Brief Report
Volume 13:02, 2025

Journal of Clinical & Medical Genomics

ISSN: 2472-128X

Open Access

Integrating Polygenic Risk Scores and Environmental Factors for Early Diagnosis of Type 2 Diabetes

Chung Fang*

Department of Post-Baccalaureate Medicine, National Chung Hsing University, Taichung, Taiwan

Introduction

Type 2 Diabetes Mellitus (T2D) has emerged as one of the most pressing global health concerns of the 21st century. Characterized by insulin resistance and relative insulin deficiency, T2D leads to chronic hyperglycemia and is associated with long-term damage to various organs, particularly the eyes, kidneys, nerves, heart, and blood vessels. The World Health Organization estimates that hundreds of millions of individuals worldwide are currently living with T2D, and this number is projected to rise sharply in the coming decades. T2D does not only carry a heavy toll in terms of morbidity and mortality but also imposes substantial economic costs on healthcare systems. The disease has traditionally been considered preventable through lifestyle interventions; however, early detection remains a crucial strategy for effective management. Delayed diagnosis often results in complications that could otherwise have been mitigated. Therefore, the scientific community continues to explore more accurate and predictive diagnostic methods. In recent years, the integration of genetic tools, particularly Polygenic Risk Scores (PRS), with traditional environmental and lifestyle factors has garnered increasing attention as a potential breakthrough in predicting and diagnosing T2D at an earlier stage [1].

Description

Polygenic risk scores are composite indicators derived from the cumulative effects of multiple genetic variants associated with a particular disease. In the case of T2D, Genome-Wide Association Studies (GWAS) have identified hundreds of Single-Nucleotide Polymorphisms (SNPs) that modestly contribute to disease risk. While each SNP may have only a small effect individually, their collective contribution can be significant. PRS synthesizes this information into a single numerical value that reflects an individual's inherited predisposition to T2D. As such, PRS can stratify individuals according to their genetic risk and potentially predict the likelihood of developing the disease before clinical symptoms emerge. The utility of PRS lies in its capacity to personalize risk prediction, which is particularly valuable for a heterogeneous disease like T2D. However, genetic risk alone does not account for the entirety of T2D susceptibility. Environmental exposures, lifestyle choices, and socio-economic factors also play substantial roles in modulating disease risk. Thus, PRS should not be viewed in isolation but rather as a complementary component of a more comprehensive risk assessment strategy [2].

Environmental and behavioral factors contributing to T2D include diet, physical activity, smoking, alcohol consumption, socioeconomic status, and exposure to pollutants. These factors interact with an individual's genetic makeup to influence metabolic processes and insulin sensitivity. For instance, a person with a high polygenic risk for T2D may never develop the disease if they maintain a healthy lifestyle and avoid environmental triggers. Conversely, individuals with a low genetic predisposition may still acquire T2D if they are exposed to significant environmental risks. The interplay between genes and the environment underscores the multifactorial nature of T2D and highlights the necessity of integrating both domains into predictive models. One promising approach to this integration is the use of the exposome framework, which captures the totality of environmental exposures over a lifetime and examines how these exposures interact with the genome. When applied to T2D, the exposome framework facilitates a more nuanced understanding of disease etiology and enables the development of risk prediction models that reflect real-world complexity [3].

Several studies have demonstrated the added predictive value of incorporating environmental data into genetic risk models. For example, research has shown that when PRS are combined with lifestyle and demographic factors, the accuracy of T2D risk prediction improves significantly. One widely cited study used data from the UK Biobank to demonstrate that individuals with high PRS who also led unhealthy lifestyles had more than tenfold higher risk of developing T2D compared to those with low PRS and healthy behaviors. Importantly, the study also showed that adherence to healthy lifestyle practices could partially offset genetic risk, providing empirical support for targeted preventive strategies. Such findings underscore the importance of considering both nature and nurture in T2D diagnostics and lend credence to the potential of integrated models for early intervention. Furthermore, early identification of high-risk individuals allows for timely lifestyle interventions, medical surveillance, and pharmacological measures that can delay or prevent disease onset [4]. From a methodological standpoint, integrating PRS and environmental factors presents several challenges. Firstly, both genetic and environmental data need to be of high quality and adequately harmonized. GWAS data require rigorous preprocessing to ensure that only robust and replicable SNPs are included in the PRS. Similarly, environmental data-often collected via self-report or indirect proxies-must be validated and standardized to ensure reliability. Secondly, statistical models must account for complex interactions between variables. Traditional regression models may be inadequate for capturing non-linear relationships and higher-order interactions. Machine learning and artificial intelligence techniques are increasingly being employed to address these limitations. Algorithms such as random forests, support vector machines, and neural networks offer enhanced capacity to model multifaceted relationships and improve predictive accuracy. These models can also incorporate time-varying data, which is particularly relevant for environmental exposures that fluctuate over an individual's lifespan [5].

Conclusion

In conclusion, the integration of polygenic risk scores and environmental factors represents a significant advancement in the early diagnosis and prevention of type 2 diabetes. This multifactorial approach reflects the complex interplay between genetic predisposition and environmental exposures that underlie disease development. By combining genomic data with lifestyle and environmental information, healthcare providers can more accurately identify individuals at risk, implement personalized prevention

***Address for Correspondence:** Chung Fang, Department of Post-Baccalaureate Medicine, National Chung Hsing University, Taichung, Taiwan; E-mail: fang.chung@gmail.com*

Received: 01 April, 2025, Manuscript No. JCMG-25-165759; **Editor assigned:** 03 April, 2025, Pre QC No. P-165759; **Reviewed:** 17 April, 2025, QC No. Q-165759; **Revised:** 22 April, 2025, Manuscript No. R-165759; **Published:** 29 April, 2025, DOI: 10.37421/2472-128X.2025.13.328

strategies, and ultimately reduce the incidence and burden of T2D. Despite methodological and ethical challenges, the potential benefits of integrated risk models are substantial. Continued investment in research, data infrastructure, and interdisciplinary collaboration will be essential to realize this potential. As the field of precision medicine evolves, the integration of PRS and environmental data stands poised to transform the landscape of T2D diagnostics and pave the way for more effective, equitable, and proactive healthcare

Acknowledgment

None.

Conflict of Interest

None.

References

1. Koyama, Satoshi, Kaoru Ito, Chikashi Terao and Masato Akiyama, et al. "Population-specific and trans-ancestry genome-wide analyses identify distinct and shared genetic risk loci for coronary artery disease." *Nat Genet* 52 (2020): 1169-1177.
2. Lewis, Cathryn M. and Evangelos Vassos. "Polygenic risk scores: From research tools to clinical instruments." *Genome Med* 12 (2020): 44.
3. O'Sullivan, Jack W., Anna Shcherbina, Johanne M. Justesen and Mintu Turakhia, et al. "Combining clinical and polygenic risk improves stroke prediction among individuals with atrial fibrillation." *Circ Genom and Precis Med* 14 (2021): e003168.
4. Gray, Michael P., Yemima Berman, Giordano Bottà and Stuart M. Grieve, et al. "Incorporating a polygenic risk score-triaged coronary calcium score into cardiovascular disease examinations to identify subclinical coronary artery disease (ESCALATE): Protocol for a prospective, nonrandomized implementation trial." *AmHeart J* 264 (2023): 163-173.
5. Saya, Sibel, Lucy Boyd, Patty Chondros and Mairead McNamara, et al. "The SCRIPT trial: Study protocol for a randomised controlled trial of a polygenic risk score to tailor colorectal cancer screening in primary care." *Trials* 23 (2022): 810.

How to cite this article: Fang, Chung. "Integrating Polygenic Risk Scores and Environmental Factors for Early Diagnosis of Type 2 Diabetes." *J Clin Med Genomics* 13 (2025): 329.