

# **Haplotype analysis of CYP 1A1 M1 (3' UTR) and M2 (exonic) polymorphisms in the susceptibility and drug response variation in vitiligo- A report from India**

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## **Abstract**

Vitiligo, a common disfiguring dermatological disorder, appears as milky white patches on the affected region of the skin. The primary pathogenic event in the causation was reported to be oxidative stress followed by autoimmunity. Cytochrome P450 (CYPs) play a major role in the metabolism of xenobiotics, drugs and other toxic intermediates and supports in cell maintenance and survival. The study aims to evaluate the association of CYP 1A1-M2 and -M1 polymorphisms in susceptibility and drug response variation. Genomic DNA was isolated from blood samples of 455 subjects of Indian origin and genotyped using PCR. The analysis revealed a significantly higher percent of patients to be fast-responders (80.6%) than slow-responders (32.0%). The genotype analysis revealed CYP1A1 M1 CC vs. CT+TT genotype in susceptibility and fast drug response and CYP1A1M1 TT vs. CT+CC was associated with protection towards vitiligo and slow drug response. Further haplotype analysis of M1 and M2 revealed CTAG block significantly associated in susceptibility and CCAA block with fast drug response. To our knowledge this is the first report that contributes to the role of CYP in vitiligo susceptibility and drug response variation. Studies in different ethnic population with large sample size are warranted to establish current findings.

## **Biography**

Surekha Tippisetty was awarded with PhD from Osmania University and credited with 5 international publications and more than 8 national and international conference presentations from academic research. She has 4 years of corporate experience in Pharmaceutical industry as Medical Writer. Currently, she is pursuing Postdoctoral study from Osmania University with high interest to expand her research activities in understanding the underlying mechanisms and various intrinsic and extrinsic factors effecting vitiligo pathogenesis and drug response variation.

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