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Exploring the Role of DRD2 and OPRM1 Gene Polymorphisms in Alcohol Dependence Risk

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Introduction

Alcohol dependence is influenced by a complex interplay of genetic, environmental, and psychological factors. Among the genetic contributors, polymorphisms in the dopamine D2 receptor gene (DRD2) and the mu-opioid receptor gene (OPRM1) have garnered considerable attention [1-5]. The DRD2 Taq1A polymorphism is associated with altered receptor density, while the OPRM1 A118G variant affects the binding affinity of beta-endorphins. Both genes play critical roles in reward processing, craving, and reinforcement. This study explores the association between DRD2 and OPRM1 polymorphisms and susceptibility to alcohol dependence, aiming to clarify genetic risk profiles and their potential application in personalized addiction treatment [6-10].

Discussion

A cohort of 300 individuals with alcohol dependence and 300 matched controls were genotyped for DRD2 Taq1A and OPRM1 A118G variants. Results showed a higher frequency of the A1 allele of DRD2 and the G allele of OPRM1 in the alcohol-dependent group. These allelic variations correlated with increased craving scores and earlier onset of alcohol use. Functional implications include reduced dopamine receptor availability and heightened opioid reward sensitivity, potentially predisposing individuals to excessive alcohol consumption. Gene-environment interactions, such as stress exposure and childhood trauma, further modulated risk. While genetic markers alone are not deterministic, they offer valuable insight into individual vulnerability and treatment response. Pharmacogenetic implications include enhanced naltrexone efficacy in OPRM1 G allele carriers.

Conclusion

DRD2 and OPRM1 polymorphisms contribute to the biological vulnerability to alcohol dependence. Genetic screening may enhance early identification of at-risk individuals and inform tailored treatment strategies, advancing the field of personalized addiction medicine.

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