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Article

Deep Neural Network-Based Nutrition-Gene Interaction Framework for Predicting Type 2 Diabetes Risk

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Abstract: Type 2 diabetes mellitus (T2DM) arises through complex mechanisms, and diet-gene interactions are not yet fully defined. We built a deep neural network (DNN) framework that combines dietary and genetic data to predict T2DM risk. Food-frequency questionnaire data from 300,000 partici-pants were merged with genome-wide association study (GWAS) data covering about nine mil-lion single-nucleotide polymorphisms. A multitask design yields two outputs: T2DM risk and individual dietary response. Dietary variables were reduced with sparse coding, and a Trans-former-based embedding layer was used to represent genetic features. In an independent mul-ticenter validation cohort (n = 15,000), the model achieved an area under the receiv-er-operating-characteristic curve of 0.92 for T2DM prediction and 78 % accuracy for die-tary-response prediction. SHAP analysis showed a significant interaction between omega-3 fat-ty-acid intake and the TCF7L2 locus. This study is the first to model nutrition-gene interactions on a large scale. It overcomes the limits of single-modality risk scores and provides a solid basis for precision-nutrition interventions aimed at preventing T2DM.

Keywords: Type 2 diabetes; deep learning; gene-diet interaction; GWAS; precision nutrition; SHAP

1. Introduction

Type 2 diabetes mellitus (T2DM) is a major global public health issue, with its pathogenesis involving complex interactions among genetic factors, environmental exposures, and lifestyle behaviors [1-3]. According to the IDF Diabetes Atlas released in 2024, the global number of adults living with diabetes has risen sharply to 620 million, with T2DM accounting for more than 92 % [4]. The disease burden shows notable regional variation [5]. In the Western Pacific, China reports a T2DM prevalence of 12.4 % among adults aged 18 and above, with the total number of patients exceeding 140 million [6,7]. In India, driven by rapid urbanization and lifestyle transitions, the diabetic population has increased by 60 % over the past decade and now exceeds 80 million [8]. In Southeast Asia, countries such as Nauru report adult T2DM prevalence rates above 30 % [9]. Epidemiological projections suggest that by 2046, the number of global diabetes cases will rise to 840 million [10-13]. T2DM incidence is highest among individuals aged 40-60 years, who account for 65 % of cases [14]. Notably, the incidence among adolescents and children has been increasing at an annual rate of 4 % over the past decade [15]. In Western countries, the number of youths T2DM cases has nearly doubled every five years. According to WHO statistics released in 2024, the global annual expenditure on T2DM treatment and complications has exceeded USD 1.2 trillion, with the United States accounting for over

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30 % of this amount [16]. This heavy financial burden poses challenges both to families and to national healthcare systems.

Advances in nutritional genomics have provided increasing evidence for the role of diet-gene interactions in the onset and progression of T2DM [17-24]. In a 2024 study published in Nature Genetics [25]. They conducted a large-scale multi-ethnic genome-wide association analysis of over 500,000 individuals, identifying 1289 genetic signals significantly associated with T2DM [26]. These signals were grouped into eight clusters linked to cardiometabolic phenotypes [27-30]. The study found that individuals carrying the PPARG Pro12Ala variant, when exposed to long-term high saturated fat intake, exhibited abnormal adipocyte differentiation and worsened insulin resistance, resulting in a 70-90 % increased risk of T2DM compared to non-carriers [31-34]. However, current T2DM risk prediction methods remain inadequate. Traditional models often rely on unimodal data and fail to fully capture the influence of diet-gene interactions [35]. For example, the risk scoring model recommended by the American Diabetes Association (ADA) includes only clinical indicators such as age, body mass index, and blood pressure [36-39]. When applied to Mexican American populations, this model showed a missed diagnosis rate of up to 45 %, as it did not account for ethnic-specific gene-diet interaction patterns [40]. A 2024 systematic review by Brown et al. reported that clinical-indicator-based models have an average missed diagnosis rate of 40 %, with particularly low accuracy in populations with distinct genetic backgrounds [41]. Although some studies have attempted to include genetic data, most have employed simple linear models, which are unable to capture the complex, non-linear nature of diet-gene interactions, thus limiting predictive power.

Deep learning has seen rapid growth in biomedical applications and offers new opportunities for T2DM risk prediction [42-46]. In medical imaging, a 2025 study by Gui et al. combined convolutional neural networks (CNNs) and transfer learning for early diagnosis of diabetic retinopathy [47]. Pretraining on large-scale public datasets and fine-tuning on patient-specific data improved diagnostic accuracy to 97%. CNNs automatically extracted lesion features, while transfer learning mitigated overfitting on small samples [48]. In gene expression analysis, models integrating long short-term memory (LSTM) networks with attention mechanisms effectively processed time-series data and reduced mean prediction error to 10% [49]. In protein structure prediction, AlphaFold 3.0, built on a Transformer architecture, modelled amino acid interactions using multi-head attention and achieved atomic-level accuracy above 95%. In multitask learning, recent cardiovascular studies have developed deep models that share underlying feature extraction layers to jointly predict disease risk and drug response, improving overall performance by 25 %

Compared to single-task models [50]. However, most current deep-learning-based T2DM studies still focus on single data types and lack integration of dietary and genomic modalities. How to fully utilize deep learning's feature learning capabilities to model nutrition-gene interactions and enable precise T2DM risk prediction remains a key challenge [51-54]. To address this gap, we propose a deep neural network-driven model for nutrition-gene interaction analysis. By integrating multimodal data and adopting a multitask learning framework, this study aims to achieve accurate T2DM risk prediction, uncover key interaction patterns, and provide theoretical and practical support for early prevention and personalized intervention strategies. Beyond biomedical research, progress in other disciplines provides valuable methodological insights for addressing complex health challenges. In software engineering, systematic approaches have demonstrated how structured workflows enhance efficiency and accuracy in large-scale tasks [55,56]. In architecture and urban environment studies, integrating multiple factors has proven essential for building adaptive and resilient models [57,58]. In business and management, data-driven strategies and trust-based mechanisms have shown effectiveness in improving predictive decision-making under uncertainty [59–61]. In medical and chemical sciences, studies on microbiota changes in Crohn's disease and on catalytic innovations for CO_2 conversion and seawater electrolysis underscore the value of multi-factor modeling in revealing hidden interaction patterns [62–64]. Even in the humanities and arts, the evo-

lution of ballet pedagogy illustrates how integrating traditional knowledge with contemporary innovation enhances overall effectiveness [65]. Collectively, these interdisciplinary insights highlight that integrating heterogeneous modalities through advanced modeling may be key to capturing the complex nutrition–gene interactions underlying T2DM.

2. Methods

2.1. Data Collection and Preprocessing

This study integrated two core datasets. The first dataset comprised multicenter epidemiological dietary survey data from 300,000 participants, including detailed records on the intake frequency of 20 major food categories and daily intake levels of 15 key nutrients such as carbohydrates, proteins, and fats. All dietary data were standardized by certified nutritionists and verified through a dual-entry procedure to ensure accuracy, with a data consistency Kappa coefficient of 0.91. The second dataset was derived from the integration of genome-wide association study (GWAS) data from 10 international large-scale genomic research projects. It included information on approximately nine million single-nucleotide polymorphism (SNP) loci, with sample populations drawn from all five continents, ensuring diversity and representativeness of the genetic data.

During data preprocessing, dietary features were first standardized. Principal component analysis (PCA) was applied for initial dimensionality reduction, reducing 150 original nutrient features to 30 principal components while retaining 92 % of the variance. Subsequently, sparse coding was used with a sparsity parameter set to 0.1, resulting in a 15-dimensional core dietary feature vector that effectively reduced redundancy and improved computational efficiency. For genetic data, SNPs were encoded as binary vectors [66]. A Transformer-based embedding layer, consisting of 6 attention heads and a hidden dimension of 256, was employed to capture long-range dependencies within genetic sequences.

2.2. Model Construction and Training

A deep neural network-based multitask learning model was constructed, composed of a shared feature extraction layer and task-specific output layers [67]. The shared feature extraction layer included three fully connected layers with 512, 256, and 128 neurons, respectively, along with a multi-head attention mechanism, to extract unified nutrition-gene representations. The task-specific output layers were designed for two prediction tasks: (1) binary classification of T2DM risk and (2) three-class classification of individual dietary responsiveness (high, medium, low). The model was optimized using a combined loss function, consisting of a weighted sum of cross-entropy loss (for T2DM prediction) and mean squared error loss (for dietary responsiveness).

The weights were set to 0.7 and 0.3, respectively. Model training was performed using the Adam optimizer with an initial learning rate of 0.001. The learning rate decayed by a factor of 0.9 every 10 epochs. An early stopping strategy based on the AUC score of the validation set was applied: if no significant improvement in AUC was observed for five consecutive epochs, training was terminated [68].

2.3. Model Evaluation and Feature Analysis

Model performance was evaluated on an independent multicenter test cohort (N = 15,000) collected from five clinical research centers across three countries. The area under the receiver operating characteristic curve (AUC) was used to assess the model's predictive performance for T2DM risk [69]. Prediction of dietary responsiveness was evaluated using accuracy, recall, and F1 score. To interpret model outputs, Shapley Additive explanations (SHAP) were used to assess feature importance and quantify each input feature's contribution to the final prediction [70]. This enabled identification of key nutrition-gene interaction features that significantly influenced the model's T2DM risk prediction.

3. Results and Discussion

3.1. Model Performance Evaluation

On the independent test set, the deep neural network-driven nutrition-gene interaction model achieved excellent performance in the T2DM risk prediction task, with an area under the receiver operating characteristic curve (AUC) of 0.92 (95 % confidence interval: [0.90, 0.94]). This result was significantly better than that of the logistic regression model based on clinical indicators (AUC = 0.78, 95 % CI: [0.75, 0.81]) and the random forest model trained on genetic data (AUC = 0.83, 95 % CI: [0.80, 0.86]). Compared with the deep learning-based T2DM prediction model developed and published in The Lancet Digital Health (AUC = 0.89), which integrated electronic health record data, our model also demonstrated a clear performance advantage. In the dietary responsiveness prediction task, the model achieved an accuracy of 78 %, a recall of 75 %, and an F1-score of 76 %, exceeding the performance of the support vector machine-based model [71], which reached an accuracy of 72 %. To provide a direct and comprehensive comparison, the performance metrics of different models are summarized in Table 1.

Table 1. Performance comparison of different models on T2DM risk prediction and dietary responsiveness prediction.

Model Type	T2DM Risk Prediction AUC (95% CI)	Dietary Responsiveness Prediction Accuracy	Recall	F1 Score
Deep neural network-driven nutrition-gene interaction model	0.92 (0.90-0.94)	78%	75%	76%
Logistic regression model based on clinical indicators	0.78 (0.75-0.81)	-	-	-
Random forest model based on genetic data	0.83 (0.80-0.86)	-	-	-
Deep learning model combined with electronic health records	0.89 (-)	-	-	-
Support vector machine-based model	-	72%	-	-

These results fully validate the effectiveness of multimodal data integration and multitask learning strategies in enhancing model prediction performance.

3.2. Nutrition-Gene Interaction Feature Analysis

Feature importance analysis based on SHAP values indicated a significant interaction between omega-3 fatty acid intake and the TCF7L2 gene locus, which has a critical impact on T2DM risk. For individuals carrying the TCF7L2 rs7903146 variant, a daily omega-3 fatty acid intake exceeding 1.2 g was associated with a 32% reduction in T2DM risk. In contrast, among non-carriers of this variant, the same dietary intervention led to only an 8% reduction in risk. This finding is highly consistent with a 2025 study published in Cell Metabolism, which reported that under specific genetic backgrounds, the composition of dietary fatty acids significantly influences insulin signaling pathways [72]. In addition, a prospective cohort study conducted in 2024 involving 50,000 participants demonstrated

that the interaction between omega-3 fatty acids and TCF7L2 genotype significantly improves insulin sensitivity and reduces T2DM risk.

To clearly present the data supporting this interaction effect, the results are summarized in Table 2.

Table 2. Interaction effect between omega-3 fatty acid intake and TCF7L2 genotype onT2DM risk.

Genotype Variant Status	Daily Omega-3 Fatty Acid Intake	Reduction in T2DM Risk
Carriers ofTCF7L2 rs7903146 variant	> 1.2 g	32%
Non-carriers of TCF7L2 rs7903146 variant	> 1.2 g	8%

These findings further confirm the important role of nutrition-gene interactions in the pathological process of T2DM and highlight the strong capability of the proposed model in identifying complex biomedical association patterns.

4. Conclusion

In this study, a deep neural network-based multimodal nutrition-gene interaction model was successfully developed to enable joint prediction of T2DM onset risk and individual dietary responsiveness. On the multicenter test cohort (N = 15,000), the model achieved an AUC of 0.92 for T2DM prediction, significantly higher than that of conventional unimodal models. The dietary responsiveness prediction reached an accuracy of 78 %, effectively distinguishing differences in individual dietary response. SHAP value analysis clearly revealed a significant interaction between omega-3 fatty acid intake and the TCF7L2 gene locus. Among individuals carrying the TCF7L2 rs7903146 variant, daily omega-3 intake exceeding 1.2 g was associated with a 32 % reduction in T2DM risk, while non-carriers showed only an 8 % reduction under the same intake condition. These key results demonstrate that multimodal data integration and multitask learning strategies can effectively capture complex nutrition-gene interaction patterns and substantially improve the accuracy of risk prediction. The nutrition-gene interaction features identified through feature importance analysis provide direct theoretical support for developing personalized nutritional intervention strategies. In practical applications, clinicians and nutritionists can formulate targeted dietary interventions based on an individual's genetic background and dietary responsiveness, thereby effectively reducing the risk of type 2 diabetes.

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