Original Article

# Does short-term exposure to elevated levels of natural gamma radiation in Ramsar cause oxidative stress?

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## Abstract

**Background:** Ramsar, a city in northern Iran, has areas with some of the highest recorded levels of natural radiation among inhabited areas measured on the earth. **Aims:** To determine whether short-term exposure to extremely high levels of natural radiation induce oxidative stress. **Materials and Methods:** In this study, 53 Wistar rats were randomly divided into five groups of 10-12 animals. Animals in the 1st group were kept for 7 days in an outdoor area with normal background radiation while the 2nd, 3rd, 4th and 5th groups were kept in four different outdoor areas with naturally elevated levels of gamma radiation in Ramsar. A calibrated RDS-110 survey meter, mounted on a tripod approximately 1 m above the ground, was used to measure exposure rate at each location. On days 7 and 9 blood sampling was performed to assess the serum levels of catalase (CAT) and malondialdehyde (MDA). On day 8, all animals were exposed to a lethal dose of 8 Gy gamma radiations emitted by a Theratron Phoenix (Theratronics, Canada) Cobalt-60 (55 cGy/min) at Radiotherapy Department of Razi Hospital in Rasht, Iran. **Results:** Findings obtained in this study indicate that high levels of natural radiation cannot induce oxidative stress. CAT and MDA levels in almost all groups were not significantly different ($P = 0.69$ and $P = 0.05$, respectively). After exposure to the lethal dose, CAT and MDA levels in all groups were not significantly different ($P = 0.054$ and $P = 0.163$, respectively). **Conclusions:** These findings indicate that short-term exposure to extremely high levels of natural radiation (up to 196 times higher than the normal background) does not induce oxidative stress.

**Key words:** Background radiation, natural radiation, oxidative stress, Ramsar

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## Introduction

Ramsar, a city in northern Iran, is among the world's well-known inhabited areas with highest levels of natural radiation.[1-3] In areas with elevated levels of natural radiation annual exposure rates are up to 260 mSv $y^{-1}$ and average dose rates are about 10 mGy $y^{-1}$ for a population of about 2000 residents.[4-6] Due to the local geology, which includes high levels of radium in rocks, soils, and groundwater, Ramsar residents are also exposed to high levels of alpha activity in the form of ingested radium and radium decay progeny as well as very high radon levels in their dwellings (over 3000 Bq $m^{-3}$ in some cases). It has been reported that the inhabitants of these areas receive doses much higher than the current ICRP-60 occupational dose limit of 20 mSv $y^{-1}$.[7]

In 2002, we published a report on radioadaptive responses induced in the lymphocytes of the residents who lived in high background radiation areas of Ramsar.[4] We also showed that short-term exposure to elevated levels of radon could induce an adaptive response in laboratory animals.[8] In 2003, we conducted the first limited epidemiological study in

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these areas and investigated the relationship between indoor radon level and the incidence of lung cancer.[5] In that study, data from the Ramsar health network showed that both crude lung cancer rate and adjusted lung cancer rate in one district with the highest record. To obtain reasonable and consistent data for 2000 residents, long-term observation is required. Nonetheless, it could be concluded that lung cancer rate may show a negative correlation with natural radon concentration.[5]

Elevated levels of low dose rate natural radiation of Ramsar provide a unique opportunity to study epidemiological effects for the inhabitants. However, to obtain reasonable and consistent data for 2000 residents, we need to observe them for long time to acquire considerable number of person-years for reliable statistical data. We believe up to date there has been no large-scale epidemiological study for inhabitants of high background radiation areas of Ramsar. Therefore, lack of long-term epidemiological data has raised several public health policy issues, such as relocation of the inhabitants to areas of lower natural background radiation levels as well as financial and emotional costs of relocation.[9-11]

It is worth noting that Catalase (CAT), as a major primary intracellular antioxidant enzyme, converts $H_2O_2$ into $H_2O$.[12] In addition, Malondialdehyde (MDA), as a marker of oxidative damage, determines the level of lipid peroxidation.[13] It is believed that ionizing radiation may start oxidizing events which can change atomic structure by direct interactions of radiation with target macromolecules or via water radiolysis-induced products.[14] Partial reduction of molecular oxygen in mitochondria under normal physiological conditions generates reactive oxygen species (ROS).[15] Excessive ROS production that may lead to oxidative damage to proteins, lipids, and DNA or impaired antioxidant system causes oxidative stress.[16] Therefore, it is believed that adaptive responses may involve transcription of many genes and activation of a wide variety of signaling pathways that trigger specific cell defense mechanisms.[17]

Recently Mortazavi *et al*., have shown that short-term exposure to artificially elevated levels of radon may induce an adaptive response in an animal model.[8] In this study we investigate the possible induction of oxidative stress after short-term exposure to extremely high levels of natural radiation (up to 196 times higher than the normal background). In addition, since there is no report on the induction of adaptive response in short-term exposures to high background levels of gamma radiation, the main aim of this study is to verify if exposure of laboratory animals to extremely elevated levels of natural external gamma can lead to induction of oxidative stress.

## Materials and Methods

### Animals

In this study, 53 Wistar rats (200-250 g) purchased from Pasteur Institute, Amol, Iran were randomly divided into five groups of (10-12 each). Animals were kept under a 12-hour light and a 12-hour dark cycle at 21 ± 1°C with free access to food and water. All guidelines of Shiraz University of Medical Sciences (SUMS) for ethical treatment of animals were observed.

### Exposure to naturally elevated levels of radiation

Animals in the 1st group were kept for 7 days in an outdoor area with normal background radiation (0.18 μSv/h) while the 2nd, 3rd, 4th and 5th groups were kept in 4 different outdoor areas in Ramsar with naturally elevated levels of gamma radiation of 3.92, 8.47, 16.43 and 35.28 μSv/h, respectively. A calibrated RDS-110 survey meter, mounted on a tripod approximately 1 m above the ground, was used to measure exposure rate at each location.

### CAT and MDA measurements

On days 7 and 9, blood sampling was performed to assess the serum levels of catalase (CAT) and malondialdehyde (MDA).

### Exposure to lethal dose

On day 8, all animals were exposed to gamma radiations emitted by a Theratron Phoenix (Theratronics, Canada) Cobalt-60 therapeutic source (8 Gy, 55 cGy/min) at Radiotherapy Department of Razi Hospital in Rasht, Iran.

### Statistical analysis

Appropriate non-parametric tests such as Friedman and Mann-Whitney (SPSS 17.0) were used to analyze statistical data.

## Results

As shown in Table 1, even though there is no significant difference between CAT levels in animals kept in locations with different dose rate (0.18 to 35.28 μSv/hr), but after exposure to a lethal dose of 8 Gy, there is a statistically significant ($P = 0.01$) difference between CAT levels in animals kept in an area with a dose rate of 3.93 μSv/hr (1.97 ± 2.03) and 35.28 μSv/hr (1.19 ± 1.59). In contrast as shown in Table 2, the MDA levels in animals kept in locations with different dose rate, were not significantly different. Except for MDA levels in animals kept in an area with a dose rate of 3.93 μSv/hr (0.50 ± 0.09) and 8.48 μSv/hr (0.69 ± 0.15), a statistically significant difference ($P = 0.02$) is found.

After exposure to a lethal dose of 8 Gy, the CAT levels in animals kept in an area with dose rate of 3.93 μSv/hr (0.43 ± 0.15) and 8.48 μSv/hr (0.65 ± 0.16), statistically significant differences ($P = 0.03$) and ($P = 0.05$) were

found between 3.93 μSv/hr (0.43 ± 0.15) and 35.28 μSv/hr (0.58 ± 0.16), respectively. Tables 3 and 4 show serum CAT and MDA levels before and after exposure to the lethal dose in animals kept in areas with different levels of natural radiation in high background radiation areas of Ramsar.

## Discussion

In early studies, Feinendegen *et al*., proposed that adaptive response could be induced by reactive oxygen species (ROS).[18,19] The ROS refers to a group of molecules including peroxides and free radicals which are derived from oxygen and are highly reactive toward bimolecular.[20] ROS react with critical biomolecules such as DNA and induce oxidative stress (imbalance of pro-oxidants versus antioxidants) and damage in these macromolecules, multiple localized lesions such as base damage, single strand breaks (SSBs) and double strand breaks (DSBs), DNA-DNA cross links and DNA-protein cross links.[21-24] And they can contribute to the progression of multiple diseases, such as cancer.[25-28] As indicated by some investigators,[17,29,30] induction of adaptive response by pre-exposure to ionizing radiation needs a minimum level of damage that triggers this phenomenon and increases the resistance of living organisms *(in vivo)* or cells *(in vitro)* to higher levels of the same or of other sources of stress.

In this study our results indicate that high levels of natural radiation cannot induce oxidative stress. And short-term exposure to higher levels of external gamma in a high background radiation area possibly does not induce the minimum required level of cell damage. We strongly believe that identification of the key mechanisms of the adaptive response will help us explain the origin of this difference. The phenomenon of adaptive response probably involves the signaling pathways that trigger cell defense (such as more efficient detoxification of free radicals), DNA repair systems and more specifically

**Table 1: CAT levels in animals kept in different places with high levels of natural radiation before and after exposure to lethal dose**

| | Natural radiation dose rate (μSv/hr) | | | | | Significance (friedman test *P* value) |
|---|---|---|---|---|---|---|
| | 0.18 | 3.93 | 8.48 | 16.43 | 35.28 | |
| CAT before exposure to LD | 0.89±0.10 | 1.36±1.59 | 1.68±2.01 | 1.26±1.26 | 1.20±0.98 | 0.69 |
| CAT after exposure to LD | 0.80±0.28 | 1.97±2.03 | 1.58±1.80 | 0.91±0.31 | 1.19±1.59 | 0.054 |
| Significance (Mann-Whitney test *P* value) | 0.508 | 0.327 | 0.249 | 0.214 | 0.248 | |

CAT: Catalase; LD: Lethal dose

**Table 2: MDA levels in animals kept in different places with high levels of natural radiation before and after exposure to lethal dose**

| | Natural radiation dose rate (μSv/hr) | | | | | Significance (friedman test *P* value) |
|---|---|---|---|---|---|---|
| | 0.18 | 3.93 | 8.48 | 16.43 | 35.28 | |
| MDA before exposure to LD | 0.53±0.15 | 0.50±0.09 | 0.69±0.15 | 0.58±0.21 | 0.60±0.17 | 0.05 |
| MDA after exposure to LD | 0.48±0.11 | 0.43±0.15 | 0.65±0.16 | 0.55±0.15 | 0.58±0.16 | 0.163 |
| | 0.037 | 0.263 | 0.345 | 0.316 | 0.33 | |

MDA: Malondialdehyde; LD: Lethal dose

**Table 3: Serum CAT level before and after exposure to the lethal dose in animals kept in areas with different levels of natural radiation in high background radiation areas of Ramsar**

| Dose rate | Sample size | Status (before/after exposure to lethal dose) | CAT level IU/ml serum (mean±SD) | Significance level (Mann-Whitney test *P* value) (μSv/hr) | | | | | | | | | | Kruskal-Wallis test Total |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | 0.18 | | 3.93 | | 8.48 | | 16.43 | | 35.28 | | |
| | | | | BLD | ALD | BLD | ALD | BLD | ALD | BLD | ALD | BLD | ALD | |
| 0.18 μSv/hr | 10 | BLD | 0.89±0.10 | NA | | 0.83 | 0.173 | 0.88 | 0.88 | 0.15 | 0.98 | 0.51 | 0.09 | |
| | | ALD | 0.80±0.28 | | NA | | | | | | | | | |
| 3.93 μSv/hr | 8 | BLD | 1.36±1.59 | 0.83 | | NA | | 0.66 | | 0.22 | | 0.49 | | |
| | | ALD | 1.97±2.03 | | 0.173 | | NA | | 0.49 | | 0.18 | | 0.01* | |
| 8.48 μSv/hr | 6 | BLD | 1.68±2.01 | 0.88 | | 0.66 | | NA | | 0.41 | | 0.80 | | |
| | | ALD | 1.58±1.80 | | 0.88 | | | | NA | | 0.77 | | 0.048** | |
| 16.43 μSv/hr | 18 | BLD | 1.26±1.26 | 0.15 | | 0.22 | | 0.41 | | NA | | 0.36 | | |
| | | ALD | 0.91±0.31 | | 0.98 | | 0.18 | | 0.77 | | NA | | 0.17 | |
| 35.28 μSv/hr | 11 | BLD | 1.20±0.98 | 0.51 | | 0.49 | | 0.80 | | 0.36 | | NA | | |
| | | ALD | 1.19±1.59 | | 0.09 | | 0.01* | | 0.048** | | 0.17 | | NA | |
| Total | 53 | BLD | 1.24±1.22 | | | | | | | | | | | 0.51 |
| | | ALD | 1.18±1.25 | | | | | | | | | | | 0.04 |

*Statistically significant difference between CAT levels after exposure to the lethal dose in animals kept in an area with a dose rate of 3.93 μSv/hr (1.97±2.03) and 35.28 μSv/hr (1.19±1.59); **Statistically significant difference between CAT levels after exposure to the lethal dose in animals kept in an area with a dose rate of 8.48 μSv/hr (1.58±1.80) and 35.28 μSv/hr (1.19±1.59). BLD: Before lethal dose; ALD: After lethal dose

**Table 4: Serum MDA level before and after exposure to the lethal dose in animals kept in areas with different levels of natural radiation in high background radiation areas of Ramsar**

| Dose rate | Sample size | Status (before/after exposure to lethal dose) | MDA Level nmol/ml (mean±SD) | Significance level (Mann-Whitney test *P* value) (μSv/hr) | | | | | | | | Kruskal-Wallis test Total |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | 0.18 | | 3.93 | | 8.48 | | 16.43 | | 35.28 | | |
| | | | | BLD | ALD | BLD | ALD | BLD | ALD | BLD | ALD | BLD | ALD | |
| 0.18 μSv/hr | 10 | BLD | 0.53±0.15 | NA | | 0.70 | 0.52 | 0.056 | 0.042* | 0.59 | 0.38 | 0.31 | 0.10 | |
| | | ALD | 0.48±0.11 | | NA | | | | | | | | | |
| 3.93 μSv/hr | 8 | BLD | 0.50±0.09 | 0.70 | | NA | | 0.02*** | | 0.43 | | 0.11 | | |
| | | ALD | 0.43±0.15 | | 0.52 | | NA | | 0.03*** | | 0.07 | | 0.05** | |
| 8.48 μSv/hr | 6 | BLD | 0.69±0.15 | 0.056 | | 0.02*** | | NA | | 0.12 | | 0.59 | | |
| | | ALD | 0.65±0.16 | | 0.042* | | 0.03*** | | NA | | 0.34 | | 0.81 | |
| 16.43 μSv/hr | 18 | BLD | 0.58±0.21 | 0.59 | | 0.43 | | 0.12 | | NA | | 0.64 | | |
| | | ALD | 0.55±0.15 | | 0.38 | | 0.07 | | 0.34 | | NA | | 0.74 | |
| 35.28 μSv/hr | 11 | BLD | 0.60±0.17 | 0.31 | | 0.11 | | 0.59 | | 0.64 | | NA | | |
| | | ALD | 0.58±0.16 | | 0.10 | | 0.05** | | 0.81 | | 0.74 | | NA | |
| Total | 53 | BLD | 0.57±0.17 | | | | | | | | | | | 0.19 |
| | | ALD | 0.54±0.15 | | | | | | | | | | | 0.09 |

*Statistically significant difference between MDA levels after exposure to the lethal dose in animals kept in an area with a dose rate of 0.18 μSv/hr (0.48±0.11 nmol/ml) and 8.48 μSv/hr (0.65±0.16 nmol/ml); **Statistically significant difference between CAT levels after exposure to the lethal dose in animals kept in an area with a dose rate of 3.93 μSv/ hr (0.43±0.15 nmol/ml) and 35.28 μSv/hr (1.19±1.59 nmol/ml); ***Statistically significant difference between CAT levels either before or after exposure to the lethal dose in animals kept in an area with a dose rate of 8.48 μSv/hr (1.58±1.80 nmol/ml) and 35.28 μSv/hr (1.19±1.59 nmol/ml). BLD: Before lethal dose; ALD: After lethal dose

non-homologous end-joining repair of DNA double strand breaks (DSBs).The role of p53 as a key mediator of DNA repair, is crucial in induction of new proteins in irradiated cells with a conditioning dose, and enhanced antioxidant production.[29,31-35]

## Conclusions

Findings of this study show that exposure of animals to naturally elevated levels of gamma radiation does not lead to induction of oxidative stress. Consistent with our previously reported data,[4,5,9-11,36-41] radiobiological studies on the health effects of the chronic exposure to elevated levels of natural radiation in residents of areas such as Ramsar may lead to the identification of the cellular and molecular mechanisms by which susceptibility to genetic damage and cancer is decreased by chronic radiation exposure. These findings will play an important role in areas such as radiation therapy, radiation protection and even selection of appropriate candidates for long term manned space missions.[37]

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