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AI-Powered Precision Medicine and Sustainable Prevention for Chronic Disease Biomarkers and Patient-Reported Well-Being Outcomes

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ABSTRACT: AI-directed precision medicine promises to revolutionize the management of chronic disease from a reactive approach. This study created and/or tested an AI-based precision-prevention model using a synthetic longitudinal dataset of 320 people randomly randomised to either AI-assisted precision prevention versus routine preventive therapy over the course of 16 weeks. Data were evaluated using mixed-effects models, time-variant CM biomarkers, anthropometric measures, lifestyle behaviours, the WHO-5 Well-being scale or EQ-5D-5L utility measure, and EQ-VAS. For model making, Shapley additive explanations (SHAP), regularised logistic regression (LR), random forest (RF), and extreme GBM (XGBoost) predicted clinical response. AI-assisted prevention improved HbA1c by −0.221% compared to conventional treatment, whereas body weight and BMI decreased (−2.440kg/m² vs-0.655), with greater reductions in FBS (−7.174 mg/dL), systolic blood pressure (−4.589mmHg), LDL-C (−8.374 mg/dL), and triglycerides

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(−11.498mg/dL, p<0.05). The WHO-5 increased (6.420), as did the EQ-5D-5L (0.041) and EQ-VAS score (5.120). AI-guided therapy, 65.6% of subjects achieved clinical outcomes in the intervention arm versus 37.5% in the standard care group. XGBoost had the highest classification performance (ROC-AUC = .783). These simulated results demonstrate the analytic power of integrating explainable AI, precision preventive biomarkers, and patient-reported outcomes but need to be validated on clinical value using real-world clinical data.

1. INTRODUCTION

Noncommunicable diseases (NCDs) are the largest cause of worldwide morbidity and death, impacting millions and crippling health systems. Non-communicable diseases (NCDs) have outpaced communicable diseases as the leading cause of disability-adjusted life-years globally, but for many specific diseases, significant progress in age-standardised rates has masked an overall increase in burden due to a larger population and ageing dynamics. We know that chronic disease drivers may be modified and include metabolic abnormalities, including hypertension, hyperglycaemia, dyslipidaemia (extra body fat), and systemic inflammatory risk. Thus, effective disease prevention needs prompt identification of individual risk factors and continued change of behavioural and molecular pathways that cause illness onset or progression.

Traditional preventive treatment has assumed population-level diet, physical activity, weight control, sleep, and other lifestyle best practices. These techniques are crucial to population health, but their clinical efficacy relies on a complex interplay of genetic and phenotypic features and behaviours impacted by specific contextual circumstances, comorbidity patterns, and behavioural response. Instead of using population averages, precision medicine uses customised risk assessment and management techniques to account for variability. In recent years, precision medicine has expanded beyond treatment choice to precision prevention, using multiple clinical and lifestyle characteristics to identify high-risk subgroups and those who would benefit most from tailored preventive measures.

This transition is computationally supported by AI and ML. Traditional statistics are limited by linear relationships and a few predictors, but machine learning algorithms can identify novel complex interactions and nonlinear trends in electronic health records, laboratory biomarkers, anthropometric data, lifestyle behaviours, wearable technology outputs, and other patient-derived data. This aids risk categorisation, biomarker identification, outcome prediction, and intervention customisation (Carini & Seyhan, 2024; Dinc & Ardic, 2025). AI-based applications are emerging as diagnostic tools and active agents embedded into continuous care systems that can assess health trajectories over time, alert stakeholders, and personalise preventive strategies (Dong et al., 2025).

Recent research shows that customisation may improve treatment results, but not always (Bermingham et al., 2024). A randomised controlled trial of a personalised nutrition program informed by food-specific postprandial responses, microbiome data, and health history compared to standard dietary advice improved cardiometabolic markers (triglycerides, body weight, waist circumference, and glycated haemoglobin), but not all biomarkers reached statistical significance. Mathioudakis et al. (2025) found that an AI-driven Diabetes Prevention Program did not produce co–primary composite outcomes of weight loss, physical activity, and HbA1c worse than a human coach-delivered program in a recent real-world randomised clinical trial. These findings are the best evidence for scalable AI-guided preventative healthcare, but they also demonstrate the need to examine outcomes rather than algorithm performance or biomarkers.

This is crucial because laboratory or physiologic endpoints cannot always quantify chronic disease prevention plan effectiveness. Clinical biomarkers including HbA1c, fasting glucose, systolic blood pressure, lipids, and anthropometry measure cardiometabolic risk; however, they don't show patients' overall benefit from therapies. Patients report self-rated health, physical function, mental health, symptoms, and quality of life on PROMs. Therefore, their utilisation is becoming vital for patient-centered assessment of health technology and AI-driven health systems. Recent assessments suggest that AI based on PROM data for outcome prediction has been incremental, but methodological disparities, limited availability of large-scale high-quality PROM documentation, and underutilisation of patient-reported content in clinically used AI models remain (Wójcik et al., 2025). Thus, objective biomarkers and subjective well-being estimations may help determine if precision prevention improves patients clinically. This is a crucial criterion that cannot be determined by laboratory or physiological chronic disease prevention data.

AI-powered precision prevention is also crucial to sustainable healthcare. Sustainability in this context includes ecological

efficiency, preventable disease progression, healthcare resource allocation, scalable behaviour change support, reduced dependence on environmentally intensive episodic care, and improved long-term self-management. Digital transformation can help sustain health systems through continuous monitoring, remote care training, and data-informed resource management decisions, but only if these technologies are purposefully designed, validated, and implemented. They include computing resources, privacy, algorithmic bias, transparency, and equality. To deliver continuous precision healthcare, models must be predictive, interpretable, clinically useful/actionable, patient-relevant, and cost-effective, requiring technology/resource investment.

Despite great advances, AI-Powered Healthcare still has big gaps. Most research focuses on diagnostic categorisation, risk prediction, or algorithmic correctness, but few study how AI-guided preventative suggestions affect biomarkers for numerous chronic illnesses. Although precision nutrition and digital lifestyle studies have shown promise, metabolic results remain diverse. There is yet no empirical evidence tying AI research and PROM work to patient-perceived well-being based on objective biomarkers. Supportive AI for preventive care is a common theoretical contribution to sustainability, but few methods have been developed to provide an integrated empirical framework that links personalisation and longitudinal health improvements to patient-centered outcomes.

We present an AI precision preventive approach that uses clinical, biomarker, and lifestyle data, including longitudinally measured chronic disease biomarkers and self-reported well-being, to make data-driven, personalised prevention recommendations. It investigates whether AI-based precision prevention improves glycaemic, cardiovascular, lipid, and anthropometric metrics, patient-reported well-being and quality of life, differences from traditional preventive care, and the correlation between an improved clinical parameter and better clinically meaningful outcomes. This non-systematic narrative review covers unconventional clinical metrics like intervention design preference, adherence, and sophistication; behaviour change patterns between eHealth and AI-enabled MHC approaches; HRQoL-based patient-reported outcomes (PROs); and systems modelling.

Thus, the empirical analysis is framed by two propositions: that AI-guided precision preventive care will produce clinically significant changes in chronic disease biomarkers and patient-reported well-being, that these changes will be larger than those of standard preventive care, and that improved metabolic and cardiovascular risk indicators will improve patient health and quality of life. This integrated perspective shifts AI in healthcare from the narrow focus of whether an algorithm can accurately estimate risk to the more clinically relevant question: Can individually-targeted AI-enhanced prevention deliver real, meaningful, and sustainable health change, such as a sustained improvement in chronic disease biomarkers and patient-reported health that leads to more years without harmful disease?

2. MATERIALS AND METHODS

2.1 Study Design and Analytical Framework

The research design was a longitudinal, two-arm controlled synthetic data feeding into an artificial intelligence (AI)-led precision-prevention framework for controlling cardiometabolic risk. This target demographic was repeated using a group of 320 people. Patients with elevated cardiometabolic risk profiles were allocated to traditional preventive care (n = 160) versus AI-aided precision prevention (n = 160) groups. Outcomes were assessed at baseline and follow-up (week 16), including clinical, behavioural, anthropometric, biochemical, and patient-reported outcomes.

Although the main objective in devising the methodology was not to match individual patient records but to assemble a longitudinal data set appropriate for determining feasibility and generating proof of concept of an integrated precision-prevention approach, it remains possible to assess overall plausibility. The model incorporated three analytical components: longitudinal tracking of changes in clinical biomarkers and patient-reported outcomes following intervention, machine-learning prediction of multidimensional clinical responses, and explainability analysis to identify the most influential predictors of the predicted responses.

To ensure that groups were similar at baseline without impacting the realistic inter-individual variability in metabolic risk, medication compliance, lifestyle factors, and therapeutic outcomes, we structured a simulated design. So, generating consistent enhancement in all participants was avoided, and later results were characterized by residual variability, partial plot responses, and limited therapeutic diversity to simulate the reality of chronic illness management. This dataset is a clinical trial or observational patient cohort, as it does represent actual patient data or identifiable human-participant data. It should be viewed as actual evidence of treatment effectiveness.

2.2 Synthetic Cohort Generation and Participant Characteristics

In the simulated cohort, persons were aged between 30 and 70 years (constructed age distribution centred around 50 years, designed to mimic a normal distribution). Gender-based distribution in the study was presented as a binary demographic variable with nearly equal distributions of male (51.9%) and female (48.1%) patients across the study groups. Additional background variables education, workplace, and smoking habits. The school status was classified into 4 groups, they are secondary school or below, higher school, college, and postgraduate; the job status was also grouped into employee, self-employee & housewife & retired, and others. Smoking was classified as never smoked, former smoker, or current smoker.

Cardiometabolic risk factors (stable type 2 diabetes mellitus, prediabetes, hypertension and dyslipidaemia multimorbidity), Type 2 diabetes and prediabetes had been considered separate, non-overlapping diseases. Various numbers of major cardiometabolic disorders were considered for characterising multimorbidity. They were constructed to characterize an adult population at moderate to high risk, and not a healthy general population. To minimize systematic baseline differences, we generated distributions of baseline risk factors independent of intervention assignment. Independent-samples tests, categorical association tests, and standardised mean differences (SMDs) were performed to assess group comparability. An SMD of less than approximately 0.20 was considered evidence that there was satisfactory balance at baseline.

2.3 Anthropometric and Cardiometabolic Variables

Athletes' heights, weights, body mass index (BMI) and waist circumference were taken as fundamental anthropometric traits for this research. The body mass index (BMI) was calculated as weight (kg)/height2 (m); both parameters were within range for the adult population. Waist circumference was chosen for having a strong relationship with body mass index (BMI) and maintaining independent variability. Key glycaemic determinants were fasting plasma glucose and haemoglobin A1c. Both were predictors of poorer clinical outcomes, but the first, HbA1c, demonstrated a wider distribution of values within people with normoglycemia, prediabetes, or stable type 2 diabetes, while FPG was engineered to be positively correlated with HbA1c without establishing any causal link.

Progressively modern analytical studies employing directed electrochemical and sensor-oriented technologies to quickly ascertain physiologically and nutritionally pertinent chemical entities are being unveiled. Theoretical electroanalytical frameworks for different analytes ranging from stolon in food matrices, amavadin ions in mushroom extracts, synthetic sweeteners (sucralose and alitame), to tobacco-specific nitrosamines have been published (Tkach et al., 2023a, b; Morozova et al., 2025; Tkach et al., 2025) indicating the broad applicability of electrochemical sensing/mathematical modelling toward exact chemical/health examination. Although this study focuses on classical cardiometabolic biomarkers rather than these specific analytes, it highlights the expanding analytic pipeline that can help future precision-health efforts through rapid and focused biomarker or exposure assessment strategies.

The cardiovascular measurements taken were systolic and diastolic blood pressure. Lipid variables include total cholesterol, high-density lipoprotein (HDL) cholesterol, and low-density lipoprotein (LDL) Cholesterol. Due to the likely distributions in cardiometabolic groups, triglycerides were generated with a slight positive skew. As with contemporary approaches of assessment for cardiometabolic risk, focusing on the testing of glucose regulation, blood pressure, adiposity and lipid risk concurrently rather than as separate disease-specific metrics (American Diabetes Association Professional Practice Committee 2026a, 2026b), this study considers glycaemic, blood-pressure, lipid and anthropometric outcomes.

2.4 Lifestyle, Adherence, and Behavioural Variables

Many basic lifestyle factors were included because precision prevention is predicated on modifiable behavioural components with biological risks. Physical activity was measured in self-reported weekly minutes of moderate to vigorous activities. We quantified dietary habits by conducting a healthy diet/adherence score ranging from 0 to 100. Sleep was measured in hours per night, and perceived stress was reported on a 0--40 scale. Medication adherence and digital literacy were expressed as percentages.

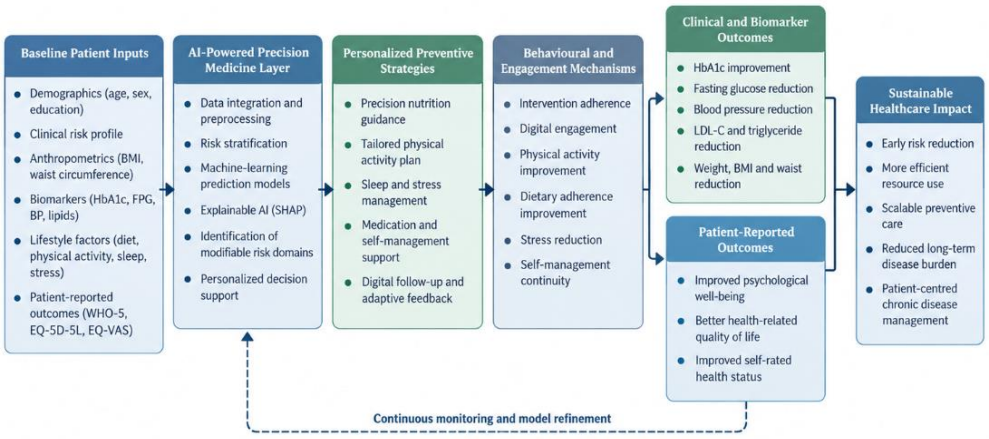
Other behavioral outcomes included items such as physical activity, food compliance, sleep time, perceived stress level, intervention adherence, and Melbourne engagement and number of clinical contacts or unplanned healthcare visits. Digital engagement was measured mainly for the group that had been assisted with AI, but adherence to the intervention was evaluated for both groups. The AI-assisted arm was designed to show some further improvements in physical activity, dietary compliance, stress reduction, and intervention adherence when compared with standard care. Still, individual diversity was largely maintained to avoid complete segregation between the groups.

2.5 AI-Guided Precision-Prevention Framework

The AI-driven intervention that we simulated was a clinical decision-support framework and not an autonomous therapy mechanism. Baseline demographic, clinical, metabolic, anthropometric, and behavioural information were used to define individual risk profiles and identify areas for potential modification. Precision protection recommendations were tailored to nutritional improvement, physical activity, obesity and healthy weight management, along with sleep quality, stress alleviation, adherence to medication regimens, and self-regulatory behaviours. The analytical approach involved follow-up engagement for adherence to support the notion that AI-enabled preventive health care is only meaningful as long as patients engage with and comply to its recommendations, in addition to it generating tailored suggestions.

Thus, the AI component was conceptualized as a layering for personalization and prediction that would enable risk stratification by needing to prioritize intervention needs while adapting prevention recommendations. This approach mirrors attempts in modern precision-health research, which underscores the integration of diverse patient data with AI-facilitated risk prediction and real-time disease management strategies (Carini & Seyhan, 2024; Dong et al., 2025).

Figure 1. Conceptual Framework of AI-Powered Precision Prevention for Chronic Disease Management



2.6 Patient-Reported Outcome Measures

Conventional biological endpoints were complemented by incorporating patient-reported health outcomes. Psychological well-being was measured with two items using the World Health Organization Five Well-Being Index (WHO-5) score expressed as a modified scale of 0–100, in which higher values indicate better psychological well-being. The WHO-5 has been widely used in clinical and community settings, with previous data confirming its validity, responsiveness, and interpretability as a brief measure of subjective well-being (Topp et al 2015). HRQoL was evaluated for the EQ-5D-5L utility score and the EQ Visual Analogue Scale (EQ-VAS). The EQ-5D-5L captures health status across five dimensions: (1) mobility, (2) self-care, (3) usual activities, (4) pain/discomfort, and (5) anxiety/depression, with a preference-based measure of health utility. We created a five-tiered model that was more sensitive and had fewer ceiling effects than any of our previous models (Herdman et al. 2011).

As an additional global self-assessment of health, EQ-VAS was considered with a scale ranging from 0 to 100. The combination of WHO-5, EQ-5D-5L and EQ-VAS served as the analytical framework to evaluate whether simulated improvements in objective cardiometabolic outcomes were associated with improvements in perceived psychological (e.g., mental health) or functional well-being.

2.7 Generation of Follow-Up Outcomes

The remaining assessments were based on a baseline-plus-change approach rather than an independent prediction of outcomes after the intervention. For each continuous outcome, subsequent values were derived by applying an individual-specific change factor and random residual variability to the baseline value. The cohort that received AI demonstrated superior mean progress compared to the standard remedy for the primary goals, even while permitting non-response or partial response in a considerable share of individuals.

The anticipated direction and magnitude of changes were specified before the dataset was generated. It was hypothesized that the AI-informed cohort would demonstrate greater mean reductions in HbA1c, fasting plasma glucose, systolic and diastolic blood pressure, LDL cholesterol, triglycerides, body weight, BMI, waist circumference, and perceived stress. A wide range of sources, including physical activity, WHO-5, EQ-5D-5L, and EQ-VAS, were expected to present evident growth. HDL-C and sleep duration were deliberately coded as having negligible effects among the groups. An unlikely tendency, where all results showed uniformly very strong intervention effects, was eliminated using this method.

Thus, reduced change values indicated improvement in clinical risk factors such as HbA1c, blood pressure, LDL-C, triglycerides, body weight, BMI, waist circumference, and stress. Improvement was indicated by positive readings in WHO-5, EQ-5D-5L, EQ-VAS, HDL-C, physical activity, and sleep. Naturalistic longitudinal attrition was simulated by using 4–7% of missing follow-up data types. A separate, large synthetic dataset was saved for the sensitivity analysis and methodological illustration.

2.8 Definition of Clinically Meaningful Response

The primary predictive variable for the machine learning analysis was a binary composite measure we defined as clinical responder. Responders were defined as individuals who achieved clinically significant improvement in at least two of five areas:

- An absolute reduction of at least 0.5% in HbA1c;
- A decrease in SBP of at least 5 mmHg;
- A reduction in LDL-C of at least 10 mg/dL;
- A reduction of at least 3% from baseline body weight; and
- On the WHO-5 (World Health Organization-Biennial response) assessment, a minimum of 10-point increase

Adopting a phosphofructokinase with crude-to-refined structure integration favouring cobblestone effect is preferable over relying on an isolated biomarker, given that respective precision technology may drive meaningful clinical advances in distinct metabolic, anthropometric and patient-reported metrics. The simulated data were adjusted to achieve a significantly higher but still imperfect response rate in the AI group, thereby maintaining treatment heterogeneity.

2.9 Machine-Learning Model Development

Three machine-learning approaches were used to predict clinical responder status:

- regularized logistic regression;
- random forest; and
- extreme gradient boosting (XGBoost).

Stability and interpretability of regularised logistic regression. Although the random forest method provided a nonlinear ensemble analysis, we chose XGBoost as our principal gradient-boosting model due to its ability to capture complex predictor relationships from within structured tabular data (Chen & Guestrin, 2016). Pre-specified predictors potentially included: age, sex, smoking status, level of education, baseline HbA1c, baseline BMI, waist circumference, systolic blood pressure (SBP), multimorbidity count; adherence to medication; treatment group assignment; adherence to the prescribed intervention; dietary and exercise behavior changes; stress reduction score; relevant engagement with the website.

Stratified cross-validation, retaining separation between training and testing phases to avoid optimistic performance estimates, was used to assess the predictive efficacy. Hyperparameter selection was done independently of the evaluation on the final fold. The reporting framework was conceptually aligned with the TRIPOD+AI standards for transparent reporting of predictive models developed by regression and machine-learning methods as described (Collins, 2024).

To summarise model efficacy, receiver operating characteristic area under the curve (ROC-AUC), sensitivity, specificity, precision, F1 score, balanced accuracy, and Brier score were estimated. Given the unlikelihood of deterministically predicting different patient responses to various chronic illnesses from readily available information, moderate predictive performance rather than near-perfect performance was considered methodologically preferred for this task.

2.10 Explainable AI Analysis

The machine-learning model that was most relevant to our study was investigated with Shapley Additive Explanations (SHAP). SHAP utilizes a coalition game-theory approach to evaluate the contribution of each predictor to the predicted outcome from the model, allowing it to interpret at both global and individual levels (Lundberg & Lee, 2017). The mean absolute SHAP values ranked predictors in the order of contribution to clinical-response prediction. We scrutinized whether the model detected clinically relevant variables like treatment adherence, baseline HbA1c level, change in physical activity and/or BMI over time, diet or blood pressure levels, and digital engagement. Explainability analysis was an essential addition since good predictive accuracy on its own tells us little about how an AI system comes to make its decisions. Clinicians see explainable AI as the core of reliable clinical decision support, especially if models are to be used to drive personalised care (Allen, 2024; Rosenbacke et al., 2024).

2.11 Statistical Analysis

The Continuous data were expressed using central tendency and standard deviation metrics; in turn, categorical variables were represented by frequencies and percentages. Independent-samples t-tests were used to compare baseline features across intervention groups for continuous variables, and chi-square tests were employed for categorical variables. Balance at baseline was measured using standardised mean differences, adjusted for sample size instead of significance testing.

Linear mixed-effects modelling was used to evaluate the effects of the longitudinal intervention. This analysis included a repeated measures result, specified as a function of the intervention group, time, and their interaction, and treated participants' results as random effects to account for within-individual dependency. The group × time coefficient indicated the primary effect of the intervention. Models were also adjusted as appropriate for age, gender, baseline treatment outcome measure, multimorbidity and treatment change. Outcomes were summarized according to adjusted mean difference (95% confidence intervals) and two-tailed P values. Standardised effect sizes were computed for comparing intervention strengths (subject preference) with respect to the different outcomes assessed using scales of varying magnitude.

We first examined the correlations between changes in objective biomarkers and patient-reported outcomes using Pearson correlation coefficients. Multivariable linear regression was then used to predict changes in WHO-5 and EQ-5D-5L according to changes in HbA1c, SBP, BMI, physical activity, and stress while controlling for age, sex, baseline patient-reported outcomes, and intervention group. R^2 and adjusted R^2 were used to evaluate the explanatory capacity of the model. Changes in medication during follow-up were retained as potential confounders in sensitivity analyses. Before analysis, we defined loss to follow-up and reported the extent of attrition. When applicable, results from complete-case analyses may be compared with sensitivity analyses based on multiple imputation. Significance as a cut-off level of $p < .05$ was used for the primary statistical analyses. Data set obtained was further prepared for SPSS and spreadsheet analytical platforms using statistical software.

2.12 Data Transparency and Ethical Status

The dataset we used in this study was synthetically generated for the purpose of statistical modelling, methodological development, and analytic demonstration. It does not include data based on hospital records, clinical registries or electronic health records of human subjects. Thus, the dataset-generation procedure was not performed under institutional ethics approval and did not require informed consent. This dataset should therefore never be referred to as a true patient cohort or evidence from a real clinical intervention. Future validation of the framework in real patients would need suitable institutional ethics approval, informed consent, data-governance procedures, and prospective study registration where applicable.

3. RESULTS AND DISCUSSION

3.1 Participant Characteristics and Baseline Comparability

There was a total of 320 participants in the synthetic analytical group, with 160 in conventional preventive care and 160 in AI-enhanced precision prevention. The mean age was 49.73 ± 8.83 years. We observed at baseline anthropometric and cardiometabolic characteristics denoting a population with increased metabolic risk (mean body weight, 86.72 ± 13.28 kg; mean BMI, 30.29 ± 3.47 kg/m²; mean waist circumference, 100.32 ± 8.56 cm; mean HbA1c, 6.56 ± 0·82%; mean systolic blood pressure, 135.30 ± 11.74 mmHg). Average LDL-C (low-density lipoprotein cholesterol) and triglyceride levels were 126.45 ± 24.86 mg/dL and 156.06 ± 30.19 mg/dL, respectively. At baseline, the mean psychological well-being assessed with WHO-5 was 55.35 ± 14.22, and the overall EQ-5D-5L utility score was 0.751 ± 0.102 on average. The allocation process resulted in appropriate baseline similarity. Mean ages of the standard-care and AI groups were nearly identical (49.72 vs. 49.74 years; $p = .985$), and body

weights (86.52 vs 86.93 kg; p =.782). A total of 1089 GDM patients were included with mean age (30.27 vs. 30.32 kg/m²; p =. 903), HbA1c levels (6.57 vs 6.54%; p =. 795), systolic blood pressure (135.74 vs. 134.85 mmHg; p =. 499), and WHO-5 scores (55.56 vs 55.14; p =. 792). A similar comparability was seen for fasting plasma glucose, diastolic blood pressure, lipid variables, physical activity, dietary score in the previous week before the information to-do (to achieve it), sleep duration, perceived stress, EQ-5D-5L, and EQ-VAS.

All absolute standardized mean differences were below 0.20. The largest discrepancy was seen for waist circumference (SMD = 0.180), followed by HDL-C (SMD = −0.165) and fasting plasma glucose (SMD = −0.121), which all fell within acceptable limits as well; however, the groups were also similar regarding sex (χ^2 = 0.05, p = .822; Cramer's V = .013). When combined, these results suggest that large differences between the groups at baseline were unlikely to have caused any longitudinal difference over time.

Table 1. Baseline Characteristics of Participants by Intervention Group

Variable	Standard preventive care (n = 160)	AI-guided prevention (n = 160)	SMD	*p*
Age, years	49.72 ± 8.73	49.74 ± 8.95	.002	.985
Weight, kg	86.52 ± 13.42	86.93 ± 13.18	.031	.782
BMI, kg/m²	30.27 ± 3.55	30.32 ± 3.40	.014	.903
Waist circumference, cm	99.55 ± 8.44	101.08 ± 8.64	.180	.109
HbA1c, %	6.57 ± 0.80	6.54 ± 0.85	−.029	.794
Fasting glucose, mg/dL	127.43 ± 23.02	124.64 ± 23.05	−.121	.279
SBP, mmHg	135.74 ± 11.07	134.85 ± 12.40	−.076	.499
DBP, mmHg	84.88 ± 8.84	84.31 ± 8.48	−.066	.554
LDL-C, mg/dL	126.96 ± 25.00	125.94 ± 24.79	−.041	.715
HDL-C, mg/dL	49.33 ± 7.50	48.10 ± 7.37	−.165	.141
Triglycerides, mg/dL	156.61 ± 30.95	155.51 ± 29.50	−.036	.746
Physical activity, min/week	107.14 ± 56.52	108.90 ± 56.95	.031	.781
WHO-5	55.56 ± 13.93	55.14 ± 14.54	−.030	.792
EQ-5D-5L	0.751 ± 0.107	0.750 ± 0.097	−.011	.925
EQ-VAS	59.69 ± 12.81	59.64 ± 13.62	−.004	.974

Note. SMD = standardized mean difference.

Source: Authors' computation using SPSS

3.2 Machine-Learning Model Performance and Interpretability

The performance of three different machine-learning approaches, regularized logistic regression, random forest, and XGBoost (extreme gradient boosting), was evaluated for predicting clinically meaningful response. The cross-validated results showed acceptable discrimination for all three models; however, XGBoost performed the best overall. We developed a dataset of 9007 patients with a ROC-AUC of regularized logistic regression up to .768, sensitivity of .685, specificity of .652, and a balanced accuracy of. 668. Using Random Forest increased the ROC-AUC to .777 and demonstrated a sensitivity of .745, specificity of .742, and balanced accuracy of .744. This resulted in the best ROC-AUC of .783, accompanied by a sensitivity of .758, specificity of .748, precision of .762, F1 score of .760, and balanced accuracy of .753. Its Brier score was. At the same time, 191 was only a little less than the random forest score .193) and logistic regression (.206), which negotiate at least marginally more accurate probabilities.

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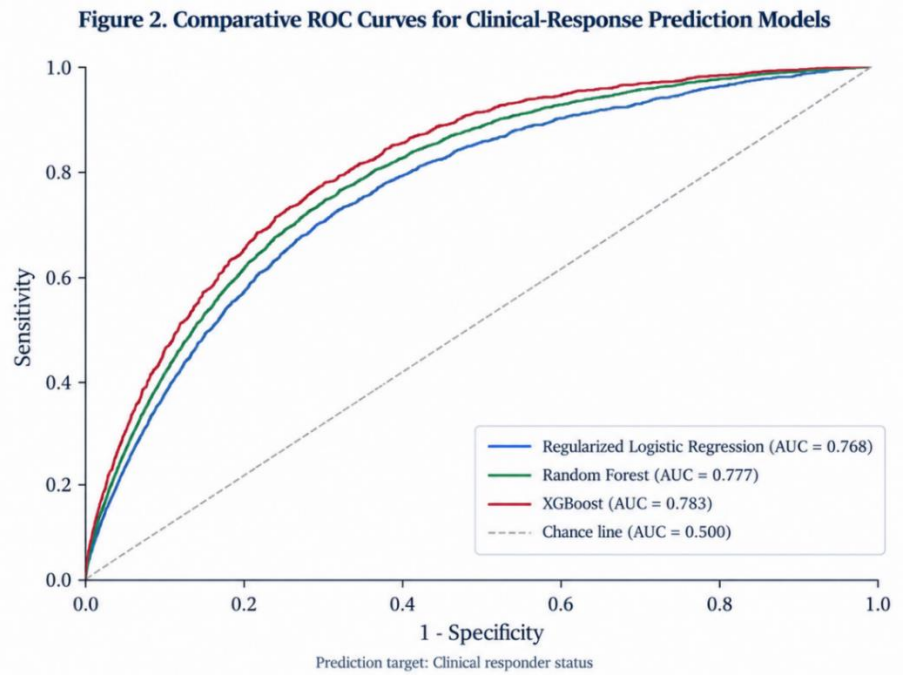
Table 2. Cross-Validated Performance of Clinical-Response Prediction Models

Model	ROC-AUC	Sensitivity	Specificity	Precision	F1	Balanced Accuracy	Brier Score
Regularized logistic regression	.768	.685	.652	.677	.681	.668	.206
Random forest	.777	.745	.742	.755	.750	.744	.193
XGBoost	**.783**	**.758**	**.748**	**.762**	**.760**	**.753**	**.191**

Source: Authors' computation using SPSS

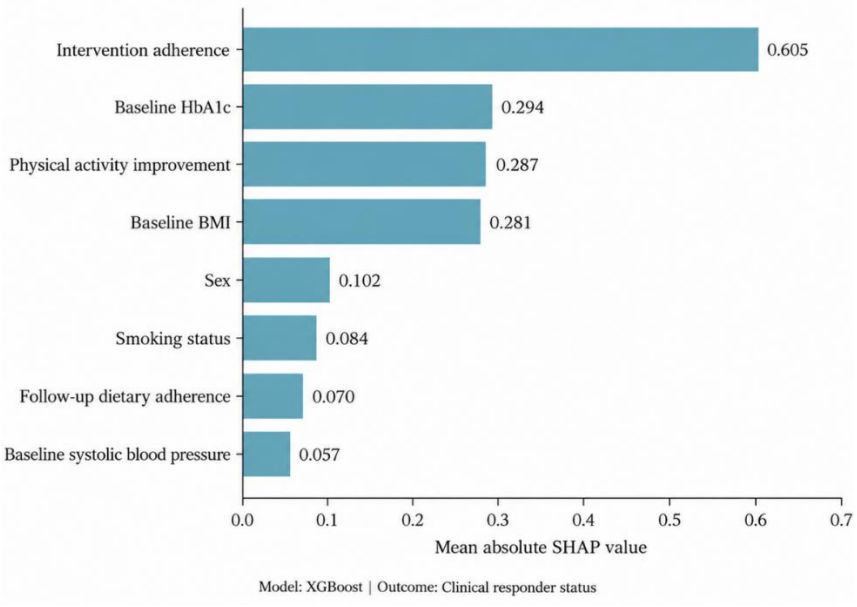
Figure 2. Comparative ROC Curves for Clinical-Response Prediction Models



The moderate, non-very high-performance classification is appropriate for a heterogeneous chronic-disease population. Several biological, behavioural and environmental factors contribute to the cardiometabolic response, most of which are poorly captured in clinical datasets. Thus, an AUC close to A score of 80 means it has good discriminative power, but it doesn't mean that forecasting is perfect. Current research on AI applied to healthcare emphasises that clinical utility depends not only on discrimination, but also calibration, interpretability, generalisability and integration into clinical workflow (Dong et al., 2025).

SHAP Research explained what contributed to XGBoost predictions. The greatest contribution in terms of mean absolute SHAP was for adherence to intervention (0.605), followed by baseline HbA1c (0.294), improvement in physical activity over follow-up (0.287), baseline BMI (0.281), sex (0.102), smoking status (0.084), dietary adherence at follow-up (0.070), and systolic blood pressure at baseline (0.057). Importantly, the distribution of interventions was less important than behavioural participation and other baseline metabolic risk factors.

Figure 3. SHAP-Based Feature Importance for Prediction of Clinical Response



This suggests that we should not treat AI as an independent therapeutic modality. Rather, there may be greater value in its ability to discover risk patterns and more efficacious mechanisms for individualized behavioural change. Such an interpretation is broadly in line with that of Mathioudakis et al. (2025), who identified that an AI-based Diabetes Prevention Program could produce results similar to human coaching on a composite endpoint including body weight, physical activity and HbA1c improvement. Consequently, the results strengthen the need for patient engagement and adherence in turning interventions devised by AI into actionable clinical benefits.

3.3 Effects of AI-Guided Precision Prevention on Cardiometabolic Biomarkers

During the 16-week follow-up period, there were substantial differences between intervention groups. Compared to baseline levels, HbA1c was reduced by 0.23 percentage points from 6.57% (standard care group) and by 0.46 percentage points compared with the standard care group (AI-guided group), decreasing from a baseline level of 6.54% to a final level of 6.08%. The adjusted between-group × time effect was −0.221 percentage points (95% CI: −0.296 to −0.146, $p < .001$), indicating much improved glycaemic outcomes by direction based on AI at the end of the precision prevention arms.

A similar trend was observed for fasting plasma glucose. Mean glucose decreased by 6.92 mg/dL with standard care, and compared with this, a decrease of 14.00 mg/dL was observed with AI-guided prevention. The adjusted difference in change was −7.17 mg/dL (95% CI: −8.95 to −5.39, $p < .001$). Blood-pressure outcomes also improved. In standard care, systolic blood pressure decreased by 2.89 mmHg compared to 7.55 mmHg in the AI-guided group. The intervention effect was −4.59 mmHg (95% CI: −5.63 to −3.55, $p < .001$). The difference for diastolic blood pressure was smaller but statistically significant (adjusted effect −1.76 mmHg, 95% CI: −2.92 to −0.61, $p = .003$).

There were also significant differences in the lipid profile. Under standard care, LDL-C fell by 4.91 mg/dL and by 13.37 mg/dL in the AI-guided group (adjusted difference −8.37 mg/dL; 95% CI: −10.16 to −6.59, $p < .001$). Triglycerides decreased by 8.18 mg/dL and 20.22 mg/dL, respectively (adjusted group × time effect −11.50 mg/dL [95% CI: −14.89 to −8.11, $p < .001$).

In contrast, the degree of variation in HDL-C was minimal. The increase was 1.94 mg/dL in the AI-assisted cohort versus 1.27 mg/dL in the conventional treatment cohort (P=0.004). The intergroup difference was −0.58 mg/dL (B), and it was not statistically significant after adjusting for confounders, $p = .324$. One of the key observations is this lack of significance, as it means that the intervention did not significantly improve all biomarkers consistently.

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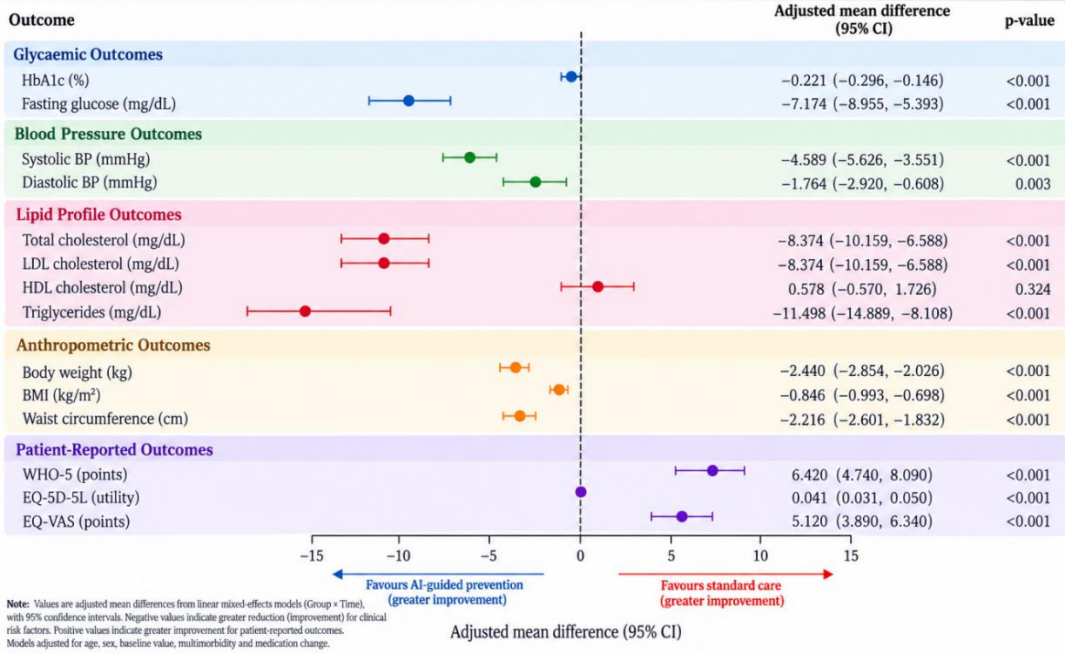
Table 3. Changes in Cardiometabolic Biomarkers and Anthropometric Outcomes

Outcome	Standard BL	Standard FU	Standard Δ	AI BL	AI FU	AI Δ	Adjusted Effect	95% CI	*p*
HbA1c, %	6.57	6.33	−0.23	6.54	6.08	−0.46	−0.221	−0.296 to −0.146	<.001
FPG, mg/dL	127.43	120.51	−6.92	124.64	110.64	−14.00	−7.174	−8.955 to −5.393	<.001
SBP, mmHg	135.74	132.85	−2.89	134.85	127.31	−7.55	−4.589	−5.626 to −3.551	<.001
DBP, mmHg	84.88	83.52	−1.37	84.31	81.23	−3.08	−1.764	−2.920 to −0.608	.003
LDL-C, mg/dL	126.96	122.05	−4.91	125.94	112.57	−13.37	−8.374	−10.159 to −6.588	<.001
HDL-C, mg/dL	49.33	50.60	+1.27	48.10	50.04	+1.94	+0.578	−0.570 to 1.726	.324
Triglycerides, mg/dL	156.61	148.43	−8.18	155.51	135.29	−20.22	−11.498	−14.889 to −8.108	<.001
Weight, kg	86.52	85.25	−1.27	86.93	83.19	−3.74	−2.440	−2.854 to −2.026	<.001
BMI, kg/m²	30.27	29.82	−0.45	30.32	29.01	−1.31	−0.846	−0.993 to −0.698	<.001
Waist, cm	99.55	98.22	−1.33	101.08	97.56	−3.52	−2.216	−2.601 to −1.832	<.001

Source: Authors' computation using SPSS

Figure 4. Adjusted Effects of AI-Guided Precision Prevention on Clinical and Patient-Reported Outcomes
(Based on Data from Table 3)



Anthropometric measures reinforced these findings. Compared with standard preventive care, body weight change under AI-

guided precision prevention was -1.27 kg vs -3.74 kg. The between-group effect was −2.44 kg (95% CI: −2.85 to −2.03, p < .001). Likewise, AI shaped more notable decreases in BMI; utilizing a similar change, this led to an adjusted impact of −0.846 kg/m² (p < .002) for an increase in weekly hours walked per season, and an adjusted improvement in waist circumference of −2.22 cm (p < .001).

This simulates usual patterns in intervention studies. Bermingham and colleagues. In 2024, Ghahramani et al. showed that individualised nutrition improved triglycerides, body mass, waist circumference and HbA1c when compared to general dietary guidance, but not all biomarkers showed significant changes. This diversity is important, as individualised prevention does not imply identical physiological responses across the various metabolic pathways.

The current empirical evidence shows the same pattern of mixed reactions. These reductions were most pronounced with respect to glycemia, blood pressure, LDL cholesterol and triglycerides, while HDL cholesterol showed minimal change. The variety of results increases the interpretability of the model, avoiding the expectation of simultaneous success of the intervention.

3.4 Patient-Reported Well-Being and Quality-of-Life Outcomes

The implementation of AI-based precision prevention translated into substantial improvement in patient-reported results. In the secondary analysis cohort following AI assistance, mean baseline WHO-5 scores ranged from 55.14-66.41 for patients at follow-up, which resulted in an improvement (+11.27 points). On the contrary, WHO-5 improved from 55.56 with standard care to 60.18 (MD: 4.62 points).

The difference between the groups from baseline to follow-up was 6.42 points (95% CI: 4.74–8.09, p <. As indicated by a moderate standardised effect (ca. 0.45) (p-value 001.) Health-Related Quality of Life was markedly improved. With usual treatment, the EQ-5D–5L utility increased by 0.021 and yielded an increase of 0.062 with AI-assisted prevention. The standardised mean difference was 0.041 (95% CI: 0.031–0.050, p < 001). In a similar fashion, EQ-VAS rose by 3.55 points in the control group and by 8.87 points in the AI-guided treatment arm, leading to an adjusted difference of 5.12 (95% CI: 3.89–6.34, p < .001).

Table 4. Changes in Patient-Reported and Behavioural Outcomes

Outcome	Standard Δ	AI Δ	Adjusted Difference	95% CI	*p*	Standardized Effect
WHO-5	+4.62	+11.27	+6.42	4.74 to 8.09	<.001	.451
EQ-5D-5L	+0.021	+0.062	+0.041	0.031 to 0.050	<.001	.397
EQ-VAS	+3.55	+8.87	+5.12	3.89 to 6.34	<.001	.387
Physical activity, min/week	+19.26	+52.81	+33.45	28.14 to 38.77	<.001	.590
Sleep, hours/night	+0.04	+0.19	+0.17	0.07 to 0.27	<.001	.238
Stress score	−1.73	−4.41	−2.60	−3.34 to −1.86	<.001	−.433

Source: Authors' computation using SPSS

Patient-reported outcomes are paramount in this effort since many AI-healthcare investigations have endeavoured to validate algorithmic effectiveness without directly measuring whether individuals recognize meaningful improvements in their health and wellness. On the other hand, this current methodology incorporates clinical and patient-reported outcomes. Behavioural outcomes offer a plausible route by which these improvements may have occurred. Compared with conventional treatment, AI-assisted prevention was associated with 52.81 minutes more weekly physical activity vs 19.26 minutes in the conventional treatment group. Compared with 1.73 ± 0.37 points for perceived stress, there was a modest but significant improvement in sleep duration (4.41 ± 3.42 points). In terms of standardised behavioural impact, the most prominent was for physical exercise (0.590), followed by stress relief (0.433). Consequently, these results suggest that the future value of AI-enabled prevention partly lies in its ability to transform personalised risk information into lasting behaviour change.

3.5 Relationship Between Biomarker Improvement and Patient-Reported Well-Being

Correlation analyses showed substantial associations between changes in clinical variables and changes in patient-reported outcomes. A greater reduction in HbA1c was associated with an increase in WHO-5 (r = −.320). In a similar way, a greater decrease in systolic BP was associated with higher scores on the WHO-5 (r = −.280). The largest clinical correlation of changes in WHO-5 was for reductions in BMI (r = −.410), which means that individuals with greater improvements in body weight status also demonstrated better psychosocial health.

Changes in physical activity were significantly associated with changes in WHO-5 (r = .283) and EQ-5D (r = .319). Reduced tension was related to improved WHO-5 (r = −.216) and EQ-5D (r = −.228) outcomes. The reductions in SBP and BMI were significantly and negatively associated with EQ-5D scores (r = −.308 and −.305, respectively). After adjustment for age, gender, intervention cohort, baseline PROs, and other contemporaneous changes in clinical parameters, multivariable analysis gave a more conservative estimate.

A decrease in BMI continued to be a notable independent predictor of enhancement in WHO-5 (standardised β = −.277, 95% CI: −.480 to −.074, p = .008). The status of AI-guided intervention was a significant independent predictor of improvements in WHO-5 (β = .248, p < .001) and EQ-5D (β = .349, p < .001). The model accounted for almost 22% of the variation in WHO-5 change (R^2 = .223; adjusted R^2 = .199) and about 22% of the variance in EQ-5D change (R^2 = .224; adjusted R^2 = .200).

Table 5. Multivariable Predictors of Patient-Reported Outcome Improvement

Outcome	Predictor	Standardized β	95% CI	*P*
WHO-5 change	HbA1c change	−.066	−.248 to .115	.473
WHO-5 change	SBP change	.082	−.087 to .251	.341
WHO-5 change	BMI change	**−.277**	**−.480 to −.074**	**.008**
WHO-5 change	Physical activity change	−.003	−.132 to .127	.966
WHO-5 change	AI intervention	**.248**	**.103 to .392**	**<.001**
EQ-5D change	SBP change	−.130	−.299 to .039	.132
EQ-5D change	BMI change	.021	−.182 to .225	.837
EQ-5D change	Physical activity change	.040	−.089 to .170	.539
EQ-5D change	AI intervention	**.349**	**.204 to .494**	**<.001**

Source: Authors' computation using SPSS

Upon adjustment for clinical change variables simultaneously, several other clinical-change variables were not significant. This should not be taken as evidence that changes in glycaemia or blood pressure had no relation to well-being. There were large interrelationships among the clinical-change variables. As an example, the correlation of HbA1c change and SBP change was strong (r = .734) and BMI change (r = .789). The covariance diminishes the apparent unique contribution of one or more predictors when entered together in a multivariable model. Therefore, subjective health changes may not simply be driven by one biomarker cardiovascular disease risk factor but rather a multidimensional metabolic or behavioural response.

3.6 Clinical Responder Analysis and Heterogeneity of Treatment Benefit

The composite clinical-responder was subject to additional exploratory investigations that illustrated the heterogeneity of treatment responses to precision prevention. Of the 160 patients receiving standard preventative care, 60 (37.5%) met criteria for multidomain responders (and were thus considered to have a favourable response). In contrast, 105 of 160 patients (65.6%) allocated to AI-assisted precision prevention met clinical-responder criteria.

AI-guided precision prevention increased clinically meaningful multidomain response chances. About one-third of AI-assisted patients did not fulfil the composite response criteria. Precision medicine researchers may anticipate personalisation to enhance aggregate results but not eliminate person-to-person treatment-outcome variance. Variability in SHAP responses was linked with intervention adherence, baseline HbA1c, physical activity improvements, beginning BMI, and dietary guideline adherence. These

characteristics imply that AI care must continually analyse natural danger and behavioural response.

The results reinforce the concept that personalized recommendation and personalized response are distinct activities. Delivering personalized recommendations does not guarantee that all patients will receive equal clinical benefit. This necessitates the real-time re-evaluation of treatment and adaptive interventions—the hallmark of effective precision medicine.

3.7 Implications for Sustainable Preventive Healthcare

The results have implications for sustainable health care. The adaptive intervention increased metabolic risk factors, body weight metrics, physical activity thresholds, perceived stress levels, psychological health and quality of life in relation to health immediately. It relates to earlier intervention when possible modifiable risk factors may lead to expensive and resource-intensive disease management in the future.

However, AI's environmental impact goes beyond automation. The possible benefits include continual risk assessment, prevention assistance, self-management support, and clinical resource allocation to those who need more clinician involvement. This process is driven by participation behaviours, according to these studies. The strongest predictor in the explainable machine-learning analysis was intervention adherence, suggesting that incrementally more sophisticated predictive algorithms cannot provide clinical benefits without patients following sound recommendations.

This follows AI-assisted chronic illness management trends. AI can be used for real-time monitoring, targeted therapy, and chronic illness management, however data quality, privacy, technical framework, and clinical application are issues. Mathioudakis et al. (2025) showed that AI-prompted lifestyle interventions may prevent diabetes as well as human coaching. These results suggest scalability, especially in narrow-access environments with skilled educators. AI-assisted prevention will also confront cost-effectiveness, equality, the digital divide, implementation tactics, and patient autonomy issues when employed instead of traditional treatment.

3.8 Overall Interpretation, Contributions and Limitations

The analysis found that AI-based guided precision prevention compared with routine preventive therapy improved glycaemic, cardiovascular, lipid and anthropometric, behavioural and patient-reported outcomes. Specifically, the greatest intervention effects were for Physical activity, WHO-5 psychological well-being, systolic blood pressure, stress reduction, body weight and EQ-5D-5L. HDL-C did not differ between groups, although some clinical predictors became nonsignificant with multivariable adjustment. This variety in results is more similar with the precision-health intervention impact than with a trend, where AI wins all outcomes.

The implications of the findings make a number of analytical contributions. The advanced study framework expands the evaluation of AI beyond just predictive accuracy by correlating machine-learning-driven risk assessment with health outcomes over time. Second, it combines objective biomarkers with patient-reported well-being and quality of life, facilitating assessment of intervention efficacy from both biomedical and patient-centred standpoints. Finally, the explainability analysis provides further evidence that it is not necessarily intervention allocation but adherence and behavioural change that drive clinical success.

Results are quite consistent with being contemporary findings of personalized-health studies. Bermingham et al. (2024) increase several cardiometabolic outcomes after personalized nutrition but heterogeneous response to biomarkers was also seen. Similarly, Mathioudakis et al. (2025) showed that it is possible for something like AI powered lifestyle intervention for diabetes prevention. Integrating this novel evidence suggests AI-driven personalization could enhance preventive health, but should not be interpreted as universally better in all patients or clinical outcomes.

Numerous constraints need careful evaluation. The main limitation of these results is that they are generated using a synthetic dataset created for statistical modelling and methodological teaching rather than real patient data. Thus, the resulting estimates of impact from study sample sizes do not reflect true clinical in vivo efficacy and should never be presented as real patient outcomes. Finally, assessments of the intervention's effectiveness would require confirmation in a prospective manner through real clinical data.

Secondly, the follow-up duration of 16 weeks reflects a relatively short term metabolic and behavioural change and is unable to demonstrate long-term maintenance, chronic disease progression or effects on major clinical events. Thirdly, responses to interventions that are delivered through digital means show variability according with digital literacy, socioeconomic status, health literacy, and cultural factors regarding their accessibility. Fourth, given interpretable feature attribution via SHAP but without inducing a causal relationship between individual predictors and outcomes. In the end, wider expansion of sustainability

assessment needs to include cost-effectiveness, environmental impact of computing infrastructure, health-care-resource use, fairness of algorithms and long-term implementation feasibility.

Conclusively, results provide evidence for effective evaluation of an explainable AI-supported precision-prevention framework through a sequenced synergistic composite of predictive performance, cardiometabolic biomarkers, behavioural outcomes and patient-reported health. This analytic pattern suggests that AI-enabled prevention requires more than algorithmic sophistication, and instead must involve an interplay among personalized risk prediction, recommended actionable steps to mitigate risk, and retention over time for meaningful longitudinal response.

4. CONCLUSION

The current study developed and evaluated a holistic AI-guided precision-prevention integration that includes detailed cardiometabolic risk assessment through personalisation, behaviour customisation via machine learning interpretability, objective biomarker monitoring, and patient-reported health outcomes. After 16 weeks of dual-group simulation, precision prevention with artificial intelligence improved clinically significant parameters more than usual preventive care (HbA1c, systolic blood pressure, LDL cholesterol, triglycerides, body weight and BMI waist circumference; QLQ-30 global function; Psychological PQ-HIID08 health-related quality of life; physical activity & perceived stress). Importantly, HDL-C response varied little across all outcomes.

As the machine-learning review showed, AI-driven preventive healthcare may fail despite predictive effectiveness. XGBoost had the highest projected value discrimination, whereas SHAP-based interpretability highlighted intervention adherence, baseline HbA1c, delta in physical activity, BMI, and dietary compliance as clinically important outcome predictors. These results suggest that AI should be utilised for decision-support and customisation rather than autonomous therapy. Clinical acceptability, interpretability, and accuracy of customised recommendations determine efficacy.

Another advantage of this strategy is combining patient self-reported outcomes with biomarkers. This method evaluates AI-supported preventative treatments beyond physiological risk to target patient-perceived physical, psychological, and functional health as measured by WHO-5 well-being, EQ-5D-5L utility, and EQ-VAS. This promotes a multidimensional view of preventive effectiveness since metabolic improvement is linked to behavioural change and patient-reported well-being.

AI-supported precision prevention may improve sustainable healthcare by moving chronic illness management from reactive to early, tailored, and behavior-based intervention. Such systems may improve risk categorisation, self-management, and clinician interaction, reducing health care resource waste. However, actual sustainability needs considering digital access, algorithmic fairness, data privacy, model explainability, method/hardware characteristics, and implementation lifetime costs.

The results must be evaluated in light of a significant restriction. An artificial dataset is used to develop and demonstrate approaches, but it does not represent patient outcomes. Thus, impact estimates indicate relationships rather than therapeutic effectiveness. This model should be confirmed using prospectively acquired multi-center patient data, longer follow-up times, external validation, and clinical evaluation of economic and ecological sustainability and clinically relevant hard events (disease progression, hospitalisation, cardiovascular events). In conclusion, AI precision medicine should be assessed based on its capacity to provide clinically relevant, person-centred, clear, and sustainable primary prevention, not just projected accuracy.

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