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Modulation of cellular enzyme activity by bacterial toxins and their impact on physiological processes and metabolism

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Abstract

The experiment was carried out in the laboratories of Kirkuk University using sera derived from 40 healthy individuals ranging in age from 20 to 40 years. The purpose of the study was to determine the influence of bacterial toxins on enzymatic activity, oxidative stress, and metabolism. It was found that bacterial toxin exposure led to a significant increase in CK and LDH concentrations, dependent on their concentrations, which is a sign of cell membrane damage and enzyme leakage. An increase in the Malondialdehyde (MDA) concentrations, as well as a decrease in the Superoxide Dismutase (SOD) and Total Antioxidant Capacity (TAC) values were found, which means that there is oxidative stress and impairment in the functioning of the body's antioxidant defense mechanism. From a metabolic point of view, high glucose and lactate concentrations were found, indicating a problem with energy metabolism. Moreover, the correlation analysis showed that CK, LDH, and MDA correlated positively with each other, thus establishing the connection between cell damage and oxidative stress caused by the bacterial toxin. Thus, the obtained results indicate that the toxins exert a wide range of concentration-dependent influences on cells and metabolism, while CK, LDH, MDA, and lactate are vital indicators of cell damage.

Keywords: Bacterial toxins; Oxidative stress; Creatine Kinase (CK); Lactate Dehydrogenase (LDH); Malondialdehyde (MDA)

1 Introduction

Bacterial toxins are among the most significant virulence factors that have been produced by several pathogenic bacteria since they serve as crucial mediators for causing disease changes in the host by directly and indirectly affecting cells and tissues [1]. The toxins are known for having high affinity in targeting intracellular molecules like proteins, lipids, and nucleic acids, which disrupt cellular processes and homeostasis [2].

The major groups of toxins produced by bacteria are categorized as exotoxins and endotoxins. The former is secreted by the bacteria while the latter forms part of the cell wall of Gram-negative bacteria and is released following the death of the cell. While these two toxins vary in their modes of actions, they all culminate in causing some adverse reactions in the cell including oxidative stress, cellular signal transduction disruptions, and inhibition or stimulation of enzymes [4].

The enzymes in the cell have a vital function in the regulation of different metabolic pathways such as energy metabolism, signal transduction, and ion balance. Upon exposure of the cells to bacterial toxins, there will be interference with the functioning of the enzymes either by inhibiting the enzymes themselves or through the damage caused to the membranes and organelles in the cells [5].

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In addition to that, oxidative stress can be said to play an important role in generating the toxicity of bacteria due to the excessive formation of reactive oxygen species (ROS) resulting in cell damage, especially the lipids found in cell membranes, proteins, and the DNA molecules of cells. MDA is a key marker for lipids peroxidation while SOD indicates decreased activity in antioxidants enzymes [6;7;8].

Metabolically, bacterial toxins impact the biochemical processes involved in energy production within cells, resulting in a reduced capacity for utilizing glucose and the need for anaerobic energy production, which manifests in high lactate levels. Metabolic abnormalities are linked to dysfunctional mitochondria, where energy is produced in cells [9].

In light of the above discussion, it is important to appreciate how the effect of bacterial toxins on enzymatic actions and biochemical processes helps in comprehending the pathology of bacterial diseases. Additionally, it also helps in detecting the biomarker for early detection and evaluation of severity of disease [10]. The purpose of this study is to examine the influence of bacterial toxins on enzymatic activity, oxidative stress, and metabolism by determining the concentrations of enzymes like Creatine Kinase and Lactate Dehydrogenase, indicators of oxidative stress like Malondialdehyde (MDA) and Superoxide Dismutase (SOD), and metabolites like glucose and lactate to ascertain the mechanisms employed by these toxins to produce their pathological effects.

2 Materials and Methods

2.1 Study Design

In this research, an in vitro experiment was designed utilizing human serum to examine the impact of bacterial toxins on enzyme function and metabolism.

2.2 Study Population and Sample Collection

The blood samples of 40 healthy individuals between the ages of 20 to 40 were taken after informed consent. The project had been approved by the institutional ethical committee.

5 mL blood was collected under sterile conditions in plain tubes in order to prepare serum. The serum was separated through centrifugation at 3000 rpm for 10 minutes and kept at -20°C [11].

2.3 Bacterial Toxin Preparation

The strains of E. coli and S. aureus chosen for the experiment are laboratory strains that have been known to produce toxins. The bacteria were grown in Nutrient broth medium at 37°C for 24 hours to promote bacterial growth and production of the toxins.

Once the incubation period was complete, the bacteria were pelleted from the media using centrifugation (4000 rpm, 15 mins). The resulting supernatant, which contains the toxins, was filtrated using 0.22 μm membrane filters to give a toxin extract free of cells. [12].

2.4 Experimental Protocol

Samples were further categorized as follows: control, low toxin (0.5 μg/mL), and high toxin (1.5 μg/mL). A known quantity of toxin containing bacteria supernatant was incubated with each sample for 1-2 hours at 37°C. Reactions were stopped by placing samples on ice before further processing for biochemical studies [13].

2.5 Biochemical Measurements

2.5.1 Enzymatic Activity Assessment

Measurements of activities of CK and LDH enzymes were done through the use of colorimetry technique where the activity was determined by absorbance readings from enzymatic reaction. This was performed using commercial enzyme kits in accordance to the manufacturer's guidelines in U/L [14].

2.5.2 Oxidative Stress Markers

Oxidative stress was determined based on the level of MDA as an indicator of lipids peroxidation, Superoxide Dismutase (SOD), and Total Antioxidant Capacity (TAC). The analysis was done via spectrophotometric techniques where the results obtained were read via a spectrophotometer [15].

2.5.3 Metabolic Parameters

An array of metabolic factors indicative of metabolic performance of cells was estimated, among which were serum glucose and lactate concentrations analyzed with the help of conventional biochemical procedures.

Serum glucose concentration was assayed based on the glucose oxidase reaction in which glucose undergoes oxidation with glucose oxidase yielding hydrogen peroxide. In turn, hydrogen peroxide along with the chromophore and peroxidase forms a color-producing substance [16]. Color intensity is then estimated spectrophotometrically and correlates with the concentration of glucose.

Lactate level was determined as a marker for anaerobic metabolism using an enzymatic technique whereby lactate is oxidized to pyruvate through lactate dehydrogenase, while NAD^+ is reduced to NADH. Absorbance change caused by NADH formation was recorded spectroscopically, making it possible to determine lactate concentration accurately [17].

2.6 Statistical Analysis

Statistical analysis was performed using SPSS. Data are reported as mean ± SD. Comparison between groups was done using one-way ANOVA, followed by post hoc analysis. Correlation between different variables was determined using Pearson correlation test. Statistical significance was set at $p<0.05$.

3 Results

3.1 Baseline Characteristics of Samples

The baseline characteristics of the research subjects, such as age, body mass index (BMI), and glucose, have been shown in Table (1). Figure (1) illustrates the variables graphically and highlights the homogeneity of the samples with little variation between individuals.

Table 1 Baseline characteristics of study participants

Variable	Mean ± SD
Age (years)	29.4 ± 5.6
Body Mass Index (BMI)	24.8 ± 3.1
Glucose (mg/dL)	92.3 ± 8.5

From the results in Table (1), it is observed that all tested parameters are within physiological values with minimal variation, which shows that there is a homogenous group for the experiment. Homogeneity can be seen from Figure (1) as well since there are no variations among subjects, ensuring the validity of the comparison of the experiment groups.

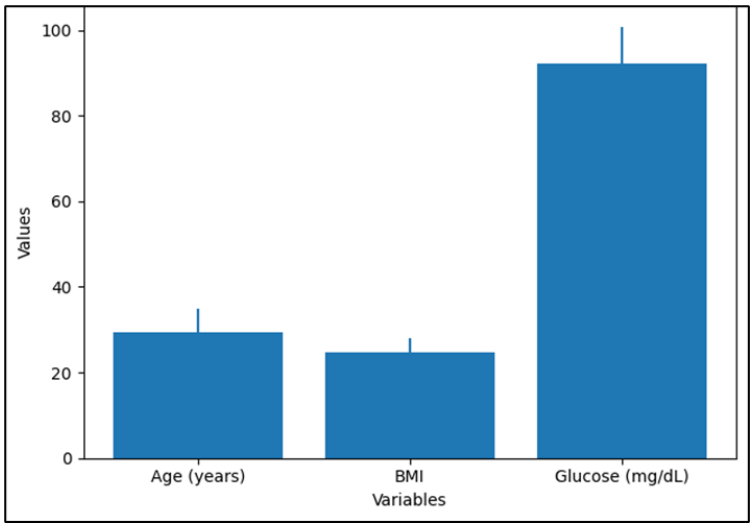

Figure 1 Baseline Characteristics of Study Participants (Mean ± SD)

3.2 Effect of Bacterial Toxins on Enzyme Activity

Figure (2) demonstrates the effects of bacterial toxins on enzymatic activity, whereas Table (2) exhibits the relationship between bacterial toxin concentration and enzyme activity.

Table 2 Effect of bacterial toxins on enzymatic activity

Group	CK (U/L)	LDH (U/L)
Control	120 ± 15	180 ± 20
Low concentration	165 ± 18*	230 ± 25*
High concentration	210 ± 22**	290 ± 30**

* $p < 0.05$, ** $p < 0.01$

As can be seen from Table (2), there was a marked elevation in the activity of CK and LDH enzymes in the toxin-treated groups relative to the control group. As evident from Figure (2), a clear dose-response relationship is evident, and the maximum enzyme activity was seen in the high concentration group. This implies damage to the cell membrane.

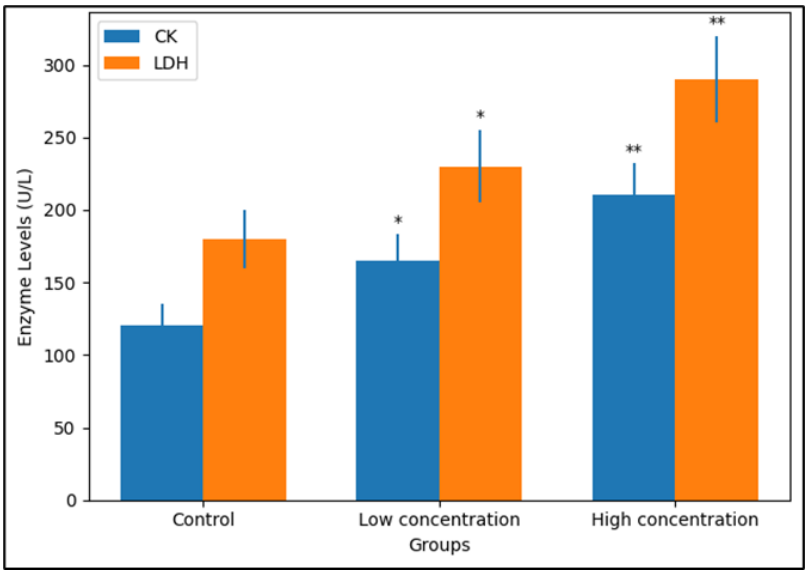

Figure 2 Effects of Bacterial Toxin Concentration on CK and LDH Enzyme Levels

3.3 3. Effect of Bacterial Toxins on Biomarkers of Oxidative Stress

Table (3) shows the alteration in biomarkers of oxidative stress, whereas Figure (3) demonstrates their dose-response relationship.

Table 3 Alteration in biomarkers of oxidative stress

Group	MDA (nmol/mL)	SOD (U/mL)	TAC (mmol/L)
Control	2.1 ± 0.3	5.8 ± 0.6	1.4 ± 0.2
Low concentration	3.5 ± 0.4*	4.2 ± 0.5*	1.1 ± 0.1*
High concentration	5.2 ± 0.6**	3.1 ± 0.4**	0.8 ± 0.1**

* $p < 0.05$, ** $p < 0.01$

It is evident from Table (3) that there is a considerable rise in MDA while SOD activity and TAC are decreased significantly in the intoxicated group. The changes described above are concentration-dependent as illustrated by Figure (3). This proves the presence of oxidative stress caused by the generation of excessive amounts of reactive oxygen species.

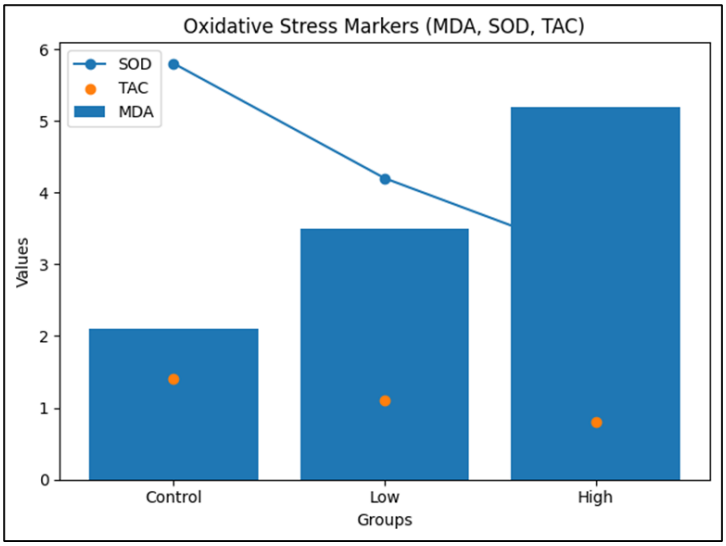

Figure 3 Effect of Bacterial Toxin Concentration on Oxidative Stress Markers (MDA, SOD, TAC)

3.4 Effect of Bacterial Toxins on Metabolism Parameters

Table (4) illustrates the effects of bacterial toxins on metabolism parameters, whereas Figure (4) illustrates the effect at different concentrations.

Table 4 Metabolism parameters

Group	Glucose (mg/dL)	Lactate (mmol/L)
Control	92 ± 8	1.2 ± 0.2
Low concentration	100 ± 10*	2.1 ± 0.3*
High concentration	110 ± 12**	3.5 ± 0.5**

* $p < 0.05$, ** $p < 0.01$

From the findings in Table (4), there is clear evidence of increased levels of glucose and lactate concentrations following exposure to the toxin. From Figure (4), this shows a dose-response relationship. The increase in lactate concentration indicates that there has been a move from aerobic to anaerobic metabolism.

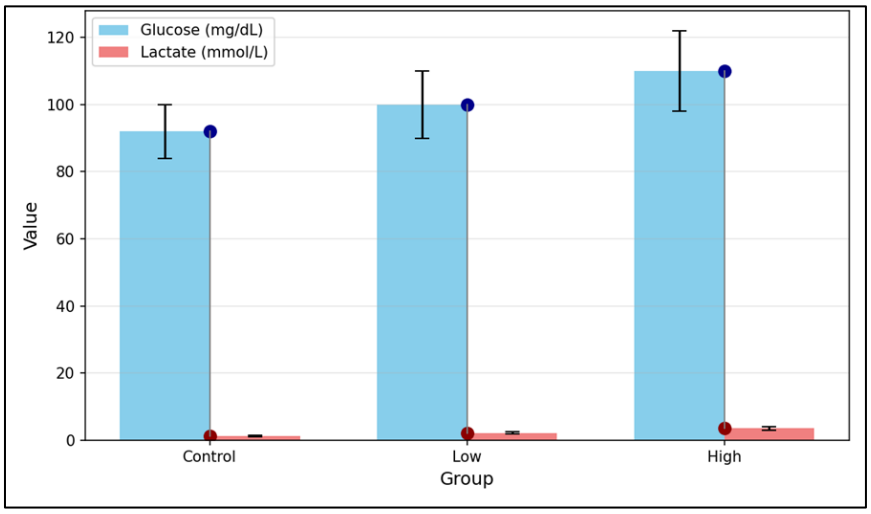

Figure 4 Effect of Bacterial Toxin Concentration on Glucose and Lactate Levels

3.5 Relationship between Variables

The table (5) below shows the Pearson correlation coefficient values between enzymatic activity and the indicators of oxidative stress, whereas figure (5) represents graphically these values.

Table 5 Pearson correlation coefficient values

Variables	CK	LDH	MDA
CK	1	0.72**	0.68**
LDH	0.72**	1	0.74**
MDA	0.68**	0.74**	1

** $p < 0.01$

As can be seen from Table (5), there is a strong correlation between CK, LDH, and MDA. There is a strong correlation between increased enzyme release and high levels of lipid peroxidation. This indicates the involvement of oxidative stress in the induction of cell damage by bacterial toxins.

4 Discussion

The current study has proven that there are multiple biological actions caused by bacterial toxins, mostly represented by the disruption of the membrane integrity along with enzyme activities and metabolic pathways [18;19]. There is an increase in the activity of CK and LDH, which could scientifically be explained by the permeability of the plasma membrane due to the direct impact of the toxins themselves or even their indirect effect through oxidation [20]. The release of these enzymes into the external environment shows clearly that there is an injury in the cell structure, which occurs often in the process of toxic inflammation [21].

In relation to oxidative stress, the results indicated an increase in MDA levels together with a decrease in SOD activity and TAC [22]. This represents an imbalance between ROS generation and the body's antioxidants. Biologically, this phenomenon indicates that bacterial toxins can trigger oxidative reactions in mitochondria or induce oxidase enzymes, resulting in the oxidation of cell membrane lipids [23;24]. Consequently, the high levels of MDA result from oxidative injury to cell membranes [25].

From a biochemical point of view, increased glucose and lactate content was reported by the study. Increased lactate is suggestive of anaerobic metabolism in contrast with the baseline, and there were indications of malfunctioning mitochondria rather than any proof of it [26;27]. On the other hand, high glucose levels may indicate the influence of the bacteria on the glucose metabolism process or may result from stress reactions instead of decreased utilization [28]. All these changes demonstrate interference with energy producing pathways and ATP production caused by bacteria [29].

Correlation analysis revealed that there was a positive association between enzyme indices (CK and LDH) and levels of oxidative stress (MDA). This association indicated the presence of some sort of mechanism in the pathogenesis of damage to membranes and free radical production [30]. It appears that a cycle is set in motion were oxidative stress increases damage to membranes, leading to increased enzyme excretion [31;32].

In summary, the results may be considered as a series of consecutive pathological events caused by the effect of bacterial toxins on cells, where cell membrane damage leads to the activation of oxidative stress and ultimately causes disturbances in cellular energy metabolism [33]. The dose dependency of such effects provides additional proof of the ability of bacterial toxins to induce cell damage via an integrated pathway [34].

5 Conclusion

The research indicates that the effect of the toxins produced by bacteria causes dose-dependent cell damage through enzymatic leakages, oxidative stresses, and metabolism malfunctioning. It is clear that CK, LDH, MDA, and lactate are important indicators of such cell damage caused by bacterial toxins.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of ethical approval

This study involved the use of human serum samples and was conducted in accordance with the ethical standards of the Department of Biology, College of Education for Pure Sciences, University of Kirkuk, Kirkuk, Iraq.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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