Vitiligo is a common skin disorder affecting approximately 1-2% of world's population. In India Gujarat and Rajasthan states have the highest prevalence of vitiligo i.e., ~8.8%. Vitiligo is characterized by circumscribed depigmented patches resulting from the loss of functional melanocytes from the epidermis. Its onset may occur at any age, but the incidence usually peaks in the second and third decades of life. Although vitiligo might be viewed as a minor disorder, its impact on patient's self-esteem and social interactions can be devastating.

The aim of this study was to assess the genetic, oxidative stress and autoimmune hypotheses in the pathogenesis of vitiligo patients of Gujarat. Oxidative stress hypothesis in vitiligo patients was assessed by estimating lipid peroxidation (LPO) levels, glutathione peroxidase (GPx) activity, catalase activity and superoxide dismutase (SOD) activity. For the genetic association of vitiligo, C⟶T SNP in glutathione peroxidase gene at codon 198, an established marker for lung cancer, and T⟶C SNP in exon 9 of the catalase gene, another established marker for vitiligo in Caucasian population were studied. Also the SOD 1 protein levels were estimated by western hybridization to establish a relation between protein levels and increased activity of this enzyme. Autoimmune hypothesis was assessed by estimating the antimelanocyte antibodies in plasma of patients as compared to controls by ELISA.

The results obtained from the study of 63 patients and 69 control subjects suggest a significantly high oxidative stress in the patients compared to controls. Erythrocyte GPx activity was found to be significantly decreased, significant increase was seen in erythrocyte SOD activity, however there was no significant change in erythrocyte catalase activity in vitiligo patients compared to controls. C⟶T SNP in glutathione peroxidase exon 2 was found to be associated with vitiligo but no association was found with T ⟶ C SNP of Catalase in Gujarat vitiligo patients. Erythrocyte SOD protein levels did not change though its activity was found to be significantly increased in vitiligo patients, suggesting that mutation/s in the exonic regions of the *sod1* gene may be responsible for the increased activity. 71% of Gujarat vitiligo patients exhibited antimelanocyte antibodies in their plasma.

Thus our results point out that oxidative stress and auto-antibodies are the possible factors that could lead to the death of melanocytes and precipitate vitiligo in patients of Gujarat.