Genetic Variants Associated with Susceptibility to Type 2 Diabetes in a South Asian Population

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Introduction

Genetic variants associated with susceptibility to type 2 diabetes (T2D) in South Asian populations have gained significant attention due to the rising prevalence of diabetes in these communities. South Asians, including individuals from India, Pakistan, Bangladesh, Sri Lanka and Nepal, exhibit unique genetic characteristics that influence their predisposition to T2D [1]. This report aims to explore the current understanding of genetic variants linked to T2D susceptibility in South Asians, highlighting key studies, genetic loci and implications for public health and personalized medicine.

T2D is a complex metabolic disorder influenced by both genetic and environmental factors. South Asians are particularly vulnerable to T2D, often presenting with the disease at a younger age and at lower levels of obesity compared to other populations [2]. This early onset and high prevalence suggest a strong genetic component in addition to lifestyle factors such as diet and physical activity. Genome-wide association studies (GWAS) have been instrumental in identifying genetic variants associated with T2D risk across different populations, including South Asians. These studies involve scanning the entire genome to pinpoint regions where genetic variations (single nucleotide polymorphisms, or SNPs) are more common among individuals with T2D compared to healthy controls.

Several GWAS conducted specifically in South Asian populations have identified significant genetic loci associated with T2D susceptibility. For instance, studies have highlighted variants in genes involved in insulin secretion (e.g., TCF7L2, CDKAL1) and insulin sensitivity (e.g., KCNQ1, PPARG). These genes play crucial roles in pancreatic beta-cell function, glucose metabolism and adipocyte differentiation, pathways central to T2D pathophysiology. An example of a notable genetic variant in South Asians is the TCF7L2 gene, which has consistently shown strong association with T2D risk across multiple studies [3]. The T allele of the rs7903146 SNP within TCF7L2 is significantly more prevalent in individuals with T2D than in healthy controls. This variant is believed to impair insulin secretion and increase susceptibility to T2D, highlighting its potential as a biomarker for risk assessment and personalized interventions in South Asian populations.

Beyond individual SNPs, studies have also explored genetic risk scores (GRS) combining multiple variants to assess overall genetic predisposition to T2D. GRS have shown promise in predicting T2D risk in South Asians, facilitating early identification and targeted interventions for high-risk individuals. Integrating genetic information with clinical and lifestyle factors could enhance risk stratification and improve preventive strategies tailored to the unique genetic profiles of South Asian populations. Despite significant progress, challenges remain in translating genetic discoveries into clinical

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practice. Variants identified through GWAS often have modest effect sizes individually, necessitating large sample sizes to achieve sufficient statistical power. Furthermore, genetic studies in South Asians have been relatively limited compared to populations of European ancestry, highlighting the need for more diverse and representative research cohorts.

Description

Ethnic diversity in genetic studies is crucial for understanding the full spectrum of genetic risk factors contributing to T2D across different populations. Studies focusing on South Asians have begun to address this gap, identifying novel genetic variants and providing insights into the underlying biological mechanisms of T2D in these populations [4]. Collaborative efforts across international research consortia have enabled meta-analyses that consolidate findings from diverse populations, enhancing the robustness and generalizability of genetic associations.

In addition to genetic research, epigenetic factors and gene-environment interactions also play pivotal roles in T2D susceptibility. Epigenetic modifications, such as DNA methylation and histone acetylation, can influence gene expression patterns related to glucose metabolism and insulin sensitivity. These epigenetic changes may be influenced by environmental factors such as diet, physical activity and exposure to pollutants, contributing to the complex interplay between genetics and environment in T2D etiology.

The clinical implications of genetic variants associated with T2D susceptibility in South Asians extend beyond risk prediction to personalized management strategies. Genetic testing could potentially identify individuals at high risk for T2D at an earlier stage, prompting targeted lifestyle interventions or pharmacological treatments to prevent or delay disease onset. However, ethical considerations regarding genetic privacy, informed consent and equity in healthcare access must be carefully addressed to ensure responsible implementation of genetic information in clinical practice [5]. Furthermore, population-specific genetic studies have the potential to uncover novel therapeutic targets for T2D tailored to the genetic profiles of South Asians. Pharmacogenomic research aims to identify genetic variants that influence individual responses to antidiabetic medications, guiding personalized treatment approaches to optimize therapeutic outcomes and minimize adverse effects.

Conclusion

In conclusion, genetic variants associated with susceptibility to type 2 diabetes in South Asian populations represent a growing area of research with important implications for public health and personalized medicine. Advances in genomics, combined with epidemiological and clinical data, are shedding light on the complex genetic architecture of T2D and informing strategies for prevention, early detection and management in diverse populations. Continued interdisciplinary collaboration and large-scale genomic studies are essential to unraveling the genetic basis of T2D and translating these discoveries into tangible benefits for individuals and healthcare systems worldwide.

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References

- Unnikrishnan, Ranjit, Ranjit Mohan Anjana and Viswanathan Mohan. "Diabetes mellitus and its complications in India." Nat Rev Endocrinol 12 (2016): 357-370.
- Kesavachandran, Chandrasekharan Nair, Vipin Bihari and Neeraj Mathur. "The normal range of body mass index with high body fat percentage among male residents of Lucknow city in north India." *Indian J Med Res* 135 (2012): 72-77.
- Phani, Nagaraja M., Prabha Adhikari, Shivashankara K. Nagri and Sydney C. D'Souza, et al. "Replication and relevance of multiple susceptibility loci discovered from genome wide association studies for type 2 diabetes in an Indian population." *PLoS One* 11 (2016): e0157364.
- 4. Sarhangi, Negar, Farshad Sharifi, Leila Hashemian and Maryam Hassani Doabsari,

et al. "PPARG (Pro12Ala) genetic variant and risk of T2DM: a systematic review and meta-analysis." *Sci Rep* 10 (2020): 12764.

 Obuchowski, Nancy A. and Jennifer A. Bullen. "Receiver operating characteristic (ROC) curves: review of methods with applications in diagnostic medicine." *Phys Med Biol* 63 (2018): 07TR01.

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