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Nutrition as a Preventative Measure Against Radiation-Related Health Risks

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Abstract

The rise in chemical, biological, radiological, and nuclear (CBRN) threats has brought significant challenges to public health, particularly concerning radiation-related diseases. Radiation exposure, whether from environmental contamination or nuclear events, can lead to numerous adverse health effects, including acute radiation syndrome, cancer, and genetic mutations. This paper explores the role of nutrition as a preventative measure against radiation-induced health risks, focusing on nutrients with antioxidant, DNA repair, and anti-inflammatory properties. The paper reviews the mechanisms by which these nutrients mitigate cellular and molecular damage caused by ionizing radiation, providing a foundation for potential dietary guidelines that can enhance resilience to radiation exposure. Traditional approaches, like the Chinese principle of "medicine and food homology," are discussed in the context of modern radiation protection, highlighting the integration of natural compounds to protect against radiation injuries.

Keywords: Nutrition, Radiation exposure, Antioxidants, DNA repair, Anti-inflammatory, Radiation-induced injury

* Introduction

Significant risks to public health are posed by the increase in unintentional and deliber ate exposure to chemical, biological, radiological, and nuclear (CBRN) agents worldwide. In regions impacted by these agents, food supplies may become contaminated, affecting seafood, freshwater, and other essential goods. Such contamination creates persistent

challenges for maintaining both public health and nutrition. A possible worldwide threat to marine ecosystems is highlighted by the recent release of radioactive wastewater into the ocean, with far-reaching implications (Nogrady, 2023). This incident could have enduring effects on marine organisms and human health, particularly in neighboring nations. Nuclear radiation exposure is linked to numerous severe health issues, including acute radiation syndrome, various cancers such as leukemia, thyroid diseases, and genetic disorders. Additionally, the interplay between radiation exposure and related stressors may exacerbate disease progression or lead to genetic harm (Hashmi et al., 2023).

The interplay between radioactive elements, radiation exposure, and lifestyle factors can greatly influence the progression of diseases (Singh, 2020). Radioactive isotopes, including tritium-3, cobalt-60, strontium-90, iodine-131, iodine-134, and cesium-137, are associated with severe health complications such as damage to hematopoietic stem cells, acute bone marrow suppression, and bone marrow failure. A notable decline in lymphocytes often follows radiation exposure, with the rate of reduction serving as a clear marker of ionizing radiation's impact (Lumniczky et al., 2021). Prolonged exposure to low levels of radiation from sources like contaminated water or radioactive particles can lead to genetic mutations and alterations, often described as "stochastic effects." (Domina et al., 2022). Managing nuclear contamination remains a pressing issue, as radioactive substances and pollutants are pervasive in ecosystems and frequently enter the human body via the food chain. To mitigate the health risks of radiation exposure, preventive strategies emphasizing lifestyle changes, including improved nutrition, are crucial (Hennig & Deng, 2020). The idea of "medicine and food homology" is emphasized in traditional Chinese medicine," which may enhance resilience and promote health when incorporated into dietary practices. Research suggests that diets aligning with this concept could help the body counteract environmental stress and minimize disease severity.

The leaf extract of Polygonum odoratum, for example, is well-known for its bioactive compounds and possesses potent antioxidant qualities that may lessen oxidative stress

and shield cells from damage caused by radiation (Kawvised et al. in 2022). Restoring metabolic balance, controlling inflammation, and treating radiation-induced injuries all depend on important biological pathways like p65/Cas3 and Nrf2/GPX4 (Tang et al. 2023, T. Yep. Wang & Co. in 2022).

bioactive phytochemicals and plant-based substances, including polyphenols and polysaccharides, have shown potential in minimizing the adverse effects of radiation by targeting specific signaling pathways. These natural substances, valued for their dual role as both nutritional and therapeutic agents, offer protection against radiation-induced cellular damage. In the aftermath of the COVID-19 pandemic, addressing radiation-related injuries and genetic disruptions caused by CBRN agents has become increasingly important. Strengthening frameworks for prevention and intervention is essential to enhance resilience to radiation and improve overall health outcomes. Current treatments for radiation injuries remain limited in effectiveness, highlighting the need for innovative approaches. Research from integrative multi-omic studies has revealed the complexity of radiation-induced injuries, with disruptions in the gut microbiome playing a potential role (W. Wang et al., 2023). Dietary interventions featuring plant-based foods, along with prebiotics and probiotics, have been shown to influence gut microbiota composition positively. These findings suggest that nutrigenetic dietary strategies may lower disease risks by modulating gut flora. Proactive measures to address the health impacts of genetic damage and mutations resulting from radiation exposure are particularly vital in high-risk regions, where radiation exposure contributes to elevated mortality rates (Zhu & Qian, 2024).

*** Mechanisms of Radiation Damage in Human Body**

Cell death is the most serious immediate effect of ionizing radiation, which mainly affects the cytology of the human body.

It is crucial to comprehend the precise processes and mechanisms of radiation-induced cell death in order to improve radiotherapy for tumor control and develop treatments for radiation injuries. Radiation-induced cell death generally falls into two main categories: interphase and proliferative death (Sia et al., 2020).

Interphase death affects cells that cease division soon after radiation exposure, with cell death occurring within hours. This is initiated by intracellular damage, activation of nucleases and proteolytic enzymes, and chromatin disintegration, resulting in nuclear shrinkage, chromatin degradation, and eventually, cell death. Membrane disruption and cellular energy imbalance are contributing factors to interphase death. Conversely, the majority of cells experience proliferative mortality as a result of mitotic catastrophe, which is induced by the accumulation of chromosomal abnormalities and inadequate repair of DNA double-strand breaks. Subsequent to one or more cell cycles, these cells forfeit their capacity to proliferate and commence apoptosis (Wang & Tepper, 2021). Cell type, cell cycle phase, and radiation dose are all documented factors that affect biological responses to radiation.

Working with the Society of Toxicologic Pathology, the classifications of cell death are updated on a regular basis by the Nomenclature Committee on Cell Death (NCCD) to maintain precision and clarity and to promote further study in this area. Radiation-induced cell death is arranged in this summary based on the molecular mechanisms outlined in the 2018 NCCD framework (Galluzzi et al. 2018).

ferroptosis, pyroptosis, autophagy-dependent cell death, and immunogenic cell death, necrosis(including parthanatos, necr optosis, and necrosis related to the mitochondrial permeability transition), necrosis (including apoptosis by intrinsic and extrinsic pathways), and non-lethal cellular responses like mitotic catastrophe and cellular senescence are some of the categories.

*** Apoptosis**

Without causing inflammation, apoptosis is a carefully regulated process of programmed cell death that eliminates damaged or superfluous cells. Originally reported by Kerr, Wyllie, and Currie in 1972, it is now recognized as a gene-regulated, energy-dependent process.

This mechanism is activated by the intrinsic and extrinsic pathways.

Internal cellular stressors that trigger the intrinsic pathway include oxidative stress, endoplasmic reticulum stress, and DNA damage. Through the pro-apoptotic proteins Bak and Bax, the Bcl-2 protein

family regulates mitochondrial outer membrane permeabilization(MOMP)

Cytochrome c and other apoptotic factors are released during this process, and they combine to form an apoptosome. Caspases 3, 6, and 7 are subsequently triggered by caspase-9, which is triggered by the apoptosome, which are executioner caspases, causing the cell to undergo apoptosis (Shichijo et al. (2023).

Conversely, extrinsic apoptosis is brought on by outside stimuli that attach to cell surface death receptors like TNF and Fas receptors. The apoptotic cascade, which frequently involves MOMP as well, can be directly started by caspase-8, which is activated by this binding. Ionizing radiation can trigger apoptosis via both pathways. In response to DNA damage, in order to regulate the cell cycle and encourage apoptosis, the P53 gene is essential.

Additionally, stress activates t he SAPK/JNK pathway, which further encourages apoptosis (C. Wang & Co. in 2020).

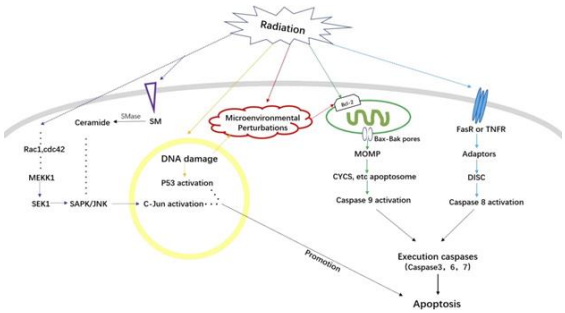


Figure 1 depicts the signaling pathways implicated in radiation-induced apoptosis.

The two main mechanisms by which radiation causes apoptosis are intrinsic and extrinsic. Both pathways come together to initiate the execution pathway, which completes the apoptotic process.

Apoptosis can also be induced by ionizing radiation via the SAPK/JNK and P53 pathways (Jiao et al. 2022).

*** Necrosis**

Necrosis is a form of cell death characterized by the uncontrolled breakdown of cellular structures, often resulting in inflammation. Unlike apoptosis, which is a regulated and programmed process, necrosis is typically considered a passive and chaotic response to severe cellular stress or damage. It can be triggered by various factors, including cytokines, ion channel dysfunction, redox reactions, and mitochondrial dysfunction (Khan et al., 2021). There are various varieties of necrosis, such as parthanatos, necroptosis, and necrosis caused by

the mitochondrial permeability transition (MPT). MPT-driven necrosis happens when calcium overload and oxidative stress cause the mitochondrial membrane to become compromised, which depletes ATP and causes pro-necrotic substances to leak into the cytoplasm. The process relies significantly on cyclophilin D (CYPD), which is essential for the formation of the permeable transition pore complex that facilitates necrosis (Jablonska et al., 2023).

Necroptosis, a type of predetermined necrosis, is triggered by specific extracellular cues. This process depends on proteins like mixed-lineage kinase domain-like pseudokinase (MLKL) and receptor-interacting protein kinases (RIPK1 and RIPK3). A necrosome forms as a result of necroptosis, which is triggered when caspase activity is suppressed.

The cell membrane is damaged by this structure, which causes it to rupture and trigger an inflammatory reaction. Parthanatos is another necrotic mechanism caused by the overactivation of poly (ADP-ribose) polymerase-1 (PARP-1) in response
to substantial DNA damage.

When parthanatos exhausts cellular energy, the mitochondria release apoptosis-inducing factor (AIF), which causes extensive DNA fragmentation inside the cell. in addition to Harreld. 2022).

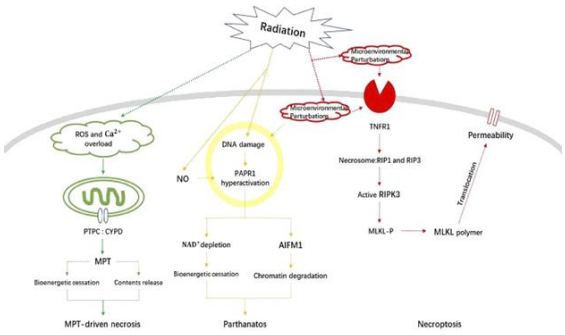


Figure 2 shows the signaling pathways that lead to radiation-induced necroptosis.

Three primary types of cell necrosis are distinguished: "parthanatos," "necroptosis," and "necroptosis-driven necrosis" are caused by the mitochondrial permeability transition (MPT). The essential molecules
that govern and carry out the process define each type (Jiao et al. in 2022).

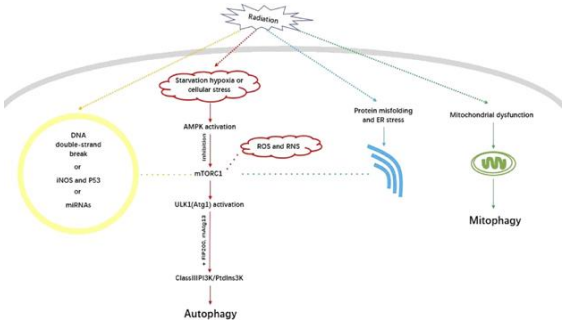
*** Autophagy-dependent cell death**

Autophagy-dependent cell death represents a regulated mechanism of cellular demise, wherein cells utilize the autophagy process to degrade their own components in response to stress or damage. In this process, damaged macromolecules or organelles are encapsulated in autophagosomes, which subsequently unite with

lysosomes to undergo destruction. Autophagy generally functions as a survival mechanism in response to stress; however, its excessive activation may result in cellular apoptosis. The regulation of autophagy initiation is predominantly governed by the ULK1 complex and the mammalian target of rapamycin complex 1 (mTORC1). When cells experience stressors such as nutrient deprivation, hypoxia, or radiation, mTORC1 activity is inhibited, allowing the activation of ULK1 and the formation of autophagosomes. Key proteins involved in this process include Atg proteins, which facilitate the formation and elongation of the autophagic vesicles (Patel et al., 2020).

Radiation exposure can induce autophagy through various pathways, particularly through DNA damage responses (DDR).

For instance, radiation-induced DNA double-strand breaks can trigger autophagy, with proteins like p53 and ATM linking DDR to autophagy activation. P53 can encourage autophagy by triggering the production of genes like DRAM and ULK1 that are essential to the autophagic process. Furthermore, endoplasmic reticulum (ER) stress, mitochondrial malfunction, and the generation of reactive oxygen species (ROS) are all common side effects of radiation exposure that might affect autophagy. In certain instances, autophagy-dependent cell death is significantly influenced by nuclear autophagy, which entails the breakdown of nuclear components (Li et al., 2022).



Autophagy-dependent cell death brought on by radiation is mediated by these signaling pathways (Figure 3).

The classical autophagy pathway is mainly regulated by the ULK1 (Atg1) complex through its interactions with the mammalian target of rapamycin complex 1 (mTORC1).

Furthermore, a particular kind of autophagy known as mitochondrial autophagy, or mitophagy, is mainly fueled by the interaction of the E3 ubiquitin ligase PARKIN and the mitochondrial kinase IPNK1. On the other hand, mitophagy can function without these molecular connections (Jiao et al. in 2022).

*** Pyroptosis**

The Gasdermin protein family is necessary for pyroptosis, a type of cell death that is mainly brought on by inflammatory caspases 1 and 11.

via both traditional and uncon ventional routes, these caspases activate and cleave Gasdermin proteins, causing the plasma membrane to become perforated. Cell death is the ultimate result of this, which also causes cell swelling. Although pyroptosis was first thought to only happen in monocyte-derived cells and to be independent of caspase-1, it has since been seen in a variety of cell types and can involve other caspases, including caspase-3, which contributes to immune defense against intracellular pathogens (Ning et al. (2024). Caspases such as caspase-1, caspase-3, and their human and mouse counterparts, caspase-4 and caspase-5, are responsible for the process, in response to diverse stimuli. Research has shown that lipopolysaccharides (LPS) from gram-negative bacteria interact with pattern recognition receptors (PRRs), often initiating pyroptosis. This activation cleaves Gasdermin proteins, particularly GSDMD, which is processed by caspase-1 to facilitate the cell death pathway. Additionally, pyroptosis increases the inflammatory response by encouraging the release of pro-inflammatory cytokines such as IL-1β and IL-18 (Cao et al. (2023).

Studies show that high-dose irradiation (10 and 20 Gy) increases caspase-1 activity and induces pyroptosis in bone marrow-derived macrophages (BMDM). The NLRP3 inflammasome has been shown to promote radiation-induced pyroptosis in these macrophages and initiate pyroptosis in lung cells exposed to radiation in mice. In addition, human umbilical vein endothelial cells (HUVECs) undergo pyroptosis when exposed to 10 Gy x-rays, leading to a decrease in Cx43 expression. When Cx43 is overexpressed, it significantly reduces pyroptosis by lowering active caspase-1 levels. These results highlight pyroptosis as a key mechanism of cell death triggered by ionizing radiation, although further research is needed to identify which cell types are most susceptible to this effect (Smith et al., 2021).

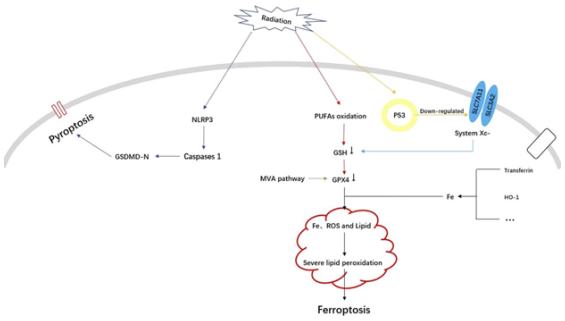


Figure 4 shows the signaling pathways that cause ferroptosis and pyroptosis when exposed to radiation.

Numerous pathways alter the activity of glutathione peroxidases (GPXs), which either directly or indirectly causes ferroptosis.

Among these pathways are heme oxygenase 1 (HO-1) and the methionine sulfur transfer pathway, voltage-dependent anion channel (VDAC) transport, system Xc-, P53, GPX4, and transferrin-mediated internal iron supply (Jiao et al. in 2022).

*** Ferroptosis**

Iron-chelating agents and lipophilic antioxidants can inhibit ferroptosis, a unique type of cell death regulated by the enzyme GPX4, which is activated by oxidative stress. This pathway is essential for tumor suppression and is mainly fueled by widespread lipid peroxidation, which needs iron and generates reactive oxygen species (ROS) (Zhang et al. in 2022). When it comes to morphology, ferroptosis and necrosis are similar. These include certain mitochondrial alterations like shrinkage, electron-dense structures, cristae loss, and outer mitochondrial membrane rupture. Ferroptosis is triggered by multiple pathways that converge on the control of GPX4 activity. These include heme oxygenase-1 (HO-1), the methionine sulfur transfer pathway, p53-mediated ferroptosis, the function of voltage-dependent anion channels (VDACs), the inhibition of the Xc- transporter, direct inhibition of GPX4 by substances such as RSL3, the function of the Xc-transporter, and the role of transferrin in supplying internal iron sources (Li et al. (2024).

By decreasing GSH-dependent lipids, GPX4 functions as a major natural inhibitor of ferroptosis and helps to prevent lipid peroxidation. According to research, ferroptosis entails the selective oxidation of particular phosphatidylethanolamines that contain polyunsaturated fatty acids (PUFAs), including adrenaline and arachidonic acid (Tang et al. 2024).

Both self-oxidation and enzymatic reactions, specifically those involving the activity of

lipoxygenases (LOXs) and cyclooxygenases (COXs), can produce lipid peroxides. Different mechanisms control the peroxidation of PUFAs during apoptosis; GPX4 indirectly inhibits lipid peroxidation, whereas LOXs directly encourage it. Ferroptosis inhibitors have shown protective effects in models of acute lung injury caused by radiation by lowering reactive oxygen species (ROS), inflammatory cytokines, and lung damage in mice. As a result of radiation-induced ROS production and AcSL4 expression activation, which results in lipid peroxidation and tumor cell ferroptosis, ferroptosis also plays a role in radiation-induced cancer cell death (Ye et al. the year 2020).

*** Immunogenic cell death**

A unique method of cellular death known as immunogenic cell death (ICD) triggers the adaptive immune response to particular antigens, whether they are endogenous (originating from cells) or exogenous (such as viral). This technique enables the immune system to react to antigens from dying cells or the host organism. In the realm of radiotherapy, ICD is especially pertinent, as it was previously believed that ICD was solely initiated by the direct demise of tumor cells within the irradiated zone. It has been noted that radiation can activate the host's immune system to attack residual tumor cells. Radiation-induced immune-mediated tumor eradication is currently acknowledged as an alternative radiosensitization mechanism, establishing it as the fifth principle in radiobiology (Liu et al., 2023).

The predominant immunosuppressive tumor microenvironment frequently limits the immunogenic benefits of radiation therapy in clinical and in vivo settings.

Through the combination of immune checkpoint inhibitors, for example, these immunosuppressive barriers can be overcome. G. Targeted radiation therapy, like anti-CTLA4 or anti-PD-1, can increase radiation therapy's immunogenic effects and lead to immune-driven tumor eradication (Yamasaki et al. (2020). Recent research suggests that low doses of radiation may alter macrophages into an M1 or iNOS+ phenotype, which would improve the immune system's ability to target tumors and draw in tumor-specific T cells. Interferon beta (IFN-κB) production can be increased by repeated low-dose radiation that does not activate

Trex1 in order to draw in BATF3-dependent dendritic cells and encourage immune activation. However, cytosolic DNA that accumulates after radiation exposure is broken down by the DNA exonuclease Trex1, which is activated by higher radiation doses (above 12–18 Gy) and reduces the immunogenic potential of tumor cells (Vaes et al. 2021).

*** Mitotic catastrophe**

The phrase "mitotic catastrophe" refers to a series of biological events that Cause premature or abnormal cell division.

These processes are frequently brought on by a variety of physical and chemical factors, and they are especially pertinent to radiation-induced cell death. The following are important causes of mitotic failure: errors in cell cycle checkpoints, microtubule instability, disruption of mitotic control, and severe DNA damage. There are two primary mechanisms that can result in a deadly mitotic disaster: (1) excessive centrosome duplication and (2) P53 inhibition, which interferes with DNA repair and G2 or M phase checkpoints (Ye et al. (2020), leading to the formation of over-duplicated centrosomes that cause multipolar division, improper chromosome segregation, and the development of abnormal nuclear structures like megakaryocytes, binucleate, or multinucleate cells. The CDK2-cyclin A or E complex is crucial for initiating centrosome amplification, a process controlled by P53, underscoring the interdependence of these pathways. Additionally, after radiation exposure, P21 fails to increase in the absence of P53, which activates CDK2 or cyclin A/E or related complexes (Bai et al., 2023).

Cell death linked to mitotic catastrophe is revealed by experiments using radiotherapy and radioimmunotherapy. The spindle assembly checkpoint, also known as the mitotic checkpoint, is triggered when cells experience a brief G2 phase arrest after radiation, which occurs before mitosis, to inhibit mitotic progression. In certain cells, a delayed apoptotic pathway is initiated during mitotic exit, involving caspase activation and mitochondrial impairment. Alternatively, cells may undergo defective mitosis, eventually leading to death through necrosis or senescence. A lag in mitotic malfunction is typically observed 2–6 days following radiation exposure (Z. Zhang et al., 2020).

*** Cell senescence**

Early on in the senescence process, cells continue to perform metabolic tasks but permanently lose their capacity to divide. They are irreversibly stopped in the G1 phase and ultimately die.

Normal cells usually experience senescence when their telomeres shorten to the point where they can no longer replicate as efficiently (Peng et al. in 2020). Radiation exposure causes a type of proliferative cell death known as radiation-induced senescence. Low levels of radiation exposure can cause the DNA damage response (DDR), which detects DNA damage and initiates a process that temporari ly halts the cell cycle. On the other hand, senescence may result from extensive damage that is left unfixed. Radiation-induced senescence is independent of telomere shortening, in contrast to replicative senescence, and research indicates that this process may impact telomere regulation by TERT and other genes (Ma et al. (2021).

After exposure to radiation, the activation of P53 plays a crucial role in promoting cell survival by inducing growth arrest and enhancing DNA repair processes. In tumor cells, P53 contributes to the onset of senescence following radiotherapy, a process that depends on the type and extent of DNA damage, often linked to P21 expression. Reduced P53 activity has been shown to impair radiation-induced senescence. Interestingly, when P53, P21, or P16 are absent, senescence occurs more rapidly, suggesting that other genes may also be involved in regulating this pathway (W. Zhu et al., 2021).

*** Protective Properties of Nutrients**

*** Antioxidants and Free Radical Scavenging**

Dhivya et al. (2023), The study conducted on Sarcostemma brevistigma, an ethnomedicinal plant commonly known as "Kodikali," aimed to evaluate its in vitro antioxidant potential through various assays. The research was motivated by the increasing interest in natural antioxidants derived from medicinal plants, which are believed to mitigate oxidative stress and its associated health risks, including cancer and cardiovascular diseases.

Sarcostemma brevistigma's ethanolic extract's antioxidant qualities were assessed using a variety of in vitro methods. Nitric oxide radical inhibition, hydroxyl radical scavenging, DPPH (1,1-diphenyl-2-picrylhydrazyl) radical scavenging, and superoxide radical scavenging activity were

among these methods, and the phosphomolybdenum assay. Each of these assays targets a distinct reactive oxygen species (ROS) and evaluates the extract's capacity to neutralize the ROS that it targets.

One popular technique for assessing an antioxidant's capacity to scavenge free radicals is the DPPH assay.

Sarcostemma brevistigma's ethanolic extract demonstrated a concentration-dependent DPPH radical scavenging effect in this investigation.

The findings showed that as the extract's concentration rose, the percentage of DPPH inhibition also increased, demonstrating the extract's potent radical scavenging ability. The reducing power assay further confirmed the antioxidant potential of the extract.

The ethanolic extract showed significant reducing capacity, comparable to that of standard antioxidants like ascorbic acid and quercetin. This suggests that the extract can act as an effective electron donor, which is a crucial mechanism in antioxidant activity. The study also quantified the total phenolic and flavonoid content in the extracts, as these compounds are known to contribute significantly to antioxidant activity. The results indicated a high total phenolic content, which correlated positively with the observed antioxidant activity. This finding aligns with previous studies that have established a link between phenolic compounds and antioxidant efficacy.

The flavonoid content was also substantial, further supporting the notion that these phytochemicals play a vital role in the antioxidant properties of Sarcostemma brevistigma.

Another significant finding was the extract's ability to inhibit nitrite formation, which is indicative of its potential to prevent oxidative damage.

The results showed a marked reduction in nitrite levels in the presence of the extract, reinforcing its role as an effective antioxidant. The study compared the antioxidant activity of Sarcostemma brevistigma with that of standard antioxidants. The ethanolic extract demonstrated comparable or superior antioxidant activity in several assays, suggesting that it could serve as a viable alternative to synthetic antioxidants in food and pharmaceutical applications.

The results of this study clearly indicate that Sarcostemma brevistigma possesses powerful in vitro antioxidant activity, attributed

to its high phenolic and flavonoid content. The encouraging findings suggest that this plant could be a valuable source of natural antioxidants, with potential applications in the prevention of oxidative stress-related diseases. The study emphasizes the need for further investigation into the specific chemical compounds responsible for the antioxidant effects and their mechanisms of action.

*** Nutrients Supporting DNA Repair**

Radiation exposure can directly damage DNA by causing breaks in its strands, leading to mutations, cellular dysfunction, and increased cancer risk.

The body relies on specific nutrients to support DNA repair mechanisms that help maintain genomic stability and mitigate the effects of this damage. Among the most important of these nutrients are folate and vitamin B12, both of which play central roles in DNA synthesis and repair (Fenech, 2019). For nucleotide synthesis, which is the process of making new building blocks for DNA, folic acid, which is a B vitamin, is absolutely necessary. Folate is also responsible for providing the methyl groups that are required for DNA methylation, which is a process that controls gene expression and safeguards the integrity of DNA. Foods rich in folate include leafy greens, legumes, and fortified cereals. A deficiency in folate may impair DNA repair mechanisms, increasing the likelihood of radiation-induced mutations (Fenech, 2019). Vitamin B12 works synergistically with folate to ensure proper DNA synthesis and repair. This vitamin helps to convert folate into its active form, which is needed for building and maintaining DNA. Without adequate B12, cells cannot efficiently use folate, leading to incomplete DNA repair processes. Vitamin B12 is mostly found in meat, fish, dairy, and eggs. It is an important part of cell division and the security of the genome as a whole. Ensuring adequate levels of both folate and B12 is especially important for those at higher risk of radiation exposure, as deficiencies in either nutrient can lead to impaired DNA repair and heightened vulnerability to radiation-induced damage (Halczuk et al., 2023).

Another critical nutrient in DNA repair is zinc, a trace mineral that acts as an essential component of many enzymes that are involved in the synthesis and repair of DNA. Zinc supports enzymes such as DNA polymerase, which helps synthesize new DNA strands, and DNA ligase,

which repairs broken DNA strands. The mineral's role in cell division and protein synthesis further underscores its importance for DNA stability and repair. Zinc is found in foods like meat, shellfish, legumes, and seeds. Because zinc deficiency can impair DNA repair enzymes, maintaining adequate zinc levels is crucial for cellular resilience against radiation-induced DNA damage (Costa et al., 2023). Niacin, which is also called vitamin B3, helps fix DNA by making NAD+, which is a coenzyme that cells need to make energy and repair DNA. NAD+ is needed for enzymes like poly (ADP-ribose) polymerases (PARPs) to work. PARPs find DNA strand breaks caused by radiation and start fixing them. Foods high in niacin include poultry, fish, whole grains, and legumes. Niacin's involvement in generating NAD+ makes it fundamental for the cellular response to DNA damage, ensuring that cells can efficiently repair breaks and maintain genetic integrity (Jung et al., 2022). Polyphenols such as resveratrol and quercetin, although not directly involved in DNA synthesis, can play supportive roles in DNA repair. These compounds, found in foods like berries, grapes, apples, and onions, act as antioxidants and reduce oxidative stress, which can cause additional DNA damage. Some polyphenols may also modulate the expression of DNA repair genes, enhancing the cell's ability to respond to and repair damage. Research suggests that a diet rich in polyphenols may improve DNA repair capacity by reducing oxidative stress and supporting cellular mechanisms that maintain DNA integrity (M. F. Fenech et al., 2023).

*** Anti-inflammatory Compounds**

Radiation exposure often triggers an inflammatory response in the body as it tries to manage and repair cellular damage. However, this inflammatory response can inadvertently contribute to further tissue damage, creating a cycle of inflammation that can worsen oxidative stress and impair cell function. Specific anti-inflammatory nutrients can help regulate this response, providing a protective effect by reducing inflammation and minimizing secondary damage (Gonfa et al., 2023).

The primary sources of omega-3 fatty acids, some of the most studied anti-inflammatory nutrients, are flaxseeds, chia seeds, and walnuts, as well as fatty fish like salmon, mackerel, and sardines. By affecting the production of prostaglandins and cytokines,

which are chemicals that regulate inflammation, omega-3 fatty acids EPA (eicosapentaenoic acid) and DHA(docosahexaenoic acid) have been demonstrated to affect the inflammatory response.

Omega-3 fatty acids can help prevent excessive inflammation in radiation-exposed tissues by balancing pro-inflammatory and anti-inflammatory signaling when taken on a regular basis. Studies suggest that these fatty acids may help reduce inflammatory markers in individuals undergoing radiation therapy, improving cellular resilience and recovery (Giacobbe et al., 2020).

Another potent anti-inflammatory is curcumin, the main ingredient in turmeric. A protein complex that is essential for controlling inflammation and frequently activated in reaction to radiation-induced damage, nuclear factor kappa B (NF-κB) is inhibited by curcumin. Curcumin can reduce inflammation at the molecular level by suppressing NF-κB, which lowers the synthesis of cytoki nes that promote inflammation. This compound has also been shown to reduce oxidative stress, further enhancing its protective effects in radiation-exposed cells. Curcumin is often combined with black pepper, which contains piperine, to enhance its absorption. Regular consumption of turmeric or curcumin supplements may offer benefits in managing inflammation associated with radiation exposure (Peng et al., 2021). Strong anti-inflammatory and antioxidant qualities are exhibited by flavonoids, a class of polyphenolic substances present in a variety of fruits, vegetables, tea, and dark chocolate. Inhibiting inflammatory enzymes like cyclooxygenase (COX) and lipoxygenase (LOX), which generate inflammatory mediators, is how flavonoids like quercetin, which are present in apples, onions, and berries, function. Quercetin also stabilizes mast cells, which release histamine and other inflammatory substances in response to cellular damage. The combined anti-inflammatory and antioxidant effects of flavonoids help protect cells from radiation-induced oxidative stress and inflammation, supporting tissue health and reducing the risk of chronic inflammatory conditions (Rakha et al., 2022).

Vitamin D is another essential nutrient that has anti-inflammatory qualities and regulates immune function. Vitamin D regulates the activity of immune cells that contribute to inflammation, including macrophages and T-cells. Sufficient

vitamin D levels can help prevent an overactive immune response, reducing inflammation and promoting immune balance. Since radiation exposure can disrupt immune function and provoke an inflammatory response, adequate vitamin D levels are essential for mitigating these effects. Sunlight exposure, fortified dairy products, and fatty fish are all good sources of vitamin D. Supplementation may be required to maintain optimal levels in populations with limited sun exposure (Chen et al. 2020).

Resveratrol is a polyphenol with strong anti-inflammatory and antioxidant qualities that is found in red wine, berries, and grapes. Resveratrol can limit the generation of inflammatory cytokines and inhibit inflammatory pathways, including those controlled by NF-κB. Its dual role as an antioxidant and anti-inflammatory agent helps protect against oxidative damage and inflammation in cells exposed to radiation. Research indicates that resveratrol's protective effects may extend to reducing radiation-induced damage in the heart, liver, and other organs, making it a valuable nutrient in mitigating the adverse effects of radiation exposure (Meng et al., 2021).

*** Conclusion**

This study highlights the significance of nutrition in mitigating the adverse health effects of radiation exposure. Nutrients such as antioxidants, DNA repair-supporting vitamins, and anti-inflammatory compounds play crucial roles in protecting against radiation-induced cellular damage. This nutritional approach aligns with both modern preventive medicine and traditional practices, offering a complementary pathway for managing radiation risks. Future research should focus on validating these findings in diverse populations and establishing nutritional guidelines that can serve as accessible, non-invasive tools to enhance radiation resilience. These strategies are particularly relevant in a world where radiation exposure may increasingly impact public health, necessitating a proactive stance on nutritional intervention as a fundamental component of radiation protection and disease prevention.

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