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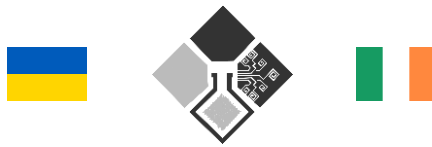
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XI INTERNATIONAL SCIENTIFIC  
AND THEORETICAL CONFERENCE**

FORMATION OF  
INNOVATIVE POTENTIAL  
OF WORLD SCIENCE

08.05.2026

WATERFORD,  
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**SCIENTIA**  
COLLECTION OF SCIENTIFIC PAPERS



Non-governmental Organization  
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with the proceedings of the  
XI International Scientific and Theoretical Conference

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## SECTION 15.

### MEDICAL SCIENCES AND PUBLIC HEALTH

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## THE ROLE OF IRON IN OXIDATIVE STRESS

Iron, or ferrum, is an essential trace element that plays a role in many biochemical processes in the human body, including oxygen transport, the respiratory chain, and enzyme function. However, its unique feature is that it can easily switch between two valence states - divalent ( $\text{Fe}^{2+}$ ) and trivalent ( $\text{Fe}^{3+}$ ). It is precisely this ability to rapidly accept and donate electrons that makes iron irreplaceable in metabolism, but at the same time potentially dangerous, as it can catalyze the formation of highly reactive (or active) forms of oxygen.

Reactive oxygen and nitrogen species (RONS) are a group of highly reactive molecules that are produced in the body as byproducts of normal metabolism. They play an important role in physiological processes, including cell signaling, the immune response, and the regulation of vascular tone [1].

Reactive oxygen species (ROS) include the superoxide anion ( $\text{O}_2^{\cdot-}$ ), hydrogen peroxide ( $\text{H}_2\text{O}_2$ ), and the hydroxyl radical ( $\text{OH}^{\cdot}$ ).

Under normal conditions, there is a balance between the formation of ROS and their neutralization by antioxidants. Antioxidants act as electron donors: they donate electrons and thereby neutralize reactive species, preventing cellular damage [1].

When the number of ROS exceeds the body's ability to neutralize them, oxidative stress occurs. This is considered one of the main causes of aging, cancer, Alzheimer's disease, and cardiovascular disease.

The primary mechanism of cellular damage during oxidative stress is the initiation of chain radical reactions. Reactive molecules steal electrons from other molecules, converting them into new radicals, which triggers a cascade of damage.

The following are primarily affected:

- proteins - they undergo denaturation and lose their functions;

- lipids - a chain reaction of membrane oxidation is triggered;
- DNA - mutations and damage to genetic material occur.

Iron is an essential trace element, but at the same time, it can significantly increase oxidative stress. This is because iron can exist in two forms –  $\text{Fe}^{2+}$  and  $\text{Fe}^{3+}$  - and easily switch between them. Normally, most of the iron in the body is in a safe form, bound to ferritin (a special storage protein) or the transport protein transferrin (which carries iron in the blood in a safe form). This bound form plays virtually no role in the formation of free radicals. Therefore, the danger comes from so-called «free» or labile iron - a small fraction of  $\text{Fe}^{2+}$  ions that are not bound to proteins and are the ones that react.

Ascorbic acid, or vitamin C, also plays a role in oxidative stress processes, but it is a double-edged role. On the one hand, it is a powerful antioxidant: it acts as an electron donor and is capable of neutralizing reactive oxygen species, thereby protecting cells from damage. On the other hand, under certain conditions, especially at elevated concentrations and in the presence of metal ions, ascorbic acid can exhibit pro-oxidant properties. Ascorbic acid reduces  $\text{Fe}^{3+}$  to  $\text{Fe}^{2+}$ , increasing the amount of divalent iron involved in the Fenton reaction (the primary mechanism through which iron becomes harmful) [2].

In the presence of divalent iron, hydrogen peroxide - which is relatively mild in its reactivity on its own - is converted into a hydroxyl radical via the Fenton reaction. This radical is one of the most aggressive in biological systems due to the presence of an unpaired electron and is extremely unstable. As a result, the hydroxyl radical reacts almost instantly with any biomolecules, showing no selectivity. Unlike other reactive oxygen species, such as the superoxide anion or hydrogen peroxide, which can be partially controlled by enzymatic systems, there are no specific detoxification mechanisms for the hydroxyl radical in the body.

It is also important to note that it has a lifespan of only nanoseconds and is therefore unable to diffuse over significant distances, acting instead directly at the site of its formation. This means that damage occurs precisely where the Fenton reaction took place, and vital cellular structures - such as DNA, proteins, or components of cell membranes - are often targeted [4].

Disruption of iron homeostasis and the accumulation of iron in its «free» state triggers a cascade of destructive processes, including lipid peroxidation and ferroptosis. If there is too much free iron in the cell or if the defense system is compromised (for example, due to glutathione deficiency), the cell literally «self-oxidizes».

Understanding these mechanisms is critical for developing new treatment strategies for neurodegenerative and cardiovascular diseases, where oxidative stress

is a leading factor in pathogenesis [3].

**Conclusions.** The results of the analysis indicate that iron plays a key role in the development of oxidative stress due to its ability to switch between  $\text{Fe}^{2+}$  and  $\text{Fe}^{3+}$  and participate in the Fenton reaction, which leads to the formation of the hydroxyl radical - the most aggressive form, capable of instantly damaging any nearby molecule. In the presence of ascorbic acid, this process can be intensified by the reduction of  $\text{Fe}^{3+}$  to  $\text{Fe}^{2+}$ . Iron is a key factor determining the intensity of free-radical processes and the extent of cellular damage. Excess iron or its release from storage during illness or injury sharply increases oxidative stress in the tissues of the liver, heart, and brain.

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