

Morphophysiology of Radiation Induced Lenticular Opacity

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Abstract— The purpose behind the study of radiation induced lenticular opacity was to know the distribution of the lens Glucose and their interaction with environmental radiation coming from the sun. Glucose is the most important free hexose sugar in the lens. It plays an important role in energy metabolism. In the lens the biological energy is necessary for the maintenance of transparency, synthesis and repair process (Kuck, 1965). This study provides information on the potential risk of cataract development due to exposure of Non-ionizing radiation i.e. UV-radiation, Infrared radiation during their occupational activities. The entire study is carried out in two phases. Phase I the normal lenses from mice have been analyzed by the standardized method in the laboratory. The biochemical analysis was carried out. Phase II the observations obtained in the laboratory were correlated with the data of normal and different cataractous lenses of other groups. These data were outcome of the standardized methodology and exposure set up for irradiation in vivo and in vitro. Animals used for these studies were the mice (*Mus musculus*) and the rat (*Rattus norvegicus*) of either sex. The Control animals as well as the Experiment animals were kept in laboratory condition. They were fed with standard animal food and water (Phase I-II). The experiment animals were kept in radiation chambers periodically to see the effect of radiation on their lenses. Each lens from control and experiment strains was subjected to biochemical analysis. The irradiation of the lens by the non-ionizing radiation was carried out by the in vivo and in vitro method.

Keywords— Lenticular opacity, cataractous lens

INTRODUCTION

The normal mice lenses were round and soft with prominent convex posterior pole. It is located between anterior aqueous and posterior vitreous humour. It is a semi-solid, elastic, avascular highly organized cellular organ with smooth, shiny surface. The lens capsule was observed to be thinner at the posterior pole. The lenses show a non-cellular capsule layer consisting of two portions. The capsule appeared homogeneous in section, suggesting that the non-cellular capsule layer uniformly surrounds the lens fibers. The cortical lens fibers lens fibers

PRINCIPLE:

The glucose of the homogenate reacts with alkaline copper tartarate in hot condition and produced cuprous oxide that reacts with arsenomolybdate to give molybdenum blue; the intense blue color is proportional to the concentration of sodium sulphate of reagent is to minimize or prevent the re-oxidation of cuprous oxide by atmospheric oxygen.

OBSERVATION:

Glucose levels in the lens of UV & IR-irradiated cultured rat lenses and their controls at different duration of exposure

were generally flattened and uniformly hexagonal in transverse section and elongated belt like cells. The nuclear fibers were highly compact and closely packed.

MATERIALS AND METHOD:

ESTIMATION OF GLUCOSE

The amount of glucose content in the lens (in vitro and in vivo) of UV-exposed and control animals were estimated using the methods of Nelson (1944) and Somogyi (1945).

are shown in the given Table I. During the early stages of UV-exposure, there was no significant change in the glucose level of the lens whereas in the later stages of UV-exposure, the lens glucose level increased when compared to control considerable increase was found only in the in vitro experiments. The glucose level in control lens was found to be slightly increased with advancing age. The increment was progressive but not significant. The changes in the glucose levels of UV-exposed lenses depend on their metabolic rate and disturbances caused by UV-radiation.

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TABLE. I. LEVEL OF GLUCOSE IN UV-INDUCED CATARACTOUS LENS

PARAMETER	EXPERIMENTAL TIME/AGE (IN WEEKS)					
Glucose ($\mu\text{g}/\text{mg}$ wet wt.)	7/11		15/19		25/29	
	CONTROL	TREATED	CONTROL	TREATED	CONTROL	TREATED
	1.072 ± 0.032 (7)	1.149 ± 0.028 (7), B	1.124 ± 0.021 (7)	1.167 ± 0.011 (7), B	1.137 ± 0.0013 (7)	1.233 ± 0.011 (7), G

(Values are Mean \pm S.E. Numbers in parentheses indicates sample size, p-values: A:<0.2, B: <0.1, D: <0.1, G: <0.001)

TABLE. II. LEVEL OF GLUCOSE IN IR-INDUCED CATARACTOUS LENS

PARAMETER	CONTROL	GROUP I (600 HRS)	GROUP II (800 HRS)	GROUP III (800 HRS HAVING A PROMINENT CORTICAL CATARACT)
Glucose ($\mu\text{g}/\text{mg}$ wet wt.)	1.22 ± 0.062 (6)	1.14 ± 0.027 (6), P<0.3	0.92 ± 0.029 (6), B	0.89 ± 0.026 (6), G

(Values are Mean \pm S.E. Numbers in parentheses indicates sample size, p-values: A:<0.2, B: <0.1, D: <0.1, G: <0.001)

The level of Glucose was reported to be declined in the lenses of experimental animal groups table.II In group I animal lenses a decrease of 6.94% is found to be insignificant statistically while in Group II animal lenses the study revealed a significant decrease ($p<0.01$) of 24.75% while Group III animals show a highly significant ($p<0.01$) decrease of 27.29% when compared with the control.

DISCUSSION:

The results obtained from the investigation shows that in the UV-exposed lenses there was a considerable increase of glucose level in the lens of in vitro and in vivo. Associated with the progression of UV induced lens opacity, considerable change in the maintenance of lens glucose level was obtained.

It has shown that in IR exposed lenses there is an increase in energy demand and a high metabolic activity in the initial stages of exposure, which is depleted with cataract formation. The glucose level has shown a continuous decline throughout the IR exposure period. A steady decline in the glucose level shows that the IR initiates glucose metabolism. The hexose monophosphate shunt is activated whenever there is a demand for NADPH.

SUMMARY:

From the data studied it could be concluded that long-term, low-level radiation exposure has salient effects on the crystalline lens, which enhances cataractogenesis. The biochemical change for the different duration of UV-exposure includes increase in Glucose. When the lenses is irradiated with IR radiation there is an increase in energy demand and a high metabolic activity in the initial stages of exposure, which is depleted with cataract formation. The study suggests that radiation causes a permanent lenticular damage which leads to irreversible lens opacity and support the concept that being in closed cavity lens has no repair mechanism for damage caused by radiation.

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