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# Biochemical Changes in Mouse after Radiation Exposure

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**ABSTRACT:** In a study evidence is mounting that UV-B and UV-A radiation affect skin differently in responses as diverse as erythema and elastosis. We found in this study that collagen metabolism was also differentially affected. Albino hairless mice were irradiated with two UV-A sources: (1) UVASUN 3000 (340-400 nm) for cumulative exposures of 4000 and 8000 J/cm<sup>2</sup>; (2) a xenon solar simulator filtered to provide full spectrum UV-A (320-400 nm) and long wavelength UV-A (335-400 nm) for cumulative exposures of 3000 and 4000 J/cm<sup>2</sup> respectively. Collagen was isolated from other skin proteins by acid extraction, pepsin digestion and salt precipitation. Collagen types I and III were separated by interrupted gel electrophoresis. Ultraviolet-A rendered the collagen highly resistant to pepsin digestion. In age-matched controls only 16-18% of the total collagen remained insoluble, whereas in long wavelength UV-A-irradiated skins the insoluble fraction was as high as 87%. A dose response was noted at 4000 and 8000 J/cm<sup>2</sup> as delivered by the UVASUN. Recovery of collagen from the pepsin soluble fraction was low in all UV-A groups and the amount of type III so small that determination of ratios of type III to I collagen was unreliable. These results suggest that chronic UV-A radiation may increase cross-linking of dermal collagen.

**KEYWORDS:** radiation, exposure, wavelength, UV, wavelength, metabolism, mouse, affected, solar

## I. INTRODUCTION

In a review it was found that exposure to acute, damaging radiation may occur through a variety of events from cancer therapy and industrial accidents to terrorist attacks and military actions. Our understanding of how to protect individuals and mitigate the effects of radiation injury or Acute Radiation Syndrome (ARS) is still limited.[1,2] There are only a few Food and Drug Administration-approved therapies for ARS; whereas, amifostine is limited to treating low dose (0.7–6 Gy) radiation poisoning arising from cancer radiotherapy. An early intervention is critical to treat ARS, which necessitates identifying diagnostic biomarkers to quickly characterize radiation exposure. Towards this end, a multiplatform metabolomics study was performed to comprehensively characterize the temporal changes in metabolite levels from mice and non-human primate serum samples following  $\gamma$ -irradiation. The metabolomic signature of amifostine was also evaluated in mice as a model for radioprotection. The NMR and mass spectrometry metabolomics analysis identified 23 dysregulated pathways resulting from the radiation exposure.[3,4] These metabolomic alterations exhibited distinct trajectories within glucose metabolism, phospholipid biosynthesis, and nucleotide metabolism. A return to baseline levels with amifostine treatment occurred for these pathways within a week of radiation exposure. Together, our data suggests a unique physiological change that is independent of radiation dose or species. Furthermore, a metabolic signature of radioprotection was observed through the use of amifostine prophylaxis of ARS.

The main objective of the another investigation was to observe biochemical changes induced by radiation and cadmium individually as well as simultaneously on the testis of Swiss albino mice. Materials and Methods: For this purpose, adult male Swiss albino mice (6-8 weeks old) were divided into four groups. Group I (sham-irradiated), Group II (treated with CdCl<sub>2</sub> solution 20 ppm), Group III (irradiated with 1.25, 2.5 and 5.0 Gy gamma rays), Group IV (both irradiated with 1.25, 2.5 and 5.0 Gy gamma rays and treated with CdCl<sub>2</sub> solution). The animals were autopsied after 1, 2, 4, 7, 10, 14 and 28 days of treatment. The testis was taken out and different biochemical parameters, such as total proteins, DNA and RNA were estimated. Results: It was found that contents of Total proteins, DNA and RNA decreased. Such decline was observed with higher dose (5.00 Gy) at early intervals. Recovery started at day 10 or 14 but control level could not be attained up to the last intervals. A similar decreasing pattern was observed[5,6] with 2.5 and 1.25 Gy, but it was found lesser as compared to 5.0 Gy group. The recovery was also faster in these groups. Results indicated the infliction of radiation insults and subsequent repair in the testes to be dose-dependent. In combined treatment group the pattern of biochemical changes was similar to individual treatment but the magnitude of occurrence was statistically higher and the normal value could not be attained even at last autopsy (28 days). Thus the damage and



recovery pattern in combined treatment (radiation and cadmium chloride) indicates the synergistic effect of these two agents. These results indicate that combined treatment of cadmium and gamma radiation causes synergistic or additive effect.

In one study, the radioprotective effect of WR-2721 on late damage after whole thorax irradiation has been studied after split doses of radiation using the standard death and breathing rate assays at monthly intervals between 3 and 15 months after irradiation, as well as two biochemical measurements of injury at 15 months, hydroxyproline (HP), an indicator of tissue fibrosis, and DNA content, an indicator of tissue cellularity. [7,8] A comparison of HP/lung and breaths per minute (BPM) in each dose group in the WR-2721 and non-WR-2721-treated mice 15 months after irradiation showed that the relationship between these two assays of late lung injury was not the same. There were large dose-related increases in breathing rate corresponding to relatively small changes in HP in the lungs of mice given radiation alone. In contrast, the mice given WR-2721 before irradiation showed large dose-related increases in HP/lung, but BPM remained relatively constant independent of dose. These data suggest then that changes in breathing rate and deaths later than 9 months after whole lung irradiation may not be due to collagen accumulation in the lung. WR-2721 did protect better against late lung functional changes (protection factors (PF) = 1.6) and late deaths (PF = 1.51) than against earlier changes in these same assays (PF = 1.4 and 1.28, respectively). Although the earlier-appearing injury after whole thoracic irradiation is most likely related to lung damage with deaths and increases in breathing rate resulting from pneumonitis, the cause of the late-appearing functional injury in the lung after radiation is not clear. [9,10] Thus protection of late lung damage measured from either lethality or breathing rate is not related to the prevention of lung fibrosis.

The last decades have seen increased concern about the possible effects of low to moderate doses of ionizing radiation (IR) exposure on cognitive function. An interdisciplinary group of experts (biologists, epidemiologists, dosimetrists and clinicians) in this field gathered together in the framework of the European MELODI workshop on non-cancer effects of IR to summarise the state of knowledge on the topic and elaborate research recommendations for future studies in this area. Overall, there is evidence of cognitive effects from low IR doses both from biology and epidemiology, though a better characterization of effects and understanding of mechanisms is needed. There is a need to better describe the specific cognitive function or diseases that may be affected by radiation exposure. Such cognitive deficit characterization should consider the human life span, as effects might differ with age at exposure and at outcome assessment. [11,12]

Measurements of biomarkers, including imaging, will likely help our understanding on the mechanism of cognitive-related radiation induced deficit. The identification of loci of individual genetic susceptibility and the study of gene expression may help identify individuals at higher risk. The mechanisms behind the radiation induced cognitive effects are not clear and are likely to involve several biological pathways and different cell types. Well conducted research in large epidemiological cohorts and experimental studies in appropriate animal models are needed to improve the understanding of radiation-induced cognitive effects. Results may then be translated into recommendations for clinical radiation oncology and imaging decision making processes.

## II. DISCUSSION

To study the effect of age on the morphologic and biochemical alterations induced by in vivo exposure of ultraviolet radiation (UV). Young and old C57BL/6 mice were exposed to broadband UVB+UVA and euthanized after 2 days. Another batch of UV-exposed young mice was monitored for changes after 1, 2, 4, and 8 days. Age-matched nonexposed mice served as controls. Lens changes were documented in vivo by slit-lamp biomicroscopy and dark field microscopy photographs ex vivo. Lens homogenates were analyzed for glutathione (GSH) level, and the activities of thioredoxin (Trx), thioltransferase (TTase), and glyceraldehyde-3-phosphate dehydrogenase (G3PD). Glutathionylated lens proteins (PSSGs) were detected by immunoblotting using GSH antibody. Western blot analysis was also done for the expression levels of TTase and Trx. Both age groups developed epithelial and superficial anterior subcapsular cataract at 2 days postexposure. The lens GSH level and G3PD activity were decreased, and PSSGs were elevated in both age groups, but more prominent in the older mice. TTase and Trx activity and protein expression were elevated only in the young mice. Interestingly, lens TTase and Trx in the young mice showed a transient increase, peaking at 2 days after UV exposure and returning to baseline at day 8, corroborated by lens transparency. The lenses of old mice were more susceptible to UV radiation-induced cataract. The upregulated TTase and Trx likely provided oxidation damage repair in the young mice. [13,14]

If a group of mice is exposed to 600 to 700 rads of X-rays delivered in a brief period (the exact dose for the response described depends on strain, age, and other variables described below) and observed over several days, one is struck by the apparent lack of any gross evidence of any effect for several days. The animals appear entirely normal. Although



progressive weight loss is occurring, the blood count is declining, and various internal tissues are becoming atrophic, no clue to these derangements is given by the external appearance of the mice. About the fifth or sixth day following exposure a few of the mice may begin to appear lethargic with ruffled fur and a generally unkempt appearance. By the eighth day a few of the mice may be dead. Daily mortality increases to a peak between the 10th and 14th day then gradually subsides. After the 20th day deaths are infrequent and the surviving mice show evidence of recovery from their injury. Thirty days after exposure it is impossible to tell an irradiated survivor from a normal mouse by inspection. Because of this characteristic slow timecourse of injury and recovery a longer observation period is used in radiation studies than is commonly used in studies of response to drugs. A 30-day observation period for evaluating the acute lethal response of mice to radiation exposure is widely used.

If the radiation dose is increased to 1,000 to 1,500 rads, a somewhat different pattern of illness and death emerges. With mice from our colony, it is obvious by the third or fourth day that the animals have been severely injured. They show evidence of diarrhea and an accompanying dehydration. Loss of weight is pronounced and the animals tend to sit quietly in the cage. On the fifth day some deaths occur and on the sixth day there is a sharp peak of mortality. Those surviving beyond 6 days may show a secondary mortality peak in the 8- to 12-day period. None survive beyond about 14 days.[15,16]

As the radiation dose is further increased up to about 10,000 rads there is little, if any, effect on survival time. All the mice die in the 5- to 7-day period to produce a curious plateau in the curve relating dose and mean survival time. Other workers have found that this stable death time occurs somewhat earlier than 5 to 7 days and many reports of a 3½-day survival time have been published. We believe the basic phenomenon to be the same but that the exact survival time depends strongly on environmental factors, particularly the intestinal bacterial flora. With radiation doses higher than about 10,000 rads the survival time again becomes dose-dependent, i.e., the higher the dose the shorter the mean survival. Signs of neurological damage appear at these very high doses with convulsive seizures a prominent part of the syndrome. With extremely high exposures delivered at high dose rates the survival time decreases to a few hours or less and, in fact, it is possible to kill animals under the beam.[17,18]

Because of these dose-dependent peaks in mortality and changing clinical signs of injury, it has been suggested that there are various modes of death in mice exposed to total-body irradiation and further that each modality has its own characteristic death time. At least four such modes have been established. Deaths at the lowest doses of radiation, where the peak in mortality occurs at 10 to 14 days, are due primarily to bone marrow injury. The evidence for this is as follows. Death at these dosages can be prevented by shielding a portion of the bone marrow such as that contained in one femur or by inoculating the exposed animals with bone marrow cells. In the dose range between about 1,000 and 2,000 rads the small peak in mortality at 8 or 9 days (in those animals surviving for this period) is apparently due to damage to oral structures. Doses of about this magnitude to the head only will reproduce this survival time with adequate evidence of histological injury to the tongue and pharynx to explain the cause of death. The survival time of 5 to 6 days (3½ days in some laboratories) at doses up to about 10,000 rads is due to intestinal injury. These survival times can be produced by irradiation of the abdomen or by irradiation of the exteriorized intestine with the remainder of the animal shielded. The short survival times resulting from massive exposures can be duplicated by irradiation restricted to the head. Direct damage to the brain is responsible for death. There is a tendency to oversimplify "causes of death" in irradiated mice in view of the identified modalities discussed above. It should not be assumed, for example, that marrow damage alone is responsible for death in the low dose range. It is of major importance but there is a complex interplay of damage to a number of tissues and tissue functions leading to death. Neither should it be assumed that the tissues listed are the only ones damaged by radiation or that these are the only modes of death. There is undoubtedly a hierarchy of causes of death. For example, radiation to the kidneys only or the heart only can also be fatal if the radiation dose is sufficiently high to compromise proper function.[19,20]

The remarkable effect of environment on survival time, particularly in the gut-death range, deserves further consideration. The survival time as a function of dose for mice maintained in three different environments. Maximum survival times were found in germ-free mice. Minimum times were obtained in an early study. In this particular investigation a very great range of doses was utilized and the short survival from massive doses (central nervous system death) is illustrated. Later studies (Storer, unpublished data) with mice rigidly controlled in a closed colony but not maintained either germ-free or pathogen-free showed survival times intermediate between the other two studies. There were, of course, strain differences as well as differences in radiation sources in these three investigations. Nevertheless, it is considered highly probable that environmental differences, and specifically differences in bacterial flora, account for the somewhat dissimilar results. As animal stocks and animal-colony management improve it may be anticipated that the survival curve will shift upward toward the line representing the germ-free mice.



### III. RESULTS

Genetic constitution is one of the major factors influencing radiation resistance in mice. It is well known that there are major strain differences in sensitivity. Differences are found not only in the survival-dose  $LD_{50}$  but also in the length of survival under repeated radiation exposures. For example, it has been determined the survival time of 27 inbred mouse strains under daily exposure to 110 R. An array of mean survival times was obtained with the most resistant strain (129/J) living twice as long as the most sensitive strain (CBA/J). Since the survival times obtained by this method are known to be correlated with the single-dose  $LD_{50}$ , it seems safe to assume that a similar ordering of sensitivity in these strains would have been obtained for  $LD_{50}$  determinations. The single-dose  $LD_{50}$  values for 10 strains of mice in our laboratory. It can be seen that there are major differences in these values. Because of these genetically controlled variations in sensitivity, inbred mouse strains provide an ideal experimental tool for determining the nature of radiation resistance. The correlated characteristics so far identified have been discussed. Generally, those characters considered to be indices of vigor are positively associated with resistance. Thus, radiation resistance may be a nonspecific measure of vigor. Nevertheless, continued study to identify genetically controlled physiological factors responsible for resistance seems worthwhile.[21,22]

Hybrids are usually more resistant than either parental strain except in those cases in which parental strains of very widely divergent sensitivity are used. In this case the hybrids may fall between two parental strains though closer to the more resistant parent. Single-gene substitutions in an inbred line produce little or no effect on sensitivity unless the gene also produces major physiological disturbances. For example, the genes for short ear (se) and for obesity (ob) produce detectable shifts in sensitivity as well as other gross disturbances. Genes such as those responsible for severe anemia produce a drastic shift in sensitivity as might be expected. Bernstein has reported an  $LD_{50}$  of about 200 R for congenitally anemic mice which makes these animals at least three times as sensitive as their normal siblings. It appears, then, that given a reasonably normal phenotype, genetic control of resistance probably represents a complex interaction of a large number of gene loci as appears to be the case with other characteristics of vigor.

There are a number of other variables besides genetic constitution that result in differences in radiation resistance. Within a strain, major changes in resistance are found with increasing age. It was found higher resistance at birth than at 30 days of age. After minimum resistance at 30 days, resistance increases to a maximum in young adulthood, remains on a plateau for a variable length of time, and then declines in old age. There is not complete agreement when the plateau of maximum resistance is attained and the time might well be a function of strain. In general, resistance increases from the time of weaning to about 3 or 4 months of age and misleading results in comparisons of  $LD_{50}$ 's can be obtained if the animals are exposed during this period of rapidly changing resistance. All too often, however, investigations are conducted with mice in this age range. We recommend the routine use of animals at least 3 and preferably 4 months of age. The sex of mice has relatively little effect on resistance to single doses of radiation. Males tend to be slightly less resistant than females but many exceptions are found. Frequently there is so little difference in survival that data can be pooled for analysis. When mice are given daily exposures to radiation at levels resulting in mean survival times of 20 to 40 days, however, there is a marked sex difference in survival time with the females dying significantly earlier. This difference can be abolished by ovariectomy so it is clear that the sensitivity is related to endocrine function rather than chromosome complement.[23]

As might be expected, environmental variables are of extreme importance in influencing  $LD_{50}$  estimations. Radiation doses in this range result in severely damaged mice whose lives hang precariously in balance and relatively minor deleterious environmental factors can result in a lethal outcome. Diets deficient in essential constituents resulting in malnourished animals produce significantly lowered radiation resistance. The  $LD_{50}$  may differ, however, with changes in standard diets. Storer (unpublished data) was able to show a difference of about 50 rads in the  $LD_{50}$  of mice fed two standard commercially available laboratory mouse foods. No gross evidence of malnutrition was apparent in the more sensitive population but apparently a subclinical deficiency existed. Seasonal variations in sensitivity have also been noted. It was found that survival under daily exposure was shorter in the summer months than through the rest of the year. He attributed the difference to variations in temperature since his animal rooms were not air-conditioned. Presumably a reversal of this effect (higher sensitivity in the winter) might occur in temperature-controlled quarters if recirculation of air with the attendant hazard of airborne bacterial contamination is practiced in colder weather.

Significant "caging effects" have been reported for mice. Exposure of mice to an approximate  $LD_{50}$  dose and calculated the expected number of cages in which 1, 2, ..., 10 mice should die. The observed results deviated significantly from expected with an excess of cages with low mortality and high mortality. The cage environment, then (presumably the bacterial flora of the cage), contributed significantly to the results. Hahn and Howland tested the effects of crowding on radiation resistance in rats. Group-caged rats were found to be more sensitive than singly caged rats. Caution is therefore required in interpreting minor differences in  $LD_{50}$  values as a result of experimental



manipulation unless equal numbers of mice are present in each cage and unless treatments are randomly distributed within cages.[24]

Radiation exposure in the moderate-to-high dose range causes an extreme depression in the immunological competence of mice. For this reason it is obvious that endemic infections in a mouse population may reach epidemic proportions following radiation. Carriers of pathogenic bacteria such as *Salmonella typhimurium* are virtually worthless for radiation sensitivity studies because of the severe infection that may result after irradiation. Even mice carrying such normally nonpathogenic forms as *Pseudomonas aeruginosa* are of limited usefulness since an overwhelming bacteremia may occur and confuse the interpretation of the mortality data. Ideally, radiation studies should be performed with pathogen-free mice (with specific listings of forms considered pathogenic), with mice contaminated only with known and specified bacteria such as *Escherichia coli*, or with germ-free mice. Improvements in methods for raising and maintaining mice may ultimately make animals of these types widely available. Until such time as they come into general use, rigid control and sanitation procedures, which are advisable in any mouse colony, are mandatory in stocks used in radiation experiments.

#### IV. CONCLUSIONS

Basically there are two mechanisms by which ionizing radiation can influence reproductive performance. Transmissible genetic defects (chromosomal derangements or point mutations) may be induced which lead to failure of implantation, early death and resorption of the fetus, or stillbirths. In totally irradiated mice this mechanism is of relatively minor importance compared with the somatic effects on the breeding animals which may markedly reduce the total numbers of viable offspring. The genetic effects and will not be reviewed here. From a consideration of the histopathological effects on the testis and ovary and the relative ease with which sterility can be induced, particularly in females, it is apparent that direct somatic effects may sharply reduce numbers of offspring during the reproductive life of mice. Males are relatively more resistant than females. Moderate to large doses of radiation may produce a period of sterility by the killing of large numbers of spermatogonia which are the stem cells from which later stages in spermatogenesis are derived. This sterility is only temporary at less than lethal doses and most of the tubules regenerate and spermatozoa are again formed. The sperm count may remain slightly lower than normal since not all tubules regenerate, but apparently there normally a great excess of spermatozoa and a modest depression in numbers does not interfere seriously with the ability to inseminate successfully.

Because of the high sensitivity of primitive ova and because ova are not continuously supplied from germinal cells capable of indefinite proliferation, female mice are easily sterilized with even modest doses of radiation. With 100 rads or less the female may become permanently sterile after bearing one or two litters. (These litters early after radiation can occur presumably because the more mature ova are more resistant than the more primitive ones.) With higher doses the incidence of complete sterility is greatly increased. Impaired reproductive performance in the female could also occur if disturbances in the endocrine balance or in the ability to provide a suitable uterine environment were induced. While such changes presumably could be induced by high doses of radiation to localized regions, they are not a factor with total-body radiation because the extreme sensitivity of the mouse ovary is limiting. It might be noted that the corpora lutea and the uterine mucosa are relatively resistant structures.[25]

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