Non-governmental Organization
International Center of Scientific Research

PROCEEDINGS OF THE XI INTERNATIONAL SCIENTIFIC AND THEORETICAL CONFERENCE

FORMATION OF INNOVATIVE POTENTIAL OF WORLD SCIENCE

08.05.2026

WATERFORD,
REPUBLIC OF IRELAND

SCIENTIA
COLLECTION OF SCIENTIFIC PAPERS

Non-governmental Organization
International Center of Scientific Research

SCIENTIA
COLLECTION OF SCIENTIFIC PAPERS

with the proceedings of the
XI International Scientific and Theoretical Conference

Formation of innovative potential of world science

May 8, 2026

Waterford; Republic of Ireland

Hosted by an authorized Crossref member with the support of the
Institute of Scientific and Technical Integration and Cooperation

Published online by Primedia E-launch LLC (USA)
Published in print by LLC UKRLOGOS Group (Ukraine)

✔ ISO 2108:2005 ✔ ISO 1086:1991 ✔ ISO 7275:1985

Waterford, 2026

UDC 082:001
F 76

The conference is included in the Academic Research Index ReserchBib International catalog of scientific conferences and registered for holding on the territory of Ukraine in UKRISTEI (Certificate № 163 dated January 26th, 2026).

Chairman of the Organizing Committee:
Miriam Goldenblat
International Center of Scientific Research, Ukraine

Book layout designer:
Gabriela Torres
Primedia E-launch LLC, USA

Responsible editor:
Yuliia Babych
UKRLOGOS Group ltd., Ukraine

F 76 **Formation of innovative potential of world science**: collection of scientific papers «SCIENTIA» with Proceedings of the XI International Scientific and Theoretical Conference, May 8, 2026. Waterford, Republic of Ireland: International Center of Scientific Research.

ISBN 979-8-89660-280-4 (series)
DOI 10.36074/scientia-08.05.2026

This collection presents the proceedings of the scientific conference, bringing together research papers on a wide range of contemporary academic topics. The volume highlights diverse perspectives, innovative approaches, and practical findings that reflect the current trends and challenges in global science and education. This collection is intended for students, postgraduate and doctoral candidates, educators, researchers, and professionals from diverse disciplines.

Conference proceedings are presented in Google Books and Bookwire™ by Bowker, ensuring international availability.

UDC 082:001

ISBN 979-8-89660-280-4

www.previous.scientia.report

SECTION 15.
MEDICAL SCIENCES AND PUBLIC HEALTH

Levkivska Kvitoslava
student of Educational and Scientific Institute of Medicine, specialty I2 «Medicine»
Bogomolets National Medical University, Ukraine

Slipchuk Valentyna
Doctor of Sciences in Pedagogy, Professor,
Professor of the Department of Medical Biochemistry and Molecular Biology
Bogomolets National Medical University, Ukraine

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 (Fe^{2+}) and trivalent (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 ($O^{\bullet}{}_2^-$), hydrogen peroxide (H_2O_2), and the hydroxyl radical ($OH^{\bullet}$).

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 – Fe^{2+} and 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 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 Fe^{3+} to 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 ferroptoxis. 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 Fe^{2+} and 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 Fe^{3+} to 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.

References:

1. Fisher, A. E., & Naughton, D. P. (2004). Iron supplements: the quick fix with long-term consequences. *Nutrition journal*, *3*, 2. https://doi.org/10.1186/1475-2891-3-2
2. Kontoghiorghes, G. J., Kolnagou, A., Kontoghiorghe, C. N., Mourouzidis, L., Timoshnikov, V. A., & Polyakov, N. E. (2020). Trying to Solve the Puzzle of the Interaction of Ascorbic Acid and Iron: Redox, Chelation and Therapeutic Implications. *Medicines (Basel, Switzerland)*, *7*(8), 45. https://doi.org/10.3390/medicines7080045
3. Pizzino, G., Irrera, N., Cucinotta, M., Pallio, G., Mannino, F., Arcoraci, V., Squadrito, F., Altavilla, D., & Bitto, A. (2017). Oxidative Stress: Harms and Benefits for Human Health. *Oxidative medicine and cellular longevity*, *2017*, 8416763. https://doi.org/10.1155/2017/8416763
4. Abe, C., Miyazawa, T., & Miyazawa, T. (2022). Current Use of Fenton Reaction in Drugs and Food. *Molecules (Basel, Switzerland)*, *27*(17), 5451. https://doi.org/10.3390/molecules27175451