The Effect of UV Radiation on Nucleotide Excision Repair in Mutated Strains of *Caenorhabditis elegans*

by Zachary Leng and Alexander Hom

Abstract—The prevalence of skin cancer continues to be themost common kind of cancer and has been associated with deficiency in Nucleotide Excision Repair. All groups of C.Elegans were exposed to 15 minutes of UV radiation and the lifespan for all groups was tested and pictures of the phenotypical differences among the groups were taken. The results showed that radiation caused the lifespan of C. Elegans to decrease significantly when compared to non-irradiated groups.

I. INTRODUCTION

Many organisms are exposed to solar ultraviolet (UV) irradiation. Although the vast majority of UV light and UVC rays emitted by the sun are blocked by the earth's ozone layer, penetrating UV light, like UVB rays, can still severely damage DNA and is thought to be a major cause of skin cancer in humans [1]. Although most past experiments used UVC radiation, it was determined that UVB was more applicable and would produce similar results to UVC [2]. Nucleotide excision repair (NER) is responsible for the removal of a wide range of DNA helix-distorting damage. NER is the process of removing strands of DNA that have a cytosine bond or a thymine bond. Bonds are formed when one is exposed to UV radiation. In order to extract these bonds, the body naturally cuts out this damaged part of DNA, and replaces it with a strand of DNA that does not have the thymine or cytosine bond [2]. In humans, NER is essential to prevent DNA damage-induced mutation accumulation and cell death which can lead to cancer and aging.

II. MATERIALS AND METHODS

N2 C. elegans were used as a control. RB864, TG1660, and BJS4 strains with NER deficiency were tested as variable groups. C. elegans were chosen as a model organism due to their genome similarity to humans. All strains used E. Coli as a food source in 10 mL of Nematode Growth Medium Agar(NGM). Chunking and worm picking was used to transfer worms from one plate to another. Age-synchronized, L4 adult stage worms were either exposed to or unexposed to UVB radiation. 10μL of Fluorodeoxyuridine was also added to plates to prevent reproduction while lifespan was being tested. When C. elegans were exposed to UVB radiation they were exposed for 15 minutes and without E. coli to prevent shielding that would reduce the UV effect. Lifespan was then recorded in days after exposure for each strain. In addition, pictures were taken of exposed and unexposed strains of C. elegans to compare differences in size and length. IBM SPSS was used to calculate descriptive statistics.

III. RESULTS

Non-irradiated N2 strains were found to have the greatest lifespan of approximately 15.0 days on average. This was significantly greater than their UV-irradiated counterparts which had an average lifespan of 12.6 days. BJS4 strains were found to have average lifespans of 11.0 days while their irradiated counterparts had lifespans of 9.0 days on average. TG1660 and RB864 strains had average unexposed lifespans of 11.7 and 12.4 days respectively while exposed strains had exposed lifespans of 8.5 and 9.7 days on average. All non-irradiated strains were found to have significantly greater lifespans over their irradiated counterparts. In addition, subtle phenotypical difference in sizes and lengths of C. elegans were noticed when comparing irradiated strains with non-irradiated strains. IBM SPSS was used to calculate descriptive statistics.

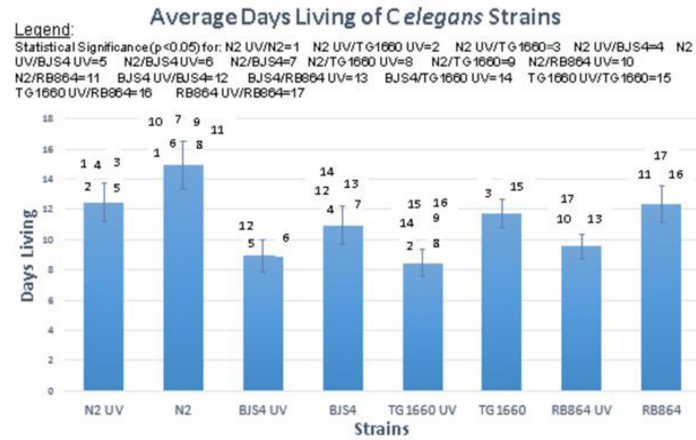

Fig. 1. Shows the results of the lifespan study for each strain of C. elegans. Numbers represent statistical significance found doing a One-Way ANOVA followed by post-hoc Scheffe ($p<0.05$) test.

IV. DISCUSSION

The alternate hypothesis was supported as significant difference were found between all irradiated and nonirradiated strains of C. elegans. Phenotypical differences were also observed between exposed and unexposed strains with irradiated strains being generally smaller than nonirradiated strains. N2 strains had significantly greater lifespans compared to all NER-deficient strains when exposed to or unexposed to Ultraviolet Radiation. This suggests that NER may have an impact in survival after radiation damage. This may be as a result of an organism's inability to repair double bonds that can lead to cancer when they are NER-deficient [3]. Overall, this study suggests NER-deficiency may impair survival after exposure to Ultraviolet Radiation [2]. The results from this experiment will help further information on skin cancer and skin related diseases like Xeroderma Pigmentosum.

V. ACKNOWLEDGEMENTS

Thank you to Alison Huenger, our mentor, for all of her guidance and support in this project.

VI. REFERENCES

[1] Zeman, Gary. "Hps.org." Health Physics Society. N.p., n.d. Web. 28 Sept. 2015. <https://www.hps.org/hpspublications/articles/uv.html>.
[2] Lans, Hannes. "Nucleotide Excision Repair in Caenorhabditis Elegans." N.p., 2011.
[3] Niedernhofer, Laura. "Tissue-specific accelerated aging in nucleotide excision repair deficiency"