INTEGRATING GENETIC SUSCEPTIBILITY AND AIR POLLUTION EXPOSURE TO PREDICT ASTHMA RISK IN AN URBAN KAZAKH POPULATION

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Background

Bronchial asthma (BA) is a complex respiratory disease resulting from interactions between genetic susceptibility and environmental exposures [1]. Air pollutants contribute to BA via oxidative stress and inflammation, especially in genetically susceptible individuals [2]. This study aimed to investigate genetic susceptibility to BA in an urban Kazakh population by integrating genome-wide SNP genotyping with environmental exposure data to enhance personalized risk prediction.

Methods

A case-control study was conducted in Almaty involving 288 participants: 144 asthma patients and 144 matched controls. Genome-wide genotyping was performed using the Infinium Global Screening Array-24 v3.0 on an iScan platform. SNP associations were analyzed using PLINK and annotated via ClinVar, dbSNP, and 1000 Genomes. Environmental exposure metrics ($PM_{2.5}$, PM_{10}, SO_2) were monitored digitally over 7 months. Gene-environment interactions were evaluated to determine pollutant-modulated SNP effects.

Results

Several SNPs demonstrated significant associations with BA risk and specific pollutant exposures. Protective variants included rs12413578 in *GSDMB* (OR = 0.24, 95% CI: 0.09- 0.67), rs907092 in *IKZF3* (OR = 0.59, 95% CI: 0.40-0.86), and rs17293632 in *SMAD3* (OR = 0.26, 95% CI: 0.09-0.75) under elevated PM_{10} exposure. Increased risk was observed for rs1837253 in *TSLP* (OR = 1.67, 95% CI: 1.03-2.69) in relation to $PM_{2.5}$, and rs1295686 in IL13 (OR = 0.22, 95% CI: 0.06-0.85) showed a protective effect under SO_2 exposure.

Conclusions

This study identified several SNPs significantly associated with asthma risk in Almaty population, including *GSDMB* rs12413578 and *IKZF3* rs907092 as protective variants, and *TSLP* rs1837253 as a $PM_{2.5}$ dependent risk marker for patients with BA. Additionally, *SMAD3* and *IL13* variants showed pollutant-specific associations with PM_{10} and SO_2, respectively. The proposed integration of gene-environment interactions into risk models may enhance asthma prediction and inform more targeted strategies in precision medicine.

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Key words: bronchial asthma, gene–environment interaction, SNP genotyping, air pollutants.

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