

Original article

Assessment of chronic genotoxic effects in radiology technicians: A comparative study using the comet assay and γ -H2AX biomarker

Maged Mostafa^{a,*} , Ebada Abd AlRaouf^b , Mona El Wassefy^a , Samir Elmasry^b 

^aDepartment of Clinical Pathology, Faculty of Medicine, Mansoura University, Egypt

^bDepartment of Molecular Biology, Faculty of Biotechnology, Sadat City University, Egypt

Abstract

Background: Healthcare workers, particularly radiology technicians, are chronically exposed to low-dose ionizing radiation (IR). While physical dosimetry monitors external exposure, biological monitoring of early genotoxic effects is crucial for risk assessment.

Objectives: To evaluate DNA damage in radiology technicians occupationally exposed to low-dose IR compared to nonexposed controls.

Methods: This case-control study included 50 radiology technicians and 50 matched controls. DNA damage in peripheral blood lymphocytes was assessed using the alkaline comet assay. A subset of participants (10 exposed and 10 controls) underwent γ -H2AX flow cytometric analysis for detection of DNA double-strand breaks.

Results: The exposed group showed significant increases in DNA damage parameters, particularly tail DNA%, olive tail moment and head intensity ($P < 0.001$). Receiver operating characteristic (ROC) analysis demonstrated excellent diagnostic performance for tail moment and head intensity (area under the curve (AUC) = 1.000). γ -H2AX analysis showed significantly higher mean fluorescence intensity and percentage of positive cells among exposed workers ($P < 0.001$). DNA damage correlated positively with occupational exposure duration.

Conclusion: Our findings suggest a link between long-term exposure to low-dose IR and DNA damage even when standard safety precautions are followed.

Keywords: Comet assay, DNA damage, ionizing radiation, occupational exposure, γ -H2AX

Ionizing radiation (IR) is an established human carcinogen and mutagen. A large number of workers in the healthcare profession, and radiologic technicians in particular, are constantly exposed to low-dose IR while performing diagnostic imaging procedures. ⁽¹⁾ Despite strict protocols and personal dosimeters for maintaining exposure limits or below the ALARA principle (As Low As Reasonably Achievable), the overall biologic effect of low-dose exposure is an area of concern for occupational health. ⁽²⁾

The major IR target in cells is DNA, with damage extending from base modifications and single to double strand breaks (DSBs), which are far more serious and

damaging to the genetic material. ⁽³⁾ Persistent biological damage can induce genomic instability, which can lead to carcinogenesis and cellular aging. ^(4,5) Biological dosimetry and/or biomonitoring involve the use of sensitive biochemical assays for measurement of molecular and cellular changes in IR-exposed individuals. However, these conventional biological screening methods with dosimetry, which use films or clinical tests, can be insensitive to subclinical effects. ⁽⁶⁾ In contrast, the alkaline comet assay (single-cell gel electrophoresis) is a versatile, quick, and highly sensitive method for measuring DNA-strand breaks and alkali-labile sites at the single-cell level, ⁽⁷⁾ widely used in occupational studies to assess genotoxic load. ⁽⁸⁾ Phosphorylation of histone H2AX to form γ -H2AX is one of the earliest and most specific cellular responses to DSB and an extremely sensitive molecular biomarker for IR-induced DNA damage. ⁽⁹⁾ While some studies have

***Correspondence to:** Maged Mostafa, Department of Clinical Pathology, Faculty of Medicine, Mansoura University, Egypt.
E-mail: magedmostafa1981@gmail.com

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used these tools, a comprehensive examination of a range of genotoxicity endpoints across carefully matched groups of radiology professionals would provide significant advantages. In this study, a comparative evaluation of DNA damage in radiology technicians with long-term occupational exposure was conducted using two complementary methods: (1) the alkaline comet assay focusing on a wide range of DNA damage endpoints and (2) a flow cytometric analysis of the γ -H2AX biomarker as a genotype-specific indicator of DSB. We hypothesized that radiology technicians would have significantly increased DNA damage levels, proportional to the duration of their occupational exposure.

Materials and methods

Study design and subjects

The proposed case-control study was approved by the Institutional Review Board, Mansoura University, Egypt. All 100 participants provided written informed consent. We split them into two groups:

- **Exposed group (n = 50):** Radiologic technicians working in different hospitals affiliated with Mansoura University, with a minimal direct personal exposure of 8 h/day, using personal dosimeters. The average yearly dosimeter reading before the study was 2,587 μ Sv/year.
- **Control group (n = 50):** Laboratory technicians working in the same university hospitals but without occupational exposure to IR. To minimize potential confounding from exposure to other genotoxic agents, individuals routinely handling cytotoxic drugs, organic solvents, formaldehyde, mutagenic chemicals, or industrial toxic substances were not eligible for inclusion. The participating laboratory technicians were primarily involved in routine clinical laboratory procedures and did not work in pathology laboratories, histopathology laboratories, cytotoxic drug preparation units, or research laboratories where exposure to known genotoxic chemicals may occur.

Common exclusion criteria were recent viral infections, chronic disease, drug consumption, recent radio-diagnostic studies, genetic disorders, and a family history of cancer.

Data and sample collection

A comprehensive questionnaire was used to obtain demographics, occupational history, smoking habit, and medical history data. A venous blood sample (10 mL) was taken from each subject after strict asepsis. Then, blood was aliquoted for complete blood count, routine biochemistry, and genotoxicity analysis.

Alkaline comet assay

The comet assay was performed on peripheral blood lymphocytes isolated through density gradient centrifugation, according to a previously described method⁽¹⁰⁾ with some modifications. Briefly, cells were mixed in low melting-point agarose and placed on precoated slides, then lysed for ≥ 1 h in a solution containing 2.5 M NaCl, 100 mM EDTA, 10 mM Tris, and 1% Triton X-100, pH 10. After alkaline unwinding in 300 mM NaOH and 1 mM EDTA, pH >13 , electrophoresis was performed at 25 V and 300 mA for 30 min. Slides were then neutralized and stained with ethidium bromide at 20 μ g/mL. Then, slides were examined under an Olympus BX53 fluorescence microscope equipped with a CCD camera. For each sample, comets were analyzed from randomly selected cells at a rate of 100 per sample using the Comet Assay IV software developed by Perceptive Instruments. Analyzed parameters were tail DNA (%), tail length (px), olive tail moment, head intensity, and total intensity.⁽¹¹⁾

Flow cytometric analysis of γ -H2AX

To further evaluate DSBs, γ -H2AX analysis was performed on a randomly selected subset of participants comprising 10 radiology technicians and 10 controls. Random selection was employed to minimize selection bias while providing a representative sample from each study group. Peripheral blood mononuclear cells were isolated, fixed, and permeabilized before incubation with an anti- γ -H2AX (Ser139) primary antibody (Catalog No. JBW301, Millipore), followed by an Alexa Fluor 488-conjugated secondary antibody. Cells were counterstained with propidium iodide and fluorescence signals acquired using a Beckman Coulter flow cytometer. Data were analyzed using FlowJo software, and the median fluorescence intensity (MFI) and the percentage of γ -H2AX-positive cells were recorded as DSB indicators.⁽¹²⁾

Statistical analysis

Statistical analyses were performed using Statistical Package for the Social Sciences (SPSS; version 25.0; IBM Corp., Armonk, NY, USA). Normally distributed data were presented as mean $\pm$ standard deviation (SD), whereas nonnormally distributed data are expressed as median (minimum–maximum). Categorical variables were presented as frequencies and percentages. Comparisons between the exposed and control groups were performed using the independent-samples *t*-test for normally distributed continuous variables and the Mann–Whitney *U* test for nonnormally distributed variables. Categorical variables were compared using the chi-square test or Fisher's exact test, as appropriate. To quantify the magnitude of between-group differences, standardized effect sizes (Hedges' *g*) with corresponding 95% confidence intervals (CIs) were calculated for the main comet assay parameters and γ -H2AX biomarkers. Receiver operating characteristic (ROC) curve analysis was performed to evaluate the ability of comet assay parameters to discriminate between radiation-exposed workers and controls. Correlations between DNA damage biomarkers and clinical or occupational variables were assessed using Spearman's rank correlation coefficient. Multivariable linear regression analyses were performed, with occupational radiation exposure as the primary independent variable, and age, sex, and smoking status as covariates. Regression coefficients (β), 95% CIs, and corresponding *P*-values were reported. All statistical tests were two-tailed, and a *P* < 0.05 was considered statistically significant.

Ethical issue

This study was approved by the Medical Research Ethics Committee, Institutional Review Board,

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Results

Demographic and basic clinical characteristics

Both exposed and control groups were equally matched in terms of age (38.3 ± 10.2 vs. 39.7 ± 9.4 , *P* = 0.5) and sex (72.0% male vs. 76.0%, *P* = 0.6). Mean duration of occupational exposure in radiological technicians was 21.2 ± 9.5 years. Routine hematological and biochemical indicators such as hemoglobin, white blood cells, alanine aminotransferase, aspartate aminotransferase and creatinine showed no differences between the experimental and control groups. However, the platelet count was significantly higher in the experimental group (253.1 ± 35.2 vs. $162.2 \pm 28.2 \times 10^3/\mu\text{L}$, *P* = 0.001).

Comet assay indices

All primary comet assay parameters significantly differed significantly between exposed and control groups (*P* < 0.001 for all comparisons, **Table 1**). Tail DNA (%), olive tail moment, tail length, and comet width demonstrated large standardized effect sizes (Hedges' *g* = 10.3, 3.7, 3.8, and 2.1, respectively), whereas head intensity showed a very large negative effect size (*g* = −8.3). These findings indicate that the observed differences are not only statistically significant but also biologically meaningful. ROC curve analysis demonstrated excellent diagnostic accuracy for key parameters. Tail moment, tail DNA%, and head intensity achieved area under the curve (AUC) values of 1.000 (*P* < 0.001), with 98.0%–100.0% sensitivity and specificity. Comet width also showed high accuracy (AUC = 0.9).

Table 1. Comparison of comet assay parameters between exposed and control groups.

Parameter	Exposed group (n = 50)	Control group (n = 50)	<i>P</i> -value	Hedges' <i>g</i>	95% CI
Tail DNA (%)	71.4 (58.3–87.5)	3.4 (0.01–9.0)	<i>P</i> = 0.001*	10.3	8.8 to 11.8
Olive Tail Moment	8.5 (2.0–13.5)	0.2 (0.0–0.3)	<i>P</i> = 0.001*	3.7	3.0 to 4.3
Head intensity	25.6 $\pm$ 8.7	88.7 $\pm$ 6.2	<i>P</i> = 0.001*	−8.3	−9.6 to −7.1
Tail length (px)	89.2 (57.1–124.6)	26.0 (7.4–42.1)	<i>P</i> = 0.001*	3.8	3.1 to 4.4
Comet width (px)	54.7 (33.4–75.5)	27.2 (7.7–54.1)	<i>P</i> = 0.001*	2.1	1.6 to 2.6

CI, confidence of interval; SD, standard deviation. Data are expressed as mean $\pm$ SD or median (minimum-maximum).

*statistically significant.

Table 2. γ -H2AX marker levels in a subset of subjects.

Parameter	Exposed group (n = 10)	Control group (n = 10)	P-value	Hedges' g	95% CI
MFI (mean $\pm$ SD)	1,850.0 $\pm$ 320.0	980.0 $\pm$ 210.0	$P=0.001^*$	4.2	2.5 to 5.8
% Positive cells (mean $\pm$ SD)	8.5 $\pm$ 2.1	3.2 $\pm$ 1.4	$P=0.001^*$	2.8	1.5 to 4.1

CI, confidence of interval; MFI, mean fluorescence intensity; SD, standard deviation.

*Statistically significant.

Table 3. Multivariable linear regression analysis of the association between occupational radiation exposure and DNA damage biomarkers.

Predictor	Tail DNA (%) β (95% CI)	Olive tail moment β (95% CI)	Tail length (px) β (95% CI)	Comet width (px) β (95% CI)	γ -H2AX MFI β (95% CI)	γ -H2AX positive cells (%) β (95% CI)
Occupational radiation exposure	69.0(66.4–71.7) $P < 0.001^*$	8.1(7.2–9.0) $P < 0.001^*$	66.3(59.3–73.3) $P < 0.001^*$	25.9(21.0–30.8) $P < 0.001^*$	820.2(619.1–1021.3) $P < 0.001^*$	4.5(2.8–6.2) $P < 0.001^*$
Age (years)	–0.1(–0.2 to 0.1) $P = 0.4$	0.0(–0.0 to 0.1) $P = 0.4$	–0.2(–0.6 to 0.2) $P = 0.3$	0.3(0.0–0.6) $P = 0.024^*$	8.2(–0.8 to 17.2) $P = 0.1$	0.1(–0.0 to 0.1) $P = 0.1$
Male sex	–1.4(–4.8 to 1.9) $P = 0.4$	0.3(–0.9 to 1.4) $P = 0.6$	2.8(–6.0 to 11.5) $P = 0.5$	–6.0(–12.1 to 0.2) $P = 0.1$	–138.8(–349.0 to 71.5) $P = 0.2$	–1.4(–3.2 to 0.4) $P = 0.1$
Smoking	0.5(–2.8 to 3.7) $P = 0.8$	0.7(–0.4 to 1.7) $P = 0.2$	–2.6(–11.1 to 6.0) $P = 0.6$	6.1(0.1–12.1) $P = 0.046^*$	84.6(–125.7 to 294.9) $P = 0.4$	0.9(–0.9 to 2.7) $P = 0.3$

All models were adjusted for age, sex, and smoking status. β , regression coefficient; CI, confidence interval; MFI, median fluorescence intensity.

*Statistically significant.

γ -H2AX flow cytometry results

γ -H2AX analysis was performed on a randomly selected subset of 10 exposed participants and 10 controls. Flow cytometric analysis demonstrated significantly higher γ -H2AX expression in the exposed group, as reflected by both MFI and the percentage of γ -H2AX-positive cells (Table 2). Both variables showed large standardized effect sizes in addition to statistical significance ($P < 0.001$). MFI showed a Hedges' g of 4.2 (95% CI: 2.5–5.8), while the percentage of γ -H2AX-positive cells demonstrated a Hedges' g of 2.8 (95% CI: 1.5–4.1), indicating substantial biological differences between the exposed and control groups (Table 2).

Correlation analyses

Within the exposed group, a significant positive correlation was observed between comet width and years of occupational exposure ($r = 0.3$, $P = 0.028$). Negative correlations were found between hemoglobin levels and both head length ($r = 0.4$, $P = 0.002$) and tail intensity ($r = -0.3$, $P = 0.045$). In the γ -H2AX subset, a positive correlation existed between MFI and work duration ($r = 0.5$, $P = 0.001$).

Multivariable linear regression analysis

To evaluate whether the observed associations were independent of potential confounding factors, multivariable linear regression analyses were performed after adjusting for age, sex, and smoking status (Table 3). Occupational radiation exposure remained an independent predictor of all DNA damage biomarkers, including tail DNA (%), olive tail moment, tail length, comet width, γ -H2AX MFI, and the percentage of γ -H2AX-positive cells (all $P < 0.001$). Among all covariates, age was independently associated only with comet width ($\beta = 0.3$, 95% CI: 0.0–0.6; $P = 0.024$), while smoking was also independently associated with comet width ($\beta = 6.1$, 95% CI: 0.1–12.1; $P = 0.046$). Neither age, sex, nor smoking showed significant independent associations with the remaining comet assay or γ -H2AX biomarkers.

Discussion

Radiology technicians occupationally exposed to chronic low-dose IR demonstrated significantly higher DNA damage than the control group. Our findings

consistently indicate an association between long-term occupational radiation exposure and increased genotoxic damage. Using these two assays strengthens the biological evidence, as the comet assay detects overall DSBs, whereas γ -H2AX specifically reflects DSBs.

The first demonstrated significant differences between exposed workers and controls, particularly for tail DNA%, olive tail moment, and tail length. These parameters are widely accepted DSB indicators extensively used in biomonitoring studies of radiation-exposed healthcare workers. Our findings are consistent with previous studies that reported significantly increased DNA damage among hospital personnel occupationally exposed to X-rays.⁽¹³⁾ Similarly, Møller *et al.* identified tail-related comet parameters as the most reliable indicators of DNA damage in occupational biomonitoring studies, which agrees with the diagnostic performance observed in our ROC analysis.⁽¹⁴⁾ Our cohort's relatively long mean occupational exposure period of 21.2 years likely explains the more noticeable DNA damage observed compared to studies with shorter exposure durations.⁽¹⁵⁻¹⁸⁾ A notable finding in the present study was a significant reduction in head intensity among exposed workers compared with controls. This observation is consistent with migration of fragmented DNA from the comet head into the tail during electrophoresis, resulting in less DNA remaining within the nucleus. Consequently, reduced head intensity correlates with increased tail-related parameters and indicates greater DNA damage rather than improved DNA integrity. Similar changes in comet head characteristics have been linked to double-strand breaks and chromatin structural alterations.⁽¹⁰⁾

Flow cytometric analysis of γ -H2AX further supported comet assay findings by demonstrating significantly higher MFI and a greater percentage of γ -H2AX-positive cells in the exposed group. As γ -H2AX is one of the earliest cellular responses to DSBs, these findings provide additional molecular evidence of persistent DNA damage among radiation-exposed workers. Similar observations have been reported in previous occupational studies evaluating healthcare professionals chronically exposed to IR.^(19, 20) Furthermore, γ -H2AX expression increases with radiation dose, and could serve as a sensitive biomarker of biological radiation exposure.^(21, 22)

Our observation of a positive correlation between γ -H2AX expression and years of occupational exposure further supports an association between cumulative occupational exposure and DNA damage. The positive correlation between comet width and years of occupational exposure also supports that cumulative exposure, rather than a single exposure event, is associated with increasing DNA damage. Previous biomonitoring studies have similarly observed that prolonged occupational exposure can lead to persistent genomic injury even when current radiation protection measures are followed.^(4, 17) These findings reinforce the importance of continuous monitoring of radiation workers throughout their professional careers.

We also detected significantly higher platelet count among exposed workers. Although previous studies reported no significant hematological changes in radiation workers,⁽²³⁾ others suggested that chronic low-dose radiation exposure may be associated with subtle biological stress responses.⁽¹⁸⁻²⁰⁾ The present study's lack of inflammatory biomarker measurement like IL-6 leaves the underlying mechanism for the elevated platelet count uncertain. Consequently, any link between platelet counts and inflammatory pathways should be interpreted cautiously and warrants further investigation. Conversely, negative correlations between hemoglobin levels and selected DNA damage parameters suggest a potential link between hematological status and genomic damage. However, due to the cross-sectional design of this study, these findings should be considered as associations rather than causal evidence. Further longitudinal studies incorporating oxidative stress and DNA repair biomarkers are needed to clarify these relationships.

Our findings differ from some recent reports that found no increase in DNA damage among occupationally exposed healthcare workers.⁽⁸⁾ Discrepancies in cumulative radiation dose, occupational exposure duration, adherence to radiation protection practices and institutional safety protocols could partly explain these differences. The relatively long occupational exposure in this study might have heightened the likelihood of detecting cumulative biological effects less noticeable in shorter employment periods.

This study has several limitations. First, its cross-sectional case-control design precludes establishing a temporal or causal relationship between occupational radiation exposure and DNA damage. Second, despite matching participants for age and sex, residual confounding by lifestyle factors, dietary antioxidant intake, genetic polymorphisms affecting DNA repair, and adherence to personal protective measures cannot be excluded. In addition, γ -H2AX analysis was performed in a randomly selected subset of participants, which may limit generalizability, despite minimizing selection bias. Future prospective studies involving larger populations and repeated biomarker measurements are warranted to validate these findings.

Conclusion

The present findings indicate that long-term occupational exposure to low-dose IR is associated with increased levels of DNA damage biomarkers in radiology technicians. The combined use of the comet assay and γ -H2AX analysis provide complementary information that may enhance the biological monitoring of radiation workers alongside conventional physical dosimetry.

Author contributions

MM and SAEM contributed substantially to the concept and design of this study, acquiring the data, reviewing the literature, and its analysis and evaluation, and interpretation. EAA contributed substantially to acquiring the data. MEW and SAEM contributed to drafting the manuscript. MM and MEW edited the manuscript critically for critical reading and intellectual content. All authors approved the final version submitted for publication and take responsibility for statements made in the published article.

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Conflict of interest statement

All authors have completed and submitted the International Committee of Medical Journal Editors Uniform Disclosure Form for Potential Conflicts of Interest. All authors declare that they have no conflicts of interest.

Data sharing statement

All data generated or analyzed in the present study are included in the published article. Further details are available for non-commercial purposes from the corresponding author upon reasonable request.

ORCID

Maged Mostafa  <https://orcid.org/0000-0001-9723-2710>

Ebada Abd AlRaouf  <https://orcid.org/0009-0000-8750-5594>

Mona El Wassefy  <https://orcid.org/0000-0002-7295-9793>

Samir El Masry  <https://orcid.org/0009-0001-1536-2587>

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