RESEARCH
www.commprac.com
ISSN 1462 2815

AWARENESS ABOUT DNA STRAND BREAKS AND CHROMOSOMAL ABERRATIONS AMONG ALLIED HEALTH SCIENCE STUDENTS

Kethan Umakanth [1], Kalpana E [2], Sivakumar Ekambaram [3], Jagadeswaran [4] and Dhanraj Ganapathy [5]*

[1] Intern, Department of Prosthodontics, Saveetha Dental College and Hospitals, Saveetha Institute of Medical and Technical Sciences, Chennai, India.
[2] IV Year, Radiotherapy Technology, Saveetha College of Allied Health Sciences, Saveetha Institute of Medical and Technical Sciences, Chennai, India.
[3] Assistant Professor and RSO, Department of Radiotherapy, Saveetha College of Allied Health Sciences, Saveetha Institute of Medical and Technical Sciences, Chennai, India.
[4] Principal and Professor, Saveetha College of Allied Health Sciences, Saveetha Institute of Medical and Technical Sciences, Chennai, India.
[5] Professor and Head of Department, Department of Prosthodontics, Saveetha Dental College and Hospitals, Saveetha Institute of Medical and Technical Sciences, Chennai, India.
*Corresponding Author Email: dhanraj@saveetha.com

DOI: 10.5281/zenodo.12069740

Abstract

Introduction: DNA is the principal target for biological effects of radiation, including cell killing, mutation and carcinogenesis. Radiation exposure causes DNA strand breaks. It may be a single strand or double strand break. Chromosomal aberrations result if a cell is irradiated early in interphase, before the chromosome material has been duplicated. If cells are exposed to radiation, DSB occurs in chromosomes. **Aim:** This survey was conducted for assessing the Awareness About DNA Strand Breaks and Chromosomal Aberrations Among Allied Health Science Students **Materials and Method:** A cross-section research was conducted with a self-administered questionnaire containing ten questions distributed amongst 100 Allied Health Science students. The questionnaire assessed Awareness about DNA Strand Breaks and Chromosomal Aberrations among Allied Health Science Students. The responses were recorded and analysed. **Results:** 74.5% of the respondents were aware of DNA Strand Breaks. 66.7% were aware of types of DNA Strand Breaks. 44% of the respondents were known that Single Strand Break is a type of DNA Strand Break. 69% of the respondents were aware of Chromosomal Aberrations. 76% were aware about the types of Chromosomal Aberrations. **Conclusion:** There is a moderate awareness amongst Allied Health Science students about DNA Strand Breaks and Chromosomal Aberrations. Enhanced awareness initiatives and educational programmes together with increased importance for curriculum improvements that further promote knowledge and awareness of DNA Strand Breaks and Chromosomal Aberrations should be initiated for further understanding and benefits.

Keywords: DNA, Strand Breaks, Chromosomal Aberrations, Radiation.

INTRODUCTION

Deoxyribonucleic acid (DNA) is a large molecule with a well known double helix structure. It consists of two strands, held together by hydrogen bonds between the bases. The “backbone” of each strands consists of alternating sugar and phosphate groups. The sugar involved is deoxyribose attached to this backbone or four bases, the sequence of which specifies the genetic code. Two of the bases are single ring groups (pyrimidines)- thymine & cytosine. Two of the bases are double ring groups (purines)- adenine & guanine. Adenine pairs with thymine and guanine pairs with cytosine[1].

DNA is the principal target for biological effects of radiation, including cell killing, mutation and carcinogenesis. Radiation exposure causes DNA strand breaks. It may

be single strand or double strand break. If the break is located on one of the strands, it is referred as single strand breaks[2]. In double strand breaks, both strands are broken, the breaks are well separated, repair occurs readily, because the two breaks are handled separately. In Such double strand breaks where breaks are opposite to each other or separated by only a few base pairs, leads to chromosomal aberrations.

Chromosomal aberrations results if a cell is irradiated early in interphase, before the chromosome material has been duplicated. If cells are exposed to radiation, DSB occurs in chromosomes[4]. The broken ends are sticky due to their unpaired bases. These broken ends may join with their original chromosome or failed to rejoin, which leads to aberration or rejoin with other broken ends[5].

The chromosomal aberrations that are lethal to cells are ring, dicentric and anaphase bridge and not lethal to cells are symmetric translocation and small interstitial deletion. The aim is to create awareness about DNA strand breaks and chromosomal aberrations among Allied Health students.

MATERIALS AND METHOD

This cross-sectional research was conducted with a self-administered questionnaire containing ten questions distributed amongst 100 Allied Health science students. The students were randomly selected across various disciplines of Allied Health Sciences. The study setting was designated in the university campus. The survey instrument was a questionnaire pre tested and evaluated for validity and reliability concerns.

The questionnaire included ten questions eliciting the demographic data through open ended responses and multiple choice questions for the other responses. The study was approved by the Institutional Ethical Committee and informed consent was obtained from the participants. The questionnaire was posted on an online platform and the identity of the respondents were kept confidential.

The questionnaire assessed the awareness about DNA strand breaks and chromosomal aberrations in medical applications. The responses were recorded and analyzed. There were no incomplete responses and no dropouts from the study. The final data obtained was organized, tabulated and subjected to statistical analysis.

The salient questions in the study are:

1) Are you aware of DNA strand breaks?
2) Do you know about types of DNA strand breaks?
3) Which of the following are types of DNA strand breaks?
4) Are you aware of chromosomal aberrations?
5) There are two types of chromosomal aberrations, lethal and non lethal?

RESULTS

74.5% of the respondents were aware of DNA Strand Breaks (Figure 1). 66.7% were aware of types of DNA Strand Breaks (Figure 2). 44% of the respondents were known that Single Strand Break is a type of DNA Strand Break (Figure 3). 69% of the respondents were aware of Chromosomal Aberrations (Figure 4). 76% were aware about the types of Chromosomal Aberrations (Figure 5).

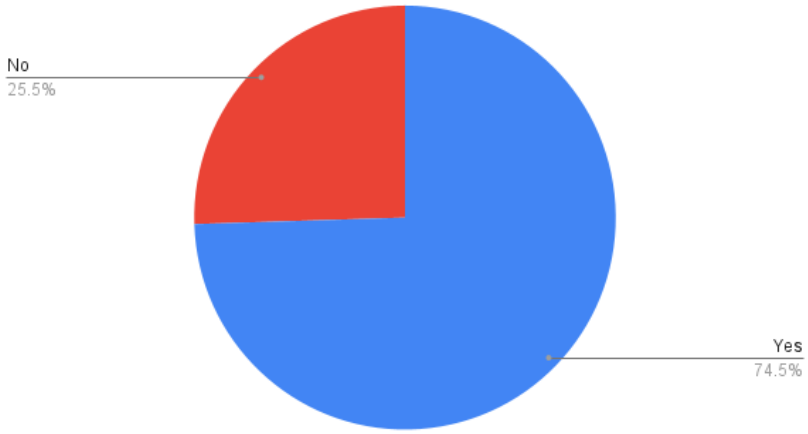

Figure1: Awareness of DNA Strand Breaks

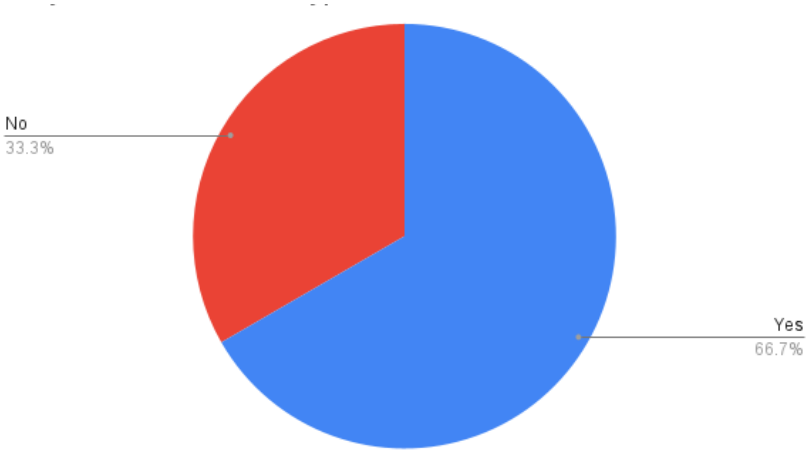

Figure 2: Awareness of types of DNA Strand Breaks

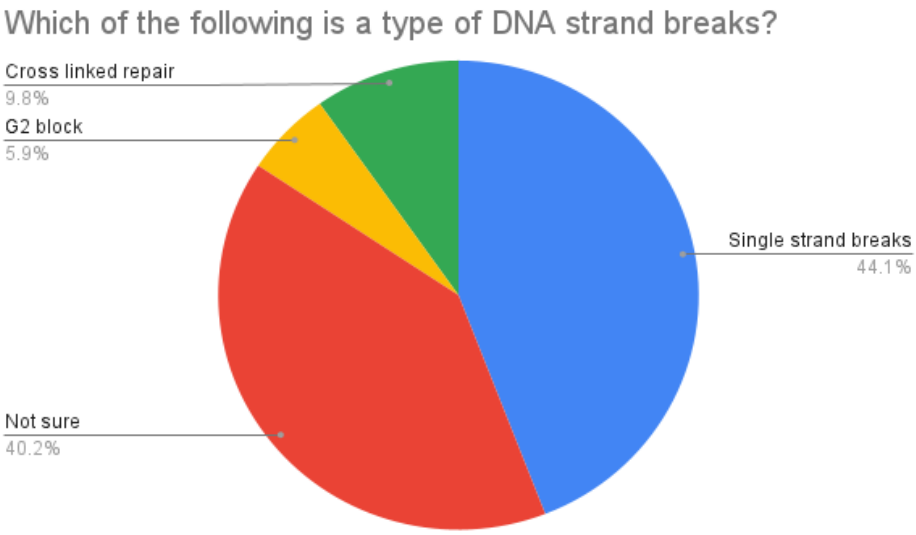

Figure 3

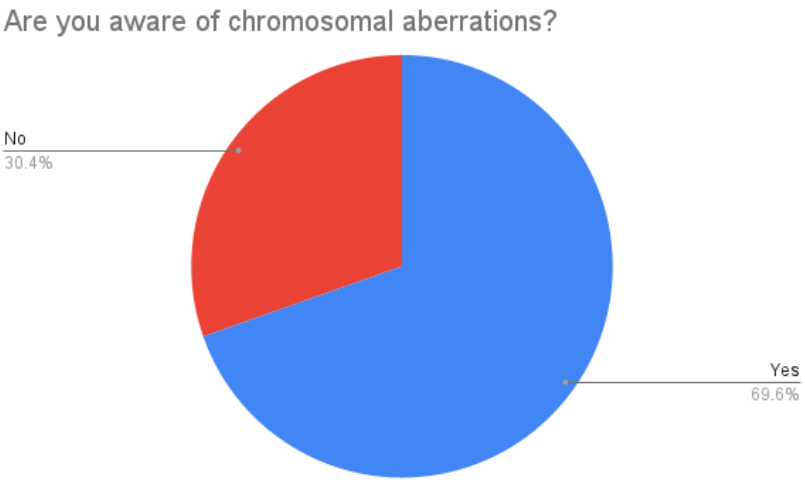

Figure 4

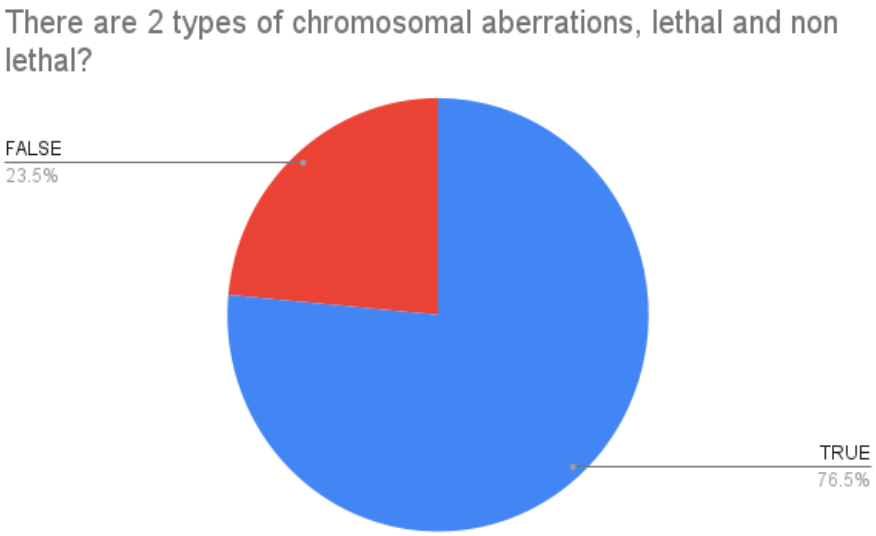

Figure 5

DISCUSSION

DNA damage can occur as a result of endogenous metabolic reactions and replication stress or from exogenous sources like radiation and chemotherapeutics. Damage comes in several different varieties: base lesions, intra- and interstrand cross-links, DNA-protein cross-links, and both single- and double-strand breaks (DSBs). Some types of damage, such as oxidative damage to DNA bases, arise, and are repaired, as often as 105 lesions per cell each day. Much less frequent are DNA DSBs, in which the phosphate backbones of the two complementary DNA strands are broken simultaneously, and these are one of the most cytotoxic forms of lesion[6].

Double-strand breaks (DSBs) in DNA form as a result of exposure to exogenous agents such as radiation and certain chemicals, as well as through endogenous processes, including DNA replication and repair[7]. In addition to these inadvertent occurrences, meiosis I entails the deliberate induction of DSBs, which triggers homologous recombination, thus helping to ensure normal chromosome segregation.

Programmed formation of DSBs also occurs during the development of somatic nuclei in protozoans, mating-type switching in yeast, T-cell receptor formation in T-lymphocytes, and immunoglobulin class switching in B-lymphocytes[8].

DNA double-strand breaks repair can be achieved by different means that are commonly grouped in two broad categories depending on the use or not of a homologous DNA sequence as a template. Repair by non-homologous end joining (NHEJ) involves direct resealing of the two broken ends independently of sequence homology. Although being active throughout the cell cycle, NHEJ is relatively more important during G1[10]. NHEJ represents the simplest and fastest mechanism to heal a DSB, thus it is the most predominant DSB repair pathway within the majority of mammalian cells, even though it may occasionally lead to loss of genetic information.

Chromosomal aberrations are one form of genetic alteration that occur during carcinogenesis. The genetic diseases ataxia-telangiectasia, Fanconi anemia, and Bloom syndrome are characterized by increased levels of spontaneous and induced chromosomal aberrations and increased susceptibility to neoplasia. Chromosomal aberrations are not only markers of genetic damage but also may have a specific role in carcinogenesis. Hence, most, if not all, cancers exhibit chromosomal alterations.[12]

These may be specific to certain cancers, supporting the concept that chromosomal abnormalities are important in the initiation and development of tumor. The study of chromosomal aberrations in peripheral blood lymphocytes, in particular, has been used to gain insight into cancer development and cancer risk in humans[13].

Tumorigenesis is a multistep process, and an increase in chromosomal aberrations or fragility does not imply that cancer will necessarily develop. However, the observation of higher aberration levels in GH-treated patients would be an indication for caution in the administration of this therapy and the need for intensive follow-up of these patients to exclude neoplasia[9]. However, the limited and contradictory data derived to date do not permit one to derive conclusions regarding the genetic effects of GH treatment with any confidence.

CONCLUSION

There is a moderate awareness amongst Allied Health Science students about Dna Strand Breaks And Chromosomal Aberrations. Enhanced awareness initiatives and educational programmes together with increased importance for curriculum improvements that further promote knowledge and awareness of DNA Strand Breaks And Chromosomal Aberrations should be initiated for further understanding and benefits.

References

1) McKinnon PJ, Caldecott KW. DNA strand break repair and human genetic disease. Annu. Rev. Genomics Hum. Genet.. 2007 Sep 22;8:37-55.

2) McKinnon PJ, Caldecott KW. DNA strand break repair and human genetic disease. Annu. Rev. Genomics Hum. Genet.. 2007 Sep 22;8:37-55.

3) Dasika GK, Lin SC, Zhao S, Sung P, Tomkinson A, Lee EY. DNA damage-induced cell cycle checkpoints and DNA strand break repair in development and tumorigenesis. Oncogene. 1999 Dec;18(55):7883-99.

4) O'connor MJ, Martin NM, Smith GC. Targeted cancer therapies based on the inhibition of DNA strand break repair. Oncogene. 2007 Dec;26(56):7816-24.

5) Rulten SL, Caldecott KW. DNA strand break repair and neurodegeneration. DNA repair. 2013 Aug 1;12(8):558-67.

6) Weinfeld M, Mani RS, Abdou I, Aceytuno RD, Glover JM. Tidying up loose ends: the role of polynucleotide kinase/phosphatase in DNA strand break repair. Trends in biochemical sciences. 2011 May 1;36(5):262-71.

7) Kurimasa A, Kumano S, Boubnov NV, Story MD, Tung CS, Peterson SR, Chen DJ. Requirement for the kinase activity of human DNA-dependent protein kinase catalytic subunit in DNA strand break rejoining. Molecular and cellular biology. 1999 May 1;19(5):3877-84.

8) Garm C, Moreno-Villanueva M, Bürkle A, Petersen I, Bohr VA, Christensen K, Stevnsner T. Age and gender effects on DNA strand break repair in peripheral blood mononuclear cells. Aging cell. 2013 Feb;12(1):58-66.

9) Obe G, Pfeiffer P, Savage JR, Johannes C, Goedecke W, Jeppesen P, Natarajan AT, Martınez-López W, Folle GA, Drets ME. Chromosomal aberrations: formation, identification and distribution. Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis. 2002 Jul 25;504(1-2):17-36.

10) Brodeur GM, Sekhon GS, Goldstein MN. Chromosomal aberrations in human neuroblastomas. Cancer. 1977 Nov;40(5):2256-63.

11) Beroukhim R, Getz G, Nghiemphu L, Barretina J, Hsueh T, Linhart D, Vivanco I, Lee JC, Huang JH, Alexander S, Du J. Assessing the significance of chromosomal aberrations in cancer: methodology and application to glioma. Proceedings of the National Academy of Sciences. 2007 Dec 11;104(50):20007-12.

12) Natarajan AT, Obe G. Molecular mechanisms involved in the production of chromosomal aberrations. Chromosoma. 1984 Aug;90(2):120-7.

13) Norppa H, Bonassi S, Hansteen IL, Hagmar L, Strömberg U, Rössner P, Boffetta P, Lindholm C, Gundy S, Lazutka J, Cebulska-Wasilewska A. Chromosomal aberrations and SCEs as biomarkers of cancer risk. Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis. 2006 Aug 30;600(1-2):37-45.