REVIEW OF LITERATURE

• **Dilek Ozmen, 2000 et al.** stated about the oxidative stress which is associated with diabetes mellitus, might play an important role in the initiation and progression of diabetic complications, it has been suggested that free oxygen radical trigger cataract, kind of the degenerative manifestations of diabetes mellitus.

• **Richard D. Wood, 2001 et al.** stated that cellular DNA is faced to continued attack, by reactive species inside cells and environmental agents as well. 130 known human DNA repair genes are described here who are taking part to minimize the Toxic and mutagenic consequences by distinct pathway of repair.

• **Gianluca Tell, 2009 et al.** explained that the DNA base excision repair pathway of all DNA lesions especially uracil, alkylated and abasic sites APE1 is the main apurinic/apyrimidinic endonucleas in eukaryotic cells, playing a central role.

• **Dong Liu, 2011 et al.** explained that 7,8-Dihydro-8-oxoguanine DNA glycosylase (OGG1) is a major DNA glycosylase, it involved in base excision repair (BER) of oxidative DNA damage to nuclear and mitochondrial DNA (mtDNA). In repairing oxidative damage to nuclear DNA under ischemic conditions, OGG1 has a crucial role, so in this way it reducing brain damage and improving functional outcomes.

• **Xiukun Cui, 2012 et al.** stated that the Heat at shock factor protein 4 (HSF4) which is expressed in the ocular lens and play an important role in the lens formation and differentiation. Any mutations in the HSF4 gene lead to congenital and senile cataract development.

• **Oyvind Osnes-Ringen, 2013 et al.** stated that they found that the observed levels of oxidized purines in cataractous lens epithelium support the theory
that if the eye comes in consistent contact with light damage and oxidative stress will cause molecular damage to the human lens epithelium. Factors responsible in pathogenesis of age-related cataract are Ultraviolet radiation (UVR) and oxidative stress, still the Damage commonly associated with UV-B irradiation was relatively low.

- **Rosana Mesa, 2013 et al.** stated that if the eye comes in more exposures to UV-B produced 6-4 PP and CPD photolesions in lens epithelial cells. Cyclobutane pyrimidine dimer (CPD) lesions were particularly prevalent and were repaired slowly if at all. It suggests that somatic mutation in lens epithelial cells may contribute to the development of cortical cataracts.

- **Xin-Xin Chi, 2015 et al.** stated that the association between the genetic polymorphisms of XRCC1 Arg399Gln G>A and XPD Lys751Gln A>C and increased possibility to age-related cataracts was statistically significant. Analysis indicated that the XRCC1 Arg399Gln G>A polymorphism was correlated nicely with the development and progression of age-related cataract in China, India, and Turkey population in the allele model and the dominant model.

- **Chen Wang, 2015 et al.** stated that if the DNA repair enzymes has any polymorphism which may affect their repair efficiency lead to diseases, for example, senile cataract, also analyze the association of single nucleotide polymorphisms in APE1, OGG1 and XRCC1 genes with the risk of age-related cataract in a Chinese population.

- **Batchimeg Norjmaa, 2015 et al.** stated that the Base excision repair (BER) is considered the most important pathway involved in removing DNA damage. BER pathway is initiated by recognition of a DNA glycosylase (OGG1-oxoguanine glycosylase 1). OGG1-O Gglycosylase 1 catalyzes the
cleavage of an N-glycosidic bond, and effectively removing the damaged base. An apurinic or apyrmidinic site (AP site) created by cleavage of DNA backbone by DNA APendonuclease/APE1. DNA polymerase XRCC1 fills in the gap with the correct nucleotide. With the progress of repair, DNA ligase completes the repair process and restores the integrity of the helix by sealing the nick.

- **Xu Zha, 2016 et al.** stated that the DNA damage of lens epithelial cells may be the primary cause of lens opacity. DNA repair efficacy affected by genetic defect, which is associated with ARC. Based on the result of genome-wide association study (GWAS), together with ARC related reports, they screened out five genes (XPD, XRCC1, OGG1, APE1, and WRN) and five corresponding single nucleotide polymorphisms (SNPs) loci lys751gln (A/C), arg399gln (G/A), ser326cys, asp148glu, and rs11574311 (C/T) related to ARC.

- **Xiaohang Wu, 2017 et al.** stated that of OGG1 and MTHFR genes polymorphism are associated with ARC susceptibility and they help to identify populations at high risk for ARC. Out of 144 polymorphisms in 36 genes were reported in the 61 previous genetic association studies. Thereby, three polymorphisms of two genes (8-oxoguanine DNA glycosylase-1[OGG1]; methylenetetrahydrofolate reductase NADPH [MTHFR]) in eight studies were included in the meta-analysis. Regarding the OGG1-rs1052133, the GG (OR = 1.925; 95% CI, 1.181±3.136; \( p = 0.009 \)) and CG (OR = 1.384; 95%CI, 1.171±1.636; \( p<0.001 \)) genotypes indicated higher risk of ARC. For the MTHFR gene, the CC+TT genotype of rs1801133 might be protective (OR, 0.838; 95%CI, 0.710±0.989; \( p = 0.036 \)), whereas the AA+CC genotype of rs1801131 indicated increased risk for the mixed subtype (OR = 1.517; 95%CI, 1.113±2.067; \( p = 0.008 \)).
• **Sbu Su, 2017 et al.** stated that the DNA damage and mal function of DNA repair are contribute to the pathogenesis of ARC. They examined the associations of 18 single nucleotide polymorphisms (SNPs) in four DNA repair genes (BLM, WRN, ERCC6, and OGG1) with ARC in Han Chinese. They also determined the possible functional consequence of the SNPs to DNA damage.

• **Nihal Yigitbasi, 2017 et al.** stated that the Increased oxidative stress in type 2 diabetes cause to the DNA damage and results diabetic complications. Xeroderma pigmentosum complementation group D (XPD) and human oxoguanine glycosylase 1 (hOGG1) are genes involved in the repair of oxidative DNA damage. There was statistically significant difference between patient and control groups in the genotype distribution of XPD Lys751Gln polymorphism (p<0.05). While the Lys/Gln was higher in patients, Lys/Lys genotype was found significantly higher in control group.

• **William Pendergrass, 2018 et al.** stated that As opposed to the clear DNA-free subcapsular and cortical areas of young adult mouse lenses, these areas in cataractous old mouse lenses were found to contain accumulations of nuclei, nuclear fragments, aggregated mitochondria, and amorphous DNA as cortical inclusions. The source of such material was a large expansion of transition nuclei in the bow region and also directs involution of surface lens epithelial cells (LECs) into the underlying cortex.