logarithms, denoted LNPIV and LNSIRI, respectively.

Three unpenalized logistic regression models were fitted. Model 0 included PSAD/0.01 alone; Model 1 added LNPIV, and Model 2 added LNSIRI. The inflammatory indices were assessed

separately because they were strongly correlated. We did not include tPSA and prostate volume alongside their ratio. Complete coefficients, units, and probability equations are provided in Supplementary Table S11 and its accompanying notes. Original-scale models and functional-form comparisons are reported in Supplementary Tables S7 and S8. Restricted cubic spline analyses assessed potential nonlinearity.[14]

Statistical analysis

Continuous variables are summarized as medians and interquartile ranges and were compared using the Mann–Whitney U test. Categorical variables were compared using the chi-squared test or Fisher's exact test when expected cell counts were below five. Discrimination was quantified by the area under the receiver operating characteristic curve (AUROC), with paired comparisons using the DeLong test.[15] Likelihood-ratio tests and the Akaike information criterion (AIC) assessed model fit. Apparent Brier scores quantified prediction error in the development sample. Internal validation used 1000 common nonparametric bootstrap resamples.[16] Each refitted model was evaluated in its bootstrap sample and the original cohort. Mean bootstrap-minus-original AUROC was subtracted from apparent AUROC to estimate optimism-corrected discrimination. Calibration intercepts and slopes were estimated jointly by logistic recalibration of bootstrap-model predictions in the original cohort and averaged across resamples.[17] Flexible calibration curves used restricted cubic splines of the prediction logit. Supplementary Methods describe curve correction, grouping, and uncertainty. The pooled cohort was not split into training and test sets, and no external validation was performed.

At sensitivity ≥90%, we selected the highest observed predicted-probability threshold meeting the constraint. At specificity ≥90%, we selected the lowest eligible threshold. Predictions at or above the threshold were classified as positive. Supplementary Table S12 provides thresholds and confusion matrices. In each of 1000 paired bootstrap samples, models were refitted, thresholds

reselected, and operating performance evaluated in that same resample. Percentile intervals quantify sampling uncertainty in paired differences; they do not correct operating performance for optimism. The 90% constraints were exploratory rather than guideline-endorsed clinical cutoffs.

Additional analyses included decision curves,[18,19] sensitivity-focused partial AUROC over sensitivities 0.90–1.00 (ref. 20) alternative predictor transformations, and the platelet component of PIV. Partial AUROC differences used 5000 paired bootstrap resamples with models refitted and evaluated within each resample. McClish standardization rescales partial area and does not remove model optimism. No multiplicity adjustment was applied to the parallel marker comparisons, which were interpreted as exploratory.

TRIPOD+AI and PROBAST+AI informed reporting and consideration of potential bias; no formal PROBAST+AI rating was undertaken.[21,22] ROC analyses used pROC.[23] The focused decision-curve display at thresholds 0.10–0.40 was selected post hoc using a published prostate-biopsy illustration.[24] This display range is not a validated recommendation for clinical thresholds. Reproducibility analyses and regenerated figures used R version 4.2.2 Patched.

Use of artificial intelligence

During manuscript preparation, ChatGPT (OpenAI) was used to assist with language editing, manuscript organization, and preparation and checking of R analysis code. ChatGPT was not used to execute the clinical analyses or independently verify the underlying patient data. All analyses were executed by the authors using the unchanged source clinical data, and all AI-assisted outputs were reviewed and verified by the authors. The authors take full responsibility for the accuracy, interpretation, and final content of the manuscript.

Data and materials availability

De-identified analytic data and analysis code may be made available to qualified investigators for methodologically sound requests, subject to ethics requirements, institutional data-protection rules, and approval by the participating institutions and corresponding authors.

RESULTS

Cohort characteristics and primary model performance

The analytic cohort included 110 men with PCa and 127 with BPH (Table 1). The PCa group had higher median tPSA, PSAD, PIV, and SIRI and lower prostate volume. Median platelet counts were identical in the two groups. PIV and SIRI were strongly correlated (Spearman $\rho = 0.91$; Supplementary Figure S2). Univariable associations are reported in Supplementary Table S1. Spline analyses found no statistical evidence of nonlinearity for the primary predictors (Supplementary Table S2 and Supplementary Figure S3).

After adjustment for PSAD, the odds ratio per one-unit increase in LNPIV was 1.929 (95% confidence interval [CI] 1.174–3.169; P = 0.009). The corresponding estimate for LNSIRI was 2.372 (95% CI 1.349–4.172; P = 0.003). A one-unit increase in either natural logarithm corresponds to an approximately 2.718-fold increase in the original index. AUROC increased by 0.013 for Model 1 and 0.011 for Model 2 relative to Model 0. Neither paired comparison reached statistical significance (P = 0.244 and P = 0.357, respectively; Table 2 and Figure 1). Both extensions had lower AIC and apparent Brier scores, with likelihood-ratio P values of 0.008 and 0.002.

Internal validation and exploratory operating points

Optimism-corrected AUROCs were 0.834, 0.843, and 0.842 for Models 0, 1, and 2, respectively. Corresponding corrected calibration slopes were 0.988, 0.968, and 0.968 (Figure 2 and Supplementary Table S13). At the high-sensitivity operating point, all three models detected 99 of 110 cancers. Specificity was 43.3%, 54.3%, and 52.8%, respectively. The increases relative to

PSAD were 11.0 percentage points (95% bootstrap interval −10.9 to 27.7) and 9.4 percentage points (−13.7 to 25.6). At specificity 90.55% (115/127), sensitivity was 60.9%, 62.7%, and 65.5%. Intervals for both sensitivity differences also included zero (Supplementary Tables S5, S6, and S12).

Partial AUROC increases also had bootstrap intervals spanning zero (Supplementary Table S9). Case-deletion stability results are presented in Supplementary Table S10. Apparent net-benefit differences varied with the threshold probability (Supplementary Table S3 and Supplementary Figures S1 and S6). Adding log-transformed platelet count to Model 2 did not improve model fit or discrimination (Supplementary Table S4 and Supplementary Figure S4).

DISCUSSION

PIV and SIRI provided additional information about biopsy-detected PCa after adjustment for PSAD in this selected cohort. However, improved model fit did not establish a reliable improvement in discrimination or clinical utility. Lower Brier scores likewise reflected apparent performance rather than independently evaluated prediction error. The distinction matters when small statistical gains are proposed as a basis for changing biopsy decisions.

Zhu et al. reported improved PCa discrimination when PIV was added to PSA in men with PSA concentrations of 4–20 ng/mL.[11] That comparison does not establish incremental value beyond PSAD within the narrower gray zone. Sulhan and Kalyenci evaluated inflammatory indices beyond an age- and PSAD-based model for clinically significant PCa and found limited incremental discrimination.[13] Their endpoint and comparator differed from those used here. Our study adds an evaluation of continuous PSAD with individual inflammatory indices in the 4–10 ng/mL range, including explicitly uncertain high-sensitivity operating-point comparisons.

The specificity point estimates at high sensitivity favored both extensions, but the paired intervals allowed either better or worse specificity than PSAD alone. Partial AUROC did not resolve this

uncertainty, and the decision curves did not show a consistent net-benefit advantage across thresholds. Each high-sensitivity rule missed 11 cancers, but equal counts do not establish that the same cancers were missed. Without cancer-grade information for these missed cases, the clinical consequences cannot be assessed. These results therefore do not establish safe biopsy avoidance. Studies in men with indeterminate MRI lesions illustrate the importance of evaluating biopsy tradeoffs within the intended diagnostic pathway.[25]

The platelet analysis addressed the component distinguishing the two indices, because PIV equals SIRI multiplied by platelet count. Adding log-transformed platelet count to PSAD plus LNSIRI did not provide evidence of additional predictive value. This statistical decomposition does not identify a biological mechanism or exclude a role for platelets in PCa.

Several limitations constrain interpretation. This retrospective complete-case analysis compared PCa only with pathologically confirmed BPH, rather than the full spectrum of biopsy-negative findings. The unavailable screening denominator prevents a complete assessment of selection and missing-data bias. Consequently, the observed disease frequency, calibrated probabilities, and decision-curve estimates should not be assumed to represent an unselected biopsy population. Missing center identifiers also prevented evaluation of hospital effects or validation across centers. Pooled internal resampling does not address these limitations.

The outcome was any PCa rather than clinically significant PCa. Standardized PI-RADS scores and several contemporary biomarkers were unavailable, leaving incremental value within an MRI-based pathway untested. PSAD alone is also a narrower comparator than a clinical model incorporating age and other established predictors. No a priori sample-size calculation supported model development.[26] Multiple exploratory comparisons and wide performance-difference intervals further limit inference.

In summary, PIV and SIRI improved model fit but did not establish a dependable clinical advantage over PSAD in this selected PSA-gray-zone cohort. Further evaluation should retain center identity and include representative biopsy-negative diagnoses, clinically significant PCa, MRI findings, and established clinical predictors. Independent assessment is needed before either marker is used to guide biopsy decisions.

AUTHOR CONTRIBUTIONS

BC performed data analysis, manuscript writing and editing, and figure preparation. YK contributed to data collection and data organization. ZM and NL reviewed and revised the manuscript and contributed to figure improvement. All authors read and approved the final manuscript and agreed to be accountable for all aspects of the work.

COMPETING INTERESTS

The authors declare no competing financial interests.

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No funding was received for this study.

SUPPLEMENTARY INFORMATION

Supplementary Tables S1–S13, Supplementary Figures S1–S6, and Supplementary Methods are provided in a separate final-format file and contain additional regression, functional-form, internal-validation, operating-point, decision-curve, partial-AUROC, platelet-component, and sensitivity analyses.

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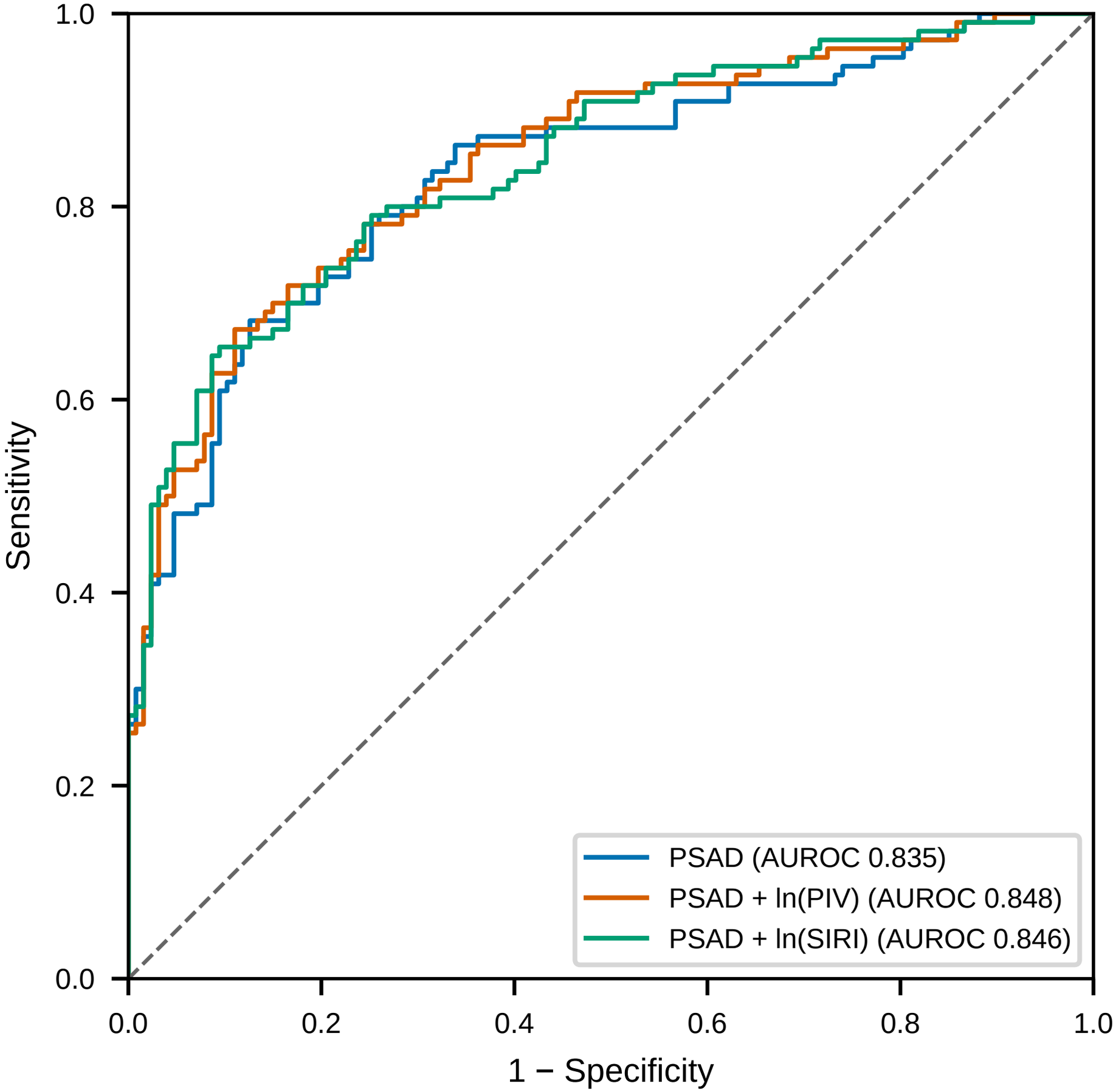
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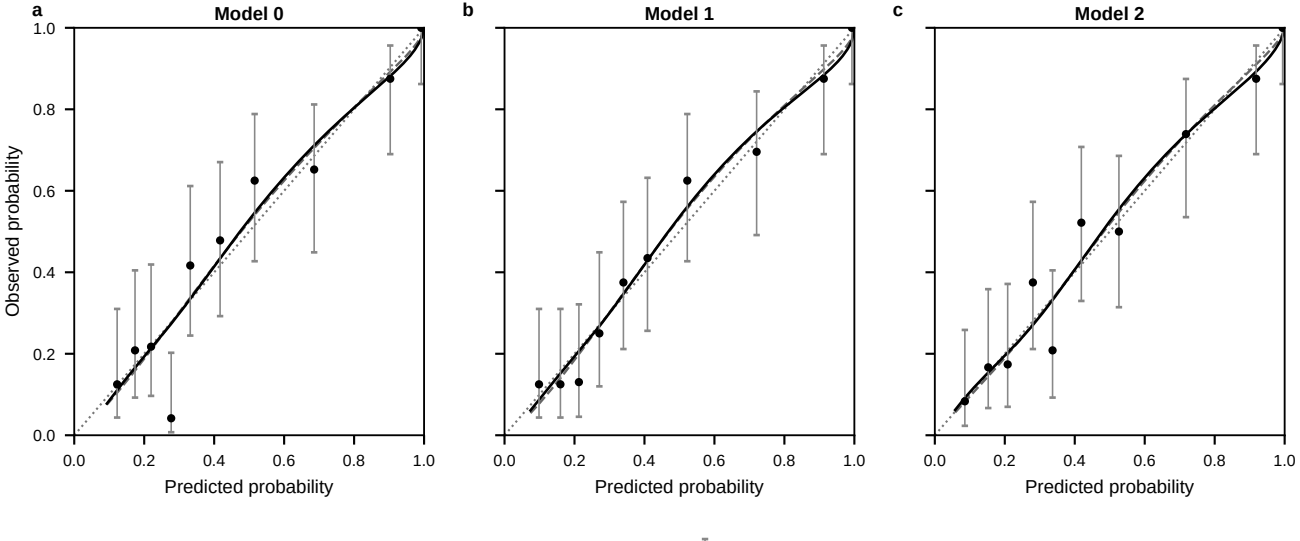
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FIGURE LEGENDS

Figure 1. Apparent receiver operating characteristic curves in 237 men undergoing initial prostate biopsy, including 110 with prostate cancer and 127 with benign prostatic hyperplasia. Model 0 uses prostate-specific antigen density (PSAD); Models 1 and 2 additionally include log-transformed pan-immune-inflammation value (LNPIV) and systemic inflammation response index (LNSIRI), respectively. The diagonal represents chance discrimination. AUROC denotes area under the receiver operating characteristic curve.

Figure 2. Flexible calibration of the three primary models in the 237-patient development cohort: (a) Model 0, PSAD alone; (b) Model 1, PSAD plus LNPIV; and (c) Model 2, PSAD plus LNSIRI. Points show observed prostate cancer proportions against mean predicted probabilities in groups defined by prediction deciles. Vertical bars are 95% Wilson score intervals. The diagonal indicates ideal calibration. Apparent curves use logistic restricted cubic splines of the prediction logit with four knots. Corrected curves subtract mean pointwise optimism from 1000 common bootstrap resamples. The bars do not represent confidence bands around the corrected curves. Supplementary Methods provide the full procedure, and Supplementary Table S13 reports scalar calibration estimates.


1.0
0.8
0.6
0.4
0.2
0.0
Sensitivity
0.0
0.2
0.4
0.6
0.8
1.0
1 − Specificity
PSAD (AUROC 0.835)
PSAD + ln(PIV) (AUROC 0.848)
PSAD + ln(SIRI) (AUROC 0.846)


a
Model 0
b
Model 1
c
Model 2
Observed probability
Predicted probability
Predicted probability
Predicted probability
0.0
0.2
0.4
0.6
0.8
1.0
Ideal
Apparent
Bootstrap-corrected
Decile estimate (95% Wilson interval)

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- Table1.xlsx
- Table2.xlsx
- SupplementaryInformation.docx