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Synergistic Role of Oxidative Stress and MIF Polymorphisms in Vitiligo Risk

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ABSTRACT

Vitiligo is characterized by melanocyte loss, where oxidative stress and genetic factors can contribute to the risk of disease. Elevated levels of oxidative stress biomarkers such as malondialdehyde (MDA) and protein carbonyl (PC) indicate lipid and protein oxidation, respectively, while macrophage inhibitory factor (MIF) gene polymorphisms may determine the individual susceptibility to disease risk. This study included 110 patients with vitiligo and 110 controls. Serum MDA and PC levels were measured using a spectrophotometer. Genetic polymorphism in the MIF-173 G>C (rs755622 G>C) gene was determined by performing PCR-RFLP. Group differences were analyzed using the Mann-Whitney U test; genetic associations were assessed by odds ratio (OR) with 95% confidence intervals (CI). Patients with GC genotype showed significantly higher MDA [1.05 (0.80-1.22) µmol/L] and PC [2.50 (1.59-3.64) nmol/mg protein] levels compared to the GG genotype [MDA: 0.62 (0.40-0.80); PC: 2.05 (1.36-3.18)]. The CC genotype exhibited the highest PC levels [5.00 (3.07-8.07)]. The GC genotype was more frequent in patients (59.1%) than in controls (24.5%), conferring an increased risk of vitiligo (OR = 4.9; 95% CI: 2.69-8.61; $p < 0.001$). However, the CC genotype association was not statistically significant. Increased oxidative damage, especially in GC and CC genotypes, together with the allelic variations in the MIF-173 gene, may contribute to enhanced vitiligo susceptibility, highlighting the interplay of oxidative stress and genetic predisposition.

Key words: Vitiligo, oxidative stress, macrophage inhibitory factor, genetic susceptibility

INTRODUCTION

Vitiligo is a chronic disorder characterized by the appearance of sharply demarcated depigmented patches on the skin. The condition affects an estimated 0.5-2% of individuals worldwide and develops as a result of multifactorial interactions among environmental triggers, metabolic imbalance, immune dysregulation, and susceptible genotypes (Rodrigues *et al*., 2017; Boniface *et al*., 2018). Among its subtypes, non-segmental vitiligo (NSV) is the most common and is characterized by symmetrical progressive lesions. Histological and immunological evidence suggests that both innate and adaptive immune responses, coupled with redox imbalance, contribute significantly to melanocyte destruction (Rashighi and Harris, 2017; Picardo and Taïeb, 2019). Oxidative stress is considered a key initiator in vitiligo pathogenesis. Elevated levels of reactive oxygen species (ROS) may damage the melanocytes, leading to lipid peroxidation and protein oxidation. Malondialdehyde (MDA), an end-product of lipid peroxidation, and protein carbonyls (PC), a stable marker of protein oxidation, are widely recognized as reliable indicators of oxidative damage (Perumal and Manoharan, 2025). Their accumulation not only damages cellular structures but also triggers inflammatory pathways that perpetuate melanocyte loss.

MIF is an upstream regulator of both oxidative and inflammatory cascades, and its overexpression has been linked to

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inflammatory conditions, such as vitiligo (Farag *et al*., 2018). Functionally relevant promoter polymorphisms in the MIF gene, such as the -173 G>C SNP, are known to influence transcriptional activity and cytokine expression (Hassan *et al*., 2022). Previous studies have reported associations between this gene variant and increased susceptibility and severity in autoimmune disorders (Castañeda-Moreno *et al*., 2018; Illescas *et al*., 2018), suggesting its potential contribution to the risk of vitiligo. Despite the growing body of data, few studies have systematically integrated oxidative stress markers and genetic susceptibility factors within the same cohort. This was the first study to simultaneously assess serum MDA and PC levels, along with the MIF (-173 G>C) polymorphism, in patients from North Indian Haryana state.

This combined approach provided new insights into the interplay between oxidative damage and host genetic background, offering a more comprehensive understanding of vitiligo pathogenesis and potential biomarkers for disease risk assessment.

MATERIALS AND METHODS

A total of 220 individuals were enrolled, comprising 110 patients with a clinical diagnosis of vitiligo, recruited from the Department of Dermatology, PGIMS Rohtak, Haryana, and 110 clinically healthy participants. None of the controls or their first-degree relative had a history of vitiligo. All participants were e" 18 years of age and included both males and females. Exclusion criteria for both groups included the presence of infectious, autoimmune, or other immunological disorders, as well as pregnancy or lactation.

Venous blood (5 ml) was drawn from each participant for biochemical and genetic analyses by a trained laboratory technician using K, EDTA-coated vacutainer tubes. All samples were transported to the laboratory under cold conditions in an ice box to ensure sample stability.

Venous blood (5 ml) was collected in plain vacutainer tubes and allowed to clot at room temperature for a few minutes. Centrifuged the samples at 3500 rpm for 12 min and the resulting serum was carefully aspirated into pre-labelled tubes. Serum aliquots were stored at -20°C until analysis, and hemolyzed samples were excluded.

For molecular analysis, 3 ml of blood was reserved for DNA extraction. Later was isolated from 600 µl of whole blood using the phenol-chloroform-isoamyl alcohol (PCI) method (24:25:1), following standard protocols. DNA quality and purity were assessed spectrophotometrically by determining the A260/A280 values within 1.8 and 2.0, considered high-purity DNA. Later, concentration was adjusted to the required working range for downstream PCR-based genotyping. The concentration of MDA in whole blood was determined spectrophotometrically by using Vineetha and Palakkal (2020) method, with a few modifications. The assay utilized trichloroacetic acid (TCA) and thiobarbituric acid (TBA) as reagents. After mixing, the samples were centrifuged at 6000 rpm for 30 seconds. The spectrophotometrically analysis of the resulting supernatant was recorded at a 530 nm wavelength.

For PC content, hydrochloric acid (HCl) was added to the test sample, followed by the addition of trichloroacetic acid (TCA) to precipitate proteins. The carbonyl groups were then quantified by measuring the absorbance of representative samples at a wavelength of 370 nm.

The MIF-173 G>C polymorphism was analyzed by performing the PCR-RFLP technique. The amplified PCR product was treated with *Alu I* restriction endonuclease by overnight incubation at room temperature. The PCR conditions and reaction mixtures were optimized (Table 1). The digested fragments were determined by gel electrophoresis to determine genotypes.

Data distribution was examined using the Shapiro-Wilk test, which revealed non-normality for MDA and PC values; therefore, results were presented as medians with interquartile ranges. Intergroup differences were tested using the Mann-Whitney U method, and genotype-related variations were assessed using the Kruskal-Wallis test. Conformity with the Hardy-Weinberg equilibrium was verified by chi-square analysis. Fisher's exact method was employed to assess genotype distributions due to the sparse data. Disease risk associated with genotypes and alleles was expected by

Table 1. Reaction conditions for the genotype MIF gene

Gene	Primer sequence	PCR condition	Restriction enzyme and reaction temperature	PCR and RFLP products size	References
-173 MIF G>Crs 755622G>C	Forward Reverse	94¯ C 5 min 94¯ C 45 sec 59¯ C 40 sec 35 cycle 72¯ C 50 sec 72¯ C 7 min	*Alu 1*37 °C for 16 h	366 bp GG: 268+ 98 GC: 268 +206+ 98+62CC: 206+ 98+62	De la Cruz-Mosso *et al*. (2014)

calculating odds ratios with 95% confidence intervals under dominant models. Statistical analysis was performed using SPSS software (version 26).

RESULTS AND DISCUSSION

A total of 110 vitiligo patients and 110 healthy controls were enrolled in this study (Table 2). The mean age of patients (34.49±12.8 years) was significantly higher than that of controls (30.60±10.2 years; p = 0.028). Age stratification a greater proportion of controls fell within the 18-30 years category (67.3%) compared with patients (45.5%), whereas the 31-45 years age group was more prevalent among patients (38.2%) than among controls (20.9%) (p = 0.004). Sex distribution analysis revealed a predominance of females in the patient cohort (59.1%) relative to controls (37.3%), while males were significantly more frequent in the control group (62.7%) (P = 0.003). No significant differences were observed between groups with respect to smoking status or dietary habits (P > 0.05). Regarding clinical subtypes, the majority of patients presented with non-segmental vitiligo (84.5%), followed by the intermediate type (11.8%) and the segmental type (3.6%), showing a very high significant difference.

Compared with the GG genotype, carriers of the GC genotype exhibited a significantly elevated vitiligo risk (OR=1.81; 95% CI: 1.05-3.12; p=0.031). An elevation in CC genotype frequency among cases relative to controls was observed (Table 3); however, this association was non-significant statistically (OR = 6.75; 95% CI: 0.32-142.6; p=0.23). In the dominant model (GC+CC vs GG), carriers of the C allele demonstrated a significantly elevated vitiligo risk (OR = 1.86; 95% CI: 1.09-3.12; p = 0.022). Allelic analysis revealed a significantly higher frequency of the C allele in patients (29.1%) compared with controls (20.5%), conferring an increased risk of vitiligo.

A Kruskal-Wallis H test revealed significant differences in malondialdehyde (MDA) concentrations across the MIF (-173 G>C)

Table 2. Demographic and clinical characteristics of controls and vitiligo patients

Variables	Vitiligo patients (N = 110)	Control (N = 110)	p-Value
Age in years(mean ± SD)	34.49 ± 12.8	30.60 ± 10.2	0.028[b*]
Age categories			
18-30	50	74	0.004[c*]
31-45	42	23	
46-60	18	13	
Gender (N/%)			
Male	45 (40.9)	69 (62.73)	0.003 [c*]
Female	65 (59.0)	41 (37.27)	
Smoking (N/%)			
Yes	13 (11.81)	5 (4.54)	0.118 [c*]
No	97 (88.18)	105 (95.45)	
Diet			
Vegetarian	94	92	0.580 [c*]
Non-vegetarian	16	18	
Types of vitiligo			
Segmental	4	-	NA
Non-segmental	93	-	
Intermediate(segmental + non segmental)	13	-	

Where, NA – Not applicable, b – Student t- test, c – Chi-square test and *P<0.05 is statistically significant.

Table 3. Genotypic, allelic and genetic model analysis of the MIF (-173 G>C) polymorphism in vitiligo patients and controls

Genotype	Patients (N = 110)	Controls (N = 110)	OR (95% CI)	p-value*
GG	48 (43.6)	65 (59.1)	1 (Reference)	-
GC	60 (54.5)	45 (40.9)	1.81 (1.05-3.12)	0.031
CC	2 (1.8)	0 (0.0)	6.75 (0.32-142.6)	0.23^
Dominant model				
GC+CC vs GG	62 (56.4)	45 (40.9)	1.86 (1.09-3.12)	0.022
Allelic				
G	156(70.9)	175(79.5)	1 (Reference)	-
C	64(29.1)	45(20.5)	1.60 (1.03-2.47)	0.036

Where: * Chi-square test and ^ Fisher's exact test.

Table 4. Median (IQR) values of serum malondialdehyde (MDA) and protein carbonyl (PC) levels according to MIF (-173 G>C) genotypes in vitiligo patients

Genotype	MDA (median, IQR; µmol/l)	PC (median, IQR; nmol/mg protein)
GG	0.62 (0.40-0.80)	2.05 (1.36-3.18)
GC	1.05 (0.80-1.22)	2.50 (1.59-3.64)
CC	0.95 (0.70-1.15)	5.00 (3.07-8.07)

genotypes (H=14.178, p=0.001) (Table 4). The genotype-dependent pattern showed the lowest MDA levels in GG homozygotes [median (IQR): 0.62 (0.40-0.80) µmol/l], while GC heterozygotes exhibited significantly elevated levels [1.05 (0.80-1.22) µmol/l]. The CC genotype demonstrated intermediate values [0.95 (0.70-1.15) µmol/l], which did not significantly differ from either the GG or GC groups, likely due to the small sample size of CC individuals. Post-hoc Dunn-Bonferroni analysis confirmed that GC carriers had significantly higher MDA levels compared to GG ($P < 0.01$), suggesting a stronger pro-oxidant effect linked to heterozygosity for the C allele (Fig. 1).

Similarly, protein carbonyl (PC) activity varied significantly among genotypes ($H = 17.550$, $P < 0.001$), indicating genotype-associated differences in oxidative protein damage (Table

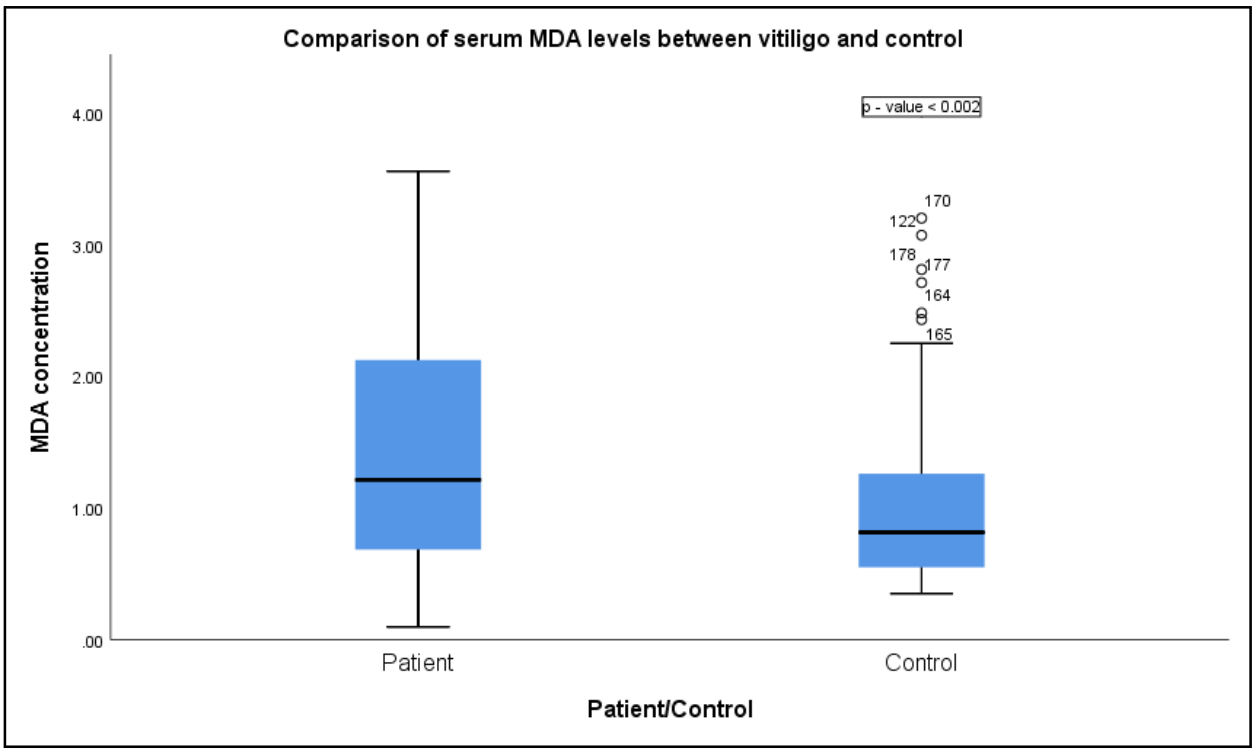

Fig. 1. Serum MDA concentrations in vitiligo patients and controls. Patients showed significantly higher levels than controls ($P < 0.003$).

4). PC levels increased progressively with the number of C alleles: GG genotype had a median (IQR) of 2.05 (1.36-3.18) nmol/mg protein, GC heterozygotes had 2.50 (1.59-3.64) nmol/mg protein, and CC homozygotes exhibited the highest levels at 5.00 (3.07-8.07) nmol/mg protein. Post-hoc analysis showed significantly elevated PC activity in CC homozygotes compared to both GG and GC groups ($P < 0.01$), with GC heterozygotes also having significantly higher levels than GG ($P < 0.05$), supporting a gene-dose effect (Fig. 2).

When genotypes were analyzed under the dominant model (GC+CC vs. GG), patients with the combined GC+CC genotypes showed higher median MDA (1.02 µmol/l; IQR: 0.78-1.20) and PC levels (2.90 nmol/mg protein; IQR: 1.75-5.10) compared to GG homozygotes (MDA: 0.62 µmol/l; IQR: 0.40-0.80; PC: 2.05 nmol/mg protein; IQR: 1.36-3.18) (Table 4). Mann-Whitney U test confirmed a significant increase in PC levels among carriers of the C allele ($P < 0.001$), while MDA levels showed an increasing trend that did not reach statistical significance. Collectively, these findings suggest that the presence of the C allele, particularly in a homozygous form, is associated with increased oxidative stress, specifically enhanced protein oxidation, in vitiligo patients.

The present study provides integrative evidence that oxidative stress contributes to the pathogenesis of vitiligo in a North Indian population from the state of Haryana. Consistent with previous research, patients with vitiligo exhibited significantly elevated malondialdehyde (MDA) and protein carbonyls (PC) compared to healthy controls, reflecting pronounced lipid and protein oxidation. The median MDA concentration of 1.21 µmol/l in patients, compared to 0.815 µmol/L in controls, aligns with findings by Farag *et al*. (2020), Abdel-Aziz *et al*. (2023) and Perumal and Manoharan (2025), reported increased lipid peroxidation in both active and stable vitiligo cases. Elevated PC levels in our cohort further confirm concurrent protein oxidative damage, strengthening the evidence for widespread oxidative stress in vitiligo. These oxidative alterations support the concept that sustained oxidative imbalance plays a central role in melanocyte dysfunction and loss (Picardo and Taïeb, 2019). However, consistent with oxidative stress, likely acts as an amplifier rather than an isolated trigger interacting with immune and genetic factors to exacerbate

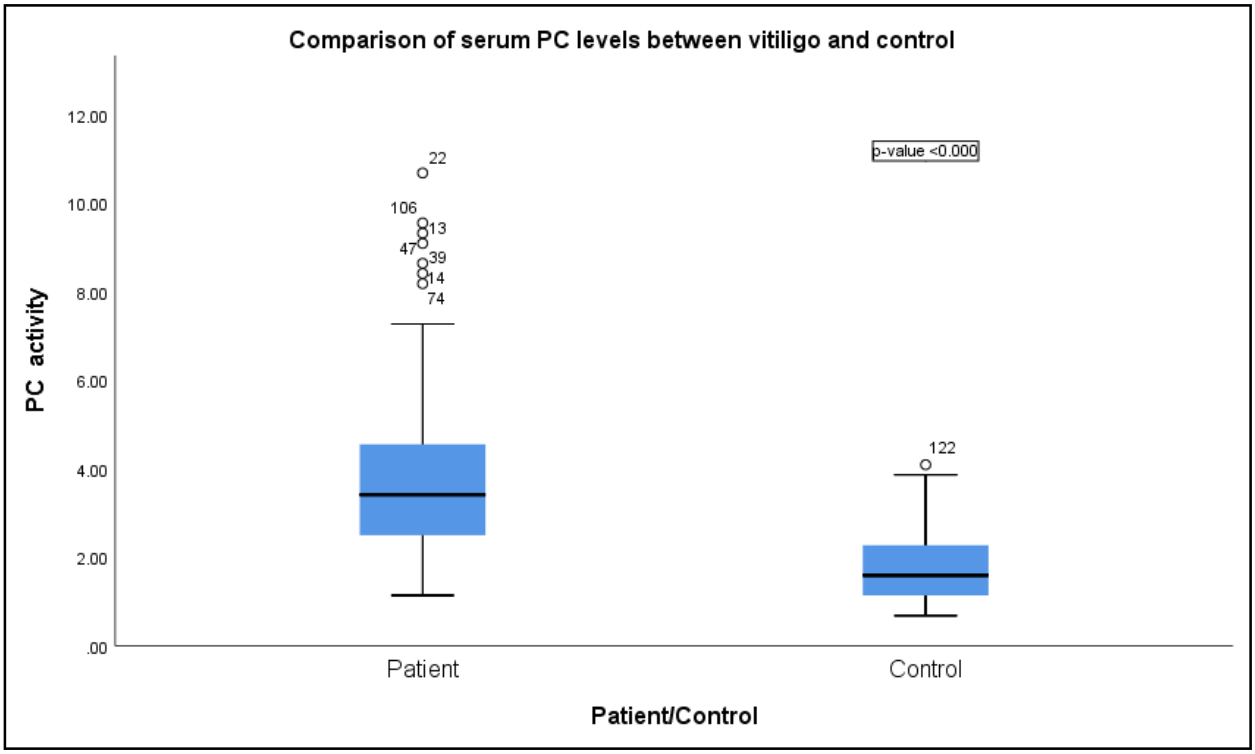

Fig. 2. Serum PC level in vitiligo patients and controls. Patients showed significantly higher levels than controls ($P < 0.000$).

melanocyte destruction. Present study's predominantly non-segmental vitiligo cohort exhibited marked oxidative imbalance.

Age emerged as a significant modifier, with vitiligo patients being older than controls and exhibiting increased oxidative stress markers with advancing age. This observation is consistent with Vineetha and Palakkal (2020), suggesting cumulative reactive oxygen species (ROS)-mediated damage and declining antioxidant defenses exacerbate disease severity over time. In contrast, gender did not significantly affect oxidative stress profiles, supporting earlier findings that oxidative imbalance in vitiligo is predominantly disease-driven rather than hormonally modulated (Farag *et al*., 2020). Additionally, comparable lifestyle factors between patients and controls reduce potential confounding influences. Genetically, the study identified a significant association between the MIF (-173 G>C) polymorphism and susceptibility to vitiligo. The GC genotype was markedly more frequent in patients and conferred an approximately five-fold increased risk of vitiligo, corroborating reports from Egyptian and Mexican populations (Ochoa-Ramírez *et al*., 2019; Garcia-Orozco *et al*., 2020). The absence of association in Chinese Han and Turkish cohorts (Aydýnöz *et al*., 2017; Wang *et al*., 2022) underscores ethnic heterogeneity and population-specific genetic effects, emphasizing the importance of region-specific genetic studies. Mechanistically, MIF influences Th1-skewed immune responses, sustaining inflammation and T-cell activation, which contribute to melanocyte destruction. Functional studies have demonstrated that MIF inhibition reduces CD8z T-cell infiltration and slows the progression of vitiligo (Chen *et al*., 2023), thereby reinforcing the biological relevance of MIF polymorphisms. The combined biochemical and genetic data suggest a synergistic effect wherein elevated oxidative stress and a pro-inflammatory MIF genotype accelerate melanocyte loss in genetically predisposed individuals.

The primary strength of this study lies in its integrative approach, which links oxidative stress biomarkers with immunogenetic susceptibility to provide a comprehensive understanding of vitiligo pathogenesis. Limitations of the study include the small number of CC homozygotes, which limit statistical power, and the cross-sectional design, which precludes causal inferences.

CONCLUSION

The study revealed that the MIF-173 G>C polymorphism, particularly the C allele, may tip the balance toward vitiligo by increasing oxidative stress in the skin. Patients carrying this allele exhibited higher levels of MDA and PC, underscoring how a minor genetic variation can amplify cellular damage. This study was among the first to integrate genetic and oxidative stress analyses in vitiligo, providing novel insights into the vulnerability of melanocytes in the population. Beyond improving understanding of the disease, these insights suggest that MIF could serve as both a biomarker and a potential target for future therapies, bringing hope for more personalized approaches to vitiligo care.

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ETHICAL APPROVAL AND INFORMED CONSENT

The study was approved by the Institutional Human Ethics Committee of Maharshi Dayanand University, Rohtak, Haryana (HEC No. 2023/39). Participation was entirely voluntary, and written informed consent was obtained from all individuals before their inclusion in the study.

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