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Original article

Alterations of Hematopoiesis Markers Induced by Exposure to Cobalt-60 or Cesium-137 in albino mice

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Abstract
Ionizing radiation from radioactive isotopes such as Cobalt -60(Co-60) and Cesium-137(Cs-137) is widely employed in medical, industrial, and scientific applications; uncontrolled exposure poses a serious hazard to biological systems. This study aimed to evaluate and compare the time -dependent toxicological effects of Co-60 and Cs-137 gamma radiation on hematopoietic dynamics and cell morphology in male albino mice. Forty- two adult male albino mice were divided randomly into seven equal groups (n = 6): one unirradiated control and six irradiated groups exposed to either Co-60 or Cs-137 for 6,12,24 hours. Hematological parameters, including red blood cell count, Hemoglobin concentration, platelet count, white blood cell counts, and leukocyte differential count, were analyzed alongside cytomorphological evaluations of peripheral blood and bone marrow smears. Platelets have the greatest radio sensitivity, exhibiting severe thrombocytopenia at all intervals, while leukocytes declined rapidly and erythrocytes showed delayed depletion. Blood and bone marrow smears revealed progressive morphological damage over time, transitioning from initial chromatin clumping to widespread stromal aplasia, ghost membrane formation, nuclear pyknosis, and karyorrhexis by 24 hours. High-energy Co-60 induced an earlier onset of cellular toxicity, whereas Cs-137 displayed a brief latent period before converging to equivalent systemic damage. Both Co-60 and Cs-137 induce severe, duration-dependent hematopoietic failure and bone marrow aplasia in male albino mice. While higher-energy Co-60 accelerates the initial onset of myelosuppression, both isotopes result in equivalent late-stage cytopathological destruction, highlighting the critical necessity for rapid therapeutic intervention following acute gamma radiation exposure.
Keywords. Hematopoiesis, Blood Parameters, Cobalt-60, Cesium-137, Albino Mice.

Introduction

Radiation is defined as energy released in the form of electromagnetic waves or subatomic particles. It encompasses various forms, including sunlight, visible light, X-rays, gamma rays, and nuclear reactor emissions. Even visible light itself constitutes a form of radiation emitted by electrons bound to atoms [1]. Radiation is classified based on its composition, energy levels, and ionizing capacity. Exposure to nuclear radiation induces chemical alterations within the tissues of living organisms, leading to substantial bodily damage. The severity of these chemical changes is directly proportional to the absorbed radiation dose. Importantly, the clinical manifestations of radiation injury may not appear immediately but rather after a latent or incubation period that can extend for years, depending on the radiation type and exposure circumstances [2]. Among the various types of electromagnetic radiation, ionizing forms, particularly alpha particles, beta particles, and gamma rays, pose the greatest danger to biological systems. Gamma rays exhibit high penetration power due to their high frequency and energy, requiring dense shielding materials such as lead or concrete for effective attenuation [3]. Cobalt and cesium are two elements that have found widespread applications across medical, industrial, and scientific domains. Their radioactive isotopes, Cobalt-60 and Cesium-137, are among the most utilized fission products, yet they also represent significant health hazards when uncontrolled exposure occurs [4].

Cobalt (Co) is a chemical element with atomic number 27. It is a hard, lustrous, silver-gray metal characterized by a high melting point (1,495°C) and remarkable resistance to oxidation. These unique properties render it valuable in the production of alloys, batteries, and industrial catalysts [4]. The global demand for cobalt has surged dramatically with the global transition toward renewable energy and electric vehicles. The Democratic Republic of the Congo currently supplies a significant portion of the world's cobalt market, raising ethical and environmental concerns regarding mining practices [4, 5]. Cobalt-60 (Co-60) is a radioactive isotope of cobalt that has gained prominence in various scientific and industrial applications since its discovery in the mid-20th century [6]. With a half-life of 5.27 years, Co-60 undergoes beta decay, emitting gamma radiation, which is a powerful form of electromagnetic radiation capable of penetrating materials and tissues [7]. It is a synthetic radioactive isotope of cobalt that does not occur naturally in significant quantities. It is produced artificially by irradiating stable cobalt-59 in nuclear reactors [6, 7]. Co-60 units deliver focused gamma radiation to tumors, typically at curative doses of 40–80 Gray (Gy), depending on cancer type and stage [8].

Exposure to Co-60 has been linked to an increased risk of developing cancer, particularly in tissues that are more sensitive to radiation. "The risk is dose-dependent; higher doses increase the likelihood of carcinogenic effects. In another study on Guinea pigs total body irradiation with Co-60 significantly affects the bone marrow, with maximum reductions in marrow nucleated cells and peripheral blood cell counts on day 9 post-irradiation [9]. Direct exposure to Co-60 can cause skin burns and damage to underlying tissues. The severity of damage depends on the duration and intensity of exposure [10]. Co-60 can contaminate the environment if not handled properly, leading to potential exposure for individuals in the vicinity. Contaminated water or soil can pose long-term health risks to communities [11].

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Cesium (Cs) is a chemical element with atomic number 55, belonging to the alkali metal group. It is a soft, silvery-gold metal that is highly reactive, igniting spontaneously in air and reacting explosively with water. Cs was discovered in 1860 by German chemists Robert Bunsen and Gustav Kirchhoff via spectroscopic analysis. Despite its relative rarity in Earth's crust. Cs has important applications in atomic clocks, photoelectric cells, catalysts, and ion propulsion engines [12].
Cesium-137 (Cs-137) is a radioactive isotope of the element cesium, which is classified as an alkali metal. It was first identified in the aftermath of nuclear fission processes and has since become one of the most prominent fission products found in nuclear reactors and fallout from nuclear weapons testing. With a half-life of approximately 30.05 years, Cs-137 is notable for its long-lived nature, allowing it to persist in the environment and pose potential health risks long after its initial release [13]. Cs-137 emits high-energy gamma radiation, which can penetrate human tissues. External exposure to large amounts of Cs-137 can cause burns, acute radiation sickness, and even death. Prolonged exposure to Cs-137 increases the risk of developing cancer due to its ability to damage cellular DNA. A previous study found associations between radioactive Cs-137 and increases in pancreatic cancer. There is a correlation between the geographical map of the incidence of and mortality from pancreatic cancer and the map of nuclear plants, related nuclear accidents, and nuclear tests with consequent radioactive fallout, of which Cs-137, with its half-life of about 30 years, is one of the most relevant components [14]. The aim of this study is to evaluate the effects of exposure to Co-60 or Cs-137 on hematopoiesis markers in albino mice, with a focus on understanding the time-dependent responses and the potential mechanisms underlying radiation-induced hematopoiesis alterations.

Methods

Ethical approval

The protocol received rigorous institutional oversight and was endorsed by two distinct entities: This study was approved by the Libyan Authority for Scientific Research, the Libyan National Committee for Biosafety and Bioethics, and the Al-Mukhtar Bioethics Committee- University of Omar Al-Mukhtar, El-Beyda, Libya. (NBC: 007. A. 25. 35). The radiation sources were approved by the Office of Analysis and Scientific Laboratories in Al-Mukhtar Center for Research Consultations and Training. (Reference number: 2.23.6.6).

Study design

Mature male albino mice weighing between 22- 36 g, obtained from the animal house of the Department of Zoology, University of Omar Al-Mukhtar, were acclimated for four weeks under standard laboratory conditions with free access to food and water. The animals were randomly divided into seven equal groups used for this study.
Group (1): The control group, the animals are not exposed to any type of radiation.
•Group (2): The group treated with radioactive Co-60 at (1.173-1.332Mev) for 6 hours.
•Group (3): The group treated with radioactive Co-60 at (1.173-1.332Mev) for 12 hours.
•Group (4): The group treated with radioactive Co-60 at (1.173-1.332Mev) for 24 hours.
•Group (5): The group treated with radioactive Cs-137 at (0.662Mev) for 6 hours.
•Group (6): The group treated with radioactive Cs-137 at (0.662Mev) for 12 hours.
•Group (7): The group treated with radioactive Cs-137 at (0.662Mev) for 24 hours.

Shielding and Containment (Concise)

Source Column: The Co-60 and Cs-137 sealed sources were fixed inside a vertical lead column (wall thickness: 8 cm for Co-60, 5 cm for Cs-137) with a 10 cm collimated aperture. Mice in PMMA cages were placed 50 cm from the aperture; dose rate was calculated using $\dot{D} = \Gamma \times A / d^2$ [6, 15].

Hematological analysis

Anticoagulated blood tubes were used to measure various haematological parameters such as red blood cell (RBC) count, haemoglobin (Hb) levels, and total white blood cell (WBC) count, monocyte count, lymphocyte count, granulocyte count, and platelet count. All complete blood count (CBC) tests were performed using an automated blood cell analyser (XP-300) [16].

Blood and Bone Marrow Smears

Peripheral blood films were prepared using standard spreading techniques. Bone marrow cells were collected from the femur by flushing the site with ice-chilled buffered saline. Both blood and bone marrow smears were air-dried, fixed in methanol for 15-20 minutes, and stained with Giemsa solution (1:2dilution in distilled water) for 15-20 minutes at ph. 7.0 [17, 18]. Morphological evaluation and a500-cell bone marrow differential count were performed using an Olympus CX41 light microscope [17].

Statistical Analysis

The statistical evaluation was conducted using Minitab software (version 17); statistical significance was evaluated using ANOVA with Tukey's multiple comparisons test after confirming the normal distribution in the data, and $P < 0.01$ was considered appropriate for statistical significance.

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Results

Radiation exposure (Co-60 and Cs-137) induced dose-and time dependent suppressions across all measured hematological parameters in male albino mice ($p<0.01$; Table 1).

Erythrocytic Parameters (RBC and HB): Red blood cell counts and hemoglobin concentration exhibited statistically significant reductions at 12 and 24 hours post-exposure for both isotopes ($p<0.01$). High-energy Co-60 exposure caused more pronounced declines in RBC count at 24 hours compared to Cs-137.

Thrombocytes (PLT): Platelet counts declined significantly across all exposure durations (6,12, and 24 hours) for both radioisotopes ($p< 0.01$). The suppression was most severe under Co-60 exposure at 24 hours versus Cs-137-treated mice.

Leukocytes and Differential Counts (WBC, Lymphocytes, Granulocytes): Total WBC counts and differential percentages (lymphocytes, monocytes, neutrophils, eosinophils, and basophils) dropped significantly ($p < 0.01$). Exposure to Co-60 initiated acute leukopenia and lymphopenia as early as 6 hours, whereas Cs-137 induced statistically significant leukocyte suppression starting primarily at 12hours. At 24 hours, Co-60 exposure almost completely depleted circulating basophils and severely depleted lymphocytes.

Table 1. Effects of Cs-137 and Co-60 Gamma Radiation on Hematological Parameters in Male Albino Mice.

Parameter (Unit)	Control (Mean ± SEM)	Time (Hours)	Cs-137 irradiated (Mean ± SEM)	Co-60 irradiated (Mean ± SEM)
RBCs Count ($X10^6$ u/L)	6.93 ± 0.15^{a}	6	6.33 ± 0.07^{b}	6.97 ± 0.11^{a}
		12	5.78 ± 0.09^{c}	5.08 ± 0.06^{d}
		24	5.13 ± 0.06^{d}	3.85 ± 0.11^{e}
Hb (g/dl)	16.50 ± 0.99^{a}	6	17.16 ± 0.80^{a}	16.00 ± 0.95^{a}
		12	7.17 ± 0.80^{b}	6.33 ± 0.76^{b}
		24	3.43 ± 0.48^{b}	4.30 ± 0.61^{b}
PLTs Count ($X10^3$ µ/L)	870 ± 14.07^{a}	6	796.83 ± 4.21^{c}	708.17 ± 8.20^{b}
		12	680.17 ± 2.59^{c}	579.50 ± 4.93^{e}
		24	626.00 ± 2.99^{d}	487.83 ± 8.83^{f}
WBCs Count ($X10^3$ u/L)	7.97 ± 0.28^{a}	6	7.82 ± 0.17^{ab}	6.48 ± 0.18^{bc}
		12	7.28 ± 0.08^{bc}	5.12 ± 0.19^{d}
		24	5.67 ± 0.15^{cd}	3.03 ± 0.10^{e}
Lymphocyte (%)	77.17 ± 1.92^{a}	6	70.00 ± 2.73^{ab}	60.00 ± 1.39^{cd}
		12	62.33 ± 1.58^{bc}	51.83 ± 1.25^{de}
		24	50.83 ± 1.38^{e}	30.33 ± 1.05^{f}
Monocyte (%)	3.17 ± 0.48^{a}	6	2.95 ± 0.22^{a}	1.40 ± 0.24^{bc}
		12	2.13 ± 0.29^{ab}	0.72 ± 0.20^{bc}
		24	0.88 ± 0.17^{bc}	0.35 ± 0.16^{c}
Neutrophils (%)	19.50 ± 1.48^{a}	6	20.00 ± 1.06^{a}	13.50 ± 1.18^{b}
		12	13.17 ± 0.75^{b}	9.50 ± 1.06^{bc}
		24	10.01 ± 0.58^{bc}	7.17 ± 0.65^{c}
Eosinophils (%)	2.83 ± 0.48^{a}	6	1.43 ± 0.15^{b}	0.93 ± 0.16^{bc}
		12	0.98 ± 0.10^{bc}	0.37 ± 0.13^{bc}
		24	0.48 ± 0.15^{bc}	0.10 ± 0.05^{c}
Basophils (%)	0.37 ± 0.13^{a}	6	0.18 ± 0.06^{ab}	0.03 ± 0.02^{bc}
		12	0.12 ± 0.05^{ab}	0.03 ± 0.02^{bc}
		24	0.07 ± 0.03^{ab}	0.00 ± 0.00^{c}

Values are expressed as Mean ± SEM (n = 6). Means with distinct superscript letters within the same row or parameter group differ significantly at ($p<0.01$).

Cytomorphological Changes in Peripheral Blood

Microscopic examination of peripheral blood smears revealed a time-dependent deterioration in cellular architecture and nuclear integrity following exposure to Co-60 and Cs-137 radiation (Figures 1 and 2). Control Group: Displayed normal, densely packed biconcave erythrocytes alongside intact leukocytes, including properly segmented mature neutrophils and morphologically sound lymphocytes. Six hours Post-Exposure: Early pathological alterations emerged, characterized by subtle RBC spacing irregularities, initial nuclear chromatin condensation in neutrophils, and early lymphocyte membrane distortion. Twelve hours Post-Exposure: Significant reduction in intact RBC density was observed, accompanied by erythrocyte overlapping and distortion. Leukocytes exhibited deep cytopathological aberrations, including neutrophil hyper- segmentation, distorted lymphocyte cell boundaries, and scattered cellular debris. Twenty-four Hours Post-Exposure: Complete collapse of peripheral blood film architecture occurred. The field was dominated by

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lysed ghost membranes. Extensive cellular fragments, severe neutrophil nuclear pyknosis (condensed, dark ink-spot nuclei), and membrane rupture leading to cytoplasm/ granule leakage.

Histopathological Dynamics in Bone Marrow

Serial evaluation of bone marrow smears reflected acute, dose- and time-dependent hematopoietic progenitor destruction and bone marrow aplasia (Figures 3 and 4). Control Group: Highly hypercellular marrow stroma populated with intact erythroid, myeloid, and megakaryocytic cell lines at various maturation stages. Six hours Post-Exposure: Moderate disruption of hematopoietic precursor pockets occurred, accompanied by early chromatin clumping and precursor cell membrane irregularity. Twelve hours Post-Exposure: Marked hypocellularity was evident, leaving prominent empty stromal spaces. Surviving progenitor populations formed dense, irregular clusters showing extensive nuclear fragmentation (karyorrhexis) and cellular degeneration. Twenty-four hours Post-Exposure: Severe bone marrow aplasia and stromal breakdown were recorded. The hematopoietic microenvironment displayed widespread destruction, characterized by massive empty stromal zones, widespread pyknotic nuclei, karyolysis, and complete loss of recognizable cellular lineage architecture.

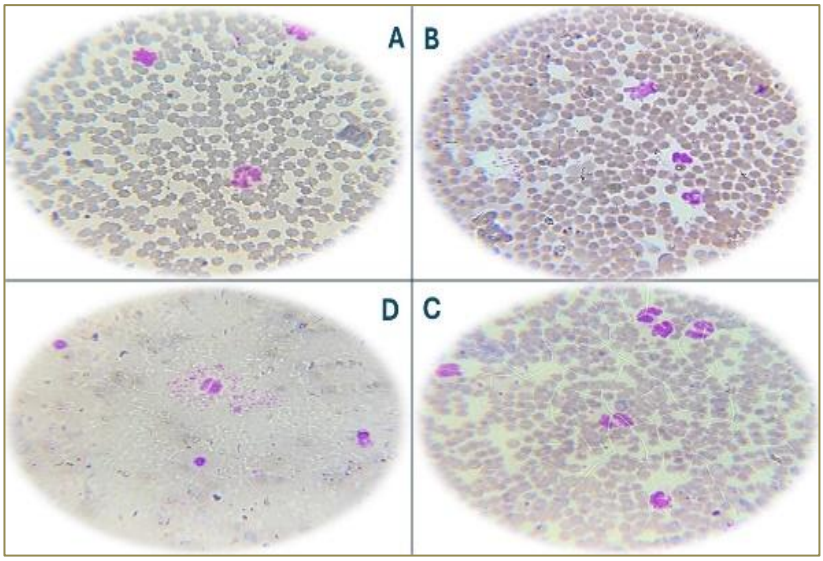


Figure 1. Representative high-power photomicrograph of peripheral blood smears illustrating the detailed cytomorphological and nuclear aberrations induced by Co-60 radiation (100× oil-immersion magnification). The panels capture the fine intracellular breakdown, progressing from intact baseline conditions in the control (A), through early structural alterations at 6 and 12 hours (B-C), to severe nuclear pyknosis and membrane collapse at 24 hours post-exposure (D).

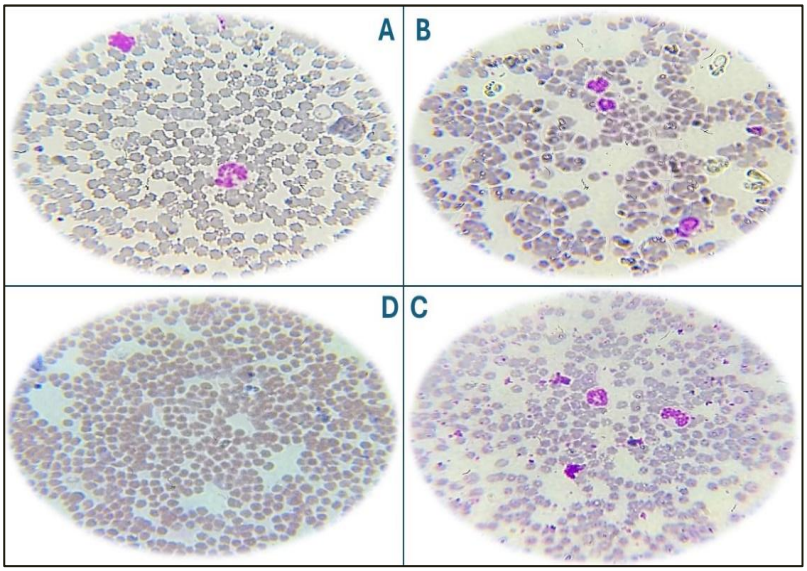


Figure 2. Representative high-power photomicrograph of peripheral blood smears illustrating the detailed cytomorphological and nuclear aberrations induced by Cesium-137 (Cs-137) gamma radiation (100 x oil immersion magnification). The panels capture the fine intracellular breakdown, progressing from intact baseline conditions in the control(A), through early structural alterations at 6 and 12 hours(B-C), to severe nuclear pyknosis and membrane collapse at 24 hours post-exposure (D).

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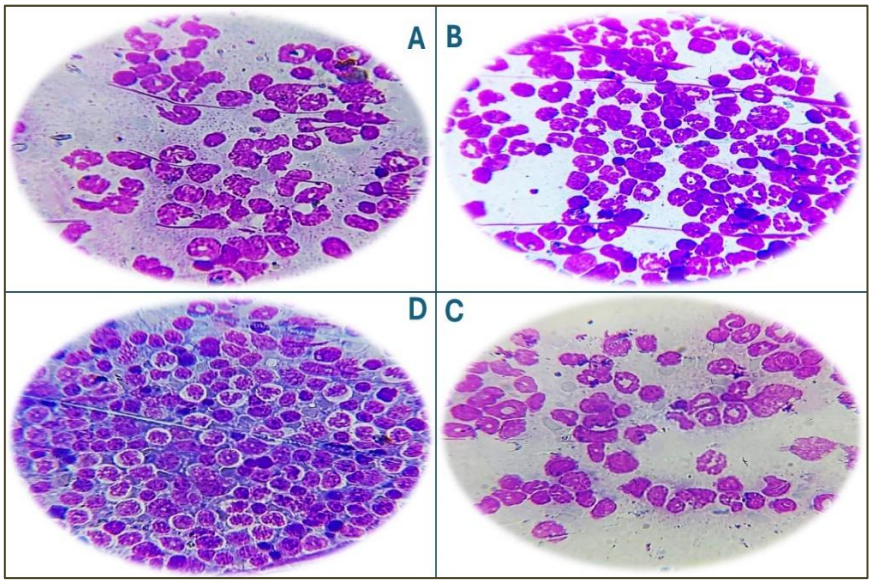


Figure 3. Representative high-power photomicrographs of bone marrow smears illustrating the detailed cytomorphological and nuclear aberrations induced by Co-60 (100x oil-immersion magnification). The panels capture the fine intracellular breakdown of hematopoietic precursors, progressing from intact nuclear and cytoplasmic architectures in the control (A), through early chromatin condensation and precursor degeneration at 6 and 12 hours (B-C), to severe nuclear pyknosis, karyorrhexis, and widespread cellular lysis by 24 hours post-exposure.

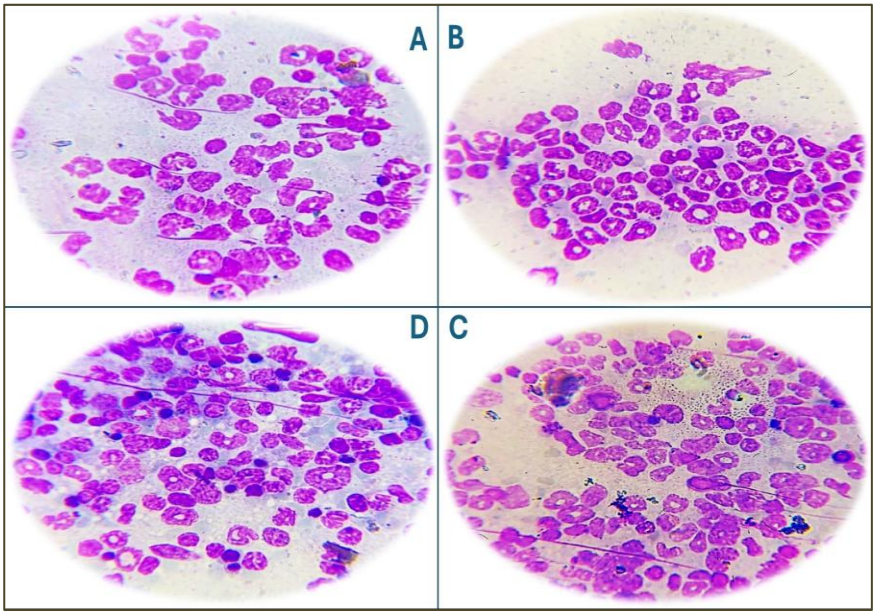


Figure 4. Representative high-power photomicrographs of bone marrow smears illustrating the detailed cytomorphological and nuclear aberrations induced by Cs-137 (100× oil-immersion magnification). The panels capture the fine intracellular breakdown of hematopoietic precursors, progressing from intact nuclear and cytoplasmic architectures in the control (A), through early chromatin condensation and precursor degeneration at 6 and 12 hours (B-C), to severe nuclear pyknosis, karyorrhexis, and widespread cellular lysis by 24 hours post-exposure.

Discussion

Exposure to gamma radiation from Co-60 and Cs-137 induced immediate, severe, and duration-dependent disruption across all hematopoietic lineages in male albino mice. The differential response observed between the two isotopes highlights the influence of energy levels and penetration dynamics on biological systems, with Co-60 exerting an earlier and more pronounced myelosuppressive effect than Cs-137.

Erythroid Lineage Depletion and Membrane Dynamics: Because mature erythrocytes lack nuclear DNA and are traditionally considered relatively radioresistant, the rapid decline in RBC counts and hemoglobin concentration, accompanied by progressive membrane ghosting- and destruction, points toward extra-medullary clearance rather than immediate genotoxicity alone. High-energy gamma radiation induces free radical oxidation of erythrocyte membrane lipids and proteins, increasing osmotic fragility and triggering splenic macrophage phagocytosis [19, 20]. The accelerated drop in RBCs observed under Cs-137 at 6 hours suggests an early membrane lysis mechanism alongside splenic sequestration. Platelets exhibited the most rapid and uniform decline ($p < 0.01$across all time points for both isotopes).

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This acute early-onset thrombocytopenia reflects the extreme radiosensitivity of megakaryocytic progenitor cells in bone marrow combined with the short circulating half-life of mature platelets [21]. Bone marrow morphology confirmed early vacuolation and degeneration of megakaryocytic precursors within 6 hours, establishing primary progenitor cell arrest as the main driver of peripheral platelet depletion rather than peripheral consumption alone [22].

Leukopenia and differential leukocyte depletion followed the classical radiobiological hierarchy. Co-60 induced significant lymphopenia and granulocytopenia starting as early as 6 hours, whereas Cs-137 required 12 hours to reach equivalent statistically significant levels. Radiation triggers interphase apoptosis in circulating lymphocytes and destroys rapidly dividing granulocytic/myeloid precursors within the marrow microenvironment [20]. Bone marrow histopathology directly mirrored these circulating cell dynamics, demonstrating rapid progression from chromatin clumping to extensive karyorrhexis, nuclear pyknosis, and ultimate stromal aplasia at 24 hours.

Both of them induced immediate, statistically significant perturbation ($P<0.01$) as early as 6 h post- exposure. Conversely, Cs-137 exhibited a latent period at 6 hours before converging to equivalent, high-grade systemic damage at 12 and 24 hours. This kinetic lag most plausibly reflects differences in photon energy and effective dose delivery rate, rather than an intrinsic difference in relative biological effectiveness. Platelets proved to be the most acutely sensitive cellular element, exhibiting severe thrombocytopenia ($P < 0.01$) at all time points for both isotopes. Lymphopenia and granulocytopenia, whereas erythrocytes exhibited a comparatively delayed depletion (significant mainly at 12-24 hours) driven primarily by membrane oxidative injury- and lipid peroxidation. And accelerated splenic sequestration. Peripheral blood and bone marrow smear analyses provide direct structural support for biochemical findings. Smears progressed from subtle erythrocyte spacing irregularities at 6 hours to widespread stromal aplasia, ghost membrane formation, nuclear pyknosis, and karyorrhexis by 24 hours, supporting early hematopoietic stem/progenitor cell apoptosis as a primary driver of peripheral toxicity.

Conclusion

In summary, this study reveals a clear time-dependent, multi-organ toxicological response to gamma radiation (Co-60 and Cs-137) in a male albino mouse Model, driven primarily by acute hematopoietic failure, systemic inflammation, and enteropathy across nearly all hematopoiesis.

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Conflicts of Interest

The authors declare no conflicts of interest.

References

1. Tanabe T. *Radiation: An Energy Carrier*. Cham (CH): Springer; 2022.
2. Nordberg GF, Nordberg M, Fowler BA, editors. *Handbook on the Toxicology of Metals*. 4th ed. Amsterdam (NL): Elsevier/Academic Press; 2015.
3. Brenner DJ, Hall EJ. Computed tomography—an increasing source of radiation exposure. *N Engl J Med*. 2007 Nov 29;357(22):2277-2284.
4. Genchi G, Lauria G, Catalano A, Carocci A, Sinicropi MS. Prevalence of cobalt in the environment and its role in biological processes. *Biology (Basel)*. 2023 Oct 12;12(10):1335. doi: 10.3390/biology12101335.
5. Wootton A. Cobalt's climate paradox. *Nat Resour J*. 2026;66(1):119-156.
6. Khan FM, Gibbons JP. *Khan's The Physics of Radiation Therapy*. 5th ed. Philadelphia (PA): Wolters Kluwer Health/Lippincott Williams & Wilkins; 2014.
7. Biswal J, Pant HJ, Sharma VK, Joseph A, Ananthanarayanan A. Preparation and characterization of cobalt-60 glass microspheres for radioactive particle tracking applications. *Appl Radiat Isot*. 2022 Jul;185:110249. doi: 10.1016/j.apradiso.2022.110249.
8. Podgorsak EB, editor. *Radiation Oncology Physics: A Handbook for Teachers and Students*. Vienna (Austria): International Atomic Energy Agency; 2005.
9. Akeem S, Lukman O, Eltahir K, Fatai O, Abiola B, Khadijat O. Bone marrow and peripheral blood cells toxicity of a single 2.0 Gy cobalt (60) ionizing radiation: an animal model. *Ethiop J Health Sci*. 2019 Mar;29(2):195-202. doi: 10.4314/ejhs.v29i2.6.
10. Brown KR, Rzucidlo E. Acute and chronic radiation injury. *J Vasc Surg*. 2011 Jan;53(1 Suppl):15S-21S. doi: 10.1016/j.jvs.2010.06.175.
11. Adeola AO, Iwuozor KO, Akpomie KG, Adegoke KA, Oyedotun KO, Ighalo JO. Advances in the management of radioactive wastes and radionuclide contamination in environmental compartments: a review. *Environ Geochem Health*. 2023 Jun;45(6):2663-2689. doi: 10.1007/s10653-022-01378-7.
12. Emsley J. *Nature's Building Blocks: An A-Z Guide to the Elements*. 2nd ed. Oxford (UK): Oxford University Press; 2011.
13. Abdel-Sabour M. Fate of cesium and strontium in soil-to-plant system (overview). *J Rad Nucl Appl*. 2022;7(2):78-83.
14. Venturi S. Correlation between radioactive cesium and the increase of pancreatic cancer: a hypothesis. *Biosfera*. 2020;12(4):242-252.
15. Valentin J. The 2007 Recommendations of the International Commission on Radiological Protection Annals of the ICRP. 2007;37(2.4):2.
16. van Dievoet MA, Louagie H, Ghys T. Performance evaluation of the Sysmex(®) XP-300 in an oncology setting: evaluation and

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comparison of hematological parameters with the Sysmex(®) XN-3000. *Int J Lab Hematol*. 2016 Oct;38(5):490-496. doi: 10.1111/ijlh.12522.
17. Bain BJ, Laffan MA, editors. *Dacie and Lewis Practical Haematology*. 12th ed. Elsevier; 2016.
18. Turgeon ML. *Clinical Hematology: Theory and Procedures*. 5th ed. Philadelphia (PA): Wolters Kluwer Health/Lippincott Williams & Wilkins; 2012.
19. Puchała M, Szweda-Lewandowska Z, Kiefer J. The influence of radiation quality on radiation-induced hemolysis and hemoglobin oxidation of human erythrocytes. *J Radiat Res*. 2004 Jun;45(2):275-279. doi: 10.1269/jrr.45.275.
20. Thiagarajan P, Parker CJ, Prchal JT. How do red blood cells die? *Front Physiol*. 2021 Mar 15;12:655393. doi: 10.3389/fphys.2021.655393.
21. Dainiak N, Waselenko JK, Armitage JO, MacVittie TJ, Farese AM. The hematologist and radiation casualties. *Hematology Am Soc Hematol Educ Program*. 2003:473-496. doi: 10.1182/asheducation-2003.1.473.
22. Meng F, Chen S, Liu C, Khan MS, Yan Y, Wan J, et al. The role of PKC in X-ray-induced megakaryocyte apoptosis and thrombocytopenia. *Blood Cells Mol Dis*. 2024 Jan;104:102798. doi: 10.1016/j.bcmd.2023.102798.

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