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# Evaluation of The Level of Interleukin 6, IL-6, Superoxide Dismutase and glutathione peroxidase in patients With Lupus systemic Lupus Erythematosus

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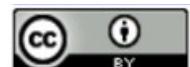
**Keywords:** Systemic lupus erythematosus, Interleukin-6, Glutathione peroxidase, Superoxide Dismutase.

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#### Citation

### ABSTRACT

**Back grounds:** Prone to flare-ups, systemic lupus erythematosus (SLE) is a chronic systemic autoimmune disease that varies in severity and progression. In SLE, both innate and adaptive immune responses are implicated. Gene-environment interactions cause a variety of immunologic alterations that lead to persistent immune responses to autologous nucleic acids. Morbidity and mortality are increased when autoantibodies or immune-complex depositions cause tissue damage to the kidneys, heart, arteries, central nervous system, skin, lungs, muscles, and joints.

**Materials and Methods:** The current study, which was conducted in the Baghdad Governorate between December 2024 and the end of March 2025, involved 90 sample patients between the ages of 10 and >60 who were severely affected by systemic lupus erythematosus and were being closely monitored by a specialist physician in terms of treatment and clinical condition observation. However, 30 individuals without SLE were included in the control group. The levels of Interleukin-6 (IL-6) and Superoxide Dismutase (SOD), glutathione peroxidase (GPX), and Source (Elk. Biotechnology-China) were measured in the lab for both groups using ELISA.

**Results:** The present study showed that the level of IL-6 serum were highly significant ( $p < 0.001$ ). As well as, there is a significant reduction in serum level of Superoxide Dismutase and Glutathione peroxidase.

**Conclusion:** There was a strong positive correlation between Superoxide Dismutase, Glutathione peroxidase and Interleukin 6 Level.

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## Introduction:

A polyclonal autoimmune response that targets several nuclear autoantigens is a characteristic of systemic lupus erythematosus (SLE), a common systemic autoimmune disease <sup>(1)</sup>. The systems typically affected encompass the integumentary system, musculoskeletal system, central and peripheral neurological systems, respiratory system, cardiovascular system, renal system, gastrointestinal system, serous membranes, and blood components. Interleukin-6 (IL-6) is a cytokine produced by monocyte macrophages that facilitates the development of B cells. It can stimulate B lymphocytes to generate immunoglobulins and engage in the progression of inflammatory responses <sup>(2)</sup>. IL-6 exerts its effects on several cell types through endocrine, autocrine, and paracrine mechanisms. IL-6 functions by binding to the membrane-associated IL-6 receptor (IL-6R), which initiates the dimerization of the signal transducing protein gp130 and activates the JAK/STAT signaling cascade <sup>(3)</sup>. Superoxide Dismutase (SOD) An essential antioxidant enzyme that catalyzes the dismutation of superoxide radicals ( $O_2^-$ ) into oxygen and hydrogen peroxide, playing a key role in protecting cells from oxidative stress.

Superoxide dismutase (SOD), the primary defense against ROS, is located in the membranes of eukaryotic cells and in extracellular fluid, where it is bound to copper or zinc or linked with manganese <sup>(4)</sup>. A robust correlation exists between SOD activity and individuals with SLE. The antioxidant enzymes Superoxide Dismutase was the focal points of elevated levels of circulating autoantibodies in the plasma and serum of SLE patients <sup>(4)</sup>.

Glutathione peroxidase is regarded as a crucial antioxidant in the defense mechanism against increased OS. The function of GPx was demonstrated by its altered activity in several illnesses. Glutathione peroxidase has selenium in its active site, which plays a crucial role in its defense activity against oxidative stress. Following the generation of hydrogen peroxide, the GPx enzyme mitigates the toxicity by reducing it to  $H_2O$ , using glutathione (GSH) and oxidizing it to glutathione disulfide (GSSG) <sup>(5)</sup>.

## Aim of the study

The study aims to evaluation of The Level of Interleukin 6, IL-6, Superoxide Dismutase and glutathione peroxidase in patients With Lupus systemic Lupus Erythematosus.

## Materials and Methods

### Study design

This study, which took place in the Baghdad Governorate between December 2024 and the end of March 2025, involved 90 sample patients between the ages of 10 and  $>60$  who had severe systemic lupus erythematosus and were being closely monitored by a specialist physician in terms of treatment and clinical condition. 30 individuals, however, were included in the control group but did not have SLE. To ascertain the levels of Interleukin-6 (IL-6) and Superoxide Dismutase (SOD), glutathione peroxidase (GPx), Source (Elk. Biotechnology-China), laboratory tests were performed for both groups using ELISA.

#### Exclusion Criteria

Individuals who suffer from these illnesses yet are not infected with SLE:  
Thyroid disease, Cancer, Kidney disease

#### Data Collection:

gel tubes coagulate at room temperature (25 °C) for 20 minutes. The serum was then separated by centrifugation for ten minutes at 3000 rpm. Serum (1 ml) was divided in half and put into 0.5 ml Eppendorf tubes. The remaining serum was moved to a different sterile 0.5 ml Eppendorf tube and kept in a deep freezer at -20 C until analysis.

#### Statistical analysis

The statistical program (SPSS) was used to perform the statistical analysis. One-way analysis of variance (ANOVA) was used to compare the groups, and the test of Duncan multiple ranges was used to test the arithmetic means for parameters in order to identify significant differences, particularly between groups. Regression plots were used to present the Pearson correlation coefficient ( $r$ ) between and other parameters. ( $P<0.05$ ) was the threshold for statistical significance.

particularly metabolic diseases, and whether or not they were taking any particular drugs. Additionally measured and documented were each person's weight, height, and systolic and diastolic blood pressure. order to gather the primary data source. The survey asked about date of birth, gender, history of any illnesses.

#### Samples Collection:

Each patient and control had five milliliters of venous blood aspirated from an appropriate vein.

The researcher used a specially created questionnaire to conduct in-depth interviews with both those without SLE and patients who had the condition in

Samples were collected between 8 and 9 a.m. after the fast. Blood samples were allowed to clot.

#### Results

The data reported in Table 1 indicates that the mean  $\pm$  standard deviation of the serum level of Interleukin 6 in patients with Systemic Lupus Erythematosus (SLE) was significantly higher than that of the control group ( $p<0.001$ ). Interleukin 6 levels in the serum are noticeably higher.

Table (2) illustrates that the mean  $\pm$  standard deviation of the blood level of Superoxide Dismutase in patients with Systemic Lupus Erythematosus (SLE) was significantly higher than that of the control group ( $p<0.001$ ). The amount of Superoxide Dismutase in the serum has significantly decreased.

Table (3) displays the mean  $\pm$  standard deviation of the glutathione peroxidase serum level in patients with systemic lupus erythematosus (SLE) in comparison to the control group. The difference was significant ( $p<0.001$ ). Serum glutathione peroxidase levels have significantly decreased.

Superoxide Dismutase and Interleukin 6 Level ( $R: + 0.09$ ) were shown to be strongly positively correlated in this study between patients with Systemic Lupus Erythematosus (SLE) and the healthy control group (Figure 1).

According to Figure (2), this study discovered a positive association between the levels of glutathione peroxidase and interleukin 6 ( $R: + 0.403$ ) in patients with systemic lupus erythematosus (SLE) and the healthy control group.

## Discussion

According to the current study, SLE patients had much greater serum IL-6 levels than healthy controls. Furthermore, the total pooled results of the current investigation demonstrated a favorable correlation between active SLE and serum IL-6 levels. The favorable association between serum IL-6 levels and SLE, however, could be explained by a number of processes. The pathophysiology of SLE is predominantly promoted by autoantibody overproduction, which can result in severe immune complex deposition. According to some theories, IL-6 may boost the generation of autoantibodies by encouraging the growth of autoreactive B lymphocytes and the conversion of naive B cells into plasma cells (6).

Research has shown that an imbalance in the Th17/Treg ratio contributes to the onset of a number of autoimmune disorders. By activating the STAT3 pathway, IL-6 has been reported to enhance the differentiation of naïve CD4+ T cells into Th17 cells. However, it has also been found to hinder Treg differentiation (7), indicating that IL-6 may possibly play a role in the development of SLE by mediating the Th17/Treg imbalance. Notably, a prior study found that IL-6 may promote inflammatory cell infiltration and immune complex deposition by causing fibroblast-like synoviocytes to secrete vascular endothelial growth factor, which in turn

may improve vascular permeability. Therefore, by compromising vascular endothelial function, IL-6 may also contribute to the development of SLE (8). All of this data points to a significant correlation between SLE disease activity and elevated blood IL-6 levels. The results of this study by Morad *et al.* (2025) might be clinically significant for the treatment of SLE. First, doctors can use the serum IL-6 level to track the activity of SLE disease. Furthermore, our research showed that IL-6-targeted treatment may be a useful approach for treating SLE. In a mouse model of SLE, it has been shown that blocking antibodies against IL-6 and IL-6R can stop the disease's onset and progression. According to certain theories, T lymphocytes, monocytes, dendritic cells, and macrophages all contribute significantly to the development of SLE, most likely through the overproduction of IL-6 (9).

According to this study survey, SOD activity was considerably lower in SLE patients than in healthy controls. According to earlier research, this rise in Superoxide Dismutase activity might only occur during the early stages of the illness and might eventually decline as disease activity increases. All of these investigations demonstrated SOD malfunction, regardless of whether SOD activity levels increased or decreased in SLE. (10,11,13). This theory is highly supported by this SOD malfunction. This study does have many limitations, though, the most evident of which is the size of the sample and the area in which it was collected. It would have been preferable to gather samples from 75–100 patients rather than just one area. Furthermore, different antigen properties of antiROS-SOD antibodies produced *in vivo* with antigens linked to SLE would support our

results even more. To sum up, this study shows that ROS causes SOD degradation in SLE, which could actively contribute to the disease's development (11,14). This study demonstrates ROS-induced SOD damage in SLE, which may play an active role in the progression and/or progress of the disease. Al-Shobaili *et al.*, study proposed that in addition to SOD in serum concentration, the quality of SOD molecules may be not only a crucial factor affecting its protective effects, but also a risk factor as a pro-oxidant in lupus patients (15).

## Conclusion

There was a strong positive correlation between Superoxide Dismutase, Glutathione peroxidase and Interleukin 6 Level.

## Recommendations

Investigate how medications used to treat Systemic Lupus Erythematosus (SLE) affect other levels of parameters. Exploring the link between Interleukin 6 levels, other variables, and metabolic anomalies can help better understand the pathogenesis of systemic Lupus Erythematosus (SLE).

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patients with systemic lupus erythematosus (SLE) and the healthy control group

Study groups	n	Serum Level of Interleukin 6 (ng/l) Mean $\pm$ SD	P. value
Patient with Systemic lupus erythematosus (SLE)	60	13.09 $\pm$ 4.5	P<0.001
Control group	30	6.3 $\pm$ 1.7	

**Table 2:** Mean  $\pm$  SD of Serum Level of Superoxide Dismutase in Patients with Systemic Lupus Erythematosus (SLE) and the Healthy Control Group

Study groups	n	Superoxide Dismutase (u/l) Mean $\pm$ SD	P. value
Patient with Systemic lupus erythematosus (SLE)	60	41.5 $\pm$ 11.4	P<0.001
Control group	30	52.2 $\pm$ 13.9	

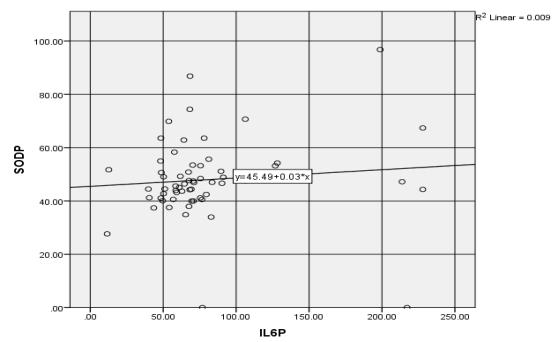
**Table 3:** Mean  $\pm$  SD of Glutathione Peroxidase Serum Levels in Patients with Systemic Lupus Erythematosus (SLE) and the Healthy Control Group

Study groups	n	Glutathione peroxidase (u/l) Mean $\pm$ SD	P. value
Patient with Systemic lupus erythematosus (SLE)	60	33.7 $\pm$ 8.7	P<0.001
Control group	30	45.3 $\pm$ 6.8	

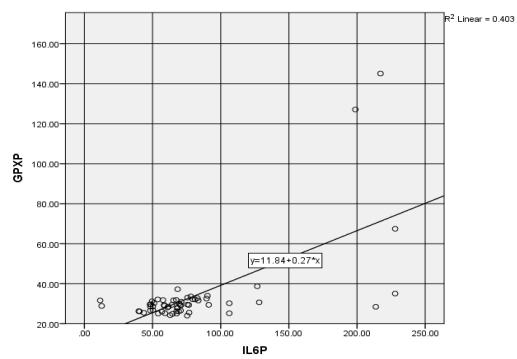
## FIGURES

## TABLES

**Table 1:** Comparison of the mean  $\pm$  SD of the serum level of interleukin 6 between



**Figure 1:** The relationship between the levels of interleukin 6 and Superoxide Dismutase in patients with systemic lupus erythematosus (SLE) and a healthy control group.



**Figure 2:** Relationship between Interleukin 6 Level and Glutathione Peroxidase in Systemic Lupus Erythematosus (SLE) Patients and a Healthy Control Group