Abstract

Over the course of an organism's life, its genome is exposed to endogenous and exogenous chemical, physical and biological agents – genotoxins. These genotoxins alter its basic structural components – sugar residues, phosphodiester bonds, and nitrogenous bases.

Organisms have therefore evolved a plethora of different strategies to both repair DNA lesions and maintain genomic stability. These DNA repair pathways are linked with several other cell pathways, including chromatin remodelling, DNA replication, transcription, cell cycle control, apoptosis – programmed cell death (PCD), thereby providing a coordinated cellular response to DNA damage.

Biochemical mechanisms of DNA repair are relatively well understood in yeast and mammals, however, far less so in plants. While these repair mechanisms are evolutionary conserved, significant differences still remain. Therefore, further investigation is required.

This thesis summarises the introduction of a novel plant model – the moss, *Physcomitrella patens* (*Physcomitrella*). As a haploid gametophyte with unique characteristics of high frequency of homologous recombination (HR), and apical growth of filaments, it is an ideal organism to study DNA repair in plants. Previous research on *Physcomitrella* regarding mechanisms of DNA lesion repair induced by radiomimetic Bleomycin, alkylating methyl methanesulfonate (MMS), and by UV irradiation has provided strong evidence of its capability to be one of the best plant models.

The combined DNA repair and induced mutagenesis study using a *Physcomitrella* culture of protonema dividing apical cells displays how the genotoxin-sensitive phenotype is not a consequence of a repair defect to eliminate induced damage. Rather this hypersensitivity is the result of rapid and effective DNA repair, thus allowing for the restoration of DNA structure at the cost of potential sequence changes prone to mutations. Mutations, particularly those occurring in essential genes, are then responsible for the sensitive phenotype.

This concept is well illustrated in the mutants, *pprad50* and *ppmre11,* of the MRN complex with an eliminated, error-free HR pathway and an enhanced, error-prone non-homologous end joining pathway (NHEJ).