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Life's Essential 8 and Type D Personality Predict Coronary Plaque Instability: An Interpretable Machine-Learning Study

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Abstract

Background: Plaque instability underlies acute coronary events, and early recognition of high-risk patients is central to secondary prevention. Current assessment relies mainly on conventional clinical indices and does not integrate modifiable health behaviours or psychological traits. We therefore developed and validated an interpretable machine-learning model combining the Life's Essential 8 (LE8) cardiovascular health metrics with Type D personality to identify coronary plaque instability.

Methods: In this retrospective observational cohort study, 1,436 patients with angiographically confirmed coronary artery disease who underwent optical coherence tomography (OCT) at a single tertiary centre between September 2022 and September 2025 were consecutively enrolled. The 1,061 patients admitted up to 31 August 2024 formed the development cohort and were randomly divided 7:3 into training and internal validation sets; the 375 patients admitted thereafter formed the temporal validation set. Candidate predictors spanned demographic, clinical, behavioural and psychological domains. Variables were selected using least absolute shrinkage and selection operator regression, and six algorithms, including extreme gradient boosting (XGBoost), were compared by the area under the receiver operating characteristic curve (AUC), calibration curves and decision curve analysis, with Shapley additive explanations (SHAP) used for model interpretation.

Results: Ten predictors were retained, eight of them behavioural or psychological: six LE8 components (nicotine exposure, sleep health, blood lipid, blood pressure, healthy diet and physical activity) and two Type D dimensions (social inhibition and negative affectivity). XGBoost achieved AUCs of 0.915, 0.905 and 0.840 in the training, internal validation and temporal validation sets, respectively, with calibration curves close to the ideal line and net benefit above both reference strategies at threshold probabilities of 0.20–0.50. Nicotine exposure, sleep health, social inhibition, blood lipid and negative affectivity ranked highest in importance. The final model was deployed as a web-based calculator for individual risk estimation.

Conclusions: Modifiable cardiovascular health behaviours, particularly nicotine exposure and sleep health, together with psychological traits, were the dominant information sources for identifying plaque instability. A model built on these dimensions requires only questionnaires, medical history and routine laboratory tests, and provides concrete behavioural targets for early screening and secondary prevention.

Keywords: Life's Essential 8; Type D personality; thin-cap fibroatheroma; optical coherence tomography; machine learning; Shapley additive explanations

1 Background

Coronary artery disease remains a leading cause of morbidity and mortality worldwide[1]. Rupture or erosion of unstable atherosclerotic plaques is the key pathological trigger of acute coronary syndromes and other acute cardiovascular events[2]. Early identification of patients at high risk of plaque instability, followed by targeted intervention, is therefore central to reducing adverse cardiovascular events and to advancing secondary prevention[3].

Current clinical assessment of plaque instability centres largely on conventional clinical and physiological biomarkers[4]. The Life's Essential 8 (LE8) framework proposed by the American Heart Association spans nicotine exposure, sleep health, physical activity, diet, blood pressure and blood lipids, and thus provides a systematic picture of an individual's cardiovascular health[5]. Type D personality, characterised by negative affectivity and social inhibition, has been shown in meta-analyses to be independently associated with adverse prognosis in cardiovascular disease[6], and the effect of psychological distress on cardiovascular events appears to be independent of LE8[7]. Integrating these behavioural, psychological and physiological dimensions may therefore capture the determinants of plaque instability more comprehensively than any single domain. However, existing prediction models for plaque instability still rely largely on age, lipid levels and inflammatory markers — biological features that either cannot be modified or are difficult to change. Such models can stratify risk, but they cannot be translated directly into preventive action.

Machine learning can capture complex non-linear relationships among multiple risk factors[8], yet its black-box nature limits clinical interpretation[9]. Shapley additive explanations (SHAP) quantify the contribution of each feature to an individual prediction and thereby improve model readability[10]. To date, however, no interpretable model for plaque instability in patients with coronary artery disease has integrated LE8 cardiovascular health metrics with Type D personality.

We therefore conducted a retrospective observational cohort study in patients with angiographically and OCT-confirmed coronary artery disease. Our aims were to assess the relative contribution of LE8 and Type D personality to the identification of plaque instability, to identify the modifiable behavioural and psychological targets within these dimensions, and to provide individual-level attribution through SHAP, thereby supporting early screening and personalised prevention in clinical practice.

2 Methods

2.1 Study design and population

This was a retrospective observational cohort study. We included 1,436 consecutive patients with a first diagnosis of coronary artery disease who were admitted to the Department of Cardiology, The Second Affiliated Hospital of Harbin Medical University, between September 2022 and September 2025 and who underwent optical coherence tomography (OCT). Coronary artery disease was diagnosed according to the 2019 ESC Guidelines for the diagnosis and management of chronic coronary syndromes[11], with angiographic stenosis ≥50% in at least one major epicardial vessel or its major branches as the confirmatory criterion. Only patients with records in the follow-up database were included.

Inclusion criteria were: (1) complete baseline clinical data, including demographic information (age, sex, history of hypertension, hyperlipidaemia and diabetes, and family history of coronary artery disease) and laboratory results; (2) complete cardiovascular health behaviour and psychological assessment data, covering all eight LE8 metrics and the Type D personality scale; (3) complete OCT imaging and plaque characterisation data; and (4) age ≥18 years, male or non-pregnant, non-lactating female.

Exclusion criteria were: (1) severe concomitant systemic disease, such as advanced malignancy, severe hepatic or renal failure, severe pulmonary disease, or active autoimmune disease; (2) missing key variables such as LE8 or Type D personality scores; (3) previous coronary artery bypass grafting or target-vessel stenting; and (4) OCT images with artefacts that precluded plaque characterisation.

The study protocol was approved by the Medical Ethics Committee of The Second Affiliated Hospital of Harbin Medical University (approval no. YJSKY2025-385) and was conducted in accordance with the revised Declaration of Helsinki. Because of the retrospective design, written informed consent was waived. All patient identifiers were removed before analysis. The model was developed and validated in accordance with the Transparent Reporting of a multivariable prediction model for Individual Prognosis Or Diagnosis (TRIPOD) statement[12].

2.2 Data collection

Data were extracted from the follow-up database and comprised three groups of variables. (1) Baseline characteristics: age, sex, monthly household income, education, residence, marital status, family history of coronary artery disease, and history of diabetes, hyperlipidaemia and hypertension. (2) Laboratory indices: fasting plasma glucose, high-density lipoprotein cholesterol and total cholesterol. (3) Scoring instruments: the LE8 health behaviour scale and the Type D Personality Scale-14 (DS-14), scored according to uniform criteria to reflect current cardiovascular health behaviours and psychological status. Because dietary patterns differ between Chinese and Western populations[13], and because body mass index

carries Asian-specific implications[14], the items were adapted according to the Asian-specific standards of the instrument. All data were extracted from records generated within 24 hours of the diagnosis of coronary artery disease.

2.3 Variable definitions

Demographic variables and family history comprised age, sex, monthly household income, education, residence and marital status. A standardised history was used to record disease in first-degree relatives (parents and siblings), including coronary artery disease, hypertension, diabetes and premature cardiovascular disease; all were dichotomised (1 = present, 0 = absent).

Laboratory indices were measured in fasting venous blood drawn on the morning after admission. Fasting plasma glucose, glycated haemoglobin, high-density lipoprotein and total cholesterol were assayed with standard reagents and instruments in the hospital laboratory, under routine internal quality control.

The LE8 score comprised eight components, each scored from 0 to 100.

(1) Nicotine exposure: never smoked, 100; quit for more than 5 years, 75; quit for 1–5 years, 50; quit for less than 1 year, 25; current smoker, 0. A further 20 points were deducted if another household member smoked indoors.

(2) Physical activity: total weekly moderate-to-vigorous activity ≥150 min, 100; 120–149 min, 90; 90–119 min, 80; 60–89 min, 60; 30–59 min, 40; 1–29 min, 20; none, 0.

(3) Sleep health: 7 to <9 hours per night, 100; 9 to <10 hours, 90; 6 to <7 hours, 70; 5–6 hours or 9–10 hours, 40; 4–5 hours, 20; <4 hours, 0.

(4) Healthy diet, assessed on 15 items: at least one tablespoon of olive oil daily; green leafy vegetables at least twice daily; at least one plate of other vegetables daily; berries at least three times weekly; at least two kinds of other fruit daily; fish or seafood at least once weekly; red meat no more than 0.5 kg weekly; fried food no more than twice weekly; soy products at least four times weekly; whole grains at least 0.5 kg daily; nuts more than four times weekly; sweets no more than four times weekly; eating out less than once weekly; alcohol no more than two bottles daily for men and one bottle daily for women (beer equivalent); butter or cream no more than four times weekly. Meeting all 15 items scored 100; 12–14 items, 80; 8–11 items, 50; 4–7 items, 25; 0–3 items, 0.

(5) Body mass index: 18.5–22.9 kg/m², 100; 23–24.9, 75; 25.0–29.9, 50; 30.0–34.9, 25; ≥35.0, 0.

(6) Blood lipids, based on non-high-density lipoprotein cholesterol: <3.4 mmol/L, 100; 3.4–4.19, 60; 4.2–4.89, 40; 4.9–5.69, 20; ≥5.7, 0. Twenty points were deducted for patients receiving lipid-lowering therapy.

(7) Blood glucose: no diabetes and glycated haemoglobin <5.7%, 100; no diabetes and 5.7–6.4%, 60; diabetes and glycated haemoglobin <7.0%, 40; 7.0–7.9%, 30; 8.0–8.9%, 20; 9.0–9.9%, 10; ≥10.0%, 0. Twenty points were deducted if normoglycaemia had been achieved with treatment.

(8) Blood pressure: <120/80 mmHg, 100; systolic 120–129 with diastolic <80 mmHg, 75; 130–139/80–89 mmHg, 50; 140–159/90–99 mmHg, 25; ≥160/100 mmHg, 0. Twenty points were deducted for patients receiving antihypertensive therapy.

Type D personality was assessed with the DS-14. The Chinese version was translated and developed by Yu Xiaonan and Zhang Jianxin and comprises two dimensions, negative affectivity and social inhibition, each with seven items. Type D personality was defined as a score ≥10 on both dimensions[15].

2.4 Outcome definition

The outcome was thin-cap fibroatheroma (TCFA) on OCT, defined as a lipid-rich plaque with a lipid core arc ≥90° and a fibrous cap thickness <65 μm[16]. All coronary vessels were analysed, not only the culprit vessel. Images were read independently by two readers blinded to clinical data and to each other's assessments, and disagreements were adjudicated by a third senior reader.

2.5 Sample size

Sample size was calculated to allow precise estimation of model parameters while minimising overfitting. We applied the events-per-variable principle, using a threshold of at least 10 events per variable in line with the methodological work of Peduzzi et al.[17]. With 20 candidate predictors, the minimum number of positive events required was 200. Drawing on previous OCT evidence[18], in which TCFA was detected in approximately 24% of patients with stable angina and 65% of those with acute coronary syndrome, we assumed a detection rate of 30%, giving a base sample size of 667. Allowing for 10% missing data and loss to follow-up, the final target was 741 patients.

2.6 Model development and validation

The 1,061 patients admitted between 1 September 2022 and 31 August 2024 formed the development cohort and were randomly divided 7:3 into training and internal validation sets. The 375 patients admitted between 1 September 2024 and 30 September 2025 formed the temporal validation set. Six models were built from the selected predictors — logistic regression, decision tree, extreme gradient boosting (XGBoost), LightGBM, support vector machine and artificial neural network — and compared using sensitivity, specificity, positive and negative predictive value, accuracy and the area under the receiver operating characteristic curve.

2.7 Statistical analysis

Analyses were performed in R 4.2.3. Overall missingness in the source data was approximately 10%, with no single variable exceeding 30%; data were assumed to be missing at random. Variables with more than 30% missing values were excluded, and remaining missing values were handled by multiple imputation by chained equations, with all candidate predictors and the outcome included in the imputation model; ten complete datasets were generated and results combined using Rubin's rules. Normally distributed continuous variables were expressed as mean ± standard deviation and compared with the t test; non-normally distributed variables were expressed as median (interquartile range) and compared with the Mann-Whitney U test. Correlations were examined with a feature correlation heatmap. Categorical variables were expressed as percentages and compared with the χ^2 test. To limit overfitting, predictors were selected by least absolute shrinkage and selection operator (LASSO) regression with 10-fold cross-validation, and hyperparameters were tuned on the training set by five-fold cross-validation with grid search. The six models were then evaluated on the temporal validation set, and the best-performing model was retained. SHAP values were used to explain how each variable contributed to the predicted risk of plaque instability. The final model was deployed as an online web calculator and evaluated across discrimination (receiver operating characteristic curves), calibration (calibration curves) and clinical utility (decision curve analysis). The calculator is accessible at http://47.94.132.110:9006/ and will be maintained for at least two years after publication.

3 Results

3.1 Study population

A total of 1,548 patients with a first angiographic diagnosis of coronary artery disease who underwent OCT in the Department of Cardiology, The Second Affiliated Hospital of Harbin Medical University, between September 2022 and September 2025 were screened (1,136 during the development period and 412 during the temporal validation period). After

applying the inclusion and exclusion criteria, 1,436 patients were enrolled, of whom 480 (33.4%) had unstable plaque. The 1,061 patients admitted between 1 September 2022 and 31 August 2024 formed the development cohort and were randomly divided 7:3 into a training set (n = 744; 248 with unstable plaque) and an internal validation set (n = 317; 98 with unstable plaque). The 375 patients admitted between 1 September 2024 and 30 September 2025 formed the temporal validation set. Eligibility criteria were identical across the three groups. The flow of participants is shown in Figure 1.

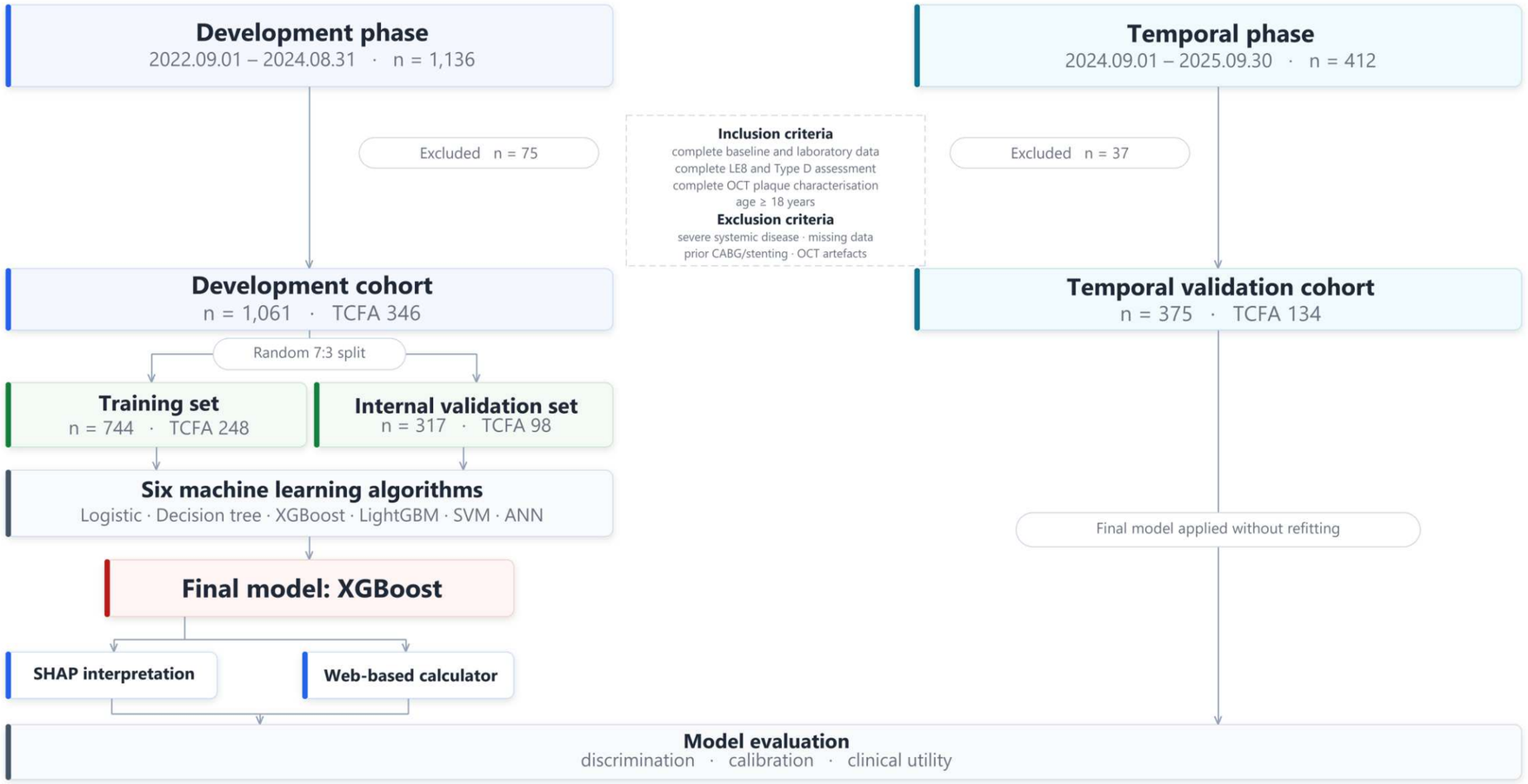


Figure 1 Flow of participants through the study.

3.2 Baseline characteristics

Table 1 compares baseline characteristics between the development and temporal validation cohorts. The two cohorts differed significantly in sex (χ^2 = 13.12, P < 0.001), nicotine exposure score (Z = 4.47, P < 0.001), body mass index score (Z = 4.27, P < 0.001) and blood glucose score (Z = 4.53, P < 0.001). No significant differences were found in age, family history of coronary artery disease, history of diabetes, hypertension or hyperlipidaemia, residence, education, income, marital status, or the negative affectivity, social inhibition, diet, sleep, blood lipid, physical activity and blood pressure scores (all P > 0.05).

Characteristics	Development cohort (n=1061)				Temporal validation cohort (n=375)					
	Total cohort (n=1061)	Stable plaque (n=715)	Unstable plaque (n=346)	P value	Total cohort (n=375)	Stable plaque (n=241)	Unstable plaque (n=134)	P value	χ^2/Z	All P value
Family history of CHD (%)										
No	822(77.47)	577(80.70)	245(70.81)	<0.001	306(81.60)	204(84.65)	102(76.12)	0.057	χ^2=2.800	0.094
Yes	239(22.53)	138(19.30)	101(29.19)		69(18.40)	37(15.35)	32(23.88)		χ^2=2.800	0.094
History of diabetes (%)										
No	728(68.61)	497(69.51)	231(66.76)	0.405	254(67.73)	165(68.46)	89(66.42)	0.771	χ^2=0.100	0.752
Yes	333(31.39)	218(30.49)	115(33.24)		121(32.27)	76(31.54)	45(33.58)		χ^2=0.100	0.752
History of hypertension (%)										
No	322(30.35)	233(32.59)	89(25.72)	0.027	119(31.73)	74(30.71)	45(33.58)	0.647	χ^2=0.250	0.617
Yes	739(69.65)	482(67.41)	257(74.28)		256(68.27)	167(69.29)	89(66.42)		χ^2=0.250	0.617
History of hyperlipidemia (%)										
No	379(35.72)	276(38.60)	103(29.77)	0.006	118(31.47)	82(34.02)	36(26.87)	0.189	χ^2=2.216	0.137
Yes	682(64.28)	439(61.40)	243(70.23)		257(68.53)	159(65.98)	98(73.13)		χ^2=2.216	0.137
Residence(%)										
Rural area	466(43.92)	320(44.76)	146(42.20)	0.105	168(44.80)	112(46.47)	56(41.79)	0.537	χ^2=1.822	0.402
County seat	273(25.73)	170(23.78)	103(29.77)		84(22.40)	50(20.75)	34(25.37)		χ^2=1.822	0.402
Urban area	322(30.35)	225(31.47)	97(28.03)		123(32.80)	79(32.78)	44(32.84)		χ^2=1.822	0.402
Education level (%)										
Junior high school or below	647(60.98)	444(62.10)	203(58.67)	0.008	220(58.67)	139(57.68)	81(60.45)	0.424	χ^2=1.552	0.67
Senior high school and secondary vocational school	260(24.51)	171(23.92)	89(25.72)		101(26.93)	68(28.22)	33(24.63)		χ^2=1.552	0.67
Junior college diploma / (Associate degree)	84(7.92)	64(8.95)	20(5.78)		26(6.93)	19(7.88)	7(5.22)		χ^2=1.552	0.67
Bachelor's degree or above	70(6.60)	36(5.03)	34(9.83)		28(7.47)	15(6.22)	13(9.70)		χ^2=1.552	0.67
Income(%)										
＜1000	117(11.03)	87(12.17)	30(8.67)	0.053	46(12.27)	33(13.69)	13(9.70)	0.083	χ^2=0.740	0.864
1000-2500	340(32.05)	240(33.57)	100(28.90)		121(32.27)	75(31.12)	46(34.33)		χ^2=0.740	0.864
2500-5000	454(42.79)	296(41.40)	158(45.66)		160(42.67)	109(45.23)	51(38.06)		χ^2=0.740	0.864
＞5000	150(14.14)	92(12.87)	58(16.76)		48(12.80)	24(9.96)	24(17.91)		χ^2=0.740	0.864

Characteristics	Development cohort (n=1061)				Temporal validation cohort (n=375)					
	Total cohort (n=1061)	Stable plaque (n=715)	Unstable plaque (n=346)	P value	Total cohort (n=375)	Stable plaque (n=241)	Unstable plaque (n=134)	P value	χ^2/Z	All P value
Gender(%)										
Male	890(83.88)	610(85.31)	280(80.92)	0.083	283(75.47)	186(77.18)	97(72.39)	0.364	χ^2=13.119	<0.001
Female	171(16.12)	105(14.69)	66(19.08)		92(24.53)	55(22.82)	37(27.61)		χ^2=13.119	<0.001
Marriage(%)										
Single	15(1.41)	10(1.40)	5(1.45)	0.419	5(1.33)	3(1.24)	2(1.49)	0.987	χ^2=2.555	0.465
Married	988(93.12)	661(92.45)	327(94.51)		341(90.93)	219(90.87)	122(91.04)		χ^2=2.555	0.465
Divorced	9(0.85)	8(1.12)	1(0.29)		5(1.33)	3(1.24)	2(1.49)		χ^2=2.555	0.465
Widowed	49(4.62)	36(5.03)	13(3.76)		24(6.40)	16(6.64)	8(5.97)		χ^2=2.555	0.465
NA score	8.00[6.00,11.00]	7.00[5.00,10.00]	10.00[8.00,13.00]	<0.001	8.00[6.00,11.00]	8.00[5.00,9.00]	10.00[7.00,12.75]	<0.001	Z=0.62	0.536
SI score	8.00[6.00,10.00]	8.00[5.00,10.00]	10.00[8.00,12.00]	<0.001	8.00[6.00,10.00]	8.00[6.00,10.00]	9.00[8.00,11.00]	<0.001	Z=-0.18	0.86
Age(years)	61.00[54.00,67.00]	62.00[55.00,68.00]	60.00[53.00,66.75]	0.002	62.00[54.00,67.00]	61.00[56.00,68.00]	63.00[51.25,67.00]	0.661	Z=0.09	0.928
Healthy diet score	50.00[25.00,50.00]	50.00[25.00,50.00]	25.00[25.00,50.00]	<0.001	50.00[25.00,50.00]	50.00[25.00,50.00]	25.00[25.00,50.00]	<0.001	Z=0.64	0.522
Physical activity score	20.00[0.00,100.00]	40.00[0.00,100.00]	0.00[0.00,80.00]	<0.001	20.00[0.00,100.00]	60.00[0.00,100.00]	0.00[0.00,80.00]	0.001	Z=-0.20	0.838
Nicotine exposure score	80.00[5.00,100.00]	100.00[5.00,100.00]	55.00[0.00,80.00]	<0.001	55.00[0.00,100.00]	80.00[0.00,100.00]	25.00[0.00,75.00]	<0.001	Z=4.47	<0.001
Sleep health score	90.00[70.00,100.00]	100.00[70.00,100.00]	70.00[40.00,90.00]	<0.001	90.00[70.00,100.00]	100.00[70.00,100.00]	70.00[40.00,90.00]	<0.001	Z=-0.70	0.485
BMI Score	75.00[50.00,100.00]	75.00[50.00,100.00]	50.00[50.00,75.00]	<0.001	50.00[50.00,75.00]	50.00[50.00,100.00]	50.00[50.00,75.00]	0.004	Z=4.27	<0.001
Blood lipid score	80.00[40.00,100.00]	80.00[60.00,100.00]	60.00[40.00,100.00]	<0.001	80.00[40.00,100.00]	80.00[40.00,100.00]	80.00[40.00,100.00]	0.073	Z=0.43	0.666
Blood glucose score	60.00[60.00,100.00]	100.00[60.00,100.00]	60.00[60.00,100.00]	<0.001	60.00[40.00,100.00]	60.00[40.00,100.00]	60.00[40.00,60.00]	0.002	Z=4.53	<0.001
BP score	25.00[5.00,50.00]	30.00[5.00,55.00]	25.00[0.00,50.00]	<0.001	30.00[5.00,55.00]	25.00[5.00,55.00]	30.00[0.00,50.00]	0.558	Z=-0.78	0.434

Within the development cohort, patients with unstable plaque differed from those with stable plaque in age (P = 0.002), family history (P < 0.001), hypertension (P = 0.027), hyperlipidaemia (P = 0.006), education (P = 0.008) and all eight LE8 scores together with negative affectivity and social inhibition (all P < 0.05). No differences were found in sex (P = 0.083), diabetes (P = 0.405), residence (P = 0.105), income (P = 0.053) or marital status (P = 0.419). Notably, all eight LE8 scores and both Type D dimensions were significantly associated with plaque instability (P ≤ 0.001), whereas among the remaining variables only family history (P < 0.001), hypertension (P = 0.027), hyperlipidaemia (P = 0.006) and age (P = 0.002) reached statistical significance.

Table 1 Baseline characteristics of the development and temporal validation cohorts

Note: Categorical variables are presented as n (%), compared with the χ^2 test. Continuous and ordinal variables are presented as median [interquartile range], compared with the Mann-Whitney U test (Z). χ^2/Z and the all P value refer to comparisons between the total development cohort and the total temporal validation cohort.

3.3 Feature selection

LASSO regression with 10-fold cross-validation (Figure 2A, B) retained ten features associated with plaque instability: nicotine exposure, sleep health, social inhibition, blood lipid, negative affectivity, blood pressure, family history of coronary artery disease, healthy diet, physical activity and diabetes. Social inhibition, negative affectivity, family history and diabetes behaved as risk factors, whereas nicotine exposure, healthy diet, physical activity, sleep health, blood lipid and blood pressure scores were protective. Pearson correlations were displayed as a heatmap of the training set (Figure 2C). Most correlations were weak; only social inhibition and negative affectivity were strongly correlated ($r = 0.83$, VIF = 3.2). All variance inflation factors were below 5, so all variables were entered into modelling. LASSO used 10-fold cross-validation, and hyperparameters were tuned by five-fold cross-validation repeated five times.

Of the ten retained features, eight came from the LE8 and Type D dimensions (six LE8 components and two personality dimensions), the remaining two being family history and diabetes. This suggests that the information available for identifying plaque instability lies mainly in modifiable behavioural and psychological domains.

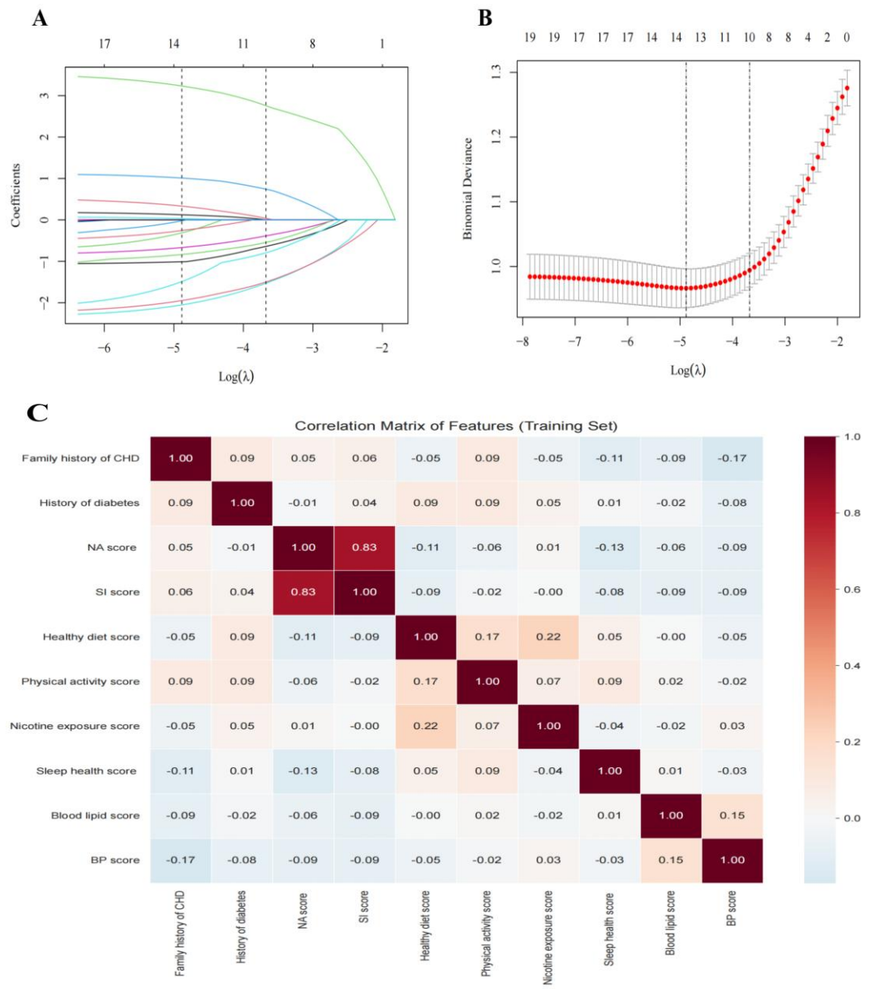


Figure 2 LASSO regression analysis and correlation of the selected features. (A) Coefficient trajectories of the LASSO model as a function of log(λ). (B) Ten-fold cross-validation curve; the left dashed line marks the optimal λ (λ.min) and the right dashed line the λ one standard error above it (λ.1se). (C) Correlation heatmap of the LASSO-selected features in the training set; red indicates positive and blue negative correlation.

3.4 Model development and performance

The ten features were entered into six models — logistic regression, decision tree, XGBoost, LightGBM, support vector machine and artificial neural network — with parameters optimised by five-fold cross-validation. Performance is summarised in Table 2 and Figure 3.

XGBoost achieved AUCs of 0.915 (95% CI 0.895–0.934), 0.905 (0.869–0.935) and 0.840 (0.801–0.879) in the training, internal validation and temporal validation sets, a decline of approximately 0.065 from internal to temporal validation. Across the six algorithms, AUCs ranged from 0.833 to 0.915 in the training set and from 0.789 to 0.840 in the temporal validation set, indicating limited differences between algorithms (Table 2). Calibration was good: Brier scores for XGBoost were 0.115, 0.119 and 0.156 in the three sets, and calibration curves lay close to the diagonal. Decision curve analysis showed that net benefit exceeded both the treat-all and treat-none reference strategies across threshold probabilities of 0.20–0.50.

Table 2 Performance of the six models across the three cohorts

Models	AUC	Accuracy	Sensitivity	Specificity	F1 score	Kappa	Youden's J	PPV	NPV
Training cohort (n=744, 248 events)									
Logistic	0.835	0.780	0.762	0.788	0.697	0.526	0.550	0.643	0.869
Decision Tree	0.865	0.761	0.839	0.722	0.700	0.510	0.560	0.601	0.899
XGBoost	0.915	0.816	0.810	0.819	0.746	0.603	0.629	0.691	0.896
LightGBM	0.905	0.809	0.827	0.800	0.743	0.594	0.627	0.674	0.902
SVM	0.833	0.780	0.770	0.784	0.700	0.528	0.554	0.641	0.872
ANN	0.897	0.798	0.810	0.792	0.728	0.571	0.603	0.661	0.893
Internal validation cohort (n=317, 98 events)									
Logistic	0.818	0.754	0.765	0.749	0.658	0.472	0.514	0.577	0.877
Decision Tree	0.856	0.760	0.827	0.731	0.681	0.498	0.557	0.579	0.904
XGBoost	0.905	0.826	0.827	0.826	0.747	0.617	0.653	0.681	0.914
LightGBM	0.897	0.833	0.837	0.831	0.756	0.630	0.668	0.689	0.919
SVM	0.812	0.748	0.745	0.749	0.646	0.455	0.494	0.570	0.868
ANN	0.879	0.776	0.776	0.776	0.682	0.513	0.552	0.608	0.885
Temporal validation cohort (n=375, 134 events)									
Logistic	0.802	0.768	0.761	0.772	0.701	0.513	0.533	0.650	0.853
Decision Tree	0.789	0.715	0.769	0.685	0.658	0.422	0.453	0.575	0.842
XGBoost	0.840	0.768	0.776	0.763	0.705	0.517	0.540	0.646	0.860
LightGBM	0.839	0.760	0.746	0.768	0.690	0.496	0.514	0.641	0.845
SVM	0.798	0.736	0.746	0.730	0.669	0.453	0.477	0.606	0.838
ANN	0.820	0.739	0.716	0.751	0.662	0.451	0.467	0.615	0.826

Note: AUC, area under the receiver operating characteristic curve; Accuracy, proportion of correctly classified patients; Sensitivity and Specificity, true positive and true negative rates; F1 score, harmonic mean of precision and recall; Kappa, Cohen's kappa agreement coefficient; Youden's J, sensitivity + specificity − 1; PPV, positive predictive value; NPV, negative predictive value. All metrics were calculated within the corresponding cohort.

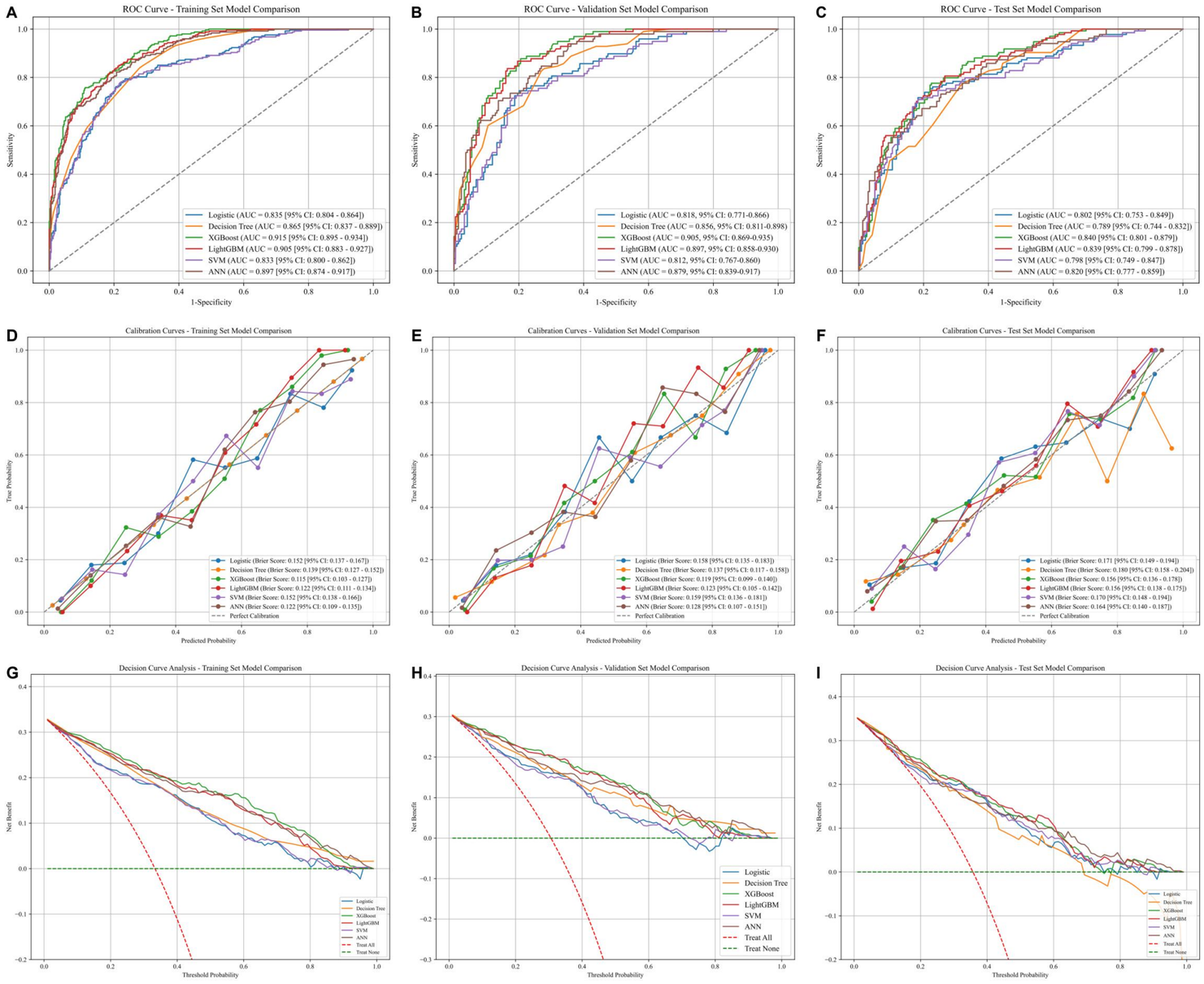


Figure 3 Performance of the six prediction models across the three cohorts. Receiver operating characteristic curves in the training (A), internal validation (B) and temporal validation (C) sets; calibration curves in the training (D), internal validation (E) and temporal validation (F) sets; decision curve analysis in the training (G), internal validation (H) and temporal validation (I) sets.

3.5 Model interpretability and clinical application

Figure 4A shows the SHAP beeswarm plot. Positive SHAP values increase and negative values decrease the predicted probability of plaque instability. Nicotine exposure, sleep, blood lipid, physical activity, blood pressure and diet scores all showed marked effects: higher nicotine exposure scores, better sleep quality, more favourable lipid status, greater physical activity, better blood pressure control and a healthier diet were associated with lower risk, whereas higher negative affectivity, greater social inhibition, diabetes and family history increased risk. Figure 4B ranks feature importance; nicotine exposure, sleep, social inhibition, blood lipid and negative affectivity occupied the top five positions.

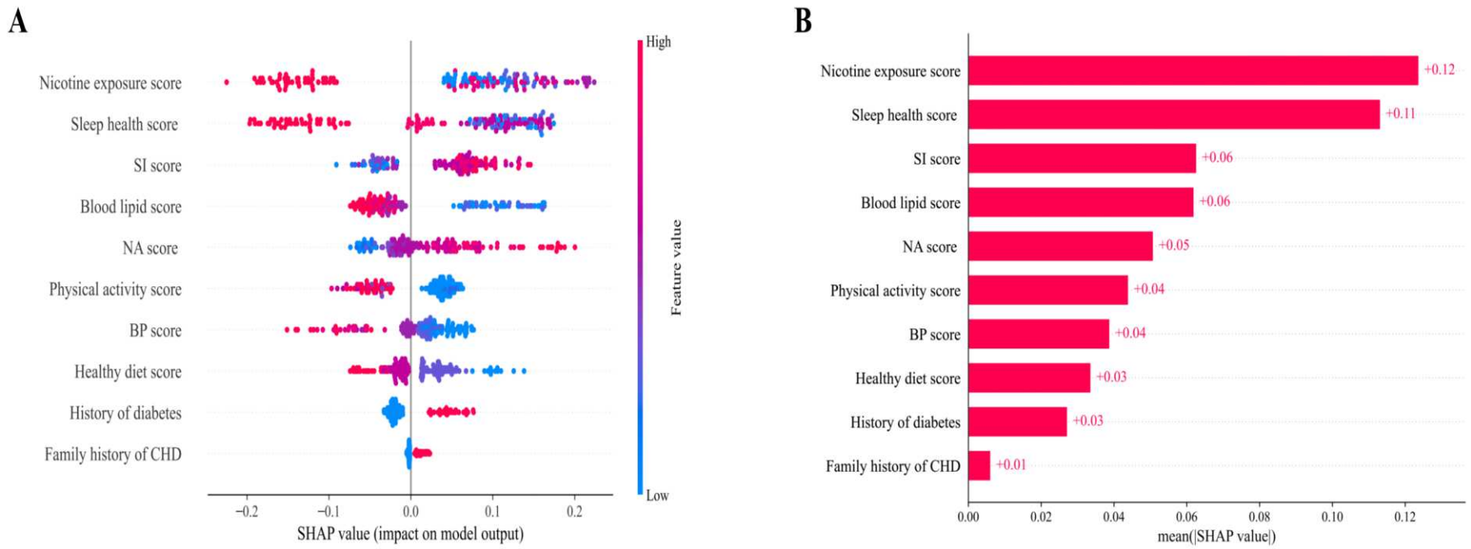
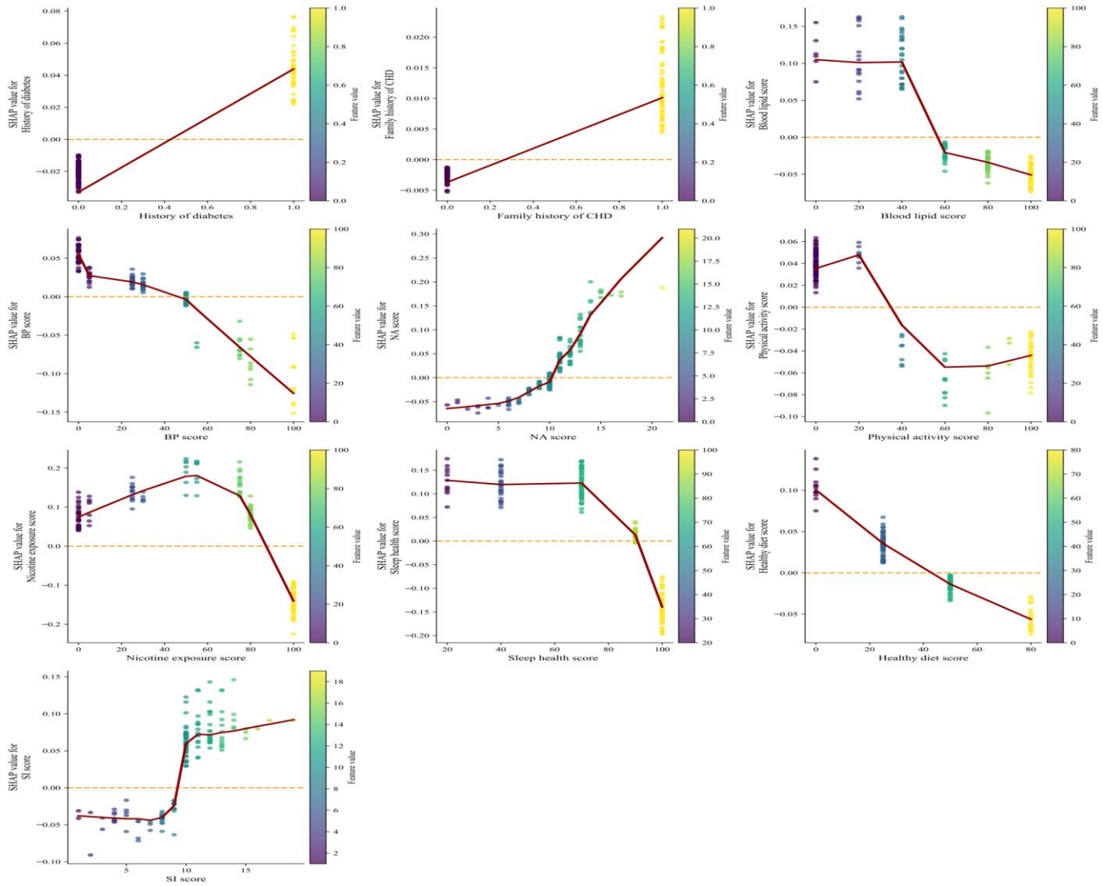


Figure 4 SHAP-based decomposition of the XGBoost model. (A) Beeswarm plot of feature attributions; the horizontal axis is the SHAP value, each row is one feature, and red and blue denote higher and lower feature values respectively. (B) Mean absolute SHAP value for each feature, ranked from highest to lowest.

Figure 5 shows SHAP dependence plots. All six health-domain scores showed protective effects at high values. Blood pressure and physical activity scores declined in an approximately linear fashion; the sleep health score was flat between 20 and 70 points and fell clearly only above 75; blood lipid and healthy diet scores declined stepwise at approximately 60 and 50 points, respectively; and the nicotine exposure score contributed positively at all values below 80 and became protective only near the maximum. Diabetes, family history, negative affectivity and social inhibition were risk-promoting, with stepwise effects for the two binary factors and non-linear thresholds at approximately 12 and 10 points for negative affectivity and social inhibition, respectively. These dependence patterns define quantifiable effect thresholds for each major factor (Table 3).



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186 Figure 5 SHAP dependence plots for the ten predictors, showing the marginal effect and non-linear association of each feature with the predicted risk of plaque instability.

187 **Table 3 Quantifiable effect thresholds of health behaviour and psychological factors**

Factor	Effect threshold	Change around the threshold
Nicotine exposure score	≥80	Shifts from a positive contribution to a protective effect
Sleep health score	≥75	Protective (negative) contribution becomes clearly stronger
Negative affectivity (NA)	≥12	Positive risk effect increases rapidly
Social inhibition (SI)	≥10	Positive risk effect increases rapidly

Blood lipid score	~60	Stepwise shift to a protective effect
Healthy diet score	~50	Stepwise shift to a protective effect

Note: The effect threshold denotes the value beyond which the direction of a feature's contribution to the predicted probability of plaque instability changes. Thresholds were read from the SHAP dependence plots and represent approximate inflection points rather than predefined clinical cut-offs. NA, negative affectivity; SI, social inhibition.

Figure 6A presents the SHAP waterfall plot for a high-risk patient (predicted probability 0.911) and Figure 6B the SHAP force plot for a patient at intermediate risk (predicted probability 0.48). Both show that the model provides individual-level attributions consistent with the overall pattern.

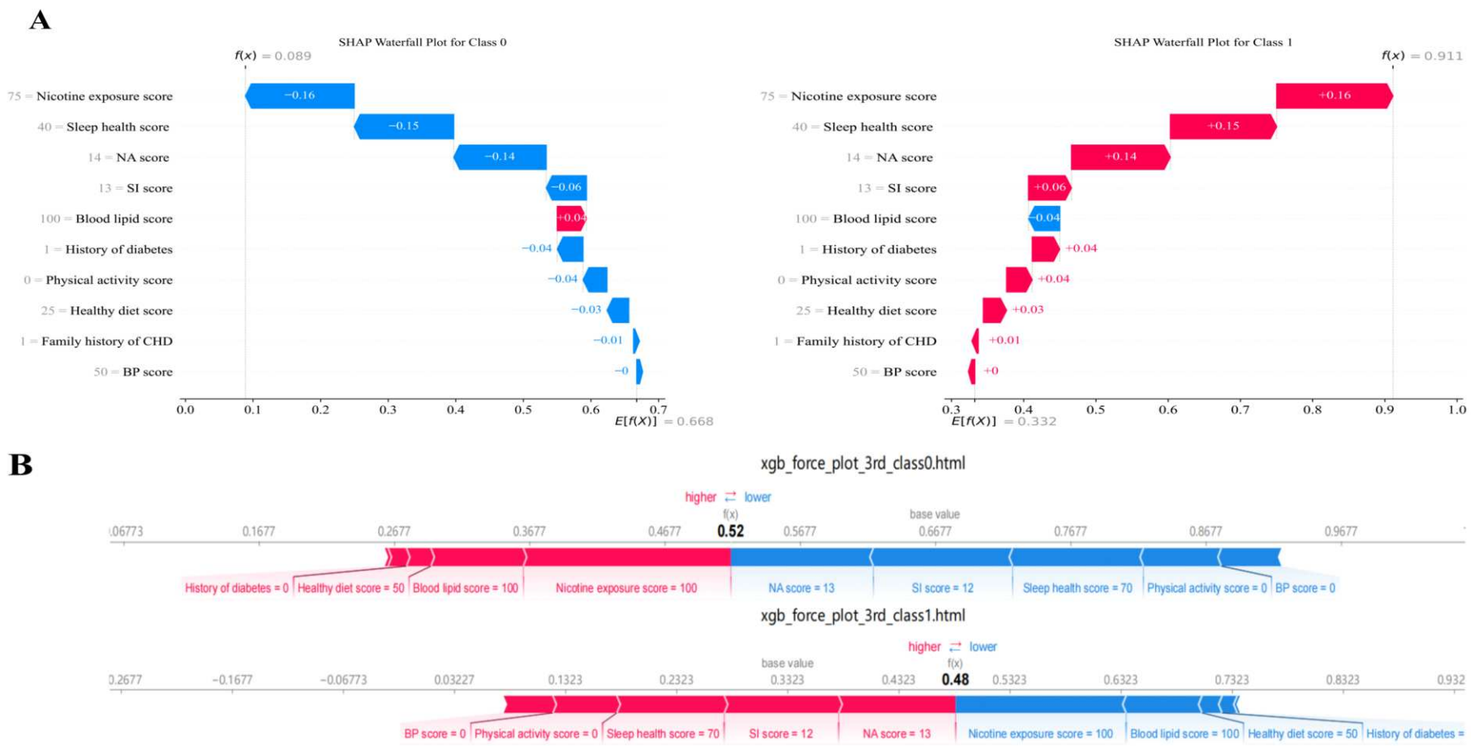

Figure 6 Individual-level SHAP explanations for two representative patients. (A) Waterfall plots for the stable plaque (Class 0) and unstable plaque (Class 1) outcomes in a high-risk patient. (B) Force plots for the same model in a patient at intermediate risk.

The prediction model was deployed as a web-based platform (http://47.94.132.110:9006/) that allows clinicians to estimate the risk of plaque instability from the clinical features entered into designated text fields.

4 Discussion

In this study, we used data from 1,436 patients with OCT-confirmed coronary artery disease to assess the relative contribution of the Life's Essential 8 (LE8) and Type D personality to the identification of plaque instability. On that basis, we developed and temporally validated a discriminative model. Established coronary risk tools such as GRACE and SCORE2 can predict cardiovascular events. However, they remain limited for identifying OCT-defined thin-cap fibroatheroma and rely mainly on conventional physiological indices — age, blood pressure and blood lipids — with little integration of modifiable health behaviours or psychosocial factors. We therefore developed a dedicated model for patients undergoing coronary angiography and OCT, incorporating standardised demographic characteristics, medical history, the eight LE8 component scores and the two Type D dimensions. The principal strength of this work lies in bringing LE8 behavioural factors and Type D psychological traits into a single machine-learning framework to identify determinants of plaque instability. Our central finding is that eight of the ten features retained by LASSO came from the LE8 and Type D dimensions, and that the top five features by SHAP importance all belonged to these dimensions. In a population with established coronary artery disease, plaque instability therefore appears to be characterised more by modifiable behavioural and psychological traits than by immutable demographic characteristics.

After LASSO selection, variables with non-zero coefficients were entered into the XGBoost model. SHAP analysis showed that higher nicotine exposure scores, better sleep health and more favourable lipid levels reduced the risk of plaque instability, whereas lower nicotine exposure scores and higher social inhibition and negative affectivity scores markedly increased it. These associations have been widely corroborated in previous studies.

We now consider each behavioural and psychological dimension in turn, together with its amenability to intervention. First, nicotine exposure. The mechanisms by which smoking promotes plaque vulnerability have been repeatedly demonstrated: nicotine and combustion products accelerate fibrous cap thinning and lipid core expansion by injuring the endothelium, activating oxidative stress and inflammatory pathways and promoting lipid oxidation. In a three-vessel OCT study of 182 participants and 465 plaques[19], TCFA was detected in 18.4% of current smokers, significantly more often than in former smokers (7.6%) or never smokers (9.9%). Our SHAP dependence curve indicates that the nicotine exposure score contributes positively at all values below 80 and becomes protective only near the maximum, consistent with the clinical observation that cardiovascular risk falls progressively with each additional

year of abstinence[20]. Second, sleep health. Since LE8 added sleep duration as its eighth metric in 2022, the link between sleep and cardiovascular events has attracted increasing attention. Ning et al., pooling six US cohorts, reported a 22–40% reduction in cardiovascular risk for every 10-point increase in LE8, with the addition of sleep further improving reclassification relative to the original Life's Simple 7[21]. In our cohort, sleep health ranked second in independent contribution, suggesting an association with coronary plaque instability that is independent of conventional risk factors in a secondary prevention population; previous work has likewise linked healthy sleep patterns to a lower risk of recurrent cardiovascular events[22]. Insufficient sleep may accelerate plaque progression through sustained sympathetic activation, hypothalamic–pituitary–adrenal axis activation and elevated inflammatory cytokines. Third, the two dimensions of Type D personality. A previous study of 177 patients undergoing OCT[23] found that patients with Type D personality had higher plasma tumour necrosis factor-α, interleukin-6 and kynurenine/tryptophan ratios, and that this inflammatory–metabolic pathway was associated with TCFA, macrophage infiltration and two-year major adverse cardiovascular events. A 2025 meta-analysis including 15 studies and 5,687 patients confirmed the association between Type D personality and adverse cardiovascular prognosis, with a prevalence of approximately 36%[24]. In a Chinese population, Li et al. followed 257 patients with acute myocardial infarction and found Type D personality to be an independent predictor of in-hospital major adverse cardiovascular events (odds ratio 3.33) and of long-term events after discharge (hazard ratio 2.27)[25]. Notably, our SHAP dependence curves place the inflection points for rising risk at approximately 10 points for social inhibition and 12 points for negative affectivity. Conventional Type D classification uses a fixed cut-off of ≥10 on both dimensions, whereas our results suggest that the contribution of these dimensions to plaque risk is continuous and begins to accumulate below those thresholds. Fourth, blood lipids. The LE8 lipid component is based on non-high-density lipoprotein cholesterol, which reflects the total burden of atherogenic lipoproteins better than low-density lipoprotein cholesterol. He et al., using paired OCT follow-up of non-culprit lesions, found that patients with a higher atherogenic lipid ratio had thinner fibrous caps and a higher incidence of TCFA, and that the ratio remained an independent predictor of TCFA progression at one year[26]. Jin et al., analysing more than 170,000 participants from China-PAR and Kailuan, further showed that the highest LE8 quintile had a significantly lower 10-year atherosclerotic cardiovascular disease risk than the lowest quintile, with blood lipids and blood pressure contributing most[27]. In our study the blood lipid score entered the top five by SHAP, consistent with these findings.

Regarding model performance, XGBoost achieved AUCs of 0.915, 0.905 and 0.840 in the training, internal validation and temporal validation sets, a decline of about 0.065 from internal to temporal validation. Given that the two cohorts differed significantly in sex, nicotine exposure, body mass index and blood glucose scores, this attenuation suggests reasonable robustness to population drift. Calibration curves lay close to the diagonal, and decision curve analysis showed net benefit superior to both reference strategies across threshold probabilities of 0.20–0.50.

Identification of vulnerable coronary plaque currently relies mainly on OCT, intravascular ultrasound or coronary computed tomography angiography[28,29]. These modalities visualise plaque morphology directly, but they are invasive or depend on large imaging systems and are therefore difficult to use routinely in outpatient or ward settings. Recent studies have attempted to predict plaque risk from imaging features or clinical variables, but the variables used have largely been confined to conventional physiological indices, with limited integration of modifiable health behaviours and psychosocial factors[30]. Our study extends this work by incorporating LE8 component scores and the two Type D dimensions, constructing and temporally validating a model against OCT-defined TCFA as the reference standard while using only questionnaires and routine laboratory tests.

This study has several distinctive features. First, the outcome was defined morphologically as OCT-defined TCFA rather than by follow-up clinical events, making plaque phenotyping direct and objective. Second, the model incorporated all eight LE8 component scores together with the two Type D dimensions in 1,436 patients, with 375 patients separated chronologically as a temporal validation set. Third, six commonly used machine-learning algorithms were compared, features were selected by LASSO with 10-fold cross-validation, and discrimination, calibration and decision-curve net benefit were all reported. Fourth, SHAP was used for both feature importance and individual attribution, and an online web tool was built for rapid clinical use.

This study adds to the evidence supporting the combined use of LE8 cardiovascular health metrics and Type D personality traits for machine-learning prediction of coronary plaque instability. Although the influence of behavioural and psychological factors on coronary prognosis is well recognised, conventional prediction methods struggle to capture their non-linear relationship with plaque morphology. Ensemble learning algorithms such as XGBoost help capture these non-linear associations, and SHAP analysis broadens our understanding of the underlying risk factors[31]. Machine learning can automatically uncover complex non-linear relationships among large numbers of variables and performs particularly well with multivariable, multilevel data[32]. The web-based tool developed here also has practical value: once a patient is identified as high risk, individualised measures — intensive smoking cessation counselling, sleep management, psychological assessment and lipid-lowering therapy — can be initiated. From a secondary prevention perspective, each target is actionable: nicotine exposure maps to smoking cessation clinics and follow-up, sleep health to sleep hygiene education and cognitive behavioural therapy for insomnia, healthy diet and physical activity to dietary modification and exercise prescription, blood pressure and lipids to target-based management, and negative affectivity and social inhibition to psychological assessment and intervention. Unlike previous models for high-risk plaque built largely on age, lipids and inflammatory or metabolic markers[33,34], the behavioural and psychological variables in our model can all be improved through behaviour change and guideline-directed treatment, making the model easier to translate into actionable prevention pathways.

5 Conclusions

In patients with coronary artery disease, modifiable cardiovascular health behaviours — notably nicotine exposure and sleep health — together with psychological traits (negative affectivity and social inhibition) constituted the dominant source of information for identifying plaque instability: eight of the ten discriminative features came from these two dimensions, and each factor had a quantifiable effect threshold. The XGBoost model built on these dimensions achieved an AUC of 0.840 in temporal validation and requires only questionnaires, medical history and routine laboratory tests, making it suitable for use in outpatient and ward settings. These findings move risk assessment for plaque instability away from immutable biological characteristics towards measurable, modifiable behavioural and psychological targets, providing concrete and practicable directions for secondary prevention of coronary artery disease. However, this was a single-centre retrospective study with a morphological OCT outcome, and multicentre external validation and prospective studies with clinical endpoints are still required.

List of abbreviations

ANN artificial neural network

AUC area under the receiver operating characteristic curve

BMI body mass index

CI confidence interval

DCA decision curve analysis

DS-14 Type D Personality Scale-14

ESC European Society of Cardiology

HDL-C high-density lipoprotein cholesterol

IVUS intravascular ultrasound

LASSO least absolute shrinkage and selection operator

LE8 Life's Essential 8

MACE major adverse cardiovascular events

NA negative affectivity

NPV negative predictive value

OCT optical coherence tomography

PPV positive predictive value

SHAP Shapley additive explanations

SI social inhibition

TCFA thin-cap fibroatheroma

TRIPOD Transparent Reporting of a multivariable prediction model for Individual Prognosis Or Diagnosis

VIF variance inflation factor

XGBoost extreme gradient boosting

Declarations

Ethics approval and consent to participate

The study protocol was approved by the Medical Ethics Committee of The Second Affiliated Hospital of Harbin Medical University (approval no. YJSKY2025-385) and was conducted in accordance with the revised Declaration of Helsinki. Because of the retrospective design, written informed consent was waived.

Consent for publication

Not applicable.

Availability of data and materials

The datasets used and/or analyzed in this study are available from the corresponding author upon reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

Xuanwei Zhao and Xinrui Ma contributed equally to this work and share first authorship. As the first author, Xuanwei Zhao led the conception and design of the study, collected and curated the data, conducted the statistical analysis, developed and validated the machine-learning models, and drafted the manuscript. Xinrui Ma contributed to the study design, data verification and manuscript preparation. Xiaotong Yu, Chunyu Zhou and Zhenjuan Zhao collected and interpreted the clinical and OCT data. Guojie Liu carried out the statistical analysis, model building and validation. Anqi Yu and Xuebing Liang contributed to the literature search and data curation. Xueqin Gao and Bo Yu supervised the study and critically revised the manuscript. All authors read and approved the final manuscript.

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