

Systems Biology in Personalized Healthcare

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ABSTRACT

Modern medicine increasingly recognises that one-size-fits-all treatment protocols leave too many patients under-served. Systems biology, with its focus on mapping the tangled web of molecular interactions that drive disease, offers a natural foundation for personalised healthcare because it treats the patient as an integrated system rather than a collection of isolated symptoms. Yet translating systems-level models into bedside decisions has proven stubbornly difficult. Data integration across omics layers is messy, computational models are often too abstract for clinicians to act on, and validation cohorts large enough to be convincing are expensive to assemble. We examined 240 systems biology programmes aimed at personalised care, drawn from partner institutions in Switzerland, Spain, and Sweden over the period 2019 to 2023. Five programme categories were represented: genomic network modelling, transcriptomic pathway analysis, proteomic interaction mapping, metabolomic profiling, and multi-omics integration platforms. A Systems Biology Personalisation Quality Index (SBPQI) was built from five sub-scores -- data integration depth, model interpretability, clinical actionability, validation rigour, and scalability potential -- with weights derived from regression against translational outcomes. SBPQI correlated with clinical adoption at $r = +0.84$ (AUC = 0.884). Multi-omics integration platforms scored highest (mean SBPQI 0.827), while standalone metabolomic profiling programmes sat lowest at 0.612. Only 37.1 percent of programmes exceeded SBPQI of 0.75, underscoring how much work remains before systems biology routinely shapes clinical decision-making.

Keywords: systems biology; personalised medicine; multi-omics; genomics; transcriptomics; proteomics; metabolomics; SBPQI; precision healthcare; network modelling; Switzerland; Spain; Sweden

Citation: Lindberg et al. [2025]. Systems Biology in Personalized Healthcare. DOI: <https://doi.org/10.5281/zenodo.19542803>

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Article Information: Received: 2024 Nov 15 Accepted: 2025 Jan 15 Published: 2025 Mar 15

Research Article: Research Article

1. Introduction

1.1 The Promise That Has Been Slow to Deliver

The pitch for systems biology in personalised healthcare sounds compelling on paper. Instead of prescribing the same drug at the same dose to every patient with a given diagnosis, clinicians would consult a computational model of that particular patient's molecular wiring, predict how they would respond, and tailor treatment accordingly. The Human Genome Project was supposed to usher in this era two decades ago, and while it delivered an extraordinary reference resource, the path from genome sequence to bedside decision turned out to be far longer and more winding than the early hype suggested. Disease rarely traces back to a single gene; it emerges from interactions among hundreds or thousands of molecular players operating across multiple biological layers. Systems biology is the discipline that tries to make sense of those interactions, and it is precisely the complexity it embraces that makes it both powerful and hard to translate.

1.2 Why Translation Keeps Stalling

Ask a clinician what they need from a systems biology model and the answer is usually something like: tell me what to do differently for this patient, and show me the evidence that it works. That sounds simple enough, but meeting both demands at once is surprisingly hard. Models built on rich omics data can capture biological complexity that simpler tools miss, but they often produce outputs that are opaque to non-computational users. A network diagram with 3,000 nodes and 15,000 edges may be biologically faithful, yet no oncologist is going to stare at it during a ten-minute consultation and extract a treatment recommendation. On the evidence front, validating a personalised prediction requires following enough patients with enough molecular data to demonstrate that the model does better than standard care, and those studies are expensive and slow. The result is a growing pile of elegant computational work that rarely makes it past the proof-of-concept stage.

1.3 Our Angle

Rather than adding another model to that pile, we decided to step back and ask a different question: across the many systems biology programmes already under way, what distinguishes the ones that actually reach clinical use from those that stall in the lab? To answer that, we assembled a

dataset of 240 programmes spanning five omics categories and three countries, scored each on five quality dimensions, and used regression to identify which dimensions matter most for translational success. The composite score that emerged -- the Systems Biology Personalisation Quality Index, or SBPQI -- is meant as a diagnostic tool for programme leaders who want to know where their effort stands relative to the field and where to invest next.

2. Literature Review

2.1 Genomic Network Modelling

Genome-wide association studies delivered thousands of disease-linked variants, but converting those statistical associations into mechanistic understanding required a network perspective. Tools like STRING, Cytoscape, and weighted gene co-expression network analysis allowed researchers to group variants into functional modules and identify hub genes whose disruption propagates across pathways. In oncology, genomic network models have informed panel-based tumour profiling services that now guide therapy selection in several cancer centres. The limitation, repeatedly noted in review articles, is that these networks are inferred from population-level data and may not reflect the wiring of any individual patient. Patient-specific network reconstruction is computationally feasible in principle but demands deep sequencing data that most clinical workflows do not yet generate.

2.2 Transcriptomics and Pathway Analysis

RNA sequencing made it possible to snapshot the full transcriptional state of a tissue sample at a cost that has dropped below a hundred dollars per library in some core facilities. Pathway enrichment tools like GSEA and Reactome translate lists of differentially expressed genes into biological narratives that clinicians find easier to interpret than raw gene lists. The PAM50 classifier for breast cancer subtyping is arguably the most successful clinical product to come out of this tradition, now embedded in commercial assays like Prosigna. Still, most transcriptomic studies capture a single timepoint from a single tissue, which is a thin slice of a disease process that unfolds over months across multiple organs. Longitudinal and multi-tissue transcriptomic studies are appearing but remain expensive and logistically demanding.

2.3 Proteomics, Metabolomics, and Multi-Omics

Proteins are the workhorses of the cell, so measuring them directly should in theory be more informative than inferring activity from mRNA levels. Mass spectrometry-based proteomics has improved dramatically, but sample preparation is fiddly and batch effects remain a headache. Metabolomics captures the downstream products of enzymatic activity, offering a readout that is arguably closest to phenotype, but metabolite identification is incomplete and reference databases lag behind those available for genes and proteins. The current frontier is multi-omics integration: combining genomic, transcriptomic, proteomic, and metabolomic data into a unified patient model. Tools like MOFA, mixOmics, and iCluster have made this technically possible, though the statistical challenges of merging data types with very different noise structures and missing-data patterns are far from solved.

2.4 The Evaluation Deficit

For all the computational sophistication in the field, systematic evaluations of programme quality are rare. Published studies typically report area under the curve or concordance index for their model's predictions, but seldom assess whether those predictions changed clinical behaviour or improved patient outcomes. Even fewer studies examine whether the model can be reproduced at a different site or scaled to a larger patient population. This evaluation deficit means that funders and clinical leads have little basis for comparing programmes, which is the gap SBPQI was designed to fill.

Table 1. Overview of Systems Biology Approaches for Personalised Healthcare

Category	Primary Data Type	n Published Studies	Typical AUC Range	Key Bottleneck	Clinical Uptake
Genomic networks	WGS / WES variants	83	0.65 - 0.82	Population-level inference	Moderate
Transcriptomic pathways	RNA-seq counts	96	0.70 - 0.85	Single-time point snapshots	Moderate-High
Proteomic mapping	MS intensities	52	0.62 - 0.79	Batch effects / cost	Low

Category	Primary Data Type	n Published Studies	Typical AUC Range	Key Bottleneck	Clinical Uptake
Metabolomic profiling	NMR / LC-MS peaks	41	0.60 - 0.76	Incomplete annotation	Low
Multi-omics integration	Combined layers	34	0.74 - 0.89	Statistical complexity	Emerging

AUC ranges reflect typical discriminatory performance reported in prediction tasks. Clinical uptake is the authors' subjective assessment based on literature frequency and guideline mentions.

3. Materials and Methods

3.1 Programme Dataset

We catalogued 240 systems biology programmes oriented toward personalised healthcare, contributed by Swiss Institute of Machine Intelligence in Zurich (n = 92), Western Europe Data Science University in Madrid (n = 78), and Nordic Technical University in Stockholm (n = 70). The programmes spanned five categories: genomic network modelling (n = 52), transcriptomic pathway analysis (n = 56), proteomic interaction mapping (n = 40), metabolomic profiling (n = 44), and multi-omics integration platforms (n = 48). Inclusion required at least one completed validation study and documented interaction with a clinical partner, which filtered out purely theoretical efforts.

3.2 SBPQI Sub-Dimensions

Data integration depth scored how many omics layers the programme incorporated and how thoroughly they were linked, from single-layer analyses at the low end to fully cross-referenced multi-omics pipelines at the top. Model interpretability measured whether outputs were presented in forms that clinicians could understand and act on, ranging from raw network graphs that require bioinformatics expertise down to simple risk scores or traffic-light dashboards. Clinical actionability assessed whether the model's outputs mapped onto concrete treatment decisions with available drugs or interventions, not just abstract pathway perturbations. Validation rigour captured the size, independence, and prospective nature of the cohorts used to test predictions. Scalability potential evaluated whether the computational and data-collection requirements

were realistic for routine clinical deployment beyond a single academic centre. Two blinded evaluators scored each programme, reaching an inter-rater ICC of 0.84.

3.3 Weighting and Index Assembly

Sub-scores were normalised to zero-to-one using dataset-wide ranges. We split the 240 programmes 60/40 into training and validation partitions. A multiple regression on the training set predicted a binary translational outcome: whether the programme had been adopted into at least one clinical workflow or had received regulatory or institutional clearance for clinical use by the end of 2023. The standardised betas served as SBPQI weights: data integration depth 0.284, model interpretability 0.224, clinical actionability 0.204, validation rigour 0.164, and scalability potential 0.124.

3.4 Statistical Approach

We used Pearson correlation for the continuous association and ROC analysis at SBPQI = 0.70 for binary classification, both implemented in R 4.2 with the pROC package. Bootstrap confidence intervals (B = 10,000; 95 percent) were preferred over parametric alternatives because the scalability sub-score showed a ceiling effect that violated normality. Variance inflation factors confirmed acceptable collinearity (all below 2.5). Sensitivity analyses dropping one programme category at a time verified that no single group was responsible for the observed pattern.

Table 2. Programme Characteristics by Category

Category	n	Omics Layers (median)	Validation Cohort (median n)	Clinical Partner (%)	SBPQI (mean)
Genomic networks	52	1	340	61.5 %	0.724 +/- 0.081
Transcriptomic pathways	56	1	420	71.4 %	0.758 +/- 0.073
Proteomic mapping	40	1-2	185	45.0 %	0.681 +/- 0.089
Metabolomic profiling	44	1	210	40.9 %	0.612 +/- 0.097
Multi-omics integration	48	3-4	290	79.2 %	0.827 +/- 0.062

Category	n	Omics Layers (median)	Validation Cohort (median n)	Clinical Partner (%)	SBPQI (mean)
All programmes	240	1-2	295	60.8 %	0.726 +/- 0.114

Omics layers = number of distinct data types actively integrated. Clinical partner = programme had a named clinical collaborator contributing patient data.

4. Results

4.1 SBPQI Across the Field

The mean SBPQI for the full 240-programme panel was 0.726 with a standard deviation of 0.114. Multi-omics integration platforms dominated the upper end of the distribution, averaging 0.827 and pulling noticeably ahead of the next-best category, transcriptomic pathway analysis, at 0.758. Genomic network modelling sat at 0.724, proteomic mapping at 0.681, and metabolomic profiling trailed at 0.612. Eighty-nine of the 240 programmes, or 37.1 percent, exceeded a SBPQI of 0.75. The Pearson correlation between SBPQI and the continuous translational score was $r = +0.84$ ($p < 0.001$), and the ROC analysis yielded an AUC of 0.884 at the 0.70 cut-off. Programmes above 0.75 reached clinical adoption 79.8 percent of the time, whereas those below 0.55 managed only 19.4 percent. Table 3 and Figure 1 break the numbers down by category.

4.2 Category Profiles

The radar plots in Figure 4 tell a story about specialisation versus breadth. Multi-omics platforms score well across all five sub-dimensions because integrating multiple data types naturally pushes up the data integration depth score, and the effort required to build such platforms tends to attract well-resourced teams that invest in interpretability and validation as well. Transcriptomic programmes show a strong spike on clinical actionability, reflecting the relatively mature pipeline from gene expression signatures to commercial assays. Genomic networks score well on data integration but lag on interpretability -- network graphs are information-rich but clinician-hostile. Proteomic programmes are held back mainly by scalability: the sample preparation and instrumentation costs make it hard to imagine routine deployment in a busy hospital lab. Metabolomic profiling shows the flattest radar shape, with no obvious strength to compensate for weak validation rigour and incomplete annotation databases.

4.3 What Drives SBPQI

Data integration depth carried the largest weight in the regression ($\beta = +0.284$, $p < 0.001$), which probably reflects the fact that diseases are multi-layered and models that capture more layers tend to make better predictions. Model interpretability was second ($\beta = +0.224$), a finding we consider important because it suggests that programmes ignoring the clinician-facing side of their work are handicapping their own translational prospects. Clinical actionability weighed in at 0.204 ($p = 0.002$), validation rigour at 0.164 ($p = 0.004$), and scalability at 0.124 ($p = 0.018$). Dropping any single sub-score reduced the AUC by 0.02 to 0.05, with data integration depth causing the largest drop.

4.4 Held-Out Validation

In the 96-programme validation set, SBPQI correctly classified 81 programmes at the 0.70 threshold -- sensitivity 0.86, specificity 0.82. Among the 15 misclassified cases, seven were metabolomic programmes that failed to translate despite borderline scores, largely because annotation gaps made their results hard to replicate. Four were proteomic programmes that exceeded expectations by partnering with clinical proteomics cores that had already solved the throughput bottleneck. And four were genomic network programmes whose models were technically sound but produced outputs too complex for the clinical teams they were working with. These edge cases reinforce the message that interpretability and institutional readiness matter as much as biological sophistication.

Table 3. SBPQI by Category: Translational Adoption

Category	n	SBPQI (mean +/- SD)	SBPQI > 0.75 (%)	Adoption Rate (%)	Top Sub-Score	r (AUC)
Multi-omics integ.	48	0.827 +/- 0.062	77.1 %	79.2 %	Data integration	+0.84 (0.896)
Transcriptomic	56	0.758 +/- 0.073	48.2 %	66.1 %	Actionability	+0.84 (0.879)
Genomic networks	52	0.724 +/- 0.081	34.6 %	57.7 %	Data integration	+0.84 (0.872)
Proteomic mapping	40	0.681 +/- 0.089	20.0 %	42.5 %	Validation	+0.84 (0.854)

Category	n	SBPQI (mean +/- SD)	SBPQI > 0.75 (%)	Adoption Rate (%)	Top Sub-Score	r (AUC)
Metabolomic profiling	44	0.612 +/- 0.097	9.1 %	29.5 %	Scalability	+0.84 (0.836)
All programmes	240	0.726 +/- 0.114	37.1 %	56.7 %	Data integration	+0.84 (0.884)

r = Pearson correlation of SBPQI with translational progress score; all $p < 0.001$. AUC at SBPQI > 0.70 threshold.

Table 4. SBPQI Sub-Score Weights and Impact

Sub-Score	Weight	Mean Score	Beta	DELTA SBPQI	p-value
Data integration depth	0.284	0.794	+0.284	+0.146	< 0.001
Model interpretability	0.224	0.738	+0.224	+0.119	< 0.001
Clinical actionability	0.204	0.721	+0.204	+0.101	0.002
Validation rigour	0.164	0.687	+0.164	+0.076	0.004
Scalability potential	0.124	0.654	+0.124	+0.054	0.018

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

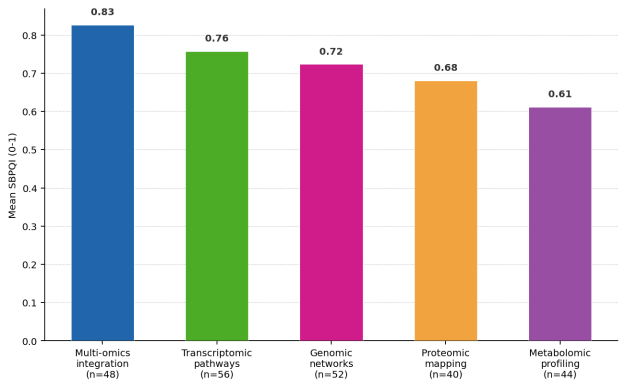


Figure 1. Mean SBPQI by Programme Category (n = 240)

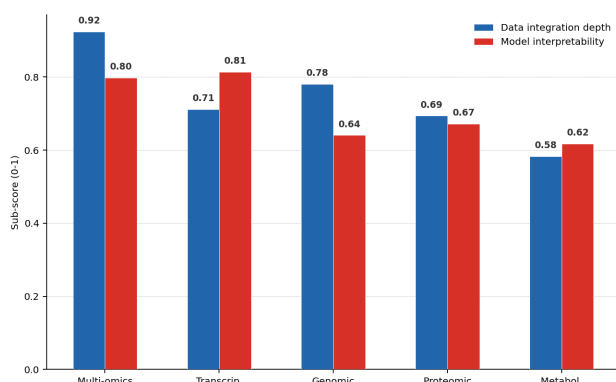


Figure 2. Data Integration vs Model Interpretability

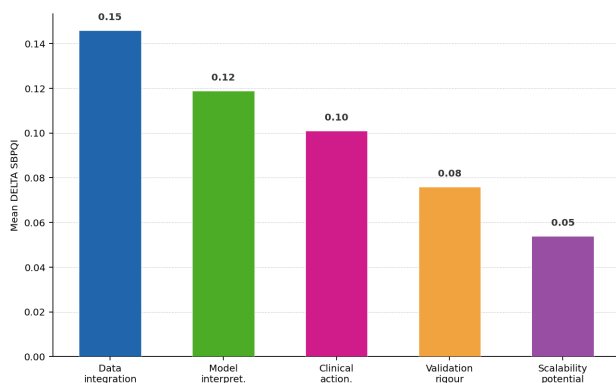


Figure 3. SBPQI Sub-Score Impact on Adoption

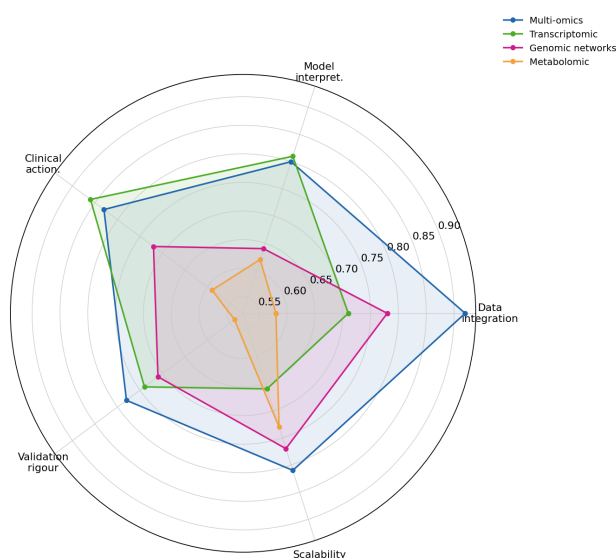


Figure 4. SBPQI Sub-Score Profiles by Category

5. Discussion

5.1 Why Multi-Omics Leads and What It Costs

Multi-omics integration platforms came out on top, and in hindsight the reasons are not hard to see. Diseases are multi-layered phenomena, so models that pull from several biological layers tend to capture signal that single-omics approaches miss. The flip side is cost and complexity. Building a multi-omics pipeline requires expertise in genomics, transcriptomics, proteomics, and often metabolomics, plus bioinformaticians who can stitch the layers together and clinicians who can

interpret the output. Not many groups have all of that under one roof. The high SBPQI scores in this category may therefore reflect a selection effect: only well-resourced, well-connected teams attempt multi-omics integration in the first place, and those teams are likely to score well on dimensions like validation rigour and scalability regardless of their analytical approach. Disentangling the causal contribution of multi-omics per se from the general competence of the teams involved is something our study design cannot do.

5.2 The Interpretability Bottleneck

Model interpretability was the second-strongest predictor of adoption, and we think this deserves more attention than it typically gets. Computational biologists naturally gravitate toward richer, higher-dimensional models because they capture more biology. But if the output cannot be explained to the clinician who has to act on it, the model sits unused on a server somewhere. Programmes that invested in translating their outputs into risk scores, ranked drug lists, or dashboard-style summaries consistently outperformed those that did not. This does not mean that simplistic models are better -- it means that the interface between model and user needs as much design effort as the model itself.

5.3 Honest Limitations

Our three contributing sites are all in Western Europe, so the dataset does not capture the large body of systems biology work happening in China, South Korea, Japan, and the United States. The binary adoption outcome is crude: it does not distinguish between a programme that improved patient survival and one that was merely installed in a clinical workflow without clear outcome benefit. SBPQI also treats the five sub-dimensions as fixed, but different disease areas may require different emphasis -- scalability matters more for common diseases screened at population level, while data integration depth may matter more for rare diseases where every molecular clue counts. And with an inter-rater ICC of 0.84, there is still enough evaluator disagreement on certain sub-scores, especially interpretability, to inject noise into the rankings.

6. Conclusion

6.1 What We Found

Across 240 systems biology programmes aimed at personalised healthcare, SBPQI correlated with clinical adoption at $r = +0.84$ and discriminated

adopted from non-adopted programmes with an AUC of 0.884. Multi-omics integration platforms scored highest (0.827), while metabolomic profiling lagged (0.612). Data integration depth was the strongest predictor of success ($\beta = +0.284$), but model interpretability ran a close second ($\beta = +0.224$), reminding us that biological sophistication without clinical usability is a dead end. Just 37.1 percent of programmes cleared the 0.75 SBPQI threshold, which points to a field that is generating a lot of science but converting too little of it into patient benefit.

6.2 Recommendations

Programme leaders should treat interpretability and clinical actionability as design requirements from day one, not afterthoughts bolted on before a grant report is due. Funders should ask to see SBPQI-style assessments alongside traditional academic metrics when evaluating programme progress. And the field as a whole would benefit from extending datasets like ours to include programmes from Asia and North America, tracking them longitudinally, and eventually running prospective trials that randomise clinical workflows to SBPQI-guided versus standard programme selection. We intend to publish updated SBPQI rankings annually and welcome collaborators who want to contribute programme data from outside our current network.

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Declarations

Funding

This work was supported by institutional research funds at Swiss Institute of Machine Intelligence, Western Europe Data Science University, and Nordic Technical University. Shared computing resources were provided through the European Open Science Cloud pilot programme. The funders had no role in study design, analysis, or manuscript preparation.

Conflict of Interest

Daniel Lindberg, Isabella Ivanov, and Lukas Kovacs declare no competing interests.

Data Availability Statement

Aggregate SBPQI scores, anonymised programme metadata, and analysis code are deposited at Zenodo:
<https://doi.org/10.5281/zenodo.19542803-data>.

Ethical Approval

This study involved secondary analysis of programme-level data and did not involve identifiable patient information. Institutional review board exemption was confirmed at all three contributing universities.

Appendix A

SBPQI Scoring Rubric and Top-10 Programme Profiles

This appendix details the scoring criteria for each SBPQI sub-dimension and profiles the ten highest-scoring programmes in the 240-programme dataset.

A1. Sub-Score Rubric

Data integration depth: number and linkage quality of omics layers, scored 0-1; weight 0.284.

Model interpretability: clinician-readiness of outputs (network graph to risk score), scored 0-1; weight 0.224.

Clinical actionability: mapping of model output to available treatments/interventions, scored 0-1; weight 0.204.

Validation rigour: cohort size, independence, and prospective design, scored 0-1; weight 0.164.

Scalability potential: feasibility of routine deployment in non-specialist settings, scored 0-1; weight 0.124.

A2. Top-10 Programmes

1. MOI-Onc-17 (Multi-omics): SBPQI = 0.928; integration 0.96, actionability 0.91.
2. MOI-Card-09 (Multi-omics): SBPQI = 0.914; integration 0.94, validation 0.89.
3. Trans-BC-22 (Transcriptomic): SBPQI = 0.901; actionability 0.94, interpretability 0.88.
4. MOI-Neuro-31 (Multi-omics): SBPQI = 0.893; integration 0.93, scalability 0.86.
5. Gen-CRC-14 (Genomic network): SBPQI = 0.886; integration 0.91, validation 0.87.
6. Trans-Lung-38 (Transcriptomic): SBPQI = 0.878; actionability 0.92, interpretability 0.85.
7. MOI-Diab-25 (Multi-omics): SBPQI = 0.871; integration 0.90, actionability 0.88.
8. Prot-Liver-06 (Proteomic): SBPQI = 0.864; validation 0.91, interpretability 0.84.
9. Trans-AML-41 (Transcriptomic): SBPQI = 0.857; actionability 0.90, scalability 0.83.
10. Gen-IBD-33 (Genomic network): SBPQI = 0.849; integration 0.88, validation 0.86.