ISSN : 2466-0744

MULTIDISCIPLINE PROCEEDINGS OF

# DIGITAL FASHION CONFERENCE

KOREA, REPUBLIC OF

# Multidiscipline Proceedings of

# DIGITAL FASHION CONFERENCE

**September 2025** *(Volume 5, No.5)*


Available at digitalfashionsociety.org
Published:
서울 합정역
파주출판도시
ISSN 2466-0744
Seoul
Rebuplic of Korea (ROK),

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# THE ROLE OF THE SOD2 GENE ALA16VAL POLYMORPHISM IN THE DEVELOPMENT OF ARTERIAL HYPERTENSION

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*Abstract: This study explores the link between the Ala16Val polymorphism of the SOD2 gene and arterial hypertension. In a case-control design 96 patients, 95 controls, the Ala/Val genotype was associated with a higher hypertension risk OR: 2.0 overall; 2.8 for grade 3, while the Ala/Ala genotype showed a protective effect. These findings enhance understanding of hypertension genetics and support personalized approaches to diagnosis and treatment.*

*Keywords: superoxide dismutase 2, Ala16Val polymorphism, arterial hypertension, genetic predisposition, oxidative stress, antioxidant enzymes, cardiovascular genetics, single nucleotide polymorphism, hypertension pathogenesis, personalized medicine*

Today, arterial hypertension is a major global health issue, affecting about one billion people and contributing significantly to cardiovascular disease and mortality. Its complex etiology involves both environmental and genetic factors, with oxidative stress playing a key role. Superoxide dismutase 2 (SOD2), a mitochondrial antioxidant enzyme, protects cells by converting superoxide radicals into hydrogen peroxide and oxygen. The Ala16Val polymorphism - an alanine-to-valine substitution in the mitochondrial targeting sequence - alters enzyme activity and mitochondrial import, reducing antioxidant defense. This variation may promote oxidative stress, vascular dysfunction, and hypertension. Understanding this association is crucial for personalized medicine and targeted therapies in cardiovascular care.

The investigation of superoxide dismutase 2 gene Ala16Val polymorphism in hypertension development reveals significant associations between specific genotypes and disease susceptibility. The heterozygous Ala/Val genotype demonstrates the strongest correlation with hypertension risk, occurring in 59.4% of hypertensive patients compared to 42.1% in healthy controls. This genotype confers a two-fold increased risk for hypertension development, with statistical significance confirmed through odds ratio analysis. The mechanism underlying this association involves altered mitochondrial targeting efficiency, where the valine substitution impairs the normal transport of the enzyme precursor across mitochondrial membranes, resulting in reduced antioxidant capacity and increased oxidative stress. The protective effect of the wild-type Ala/Ala genotype presents compelling evidence for its role in hypertension prevention. This genotype occurs significantly less frequently in hypertensive patients compared to controls, with frequencies of 21.9% versus 34.7% respectively. The protective odds ratio of 0.5 indicates that individuals carrying the Ala/Ala genotype have approximately half the risk of developing hypertension compared to those with other genotypes. This protective effect likely results from optimal superoxide dismutase 2 activity, maintaining cellular redox homeostasis and preventing oxidative damage to vascular structures. Grade-specific analysis reveals a dose-dependent relationship between the Ala/Val genotype and hypertension severity. While grade 1 hypertension shows a modest association with the heterozygous genotype, grade 3

hypertension demonstrates a pronounced correlation, with the Ala/Val genotype occurring in 66.7% of patients compared to 42.1% in controls. This represents a 2.8-fold increased risk for severe hypertension, suggesting that the genetic variant contributes not only to hypertension development but also to disease progression and severity.The relationship between genotype and disease severity indicates that oxidative stress mechanisms become increasingly important in advanced hypertensive states. The homozygous Val/Val genotype presents interesting findings, occurring less frequently in hypertensive patients than expected, suggesting a complex interaction between genetic variants and disease susceptibility. This observation indicates that the heterozygous state may represent an optimal condition for disease development, possibly through intermediate enzyme activity levels that neither provide full protection nor complete vulnerability to oxidative stress.

The SOD2 Ala16Val polymorphism contributes to arterial hypertension through oxidative stress and impaired antioxidant function. The Ala/Val genotype increases hypertension risk, especially in severe cases, while Ala/Ala appears protective. These findings highlight its role in hypertension pathogenesis and potential for risk assessment and personalized therapy. Future research should examine its molecular effects, interactions with other genes and environment, and inform targeted interventions for prevention and management.

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