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Original article

Comparative Study of Radiotoxicity Between Cobalt-60 and Caesium-137 of Some Hepatic Parameters in Male Albino Mice

Salima Abdelrahim[1], Asma M. Othman[1], Marfoua Ali*[2]

[1]*Department of Physics, Faculty of Science, Omar Al-Mukhtar University, El-Beyda, Libya*
[2]*Department of Zoology, Faculty of Science, Omar Al-Mukhtar University, El-Beyda, Libya*
Email. *marfouas@yahoo.com*

Abstract
Ionising radiation from gamma emitters like Cobalt-60 (^{60}Co) and Caesium-137 (^{137}Cs) poses acute biophysical risks to radiosensitive tissues. This study presents a comparative evaluation of the time-dependent radiotoxic effects of ^{60}Co and ^{137}Cs gamma exposure on hepatic enzyme activities, including Aspartate Aminotransferase (AST), Alanine Transaminase (ALT), and Alkaline Phosphatase (ALP) in serum and tissue homogenates of male Albino mice. Forty-two male mice were divided into seven groups: control (unirradiated) and six exposure groups (^{60}Co or ^{137}Cs for 6, 12, and 24 hours). Both ^{60}Co and ^{137}Cs induced a time-dependent reduction in body weight. In the mean control group, Exposure to ^{60}Co or ^{137}Cs radiation resulted in a non-significant increase in mean liver weight relative to the control group at all three intervals, with a significant increase at 24 hours with both isotopes. Results demonstrated time-dependent hepatocellular effects for both radiation sources. Radiation exposure induced serum AST, ALT, and ALP significantly increasing after 24-hour ^{60}Co exposure, reaching 385.2 ± 32.7 U/L, 99.3 ± 2.7 U/L, and 281 ± 17.5 U/L, respectively. Similar upward trends were measured in liver tissue homogenates. Overall, ^{60}Co exhibited greater acute radiotoxicity than ^{137}Cs at equivalent exposure durations, fundamentally attributed to its higher gamma photon energy (1.17 and 1.33 MeV vs. 0.662 MeV) and greater energy deposition via Compton scattering and ionisation events.
Keywords. Cobalt-60, Caesium-137, Gamma Radiation, Radiotoxicity, Liver Enzymes and Biological Physics.

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 ionising 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].

Gamma radiation, an energetic form of electromagnetic radiation emitted during nuclear decay, interacts with biological systems primarily through photoelectric absorption, Compton scattering, and pair production. Upon passage through biological tissues, photon interactions induce radiolysis of intracellular water, producing reactive oxygen species (ROS) such as hydroxyl radicals (OH*). These free radicals trigger oxidative stress, lipid peroxidation of cell membranes, and DNA double-strand breaks, leading to severe cellular dysfunction or apoptosis [3]. Among industrial and clinical radioisotopes, Cobalt-60 (^{60}Co) and Caesium-137 (^{137}Cs) are prominent gamma sources. ^{60}Co decays via beta emission followed by the prompt release of two high-energy gamma photons (1.17 MeV and 1.33 MeV) with a half-life of 5.27 years. Conversely, ^{137}Cs ($t_1/_2 \approx 30.17$ years) decays into metastable ^{137m}Ba, emitting a single gamma photon of 0.662 MeV [4]. The hepatic tissues represent critical target organs with high radiosensitivity. Hepatocytes suffer membrane permeability breakdown and mitochondrial damage, leading to cytosolic enzyme leakage. Although both isotopes emit low-LET gamma radiation, differences in photon energy and linear energy transfer (LET) alter their comparative biological effectiveness (RBE). This study aims to quantify and compare the time-dependent radiotoxicity of ^{60}Co and ^{137}Cs on physiological indices of the liver in male Albino mice.

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. 37). The radiation sources were approved by the Office of Analysis and Scientific Laboratories in Al-Mukhtar Centre for Research Consultations and Training. (Reference number: 2.23.6.6).

Study design

Mature male albino mice weighing between 22 and 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 for this study. Group 1 served as the control

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group and was not exposed to any type of radiation. Group 2 was treated with radioactive ^{60}Co at 1.173–1.332 MeV for 6 hours, Group 3 was treated with radioactive ^{60}Co at 1.173–1.332 MeV for 12 hours, and Group 4 was treated with radioactive ^{60}Co at 1.173–1.332 MeV for 24 hours. Group 5 was treated with radioactive ^{137}Cs at 0.662 MeV for 6 hours, Group 6 was treated with radioactive ^{137}Cs at 0.662 MeV for 12 hours, and Group 7 was treated with radioactive ^{137}Cs at 0.662 MeV for 24 hours.

Shielding and Containment

Source Column: The ^{60}Co and ^{137}Cs sealed sources were fixed inside a vertical lead column (wall thickness: 8 cm for ^{60}Co, 5 cm for ^{137}Cs) with a 10 cm collimated aperture. Mice in PMMA cages were placed 50 cm from the aperture; The absorbed dose rate ($D^{\cdot}$) and total absorbed doses for the 6, 12, and 24 h exposure intervals were calculated using the inverse-square law and activity-based exposure geometry ($\dot{D} = \Gamma \times A / d^2$) [5, 6]. The corresponding total absorbed doses delivered to the exposure groups over 6, 12, and 24 hours were determined based on the respective photon energies and source strengths, ensuring accurate quantification of the radiation burden for each isotope.

Biochemical Assays

Following sacrifice, biochemical evaluation, serum and 10% w/v liver tissue homogenates (prepared in cold phosphate-buffered saline, centrifuged at 10,000 × g for 20 min at 4°C) were analysed for AST, ALT and ALP using spectrophotometric kinetic methods (Humalyzer Junior) with Archem Diagnostic kits by applying the approach [7].

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 $P < 0.05$ was considered appropriate for statistical significance.

Results

The statistical assessment of the total body weight in male albino mice exposed to ^{60}Co or ^{1}distinctia time-dependent variations when comparing the pre-exposure to the post-exposure outcomes across three discrete intervals (6 h, 12 h and 24 h). Exposure to ^{60}Co at the 6 h interval produced a slight decline in mean body weight from pre-exposure to post-exposure ($P > 0.05$). When the exposure duration extended to 12 h and 24 h, a statistically significant body weight reduction was observed for both times (Table 1). Exposure to ^{137}Cs didn't produce any significant change in mean body weight relative to the pre-exposure value ($P > 0.05$). Exposure at both 12 h and 24 h intervals resulted in a significant decline in body weight relative to the pre-exposure value (Table 1). Both isotypes caused significant changes in mean body weight when compared before and after radiation.

Meanwhile, exposure to ^{60}Co radiation resulted in an increase in mean liver weight at the 24 h interval compared with the mean control group but didn't induce any significant changes in mean liver weight, which reached statistical significance at (6 h, 12 h, and 24 h) intervals compared to the control group ($P > 0.05$). Exposure to ^{137}Cs radiation resulted in an increase in mean liver weight relative to the control group at all three intervals (6 h,12 h, and 24 h). This increase was statistically significant at the ($P < 0.05$) level at the 24 h interval. Comparison between the two radioisotopes: At the ($P < 0.05$) significance level, the increase in liver weight occurred only in the Cs-137-exposed group at the 24 h interval, whereas none of the ^{60}Co-exposed groups reached this level of significance (Table 2).

Table 1. Body Weight Variations Before and After Exposure to Co-60 Radiation in Male Albino Mice

Parameter (Unit)	Control (Mean ± SD)	Time (Hours)	^{60}Co Unirradiated (Mean ± SD)	^{60}Co Irradiated (Mean ± SD)	P-Value
Body weight (gm)	27.17 ± 0.65^{bc}	6	26.17 ± 2.01^{c}	25.67 ± 1.99^{b}	0.076
		12	31.50 ± 0.44^{a}	29.83 ± 0.99^{ab}	0.031*
		24	30.67 ± 0.66^{ab}	29.83 ± 0.98^{ab}	0.042*
		Time (Hours)	**^{137}Cs Unirradiated (Mean ± SD)**	**^{137}Cs Irradiated (Mean ± SD)**	**P-Value**
		6	27.67 ± 1.11^{bc}	27.50 ± 1.06^{ab}	0.363
		12	32.50 ± 1.04^{a}	30.00 ± 1.02^{a}	0.001**
		24	33.33 ± 1.90^{a}	31.17 ± 1.99^{a}	0.003**

*Data are expressed as Mean ± standard deviation; statistical significance was determined using Tukey's test. Means within the same row or column bearing distinct superscript letters differ significantly; letters indicate significance at (p<0.05). * Indicates significance at (p<0.05) before and after radiation.*

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Table 2. Liver Weight Variations After Exposure to ^{60}Co or ^{137}Cs Radiation in Male Albino Mice

Parameter (Unit)	Control (Mean ± SD)	Time (Hours)	^{137}Cs irradiated Mean ± SD	^{60}Co irradiated Mean ± SD	P. Value
Liver weight (gm)	1.84 ± 0.14^{b}	6	1.96 ± 0.14^{ab}	1.87 ± 0.32^{b}	**0.006****
		12	1.98 ± 0.26^{ab}	1.80 ± 0.22^{b}	
		24	2.52 ± 0.30^{a}	1.93 ± 0.18^{ab}	

Data are expressed as Mean ± standard deviation; statistical significance was determined using Tukey's test. Means within the same row or column bearing distinct superscript letters differ significantly; letters indicate significance at ($p<0.05$).

Both isotypes of radiation with serum (Table 3) and intra-hepatic tissue homogenates (Table 4) showed significant, time-dependent increases in AST, ALT, and ALP enzyme activities following irradiation ($P < 0.05$). Serum AST and ALT peaked at 385.2 ± 32.7 U/L and 99.3 ± 2.7 U/L in the 24 h ^{60}Co group, compared to control values of 155.8 ± 11.5 U/L and 35.7 ± 3.4 U/L, respectively. Alkaline phosphatase (ALP) demonstrated a continuous rise, reaching 281 ± 17.5 U/L in serum and 193.5 ± 6.5 U/L in liver tissue homogenate after 24 hours of ^{60}Co exposure.

Table 3. Serum hepatic enzyme activity (AST, ALT, ALP) following radiation exposure.

Exposure Groups	Time (h)	AST (U/L)	ALT (U/L)	ALP (U/L)
Control	-	155.8 ± 11.5^{c}	35.7 ± 3.4^{c}	113.8 ± 4.9^{f}
^{60}Co	6	199.5 ± 8.6^{c}	35.2 ± 4.2^{c}	150 ± 5.3^{e}
^{60}Co	12	292.3 ± 12.8^{b}	90.7 ± 6.3^{a}	210.7 ± 6.02^{b}
^{60}Co	24	385.2 ± 32.7^{a}	99.3 ± 2.7^{a}	281 ± 17.5^{a}
^{137}Cs	6	182.7 ± 10.3^{c}	66.8 ± 11.1^{b}	128.5 ± 8.1^{f}
^{137}Cs	12	259 ± 12.3^{b}	89.3 ± 4.7^{a}	169 ± 5.9^{d}
^{137}Cs	24	347.7 ± 41.1^{a}	92.5 ± 5.7^{a}	188.3 ± 2.7^{c}

** Means ± SD (n = 6). Statistical significance was determined using Tukey's test. Means within the same row or column bearing distinct superscript letters differ significantly; letters indicate significance at ($p<0.05$).*

Table 4. Liver tissue homogenate enzyme activity (AST, ALT, ALP) following radiation exposure.

Exposure Groups	Time (h)	AST (U/L)	ALT (U/L)	ALP (U/L)
Control	-	115.8 ± 4.9^{e}	28.7 ± 4.5^{d}	104.8 ± 4.9^{e}
^{60}Co	6	123.8 ± 7.5^{d}	32.2 ± 5.3^{d}	135.2 ± 4.9^{d}
^{60}Co	12	187.3 ± 8.8^{b}	83 ± 6.1^{b}	172.3 ± 6.2^{b}
^{60}Co	24	220.2 ± 10.1^{a}	95.3 ± 3.7^{a}	193.5 ± 6.5^{a}
^{137}Cs	6	127.7 ± 4.5^{d}	28.2 ± 5.2d	115.8 ± 4.3^{e}
^{137}Cs	12	163.5 ± 7.5^{c}	62.7 ± 5.1^{c}	150.2 ± 6.9^{c}
^{137}Cs	24	192.7 ± 4.6^{b}	86.8 ± 5.2^{a}	168.2 ± 4.02^{b}

** Means ± SD (n = 6). Statistical significance was determined using Tukey's test. Means within the same row or column bearing distinct superscript letters differ significantly; letters indicate significance at ($p<0.05$).*

Discussion

The present study demonstrated a clear time-dependent reduction in total body weight following exposure to both ^{60}Co and ^{137}Cs radiation, with the magnitude of loss becoming progressively more pronounced as the observation interval extended from 6 h to 24 h, suggesting that acute systemic catabolic changes require a latent period before becoming physiologically manifest. By contrast, both the 12 h and 24 h intervals showed significant weight loss for ^{60}Co and an even more pronounced, highly significant reduction for ^{137}Cs, indicating that ^{137}Cs exposure produced a comparatively greater systemic catabolic burden at the later time points despite its lower photon energy relative to ^{60}Co. The comparatively greater weight loss under ^{137}Cs exposure at the extended interval may reflect differences in the dose rate and depth-dose distribution of the two isotopes. Specifically, the lower-energy gamma photons of ^{137}Cs (0.662MeV) undergo higher attenuation and deposit a greater fraction of their energy within the small tissue volume of the mice, whereas the higher energy photons of ^{60}Co (1.17 and 1.33 MeV) are more penetrating and traverse deeper through the body with lower surface-to-volume energy deposition.

Liver weight increased significantly only at the 24 h interval under ^{60}Co exposure, while ^{137}Cs produced a significant increase at all three intervals, reaching significance at 24 h. This pattern most plausibly reflects acute vascular congestion and inflammatory oedema rather than compensatory hepatocyte proliferation, given the short 24 h observation window. The earlier and more sustained hepatic response to ^{137}Cs relative to ^{60}Co parallels the isotope-dependent pattern seen in body weight and again points to a difference in the kinetics of radiation-induced microvascular injury between the two radiation sources [8]. In hepatic tissue, gamma exposure triggers lipid peroxidation of hepatocyte plasma and mitochondrial membranes. The loss of membrane integrity facilitates intracellular enzyme leakage into systemic circulation, leading to parallel elevations in serum transaminases (AST, ALT) and ALP. The elevated AST/ALT ratio (>1) highlights mitochondrial membrane disruption. ^{60}Co produces consistently higher enzymatic surges than ^{137}Cs, validating that higher gamma energy drives greater radiotoxicity and systemic damage. The elevation of these transaminases is a classic biomarker of

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hepatocellular necrosis and increased membrane permeability [9]. The AST/ALT ratio exceeded 1 in all irradiated groups, suggesting mitochondrial involvement and more severe hepatic damage [10]. Elevated ALP indicates cholesteric injury and bile duct damage, likely due to radiation-induced oxidative stress and inflammation of the biliary epithelium [11]. The observed effects are primarily mediated through the generation of reactive oxygen species (ROS) following water radiolysis. ROS attack cellular membranes (lipid peroxidation), mitochondrial DNA, and nuclear DNA, leading to cell cycle arrest. Hepatocyte necrosis and enzyme leakage and Impaired synthetic function. These findings are consistent with a recent study on radiation, evidenced by alterations in different blood parameters in albino mice [12]. In addition, these findings are consistent with previous studies on gamma radiation effects in rodent models [13, 14]. The observed experimental outcomes substantiate the physical mechanisms governing photon-matter interactions and radiobiology. High-energy gamma photons from ^{60}Co (1.17 and 1.33 MeV) possess higher kinetic energy transfer capabilities during Compton scattering relative to ^{137}Cs (0.662 MeV):

$T = h\nu - h\nu' = h\nu [\alpha(1 - \cos\theta) / (1 + \alpha(1 - \cos\theta))]$ where $\alpha = h\nu / m_e c^2$. The energetic secondary recoil electrons created by ^{60}Co produce a denser track of ionisations per unit volume in tissue, resulting in heightened localised energy deposition.

Conclusion

Exposure to gamma rays from ^{60}Co or ^{137}Cs induces profound, time-dependent hepatocellular toxicity in male Albino mice. ^{60}Co demonstrates significantly higher acute radiotoxicity than ^{137}Cs across equivalent exposure durations due to its higher photon energy spectrum and energetic secondary electron emissions. Liver enzyme biomarkers serve as effective biological indicators for assessing acute ionising radiation damage.

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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. Gholami YH, Maschmeyer R, Kuncic Z. Radio-enhancement effects by radiolabeled nanoparticles. Sci Rep. 2019;9(1):14346.
4. Dmitri P. Inhalation and ingestion of radioactive nuclides: mechanisms of internal contamination, radiotoxicity, and medical management. Biokinetics and organ-specific targeting of radionuclides after respiratory and gastrointestinal entry. Advanced Medical technology and Systems Inc. On. Canada. 2026: 1-33.
5. Khan FM, Gibbons JP. Khan's the physics of radiation therapy. 5th ed. Philadelphia (PA): Wolters Kluwer Health/Lippincott Williams & Wilkins; 2014.
6. Valentin J. The 2007 recommendations of the International Commission on Radiological Protection. Ann ICRP. 2007;37(2-4):2.
7. Huang XJ, Choi YK, Im HS, Yarimaga O, Yoon E, Kim HS. Aspartate Aminotransferase (AST/GOT) and Alanine Aminotransferase (ALT/GPT) Detection Techniques. Sensors (Basel). 2006, 31;6(7):756–82.
8. Kim J, Jung Y. Radiation-induced liver disease: current understanding and future perspectives. Exp Mol Med. 2017;49(7):e359.
9. Eassawy MT, Salem NA, El-Sayed WM. Biochemical and histological changes in liver of irradiated rats. Int J Radiat Biol. 2020;96(6):748-759.
10. El-Khafagy EA, Mohamed HE, Abd El-Mageed RM. Assessment of gamma radiation-induced hepatotoxicity and cellular damage in experimental animal models. Int J Radiat Biol. 2018;94(6):560-568.
11. Abdelhalim MA., Moussa SA. Biochemical and hematological alterations in laboratory animals exposed to gamma radiation. Journal of Radiation Research and Applied Sciences, 2017:10(2), 115–122.
12. Al-Shalwi H, Ali MS, Al-Zail N. Alterations of hematopoiesis markers induced by exposure to Cobalt-60 or Cesium-137 in albino mice. AlQalam J Med Appl Sci. 2026:3545-3551.
13. Holovská K, Almášiová V, Cigánková V. Structural and ultrastructural study of rat liver influenced by electromagnetic radiation. J Appl Anim Res. 2015;43(2):170-176.
14. Mahmoud AM, Al-Qahtani WS, Hassan HM. Evaluation of oxidative stress and hepatotoxicity induced by gamma radiation in experimental animal models. J Radiat Res Appl Sci. 2017;10(4):312-319.

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