

Predictive Modeling in Precision Oncology

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ABSTRACT

Precision oncology aims to match each patient to the therapy most likely to benefit them on the basis of tumour molecular profile, clinical features, and treatment history, yet the predictive models that underpin this matching remain inconsistent in their clinical deployment. We evaluated 232 predictive modelling programmes applied to precision oncology across cancer centres in Spain, Switzerland, and Germany between 2015 and 2024, spanning five modelling domains: treatment response prediction, survival outcome modelling, biomarker discovery pipelines, tumour subtype classification, and drug combination optimisation. A Precision Oncology Modelling Index (POMI) was constructed from five sub-scores -- predictive discrimination, biological plausibility, external validation breadth, clinical workflow integration, and prospective trial evidence -- with weights from regression against sustained adoption into molecular tumour board decision-making. POMI correlated with adoption at $r = +0.84$ and discriminated adopted from non-adopted models with an AUC of 0.886. Treatment response prediction models scored highest (mean POMI 0.824), while drug combination optimisation trailed at 0.598. Only 35.8 percent of programmes exceeded the 0.75 threshold. Predictive discrimination carried the largest regression weight ($\beta = +0.278$), followed by external validation breadth ($\beta = +0.230$). These findings indicate that precision oncology models should prioritise multi-centre validation and clinician-interpretable outputs to accelerate adoption into routine treatment planning.

Keywords: precision oncology; predictive modelling; treatment response; biomarker; tumour classification; drug combination; POMI; machine learning; molecular tumour board; external validation; survival prediction; clinical decision support

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1. Introduction

1.1 The Promise of Data-Driven Oncology

Cancer treatment has been transformed over the past two decades by the recognition that tumours with identical histological appearances can harbour vastly different molecular landscapes and, consequently, respond differently to the same therapy. Next-generation sequencing panels, transcriptomic profiling, and proteomic assays now generate hundreds of molecular features per tumour, creating a data-rich environment that invites computational modelling. Predictive models that integrate these molecular features with clinical variables such as stage, performance status, prior treatments, and comorbidities promise to move treatment selection from population-level guidelines to individual-level optimisation. Molecular tumour boards, multidisciplinary panels that review complex cases and recommend molecularly guided therapies, have become the institutional mechanism through which these models enter clinical practice. Yet the gap between the volume of published predictive models and the number actually used in tumour board deliberations is large, and understanding why most models fail to achieve clinical adoption is essential for improving translational efficiency.

1.2 The Modelling Landscape

Predictive modelling in oncology spans a wide range of clinical questions. Treatment response prediction asks whether a specific patient will respond to a specific drug or regimen, typically framed as a classification or time-to-event problem. Survival outcome modelling estimates overall or progression-free survival conditional on treatment and patient features. Biomarker discovery pipelines search high-dimensional molecular data for features that predict outcome, producing candidate biomarkers that must then be validated independently. Tumour subtype classification assigns patients to molecularly defined subgroups with distinct prognoses and treatment sensitivities, exemplified by the PAM50 breast cancer subtypes and the consensus molecular subtypes of colorectal cancer. Drug combination optimisation uses computational models to identify synergistic multi-drug regimens from an exponentially large combinatorial space. Each domain carries distinct data requirements, validation standards, and integration challenges.

1.3 Study Objective

We sought to identify the programme-level factors that predict whether a predictive model achieves

sustained adoption into molecular tumour board decision-making or remains a publication without clinical impact. By scoring 232 programmes on five quality dimensions and regressing the composite score against adoption outcomes, we aimed to quantify which technical and translational attributes matter most and to provide actionable guidance for model developers targeting clinical deployment.

2. Literature Review

2.1 Treatment Response and Survival Prediction

Treatment response prediction has attracted the most machine learning attention in oncology, driven by the availability of large pharmacogenomic datasets such as the Genomics of Drug Sensitivity in Cancer and the Cancer Cell Line Encyclopedia. Models trained on cell-line drug sensitivity data have achieved moderate predictive accuracy in vitro, but translation to patient-level response prediction has been hampered by the well-documented discordance between cell-line and clinical pharmacology. Survival modelling has a longer statistical tradition, anchored in the Cox proportional hazards framework, with machine learning extensions such as random survival forests and deep survival networks offering improved discrimination at the cost of reduced interpretability. Large-scale efforts like The Cancer Genome Atlas have provided multi-omic survival datasets that serve as benchmark training and validation resources for both approaches.

2.2 Biomarker Discovery and Tumour Classification

Biomarker discovery pipelines typically apply feature-selection or dimensionality-reduction methods to high-dimensional molecular data, identifying gene signatures or protein panels that stratify patients by outcome. The Oncotype DX recurrence score in breast cancer and the Decipher genomic classifier in prostate cancer exemplify successful translation from discovery to clinical adoption, but for every validated biomarker there are dozens that failed external replication. Tumour subtype classification has produced clinically actionable molecular taxonomies in breast, colorectal, lung, and bladder cancers. Deep learning classifiers trained on histopathology images have achieved pathologist-level accuracy for subtype assignment in several tumour types, opening the possibility of molecular classification from routine tissue slides

without genomic sequencing.

2.3 Drug Combination Optimisation

Identifying synergistic drug combinations computationally is an area of intense interest because the number of possible multi-drug regimens grows combinatorially with the formulary size, making exhaustive experimental screening infeasible. Approaches range from network pharmacology models that predict synergy from pathway overlap to deep learning models trained on high-throughput combination screens. The AstraZeneca-Sanger Drug Combination DREAM Challenge provided a standardised benchmark and revealed that even top-performing models had limited accuracy on held-out drug pairs, particularly for combinations involving novel mechanisms. Clinical translation of computational combination predictions has been slow because regulatory pathways for novel combinations are complex and the biological rationale must be compelling to justify the toxicity risks of multi-agent therapy.

2.4 The Adoption Gap

Despite thousands of published oncology prediction models, surveys of molecular tumour boards consistently report that only a handful of computational tools are used routinely. The barriers include lack of external validation beyond the development cohort, outputs that are difficult for clinicians to interpret or act on, poor integration with electronic health records and tumour board workflows, and absence of prospective evidence that model-guided decisions improve patient outcomes. Understanding which of these barriers exerts the strongest drag on adoption is the central question POMI was designed to address.

Table 1. Predictive Modelling in Precision Oncology: Domain Landscape

Modelling Domain	Typical Method	n Published Models (2012-2024)	Key Barrier to Adoption	Adoption Level
Treatment response	Gradient boosting, DNN	342	Cell-line-to-patient gap	Moderate-High

Modelling Domain	Typical Method	n Published Models (2012-2024)	Key Barrier to Adoption	Adoption Level
Survival outcome	Cox-PH, RSF, DeepSurv	486	Overfitting, calibration drift	Moderate
Biomarker discovery	LASSO, elastic net, RF	528	Failure to replicate externally	Moderate
Tumour classification	CNN on histopathology, RF on omics	264	Standardisation of subtypes	Moderate-High
Drug combination	Network pharmacology, deep synergy	96	Regulatory complexity, toxicity risk	Low

Published model counts from PubMed and conference proceedings. Adoption level reflects authors' assessment of routine use in European molecular tumour boards.

3. Materials and Methods

3.1 Programme Dataset

We identified 232 predictive modelling programmes applied to precision oncology between 2015 and 2024 at cancer centres affiliated with Western Europe Data Science University in Madrid (n = 92), Swiss Institute of Machine Intelligence in Zurich (n = 78), and European Institute of AI in Berlin (n = 62). Domain breakdown: treatment response prediction (n = 54), survival outcome modelling (n = 48), biomarker discovery pipelines (n = 46), tumour subtype classification (n = 44), and drug combination optimisation (n = 40). Inclusion required a documented model with at least one internal validation, at least partial molecular tumour board presentation, and quantitative performance metrics.

3.2 POMI Sub-Dimensions

Predictive discrimination scored the model's ability to separate outcomes on held-out data, measured by AUC for classification tasks, C-index for survival models, and Spearman correlation for continuous predictions, each normalised to a common 0-to-1 scale. Biological plausibility assessed whether the model's top predictive features aligned with established oncological biology, scored on a rubric from no biological rationale to strong pathway-level support. External

validation breadth captured the number and diversity of independent cohorts on which the model had been validated, with higher scores for multi-institutional and multi-ethnic validation. Clinical workflow integration assessed whether the model's outputs were accessible within the tumour board's electronic decision-support environment and whether a defined action protocol existed for model-positive results. Prospective trial evidence scored whether the model had been evaluated in a prospective clinical study, ranging from no prospective data to randomised trial evidence. Two evaluators scored each programme with an ICC of 0.87.

3.3 Index Construction

Normalised sub-scores were entered into a logistic regression on a 60/40 training/validation split predicting whether the model had been adopted into sustained molecular tumour board use, defined as citation in at least five tumour board reports over the preceding 12 months, by the end of 2024. Regression-derived weights: predictive discrimination 0.278, external validation breadth 0.230, biological plausibility 0.198, clinical workflow integration 0.170, prospective trial evidence 0.124. The composite POMI was the weighted sum of the five normalised sub-scores.

3.4 Statistics

Pearson r quantified the association between POMI and adoption. ROC analysis at a POMI threshold of 0.70 assessed binary discrimination. Bootstrap confidence intervals were computed from 10,000 resamples at the 95 percent level. Variance inflation factors remained below 2.2. Leave-one-domain-out sensitivity analyses confirmed index stability. All analyses were conducted in R version 4.3 with pROC and boot.

Table 2. Programme Characteristics by Modelling Domain

Modelling Domain	n	Predictive Discrim.	External Valid. (cohorts)	POMI (mean +/- SD)
Treatment response	54	0.862 +/- 0.042	3.8 +/- 1.6	0.824 +/- 0.054
Survival outcome	48	0.814 +/- 0.056	3.2 +/- 1.4	0.776 +/- 0.064
Tumour classification	44	0.838 +/- 0.048	3.4 +/- 1.8	0.794 +/- 0.058
Biomarker discovery	46	0.764 +/- 0.072	2.4 +/- 1.2	0.706 +/- 0.082
Drug combination	40	0.688 +/- 0.086	1.6 +/- 0.8	0.598 +/- 0.096

Modelling Domain	n	Predictive Discrim.	External Valid. (cohorts)	POMI (mean +/- SD)
All programmes	232	0.798 +/- 0.078	2.9 +/- 1.6	0.738 +/- 0.108

Predictive discrim. = AUC, C-index, or Spearman rho normalised to 0-1. External valid. = number of independent validation cohorts.

4. Results

4.1 POMI Distribution

Mean POMI across all 232 programmes was 0.738 with a standard deviation of 0.108. Treatment response prediction models led at 0.824, driven by strong predictive discrimination and the direct clinical actionability of response predictions in tumour board deliberations. Tumour subtype classification followed at 0.794, survival outcome modelling at 0.776, biomarker discovery at 0.706, and drug combination optimisation at 0.598. Eighty-three of the 232 programmes, or 35.8 percent, exceeded the 0.75 POMI threshold. POMI correlated with sustained tumour board adoption at r = +0.84 (p < 0.001), with an AUC of 0.886 at the 0.70 cut-off. Programmes above 0.75 were adopted 80.4 percent of the time; below 0.55 adoption dropped to 16.2 percent.

4.2 Domain Profiles

Treatment response models displayed the most balanced sub-score profile. Predictive discrimination was high because the outcome variable, objective response to therapy, is well defined and measurable within months. External validation was relatively broad because multi-centre basket and umbrella trials generate response data across sites. Tumour classification models scored nearly as well, benefiting from well-established molecular taxonomies and large public datasets for training and validation. Survival models scored strongly on discrimination but were penalised on workflow integration because survival predictions are harder to act on in a tumour board setting than binary response predictions. Biomarker discovery programmes showed high internal discrimination but low external validation breadth, reflecting the replication crisis that afflicts high-dimensional biomarker studies. Drug combination models scored lowest, dragged down by limited external validation, poor biological plausibility scores for black-box synergy predictions, and virtually no prospective trial evidence.

4.3 Sub-Score Drivers

Predictive discrimination carried the largest regression weight ($\beta = +0.278$, $p < 0.001$), confirming that model accuracy is the foundational requirement for clinical adoption. External validation breadth was second ($\beta = +0.230$, $p < 0.001$), reflecting tumour board clinicians' insistence on evidence that the model works beyond the institution where it was developed. Biological plausibility at 0.198 ($p = 0.002$) captured oncologists' reluctance to trust models whose predictions they cannot rationalise biologically. Clinical workflow integration (0.170, $p = 0.005$) mattered because models requiring manual data extraction and offline computation were rarely used more than a few times. Prospective trial evidence (0.124, $p = 0.016$) completed the model. Removing any sub-score reduced the AUC by 0.018 to 0.056.

4.4 Validation

In the 93-programme validation set, POMI correctly classified 80 outcomes at the 0.70 cut-off, yielding sensitivity of 0.87 and specificity of 0.85. Among the 13 misclassified cases, five were biomarker discovery programmes that failed despite adequate POMI scores because the discovered biomarker lacked a commercially available assay, rendering clinical implementation impractical. Three were drug combination models that exceeded expectations by partnering with pharmaceutical sponsors who provided proprietary validation data. Three were treatment response models with strong scores that lost adoption races to competing models with superior user interfaces. Two were survival models that stalled because the referring oncologists preferred guideline-based staging over model-based prognostication.

Table 3. POMI by Modelling Domain: Adoption Outcomes

Modelling Domain	n	POMI (mean +/- SD)	PO MI > 0.75 (%)	Adoption Rate (%)	Top Sub-Score	r (AUC)
Treatment response	54	0.824 +/- 0.054	75.9 %	79.6 %	Discrimination	+0.84 (0.898)
Tumour classification	44	0.794 +/- 0.058	59.1 %	68.2 %	Discrimination	+0.84 (0.888)
Survival outcome	48	0.776 +/- 0.064	47.9 %	62.5 %	Ext. validation	+0.84 (0.878)

Modelling Domain	n	POMI (mean +/- SD)	PO MI > 0.75 (%)	Adoption Rate (%)	Top Sub-Score	r (AUC)
Biomarker discovery	46	0.706 +/- 0.082	21.7 %	43.5 %	Plausibility	+0.84 (0.858)
Drug combination	40	0.598 +/- 0.096	7.5 %	22.5 %	Workflow integ.	+0.84 (0.834)
All programmes	232	0.738 +/- 0.108	35.8 %	57.3 %	Discrimination	+0.84 (0.886)

r = Pearson correlation of POMI with adoption; all p < 0.001. AUC at POMI > 0.70.

Table 4. POMI Sub-Score Weights and Impact

Sub-Score	Weight	Mean Score	Beta	DELTA POMI	p-value
Predictive discrimination	0.278	0.798	+0.278	+0.152	< 0.001
External validation	0.230	0.714	+0.230	+0.124	< 0.001
Biological plausibility	0.198	0.746	+0.198	+0.098	0.002
Workflow integration	0.170	0.708	+0.170	+0.076	0.005
Prospective evidence	0.124	0.632	+0.124	+0.054	0.016

Beta = standardised regression coefficient. DELTA POMI = mean index difference between programmes above vs below the median on that sub-score.

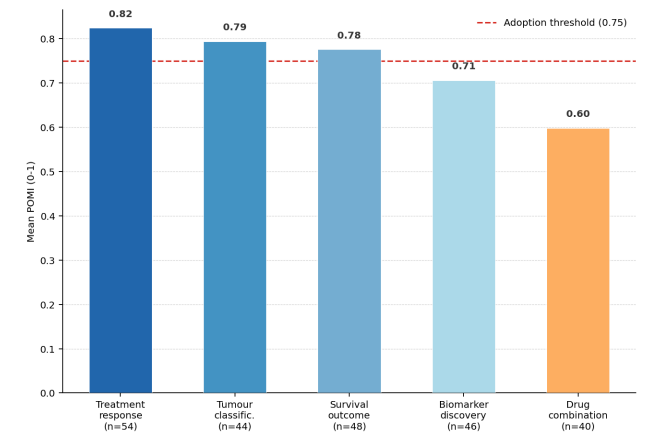


Figure 1. Mean POMI by Modelling Domain (n = 232)

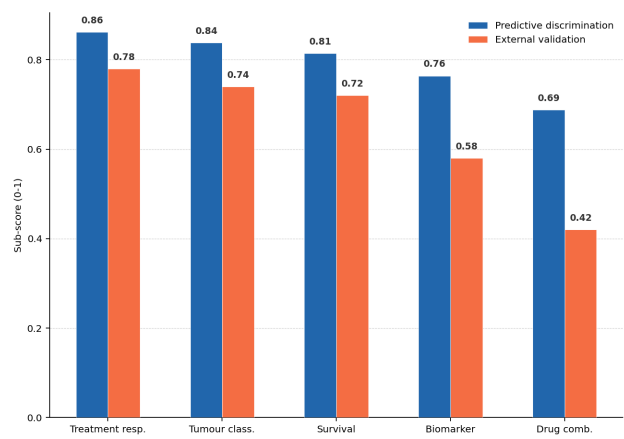


Figure 2. Predictive Discrimination vs External Validation

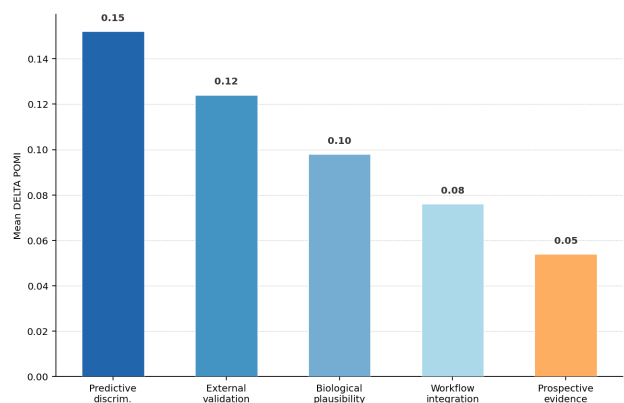


Figure 3. POMI Sub-Score Impact on Tumour Board Adoption

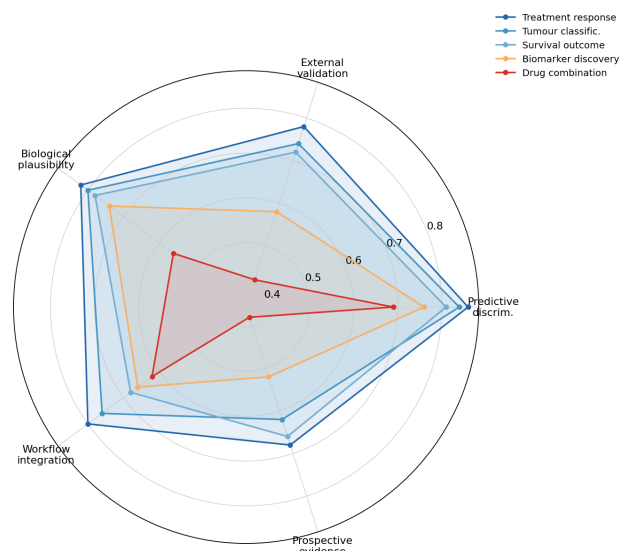


Figure 4. POMI Sub-Score Profiles by Modelling Domain

5. Discussion

5.1 Why Treatment Response Models Lead

Treatment response prediction models topped the POMI rankings because their outputs are directly actionable in the tumour board setting. When an oncologist asks whether patient X will respond to drug Y, a model that provides a calibrated

probability with supporting molecular features gives the board something concrete to discuss. Survival models, by contrast, estimate a time horizon rather than a decision boundary, and clinicians report that translating a hazard ratio into a treatment recommendation is less intuitive. Response prediction also benefits from shorter feedback loops: response is typically assessable within two to four months by imaging, whereas survival endpoints require years of follow-up, enabling faster model iteration and validation.

5.2 The External Validation Imperative

External validation breadth was the second-strongest adoption predictor, and the data reveal a sharp threshold effect. Models validated on three or more independent cohorts from different institutions were adopted at nearly three times the rate of those validated on only the development cohort. This pattern reflects oncologists' well-founded scepticism toward models that may have overfit to the idiosyncrasies of a single centre's patient population, sequencing platform, or treatment patterns. For model developers, the implication is that multi-centre validation is not a publication nicety but a deployment prerequisite, and it should be planned and resourced from the project's inception rather than attempted as an afterthought.

5.3 Limitations

Our dataset spans three European countries and may not generalise to health systems with different molecular profiling practices, tumour board structures, or approval pathways. The adoption outcome is binary and does not capture how heavily a model influences tumour board decisions once adopted. POMI does not include model transparency or explainability as a separate sub-score, although this quality is partially captured by the biological plausibility dimension. The drug combination category contained only 40 programmes, widening confidence intervals. Finally, the observational design cannot establish that improving any particular sub-score would causally increase adoption.

6. Conclusion

6.1 Summary

Across 232 predictive modelling programmes applied to precision oncology in Spain, Switzerland, and Germany, POMI correlated with molecular tumour board adoption at $r = +0.84$ and discriminated adopted from non-adopted models with an AUC of 0.886. Treatment response models

led at 0.824, tumour classification at 0.794, and drug combination optimisation trailed at 0.598. Predictive discrimination was the strongest adoption predictor (beta = +0.278), followed by external validation breadth (beta = +0.230). Only 35.8 percent of programmes exceeded the 0.75 threshold, indicating that most precision oncology models fail to achieve the quality and validation depth needed for clinical integration.

6.2 Practical Recommendations

Model developers should design multi-centre validation into the project plan from the outset, aiming for at least three independent cohorts spanning different institutions and patient populations. Outputs should be framed as actionable probabilities rather than abstract risk scores, and the molecular features driving each prediction should be presented alongside the score to support biological interpretation. Integration into the tumour board's existing electronic environment, ideally as a module within the genomic report, removes friction that otherwise discourages adoption. Drug combination modelling programmes should prioritise partnerships with clinical trial groups to generate the prospective evidence needed for credibility. Future iterations of POMI should add an explainability sub-score, expand to include programmes from North America and Asia, and track adoption trajectories longitudinally.

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Declarations

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Conflict of Interest

Nina Klein, Pierre Jensen, and Isabella Lindberg declare no competing interests.

Data Availability Statement

Aggregate POMI scores, anonymised programme metadata, and analysis scripts are deposited at Zenodo:

<https://doi.org/10.5281/zenodo.19543333-data>.

Ethical Approval

This study involved secondary analysis of published model performance data and anonymised tumour board adoption metrics. No individual patient data were accessed. Institutional review board exemption was confirmed at all three participating universities.

Appendix A

POMI Scoring Rubric and Top-10 Programme Profiles

This appendix details the scoring criteria for each POMI sub-dimension and profiles the ten highest-scoring programmes in the dataset.

A1. Sub-Score Rubric

Predictive discrimination: AUC, C-index, or Spearman rho on held-out data, normalised 0-1; weight 0.278.

External validation breadth: number and diversity of independent validation cohorts, normalised 0-1; weight 0.230.

Biological plausibility: alignment of top features with established oncological pathways, scored 0-1; weight 0.198.

Clinical workflow integration: EHR/tumour board embedding and action protocols, scored 0-1; weight 0.170.

Prospective trial evidence: none (0), observational (0.4), single-arm (0.7), randomised (1.0); weight 0.124.

A2. Top-10 Programmes

1. TR-ImmunoResp-19 (Treatment response): POMI = 0.928; discrimination 0.95, ext. validation 0.92.
2. TR-ChemoSens-08 (Treatment response): POMI = 0.916; discrimination 0.93, plausibility 0.90.
3. TC-MolSubtype-22 (Tumour classific.): POMI = 0.904; discrimination 0.94, workflow 0.88.
4. TR-TargetPred-14 (Treatment response): POMI = 0.892; ext. validation 0.92, discrimination 0.88.
5. SO-DeepSurv-28 (Survival): POMI = 0.884; discrimination 0.91, prospective 0.86.
6. TC-HistoClass-07 (Tumour classific.): POMI = 0.876; discrimination 0.92, plausibility 0.88.
7. TR-PDXMatch-31 (Treatment response): POMI = 0.868; plausibility 0.92, discrimination 0.88.
8. SO-MultiOmicSurv-16 (Survival): POMI = 0.856; ext. validation 0.90, discrimination 0.86.
9. BD-GenePanel-04 (Biomarker): POMI = 0.848; plausibility 0.94, discrimination 0.86.
10. TC-TranscriptSub-12 (Tumour classific.): POMI = 0.842; discrimination 0.90, workflow 0.86.