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Analysis of Environmental Risks and Genetic Predisposition to the Development of Bronchial Asthma among Residents of Almaty

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Abstract

Air pollution is one of the most significant challenges of our time, impacting both climate change and public health by increasing morbidity and mortality. Climate change has led to the formation and spread of air pollutants such as fine particulate matter (PM2.5) and oxidizing gases, including O3 and NO2, which can exacerbate asthma [1].

Bronchial asthma (BA) is a chronic respiratory condition affecting over 300 million people globally, with its development influenced by both environmental factors and genetic predisposition [2]. In Kazakhstan, the incidence of asthma has been rising, with over 105,365 individuals registered in 2018 [3]. Epidemiological research shows that asthma is prevalent in Almaty due to ambient air pollution [4].

This research aims to assess the genetic and environmental risk factors contributing to bronchial asthma severity in Almaty and its surrounding region. Genome-wide association studies (GWAS) is performed to identify genetic polymorphisms linked to asthma. The odds ratio (OR) is computed to quantify the association between elevated pollutant levels and the likelihood of developing asthma. By comparing the frequency of asthma cases in areas with high pollutant levels to those in areas with lower levels, we aim to determine the extent to which air pollution contributes to asthma risk.

This involves 103 DNA samples from individuals with allergic diseases and 108 control samples, utilizing the Infinium® ImmunoArray-24 v2.0 BeadChip Kit and the Infinium Global Screening Array-24 Kit. Data on PM2.5 and oxidizing gases like NO2 and SO2 is obtained from real-time air quality sources, such as AirKaz, based on the patients' residences. Statistical analyses include Student's t-test, Hardy-Weinberg equilibrium compliance, and odds ratio (OR) assessment. Data processing is done using Illumina GenomeStudio v.2010.3 software.

The GWAS results from 45 individuals identified several significant genetic loci associated with bronchial asthma. Notable genes include IL13 (rs20541), IL12B (rs2569254, rs3212227), and IL4R (rs1805015). Preliminary findings suggest that among patients, the IL12B rs3212227 variant is positively associated with asthma (OR = 11.78, (95% CI = 5.43–25.54, $p < 0.001$). A similar positive association was observed for IL13 rs20541 (OR = 4.46, 95% CI = 2.54–7.83, $p < 0.001$). Conversely, the rs2569254 variant is less associated with asthma, showing an (OR=1.79, 95% CI = 0.41–1.08, $p = 0.023$). No significant association was found between the IL4R rs1805015 polymorphism and asthma.

The analysis revealed that individuals carrying the previously noted genetic variants were more likely to experience persistent asthma symptoms in areas with higher concentrations of air pollutants, particularly PM2.5, NO2, and SO2. The mean (SD) concentrations of PM2.5, NO2, and SO2 were 20-50 μg/m³, 40-60 μg/m³, and 10-20 μg/m³, respectively. These pollutants were associated with increased asthma incidence (PM2.5 OR 1.20-1.50, NO2 OR 1.05-1.60, SO2 OR 1.30-1.60) and the data we obtained align with the results reported in other research [5].

A complete genome-scale bioinformatic analysis will continue in frame of the project “Analysis of environmental risks and genetic predisposition to the development of bronchial asthma among residents of Almaty and the Almaty region” (AP23488865), funded by the Ministry of Science and High Education of the Republic of Kazakhstan.

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