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Review Article

Ionizing and non-ionizing electromagnetic radiation in modern medicine

Paweł Sowa^a, Joanna Rutkowska-Talipska^b, Urszula Sulkowska^c,
Krzysztof Rutkowski^d, Ryszard Rutkowski^{e,*}

^aFaculty of Public Health, Stanislaw Staszic College of Public Administration, Białystok, Poland

^bDepartment of Rehabilitation, Medical University, Białystok, Poland

^cFaculty of Biology and Chemistry, University of Białystok, Poland

^dDepartment of Allergy, Cambridge University Hospital, Cambridge, United Kingdom

^eDepartment of Respiratory Diagnostics and Bronchoscopy, Medical University, Białystok, Poland

ARTICLE INFO

Article history:

Received 3 June 2012

Accepted 10 July 2012

Keywords:

Ionizing
Non-ionizing
Radiation
Medicine
Modern

ABSTRACT

Introduction: Electromagnetic radiation (EMR) has been successfully employed in modern medicine for many years. The medical community, however, often lacks in-depth knowledge concerning different types of radiation, their mechanisms of action and clinical applications.

Aim: Our review offers a comprehensive overview of the biological action of ionizing radiation (IR) and non-ionizing radiation (NIR) and their applications in modern medicine. Chronic exposure to a high frequency electromagnetic field (EMF) as a potential public health risk is also discussed.

Materials and methods: Current literature on IR and NIR has been reviewed and grouped thematically.

Results and discussion: Biological effects of EMR depend on its physical properties. IR is a potentially lethal stream of high energy particles. NIR carries less energy. EMR can damage DNA directly or indirectly via reactive oxygen/nitrogen species. It has been, however, successfully used in oncology (radiotherapy), physiotherapy (microwaves), rheumatology and endocrinology. Effective communication and data transmission are possible thanks to radio-, micro- and infrared waves. Cybernetics and modern forms of communication have been instrumental in the development of telerehabilitation and telemedicine. Evidence for the detrimental effects of cell phones, the most common source of EMR, on the developing central nervous system is scarce, but concerns have been raised about their carcinogenicity.

Conclusions: Modern medicine cannot function without IR and NIR. However, their potentially undesirable biological side effects need to be taken into account.

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*Correspondence to: Prof. Ryszard Rutkowski, Starobojarska 20/6, 15-073 Białystok, Poland. Tel.: +48 608 255 565.

E-mail address: rutkowski@csk.pl (R. Rutkowski).

1. Introduction

Biological effects of electromagnetic radiation (EMR) vary depending on its physical properties and, in particular, ionizing potential. They have been described in greater detail in part I of our review.

Ionizing radiation (IR) creates a stream of high-energy photons or alpha particles, protons and neutrons which in high doses can be lethal to living organisms.^{18,42,43} Acute postradiation syndrome triggered by excessive IR often leads to organ failure or death. Early changes include lymphocytes deficiency, impaired cellular immunity, anemia, transient infertility, acute radiation dermatitis, hair loss, lens opacity, and acute intestinal inflammation. Long-term (late) effects may lead to malignancies including leukemia.^{41,43}

A relatively low energy non-ionizing radiation (NIR) does not cause lethal ionization of atoms and molecules of matter.^{18,27,37}

On entering the body, IR and NIR damage nitrogenous bases of the deoxyribonucleic acid (DNA) directly or indirectly through reactive oxygen species (ROS) or nitrogen species (RNS). ROS (free radicals) include hydroxyl (OH^\bullet), hydroperoxide (HO_2^\bullet) and superoxide ($\text{O}_2^{\bullet-}$) radicals, hydrogen peroxide H_2O_2 and singlet oxygen ($^1\text{O}_2$) (Fig. 1).

OH^\bullet produced in the Fenton reaction are the most reactive of all ROS. HO_2^\bullet and $\text{O}_2^{\bullet-}$ have a long half-life, but are rare. They can, however, transform into highly reactive OH^\bullet radicals in the iron-catalyzed Haber-Weiss reaction. RNS family encompasses nitric oxide (NO) and its metabolites: nitrosonium cation (NO^+), nitroxyl anion (NO^-) and peroxy-nitrite (ONOO^-). Oxidative DNA damage is induced mainly by

hydroxyl radicals; $\text{O}_2^{\bullet-}$ and H_2O_2 do not cause direct DNA changes. H_2O_2 penetrates easily into the nuclear membrane of the nucleus and becomes a substrate in the Fenton reaction, producing highly damaging OH^\bullet . Interaction of OH^\bullet with DNA damages deoxyribose, breaks phosphodiester bonds between nucleotides and cross-links with nuclear proteins and DNA. Interaction of hydroxyl radicals with deoxyribose residues in turn breaks single or double-stranded DNA.

Hydroxyl radicals alter amino acid residues and prosthetic groups of enzymes and cause fragmentation and aggregation of proteins. Proteins with aromatic amino acids and sulphur (cysteine, methionine, etc.) are the most susceptible. ROS and RNS contribute to lipid peroxidation, change physicochemical properties of cell membranes and their liquidity and disturb transmembrane transport in the respiratory chain and intracellular signal transduction.^{7,17,20,34,37,38}

In cancerous cells, ROS and lipid peroxidation products act directly on the nuclear transcription factor κB (NF- κB ; REL oncogene family) and alter the expression of Bcl-2, Bax and p53. These molecules regulate apoptosis and the rate of telomerase shortening and inhibit the growth and apoptosis of malignant cells. In healthy cells, ROS activate oncogenes and inactivate tumor suppressor genes, which may initiate carcinogenesis and tumor progression.^{8,12,20,28,33,42}

2. Aim

Our review endeavors to familiarize healthcare professionals with the biological action of IR and NIR and their ever

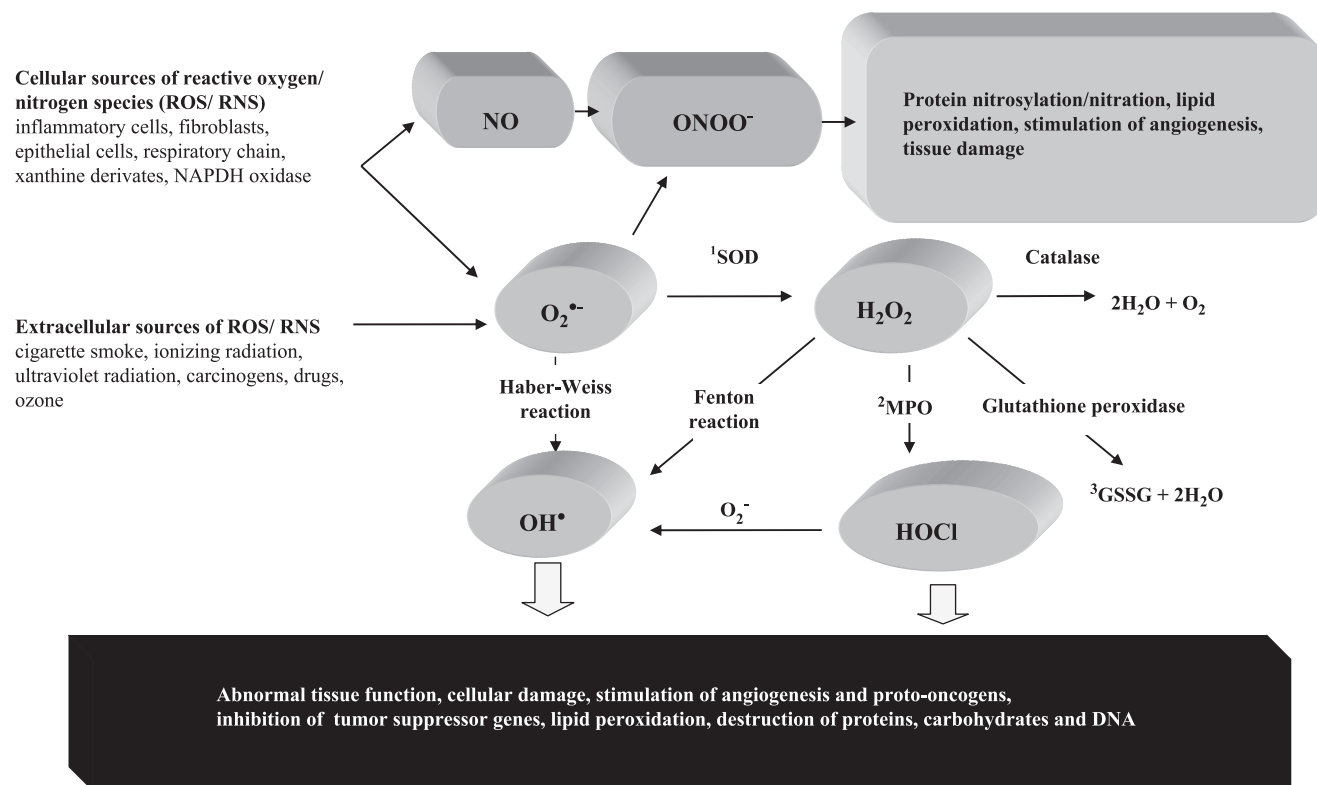


Fig. 1 – Reactive oxygen and nitrogen species (ROS/RNS) – mechanism of action:³⁴ ¹SOD – superoxide dismutase, ²MPO – myeloperoxidase, ³GSSG – glutathione disulfide.

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