

Original Article

Detection of Mitochondrial DNA Deletion in Medical Workers Occupationally Exposed to Ionizing Radiation in Tripoli, Libya

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Abstract

Occupational exposure to low-dose ionizing radiation poses chronic health risks to radiology personnel. While traditional monitoring captures recent damage, modern molecular epidemiology focuses on mitochondrial DNA (mtDNA) alterations. Due to limited repair mechanisms, the mitochondrial genome is highly sensitive to radiation, making mtDNA alterations valuable biomarkers for cumulative genetic injury. This study aimed to assess the potential induction of the mtDNA4977 deletion and variations in mtDNA-CN in the blood of staff members working in radiology departments across three medical facilities in Tripoli, Libya. A total of 72 participants were recruited from Metiga Military Hospital, Alkhadra General Hospital, and the National Center for Disease Control–Libya between July and October 2024. The study population included 50 radiation-exposed workers (Group A) and 22 non-exposed individuals (Group B). Data were obtained using a structured questionnaire and peripheral blood sampling. Quantitative real-time PCR was applied to evaluate mtDNA-CN and detect the mtDNA4977 deletion. ΔCt values for mtDNA4977 were significantly lower in the radiation-exposed group than in controls, indicating higher levels of mitochondrial DNA deletions among exposed workers. mtDNA-CN was also significantly reduced in the exposed group, suggesting possible radiation-induced mitochondrial depletion. Multivariable linear regression models further confirmed that occupational radiation exposure remained an independent and highly significant predictor of both mitochondrial alterations, even after adjusting for age and smoking status. These findings demonstrate a clear association between occupational exposure to ionizing radiation and alterations in mtDNA. Increased mtDNA4977 deletions and reduced mtDNA-CN among exposed workers highlight their potential as biomarkers of chronic radiation exposure. Consequently, these results underscore the critical importance of continuous biological monitoring and reinforcement of protective measures for radiology staff.

Keywords. mtDNA4977 Common Deletion, Mitochondrial DNA Copy Number, Ionizing Radiation, Tripoli.

Introduction

Humans are continuously exposed to low levels of natural ionizing radiation originating from cosmic sources, the atmosphere, and the Earth's rocks and soil [1]. In addition, individuals encounter ionizing radiation from artificial sources such as X-ray devices and radioactive materials used in medical, industrial, and scientific applications [2]. In the medical field, ionizing radiation, including X-rays, gamma rays, and beta particles, is widely used for diagnostic and therapeutic purposes. X-rays remain the most commonly applied due to their ability to produce rapid, non-invasive, and detailed images of internal structures [1].

Globally, medical use of ionizing radiation is substantial, with an estimated one million radiation therapy procedures and 3.6 billion diagnostic and interventional radiological procedures performed in 2008 [3]. Consequently, healthcare workers represent the largest occupational group routinely exposed to low-dose ionizing radiation [4]. Personnel working in radiology and fluoroscopy units are particularly at risk due to chronic exposure to low levels of X-rays. Long-term exposure has been associated with cataract formation, skin injuries, reproductive effects such as reduced fertility, and genetic alterations, including mutations and deletions in nuclear DNA [5]. Numerous studies have investigated genotoxicity biomarkers in medical workers exposed to ionizing radiation. According to a systematic review and meta-analysis study, chromosome aberrations, mainly dicentrics, and micronuclei, are the most reliable biomarkers for identifying genotoxicity in medical workers exposed to low-dose ionizing radiation. These biomarkers consistently show elevated levels in exposed workers compared to those unexposed. However, these are well-established indicators of recent ionizing radiation exposure but decrease over time. Other markers, including stable translocations, sister chromatid exchanges, and comet assay parameters, show potential but require further validation [4].

More recent research has shifted attention toward mitochondrial DNA (mtDNA), which is highly susceptible to radiation-induced mutations, insertions, and deletions [6]. The accumulation of mtDNA alterations suggests its value as a biomarker of radiation exposure [7,8]. One of the most prominent alterations is the 4977-bp deletion, known as the “common deletion” (mtDNA4977), which removes nearly one-third of the mitochondrial genome (positions 8470–13447) [9]. Due to its characteristic formation mechanism, this deletion is highly sensitive and easily detectable [10]. A study in Italy found significantly increased levels of the mtDNA4977 deletion among catheterization laboratory staff chronically exposed to low-dose radiation and higher mtDNA copy number (mtDNA-CN) among workers with lower exposure, supporting their potential as biomarkers of radiation-induced mitochondrial damage [7].

In Libya, occupational radiation safety remains a critical concern due to inadequate monitoring systems and insufficient availability of protective equipment. Previous studies have reported considerable deficiencies in radiation protection practices, including limited use of dosimeters, weak enforcement of safety regulations, and limited access to personal protective equipment [11–14]. In light of these ongoing challenges, it is essential to assess the biological consequences of occupational exposure among healthcare professionals. This study therefore, examines the potential induction of mtDNA4977 and variations in mtDNA-CN among radiology staff in selected healthcare facilities in Tripoli, Libya. This approach enables the generation of molecular-level insights into genotoxic effects that may arise from insufficient adherence to radiation safety practices.

Materials and Methods

Study design

This study adopted an observational cross-sectional design to assess the potential induction of the common mitochondrial deletion (mtDNA4977) and variations in mitochondrial DNA copy number (mtDNA-CN) among medical staff working in diagnostic and interventional radiology departments. The study was conducted across two hospitals and one medical center in Tripoli, Libya. Blood samples were collected from all participants to analyze mitochondrial DNA alterations.

Study population and settings

The study was carried out in three major healthcare facilities in Tripoli, Libya—Metiga Military Hospital, Alkhadra General Hospital, and the National Center for Disease Control, chosen for their high radiology workloads. A purposive sampling method was used to recruit 72 participants. Group A consisted of 50 workers occupationally exposed to ionizing radiation in diagnostic and interventional radiology departments. Group B included 22 age-matched healthcare workers from non-radiology departments within the same institutions who had no occupational radiation exposure. Inclusion and exclusion criteria ensured that participants had comparable work environments but differed specifically in radiation exposure status.

Inclusion and Exclusion Criteria

Eligible participants included radiology staff with more than one year of employment in diagnostic or interventional radiology departments, as well as non-radiology staff from the same hospitals who had at least one year of service and no previous occupational exposure to ionizing radiation. All participants were required to be between 18 and 65 years old. Individuals with a history of cancer or cardiovascular diseases were excluded to avoid confounding health-related factors.

Ethical approval

This study was approved by the National Center for Disease Control's ethics committee (NBC: 002.H-24.5). Written informed consent was obtained from each study participant before data collection.

Data collection

Data were collected from July to October 2024 using two primary methods. The first method involved administering a structured, multiple-choice questionnaire to assess participants' clinical and demographic characteristics, as well as their radiation protection practices.

The second method involved blood sample collection for the detection and analysis of mtDNA-CN and the mtDNA4977 using quantitative polymerase chain reaction (qPCR), as previously described by [7].

Sample Size and Statistical Power

A post-hoc power analysis was conducted using G*Power 3.1 to evaluate the statistical adequacy of the study's sample size ($N = 72$, comprising $n = 50$ radiation-exposed and $n = 22$ control subjects) adapted for the non-parametric Wilcoxon-Mann-Whitney framework. Based on an alpha level of 0.05 and incorporating the substantial distribution shifts corresponding to the observed large rank-biserial correlation effect sizes (0.72 for mtDNA4977 and -0.56 for mtDNA-CN), the study achieved a calculated statistical power exceeding 0.99 for both mitochondrial markers. These values are well above the conventional threshold of 0.80, confirming that the sample size provided exceptional statistical resolution to detect genuine variances between the study groups [15].

Questionnaire design

The questionnaire consisted of two sections; the first section provided the demographic information, including age, gender, educational background, work experience (years), total working hours per week, type of hospital they work in (private/public or both), and their smoking habits. The second section consisted of questions on awareness and knowledge about ionizing radiation: the habitual use of personal protective equipment, such as a lead apron, eye goggles, and a thyroid shield, when working in a radiation-exposed environment; the maximum permissible dose of radiation per year for workers; the use of a dosimeter; and the damage that may be induced due to long-term exposure.

Collection of samples

Blood samples were collected from all participants using (EDTA) anticoagulant tubes and were stored at -20°C until analysis.

Laboratory Analysis

Genomic DNA Extraction from Blood

Genomic DNA was extracted from whole blood using the QIAamp DNA Mini Kit (Qiagen, USA) according to the manufacturer's instructions. The concentration and purity of the isolated DNA were assessed using a NanoDrop Lite spectrophotometer (Thermo Scientific, USA). Absorbance purity ratio (A_{260}/A_{280}) of ≥ 1.7 is considered to be appropriate for qPCR. All DNA samples were stored at -80°C until further analysis.

Analysis of mtDNA 4977 bp deletion and mtDNA-CN by quantitative PCR

Quantification of the mtDNA4977 deletion and mtDNA-CN was performed using qPCR following the method described by [16]. Amplification reactions were carried out on a CFX96 Touch Real-Time PCR Detection System (Bio-Rad, USA) using SYBR® Premix Ex Taq™ (TaKaRa, Japan). Each qPCR reaction (20 μL) contained: 10 μL of 2 $\times$ SYBR Green premix, 0.4 μL each of forward and reverse primers (10 pmol/ μL ; Table 1), 2 μL of DNA template (50 ng), and 7.2 μL of nuclease-free water.

Thermal cycling conditions consisted of an initial denaturation cycle at 95°C for 3 min, followed by 40 cycles of denaturation at 95°C for 10 s, annealing at 60°C for 20 s, and extension at 72°C for 30 s.

Primers and Target Regions

Three regions were amplified, including: the mitochondrial ND1 (mtND1) gene (a conserved, non-deleted mitochondrial region serving as an internal mtDNA control), the mtDNA4977 deletion fragment (spanning the remaining junction region post-deletion), and the human β -globin gene (nuclear DNA marker utilized for normalization). These targets enabled quantification of both mtDNA deletion levels and mtDNA copy number.

Table 1. Regions and primers amplified in qPCR

Fragments	Nucleotide Position	Product (bp)	Primer sequence
Conserved region (ND1 gene-mtDNA)	3307-3458	152	F- 5'- ATACCCATGGCCAACCTCCTA-3' R- 5'- TCAGCGAAGGGTTGTAGTAGC -3'
mtDNA4977 deleted region	8388-13646	281	F- 5'- CCATAATTACCCCCATACTCC-3' R- 5'- AAGCGAGGTTGACCTGTTAGG-3'
human β -globin gene	61991-62259	268	F- 5'-GAAGAGCCAAGGACAGGTAC-3' R- 5'- CAACTTCATCCACGTTCCACC-3'

Calculation of mtDNA4977 Deletion Levels and mtDNA Copy Number

Relative quantification was performed using the ΔCt method. The level of mtDNA4977 deletion was calculated as: $\Delta\text{Ct} = \text{Ct (mtDNA4977)} - \text{Ct (mtND1)}$. Because Ct values are inversely proportional to the amount of starting template, lower ΔCt mtDNA4977 deletion values indicate a higher relative abundance of deleted mtDNA molecules. Relative mitochondrial DNA copy number (mtDNA-CN) was determined by normalizing the mitochondrial genome against nuclear DNA, calculated as $\Delta\text{Ct} = \text{Ct (mtND1)} - \text{Ct } (\beta\text{-globin})$. For this copy number index, higher ΔCt copy number values indicate a lower relative mitochondrial DNA copy number. Calculations were based on methods previously described [7,16,17].

Statistical analysis

Data curation was performed using Microsoft Excel, and statistical analyses were conducted using Jamovi software (version 2.3). Data were expressed as numbers (n) and percentages (%) for categorical variables, and as Medians accompanied by Interquartile Ranges (IQR) for continuous variables. The Shapiro-Wilk test was used to assess the normality of data distribution. Group comparisons were conducted using the Mann-Whitney U test. The significance level was set at a pvalue ≤ 0.05 .

Results

Demographic and clinical characteristics

The demographic and clinical characteristics of the study population reveal important similarities between medical workers occupationally exposed to ionizing radiation (N=50) and unexposed individuals (N=22). The two groups were well-matched in terms of age, with a median age of 35.0 years (IQR: 14.5) for the exposed group compared to 33.5 years (IQR: 11.8) for the unexposed controls ($p = 0.966$). Sex distribution was also virtually equivalent between the study groups, with males comprising 76.0% of the exposed group and 68.2% of the unexposed group ($p = 0.687$). Occupational profiles were highly comparable apart from radiation exposure status; the groups exhibited identical median weekly working hours (30.0

hours [IQR: 11.5] vs. 30.0 hours [IQR: 15.2], $p = 0.720$) and displayed a statistically comparable distribution across categories of professional work experience ($p = 0.932$), with approximately half of each sample possessing 1–5 years of experience. No statistically significant difference was observed in smoking prevalence between the unexposed and radiation-exposed groups (45.5 % vs. 24.0 %, respectively; $p = 0.096$). Among active smokers within both groups, the median smoking duration was 3.0 years (IQR: 8.8) for the exposed individuals and 8.5 years (IQR: 5.3) for the unexposed controls ($p = 0.485$), with cigarette consumption patterns remaining predominantly ≤ 1 pack/day across the entire study population (Table 2).

Table 2. Clinical and demographic characteristics of the study population

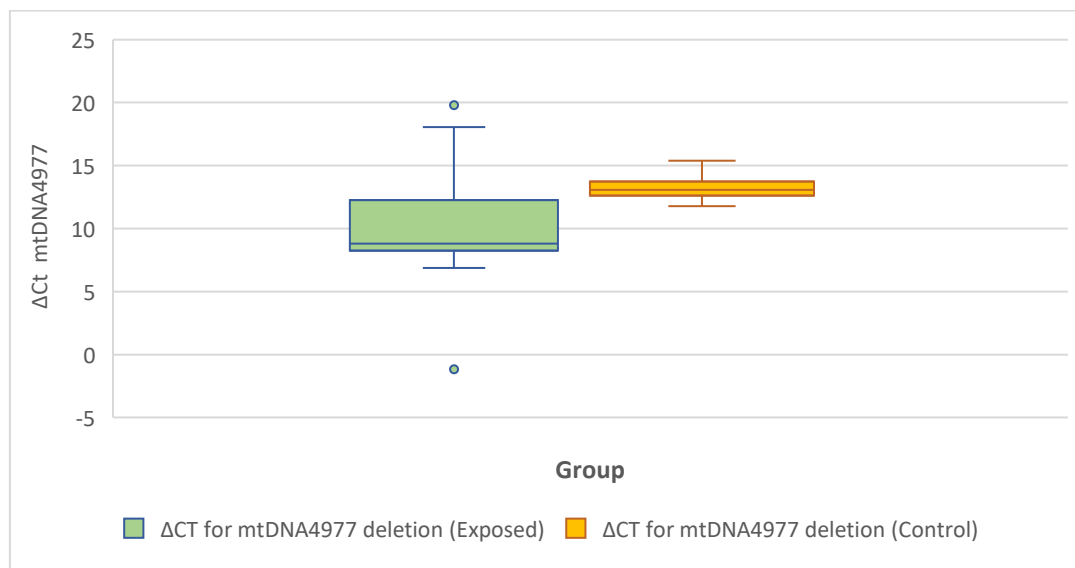
Characteristics	Exposed Group (N=50)	Unexposed Group (N=22)	p-value
Age (years), Median (IQR)	35.0 (14.5)	33.5 (11.8)	0.966
Sex, n (%)			
Male	38 (76.0%)	15 (68.2%)	0.687
Female	12 (24.0%)	7 (31.8%)	
Work Experience (years), n (%)			
1-5 years	24 (48.0%)	11 (50.0%)	0.932
6-10 years	10 (20.0%)	5 (22.7%)	
11-15 years	8 (16.0%)	2 (9.1%)	
More than 16 years	8 (16.0%)	4 (18.2%)	
Weekly Working Hours, Median (IQR)	30.0 (11.5)	30.0 (15.2)	0.720
Smoking Status, n (%)			
Smoker	12 (24.0%)	10 (45.5%)	0.096
Non-Smoker	38 (76.0%)	12 (54.5%)	
Smoking Duration (years), Median (IQR)	3.0 (8.8)	8.5 (5.3)	0.485

IQR = Interquartile Range.

Comparative analysis of mitochondrial DNA copy number (Δ Ct mtDNA-CN) and mtDNA4977 deletion (Δ Ct mtDNA4977) between the radiation-exposed group and the unexposed group.

Δ Ct mtDNA4977 level

The analysis of Δ Ct mtDNA4977 levels revealed a highly significant difference between radiation-exposed and control groups ($p = 1.51 \times 10^{-6}$), evaluated via the non-parametric Mann-Whitney U test (Figure 1). The radiation-exposed group demonstrated a median Δ Ct value of 8.82 (Range: -1.17 to 19.79), whereas the unexposed control group showed a higher median value of 13.07 (Range: 11.77 to 15.39). This pronounced downward shift in Δ Ct values reflects an earlier PCR cycle amplification, directly demonstrating a significantly higher relative abundance of the mt DNA4977 deletion among exposed personnel. The large effect size (rank-biserial correlation = 0.72) further underscores the substantial magnitude of this biological difference between the groups.

**Figure 1. Distribution of Δ Ct mtDNA4977 deletion levels between groups.**

Data were presented as box-and-whisker plots showing the median (horizontal line), interquartile range (IQR), whiskers extending to $1.5 \times$ IQR, and outliers. The radiation-exposed group had significantly lower Δ Ct values than the control group, consistent with a higher relative abundance of the mtDNA4977 common deletion (Mann-Whitney U test, $p = 1.51 \times 10^{-6}$).

Δ Ct mtDNA-CN level

The analysis revealed a statistically significant difference in Δ Ct mtDNA-CN levels between radiation-exposed and control groups, as demonstrated by Mann-Whitney U test results ($p = 1.70 \times 10^{-4}$; Figure 2). The radiation-exposed group showed a higher (less negative) median Δ Ct mtDNA-CN value of -6.73 (Range: -12.07 to -2.71), while the unexposed control group exhibited a lower (more negative) median value of -8.48 (Range: -19.49 to -7.12). Because Ct values are inversely proportional to initial template abundance, this significant upward shift closer to zero in the exposed group indicates a

substantial reduction of relative mitochondrial DNA copy number compared to the controls. This observation is supported by a large effect size (rank-biserial correlation = -0.56), confirming a robust magnitude of difference between the groups.

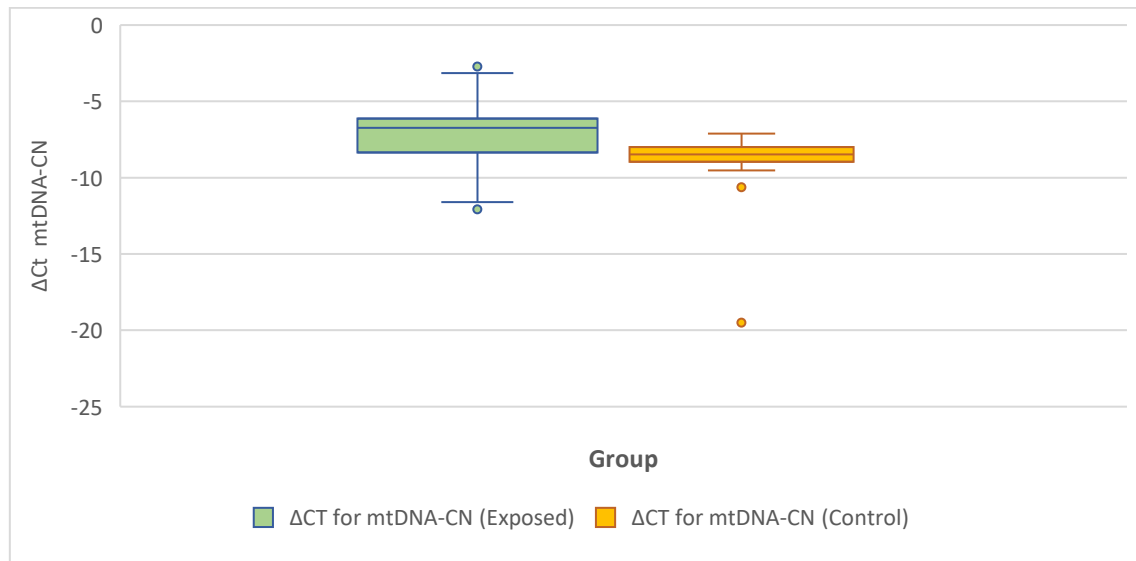


Figure 2. Distribution of relative ΔCt mtDNA-CN levels between groups.

Data were presented as box-and-whisker plots showing the median (horizontal line), interquartile range (IQR), whiskers extending to $1.5 \times \text{IQR}$, and outliers. The radiation-exposed group exhibited significantly higher (less negative) ΔCt values than the control group, consistent with a reduced total mitochondrial DNA copy number (Mann–Whitney U test, $p = 1.70 \times 10^{-4}$).

Multivariable regression analysis

In multivariable linear regression models adjusted for age and smoking status, occupational radiation exposure remained a highly significant independent predictor of both ΔCt mtDNA4977 deletion cycles ($\beta = -3.41$, $\text{SE} = 0.72$, $t = -4.73$, $p < 0.001$) and relative ΔCt mtDNA-CN values ($\beta = 1.59$, $\text{SE} = 0.57$, $t = 2.80$, $p = 0.007$). Notably, confounding factors including age ($p = 0.813$ and $p = 0.909$, respectively) and smoking status ($p = 0.958$ and $p = 0.185$, respectively) showed no statistically significant associations with either biomarker (Table 3). This confirms that the observed deletion accumulation and copy number depletion are independently associated with occupational exposure profiles rather than demographic or lifestyle variations.

Table 3. Multivariable Linear Regression Models for Mitochondrial Biomarkers

Dependent Variable / Predictor	Unstandardized Coefficient (β)	Standard Error (SE)	t-value	p-value
Model 1: Δ Ct mtDNA4977 Deletion				
Intercept (constant)	12.92	1.64	7.88	< 0.001
Radiation exposure group	-3.41	0.72	-4.73	< 0.001
Age (Years)	0.01	0.04	0.24	0.813
Smoking Status (Yes vs. No)	-0.04	0.74	-0.05	0.958
Model 2: Δ Ct mtDNA Copy Number				
Intercept (constant)	-8.58	1.29	-6.65	< 0.001
Radiation exposure group	1.59	0.57	2.80	< 0.007
Age (Years)	-0.004	0.03	-0.12	0.909
Smoking Status (Yes vs. No)	-0.78	0.59	-1.34	0.185

Note: For the Radiation Exposure variable, "Unexposed" is the reference baseline. For Smoking Status, "No" is the reference baseline.

Assessment of participant awareness, knowledge, and safety practices in ionizing radiation protection

Evaluation of participant awareness and knowledge of ionizing radiation

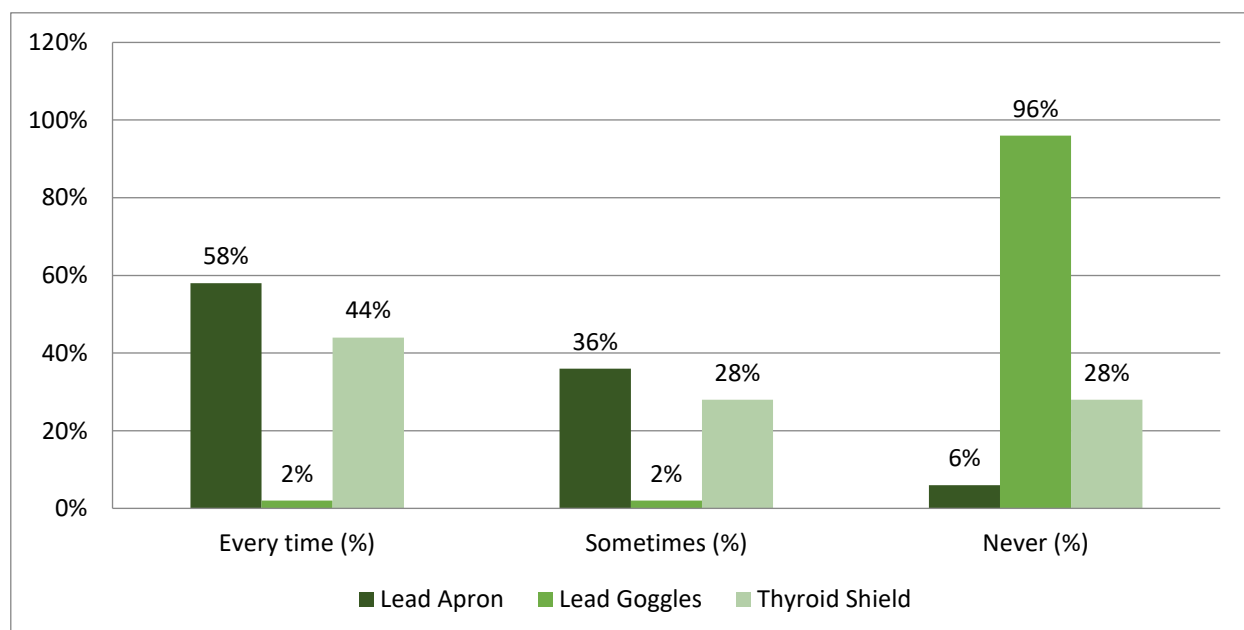
The results highlight that despite a significant gap in formal training, where the majority of participants had not attended a course or workshop, they still demonstrated a high level of awareness and knowledge regarding radiation hazards and proper shielding protocols. As shown in (Table 4).

Table 4. Participant awareness and knowledge of ionizing radiation

Question	Response	N	%
Attended course/workshop?	Yes	19	38%
	No	31	62%
Harmful effects of radiation?	Yes	47	94%
	No	0	0%
	I don't know	3	6%
Best shielding for X-rays?	Lead	49	98%
	Aluminum	0	0%
	I don't know	1	2%

Personal protective equipment

The data on personal protective equipment (PPE) usage highlights critical deficiencies in radiation safety practices among workers, as shown in (Figure 3).

**Figure 3. Personal protective equipment shield material for X-rays**

Knowledge of radiation protection principles and dose limits

The data reveals significant knowledge gaps in radiation safety fundamentals. Only 38% of participants correctly identified ALARA (As Low as Reasonably Achievable) as the ICRP radiation protection principle, while just 14% knew the correct maximum permissible annual dose limit (50 mSv) for radiation workers (Figure 4).

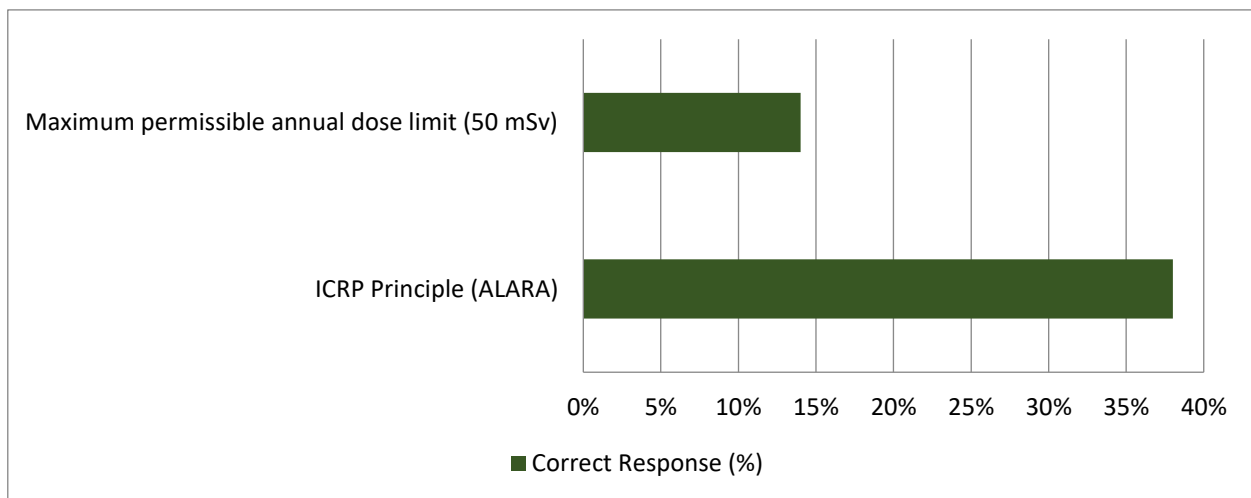


Figure 4. Participant knowledge of ICRP radiation protection principle and dose limit.

Assessment of personal dosimeter use and monitoring practices

The data on personal dosimeter usage highlights serious deficiencies in radiation exposure monitoring practices among the surveyed workers, as shown in Table 5. A significant majority of the personnel do not use any form of exposure monitoring, leaving only a small minority who actively utilize dosimeters. Among the sub-population of workers who do use monitoring devices, film badges represent the primary type employed, with electronic personal dosimeters (EPDs) being rarely utilized. Furthermore, monitoring management appears compromised, as only a fraction of these individuals reported that their personal dosimeters had been checked within the past year.

Table 5. Participant responses on dosimeter usage, type, and monitoring frequency

Question	Response	N	%
Use dosimeter?	Yes	12	24%
	No	38	76%
Dosimeter Type	Film badge	11	22%
	EPD	1	2%
Last checked	Within past year	9	18%
	Never checked	2	4%
	Other (month/3 months)	1	2%

Health Effects

The data reveals ongoing gaps in participant awareness regarding the health impacts of ionizing radiation (Figures 5,6).

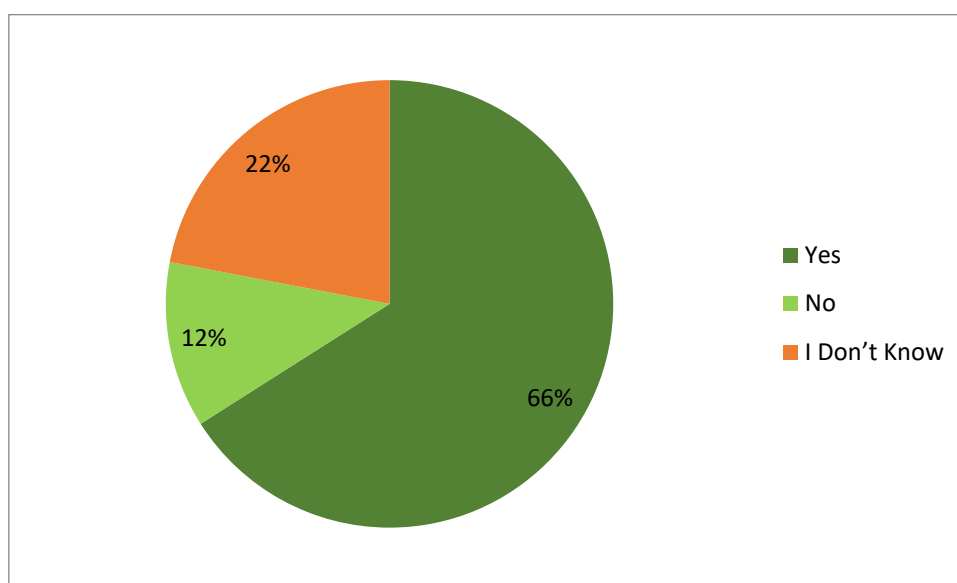


Figure 5. Participant awareness of genetic damage

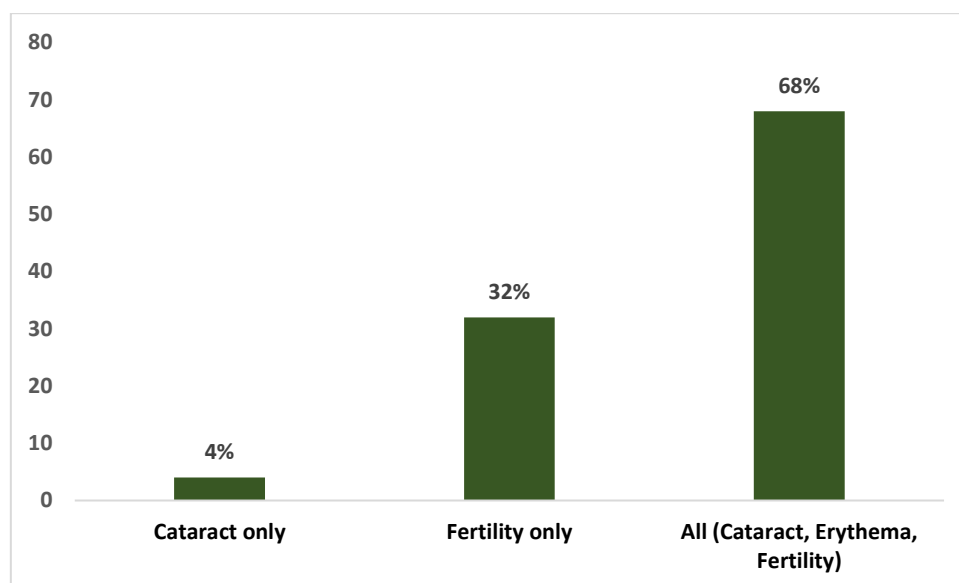


Figure 6. Awareness of biological outcomes of radiation overexposure

Discussion

X-Ray exposure and mitochondrial genomic alterations

This study presents the first molecular evidence indicating notable mitochondrial DNA damage among Libyan radiology professionals who are chronically exposed to low-dose X-ray radiation. The findings demonstrate a clear and statistically significant link between occupational exposure and changes in both mitochondrial DNA integrity and copy number.

Our results are consistent with previous experimental and human studies demonstrating that ionizing radiation can compromise mitochondrial genomic integrity. The significant decrease in ΔCt mtDNA 4977 observed among exposed participants indicates higher levels of mtDNA 4977 deletions, suggesting radiation-induced mitochondrial damage. This finding aligns with the results reported by Borghini et al. [7], who reported significantly elevated mtDNA 4977 deletions among catheterization laboratory workers with chronic occupational exposure. In the same study, workers with accurately reconstructed lifetime occupational doses showed higher mtDNA 4977 deletions in those with a median lifetime dose of 16.8 mSv. Interestingly, Borghini et al. [7] also observed higher mtDNA-CN in low-exposure groups, possibly reflecting an early adaptive response, whereas the present study demonstrated a significant reduction in mtDNA-CN that may represent a more advanced stage of mitochondrial dysfunction resulting from chronic occupational exposure.

In line with these observations, experimental studies in animal models and cell cultures have demonstrated dose- and time-dependent increases in mtDNA deletions following exposure to X-rays [10,18]. Wang et al. [19] reported transient increases in mtDNA deletions in HepG2 cells exposed to 5–10 Gy X-rays, peaking at 24–48 hours and returning to baseline by day 10, along with a short-term elevation in mtDNA copy number. In contrast to these short-lived in vitro effects, the current human data indicate sustained mitochondrial injury, likely resulting from chronic or cumulative radiation exposure. Consistent with this pattern, Antipova et al. [18] observed significant increases in mtDNA deletions in mouse brain and spleen tissues following 5 Gy X-ray exposure, with damage persisting for at least four months post-irradiation. Their tissue-specific findings support the idea that radiation-induced mitochondrial damage can accumulate over time in vivo.

Taken together, these findings confirm that ionizing radiation induces mtDNA deletions across different models, with the most persistent effects observed in humans. This supports the potential use of mtDNA alterations as biomarkers of long-term radiation exposure.

A significant reduction in mtDNA-CN was observed among radiation exposed healthcare workers compared with unexposed controls, suggesting mitochondrial genome instability or impaired biogenesis associated with chronic low-dose exposure. Experimental studies, however, have often shown transient increases in mtDNA-CN following acute irradiation. Seino et al. [20] reported elevated mtDNA-CN in both cancerous (HeLa) and non-cancerous (BJ1-hTERT) cells 24 hours after X-ray exposure, while Zhou et al. [21] observed similar time- and dose-dependent increases within 72 hours in MCF-7 cells, particularly at low doses (0.05–0.5 Gy). In vivo experiments in mice showed increased peripheral blood mtDNA-CN levels one day after exposure to 2 Gy X-rays, returning to baseline by day seven [20]. These findings are interpreted as compensatory mitochondrial responses to acute oxidative stress.

In contrast, the persistent mtDNA-CN reduction seen in the current study likely reflects the cumulative effects of chronic occupational exposure. Notably, Seino et al. [20] also reported decreased mtDNA-CN in the offspring of irradiated mice, reinforcing the potential for long-term mitochondrial impairment following prolonged or repeated radiation exposure.

Therefore, the alterations detected in mtDNA4977 and mtDNA-CN, along with new markers such as cell-free mtDNA, may serve as valuable tools for identifying the biological impact of occupational radiation exposure [7,8]. These findings highlight the potential of molecular biomarkers as sensitive and practical indicators. Integrating such markers into regular

health surveillance could enhance the early detection of radiation-related effects and reinforce protective measures for radiology personnel.

While these molecular findings highlight the biological consequences of radiation exposure and suggest a need for increased attention to occupational risks, assessing personnel awareness of radiation risks and protective measures is equally important for minimizing long-term health effects related to occupational exposure.

Radiation protection knowledge and awareness among medical workers using X-Ray radiation

In addition to the analysis of mitochondrial alterations caused by exposure to ionizing radiation, this study also assessed radiation safety awareness and adherence to protective measures among Libyan radiology personnel, as such practices constitute the primary defense against occupational exposure. The results revealed significant deficiencies, including inconsistent PPE usage, poor dose monitoring, and a limited understanding of radiation-related health risks, similar deficiencies have been documented in earlier studies.

A critical concern identified in this study was the low utilization of personal dosimeters among radiology personnel, with only 24% reporting active use and a striking 76% operating without any form of exposure monitoring. This absence of regular exposure monitoring violates the principle of dose limitation and leaves workers unaware of their cumulative radiation exposure, contrary to international radiation protection guidelines [22].

These findings align with previous studies in Libya. Yousef et al. [14] found that 87% of participants lacked dosimeter availability, and only 8% of radiology technicians were informed about their radiation exposure levels. Similarly, Abdulla et al. [11] reported that just 9% of medical centers in Al-khoms City had dosimeters available, while Musa [12] observed that only 40% of hospitals on the western coast implemented radiation monitoring, and even then, coverage was inconsistent and poorly managed.

Internationally, studies corroborate a discrepancy between awareness and practice. For instance, Salah Eldeen and Farouk [23] found that while 84.1% of healthcare workers were aware of personal dosimeters, only 34.4% consistently used them, and just 28.2% reported that their departments enforced dosimeter use. In Cyprus, Zervides et al. [24] observed high dosimeter usage among radiographers, yet around 30% were unaware of correct device placement. In Turkey, Zekioglu and Parlar [25] reported 89.6% dosimeter use among healthcare workers, but 10.4% did not use one because of forgetfulness, inaccessibility, or distrust in the device, highlighting that behavioral and systemic issues remain even in countries with stricter policies.

In Libya, the issue is more severe, driven by limited availability and weak institutional enforcement. This underuse of dosimeters reflects a major gap in occupational radiation safety. The ICRP emphasizes the importance of calibrated and regularly reviewed dosimeters to support the core principles of radiation protection, justification, optimization, and dose limitation [22]. The lack of such practices in Libyan facilities not only violates international safety standards but also places healthcare workers at risk of unmonitored radiation exposure. Addressing these gaps requires national policies, routine dose monitoring, and alignment with international protocols to ensure both safety and compliance.

The use of PPE was inconsistent, revealing major gaps in radiation safety practices. Only 58% of participants consistently wore lead aprons, while the use of lead goggles and thyroid shields remained notably low. Similar patterns were observed in other Libyan studies, where limited availability and discomfort were cited as key barriers to PPE use [13,14]. Internationally, Salah Eldeen and Farouk [23] reported high awareness but poor compliance, with only 13.9% of workers using lead aprons despite 82.1% awareness. In contrast, higher adherence was noted in Portugal, where over half of the staff regularly used lead aprons and thyroid shields [26]. These findings underscore the urgent need for improved PPE access, regular training, and institutional enforcement to enhance radiation protection practices.

The results of this study, consistent with previous studies [12–14], reveal a major lack of formal radiation safety training among radiology professionals in Libya, with only 38% having attended relevant courses, and most medical centers lacking structured programs. This deficiency, together with low dosimeter usage, inconsistent PPE application, and weak institutional commitment to safety standards, increases the risk of occupational radiation exposure.

The observed deficiencies in radiation protection practices may help explain the molecular alterations identified in this study, providing biological confirmation of the long-term impact of inadequate safety measures.

This study presents the first Libyan investigation linking occupational radiation exposure with molecular biomarkers of chronic genotoxicity, specifically mtDNA4977 deletion and mtDNA-CN variation. It provides concrete molecular evidence of biological damage among healthcare workers, transforming theoretical concerns into measurable reality and emphasizing the urgent need to strengthen national radiation safety practices through training, monitoring, and proper PPE use. Globally, the study highlights the value of mitochondrial biomarkers as sensitive indicators of cumulative low-dose radiation effects, supporting their integration into occupational health surveillance programs.

A notable limitation of the study is that there is a lack of reliable dosimetry data, as many participants did not use personal dosimeters consistently, and those who did rarely had their devices checked. Consequently, we were unable to establish a direct correlation between accumulated radiation dose and mitochondrial DNA alterations (mtDNA4977 deletions and mtDNA copy number). This limitation highlights the need for improved radiation dose monitoring in future research to better understand dose-response relationships.

Conclusion

The present study indicates that long-term occupational exposure to low-dose ionizing radiation is associated with significant mitochondrial DNA alterations in radiology personnel. The observed increases in mtDNA4977 deletions and reductions in mtDNA-CN among exposed individuals suggest impaired mitochondrial function, which may serve as early biomarkers of radiation-induced cellular damage. These molecular changes, alongside poor radiation safety practices, highlight a serious occupational health concern. Overall, the findings underscore the urgent need for systemic improvements in radiation protection measures, continuous biological monitoring, and stricter reinforcement of safety protocols within healthcare facilities in Libya.

Author contributions (Credit)

Nisreen Alaribi Aqeelah: Conceptualization, Methodology, Investigation, Writing – original draft.

Mustafa Mohamed Drah: Methodology, Writing – review & editing.

Abdulla Masood Bashein: Supervision, Writing – review & editing.

Taher Ahmed Shaibi: Validation.

Hanan Alaribi Aqeelah: Visualization, Formal analysis.

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Conflict of interest

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Authors' contributions

The authors have equal participation in conceptualization, preparation of the original draft, review, and editing.

Data availability

All analyzed data are included in this study.

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