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DNA Repair Mechanisms in Algae

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ABSTRACT

DNA repair mechanisms have been a remarkable subject in algae, as in every living organism. Continuous exposure to mutagens has led to the evolution of protection mechanisms in primary producer algae of aquatic ecosystems and various pathways have developed in multiple algae. These pathways are direct and indirect. Undoubtedly, the most studied pathway is photoreactivation and photolyase enzyme. Photoreactivation is a repair mechanism that recycles thymine dimers by occuring UV radiation in both prokaryotic and eukaryotic organisms. Apart from this, there are limited findings in algae regarding O6 methyl guanine repair, another direct pathway. Mismatch repair, one of the indirect pathways, contains homologs of well-defined *Echerichia coli* enzymes in algae. In addition, homologs of enzymes belonging to base excision repair and nucleotide excision repair mechanisms defined in higher eukaryotes have been investigated in various algae, especially in *Chlamydomonas* sp. Recombinational repair is one of the indirect repair types seen in algae. In this study, a general overview of the repair mechanisms found in algae were presented.

Keywords: photoreactivation, O6-methylguanine repair, mismatch, recombinational repair, BER, NER

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Alglerde DNA Tamir Mekanizmaları

Öz: DNA onarım mekanizmaları, her canlı organizmada olduğu gibi alglerde de dikkat çekici bir konu olmuştur. Mutajenlere sürekli maruz kalma, sucul ekosistemlerin birincil üretici alglerinde koruma mekanizmalarının evrimleşmesine yol açmış ve birçok algde çeşitli yollar gelişmiştir. Bu yollar doğrudan ve dolaylıdır. Kuşkusuz en çok çalışılan yol fotoreaktivasyon ve fotoliyaz enzimidir. Fotoreaktivasyon, hem prokaryotik hem de ökaryotik organizmalarda UV radyasyonu oluşturarak timin dimerlerini geri dönüştüren bir onarım mekanizmasıdır. Bunun dışında, alglerde bir diğer doğrudan yol olan O6 metil guanin onarımı ile ilgili sınırlı bulgular vardır. Dolaylı yollardan biri olan yanlış eşleşme onarımı, alglerde iyi tanımlanmış *Echerichia coli* enzimlerinin homologlarını içerir. Ayrıca, yüksek ökaryotlarda tanımlanan baz çıkarma onarımı ve nükleotid çıkarma onarımı mekanizmalarına ait enzimlerin homologları, özellikle *Chlamydomonas* sp.'de olmak üzere çeşitli alglerde araştırılmıştır. Rekombinasyonel onarım, alglerde görülen dolaylı onarım türlerinden biridir. Bu çalışmada, alglerde bulunan onarım mekanizmalarına genel bir bakış sunulmuştur.

Anahtar kelimeler: fotoreaktivasyon, O6-metilguanin onarımı, yanlış eşleşme, rekombinasyon onarımı, BER, NER

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Introduction

DNA repair mechanisms are the responses to damage caused by exogenous and endogenous factors in the genetic material of organisms (Iyama and Wilson, 2013). Cell cycle arrest, gene expression change, stimulation of DNA repair, and programmed cell death may occur depending on the intensity and type of damage (Roos and Kaina, 2013). DNA damage can occur in different ways, such as double and single-strand breaks, insertions and deletions, abasic sites and DNA-protein cross-link formation (Friedberg, 2003). Understanding these types of mutations, which result from spontaneous or environmental DNA damage, is essential to explain the repair mechanisms specific to the type of mutation. DNA repair mechanisms are divided into two as: direct and indirect repair. Direct repair includes photoreactivation and repair with O6-Methylguanine-DNA-Methyltransferase, while indirect repair includes Base Excision Repair (BER), Nucleotide Excision Repair (NER), Double-Strand Break Repair (SSBR) and Mismatch Repair (MMR) recombinational repair (Chatterjee and Walker, 2017; Kurtoğlu and Tekedereli, 2015)

Photoreactivation

Photoreactivation, a frequently studied DNA repair mechanism in algae, is based on the repair of damage by cleavage of cyclobutane pyrimidine dimers (CPDs) by the photolyase enzyme (Figure 1). Van Baalen (1968) studied the survival curve of a blue-green alga, *Agmenellum quadruplicatum*, under UV irradiance (254 mμ) and reported evidence that it exhibited strong photoreactivation at around 430 mμ. Baalen and O'donnell (1972) reported that photoreactivation occurs at 240-300 nm in the alga *Agmenellum quadruplicatum*. Amla (1979) studied the photoreactivation of the alga *Anacystis nidulans* upon UV exposure and showed that black, blue, and white light can photoreactive UV exposure but green, yellow, and red light cannot. After Sancar (1994) discovered that the enzyme photolyase recycles thymine dimers induced by UV light, this enzyme was also included in photoreactivation studies in algae. Asimgil and Kavaklı (2012) characterized five genes in the photolyase/cryptochrome family from the red alga *Cyanidioschyzon merolae*. Phylogenetic analysis showed that one gene (6-4) was close to photolyase, three of them were close to cryptochrome-dash (CRY-DASH), and one gene was an independent clade. They also isolated the first known (6-4) photolyase from the most primitive photosynthetic organism, *C. merolae*. Fortunato et al. (2015) revealed the cryptochrome/photolyase family in photosynthetic organisms. They reported that this family consists of photolyases, blue light-activated enzymes that repair ultraviolet light-induced DNA damage, and cryptochromes, known for their photoreceptor functions in terrestrial plants. Li et al. 2015 expressed the *Chlamydomonas* sp. ICE-L photolyase gene for the first time in *Echerichia coli* and found the PHR2 gene plays a role in vitro photoreactivation. Hull et al. (2017) quantified UV-induced DNA lesions in the alga *Tetraselmis suecica* by enzyme-linked immunosorbent assay to molecularly assess DNA damage and repair kinetics. They found that most DNA repair occurred within 6 h and was complete within 24 h. Photoreactivation of DNA damage indicates that the enzymatic DNA repair kinetics are not affected by culture conditions in *T. suecica*. König et al. (2017) studied cryptochrome-related photolyases in diatoms. Genome sequences of all photolyases were revealed in diatoms. They reported that cryptochromes CPF1 and CryP have 6-4 photolyase activity. Kottke et al. (2017) studied the functional analysis of cryptochrome photoreceptors from green algae *Chlamydomonas reinhardtii* (plant and animal-like cryptochromes) and *Ostreococcus tauri* (CPF1) in terms of their biological significance and spectroscopic properties. Plant cryptochrome and CPF1 function as UVA/blue light receptors. An et al. (2018) studied the presence of ICE-L (6-4) photolyase enzyme in *Chlamydomonas* sp. for the first time and found that there was 6-4PP repair activity in the presence of UV-B. Pescheck (2019) studied photoreactivation in two co-occurring green macroalgae, *Cladophora* sp. and *Ulva intestinalis*, in the Baltic Sea. He found that due to regular exposure to high sunlight in their habitats, both species require resistance mechanisms to protect themselves against ultraviolet-B (UV-B)-induced DNA damage. Wang et al., (2022) observed the physiological and transcriptome level responses of desert green algae *Chlorella* sp. to high-dose UV-B radiation to study the tolerance mechanism, and found that DNA damage and chlorophyll and soluble protein contents did not undergo significant changes at the early irradiation stage. Haney et al. (2022) manipulated putative photolyase-encoding genes in *Synechococcus* sp. strain RS9916 and discovered that each gene contributed to the overall capacity of this organism to survive UVR. Additionally, each conferred increased survival after UVR exposure when transformed into *Escherichia coli* lacking photolyase and the SOS response. Wu et al. (2023) found six different CPF members in the genome of the alga *Saccharina japonica*.

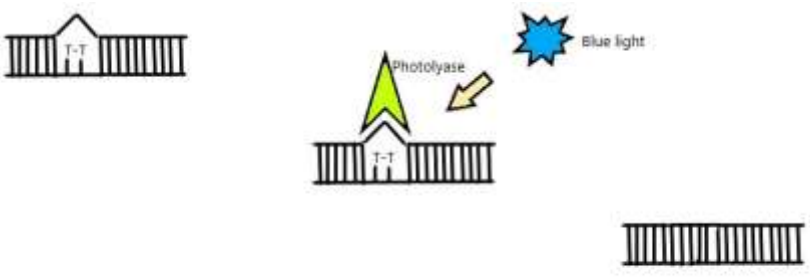

Figure 1. Photoreactivation in algae with photolyase enzyme

O6-Methylguanine repair

The O6-Methylguanine repair (O6-MeG) is created by methylation agents in the cell DNA. If it is not repaired, thymine is matched as a complementary base against O6-MeG at the end of the first replication of the cell. O6-methylguanine (O6-MeG) partially blocks the elongation function of RNA polymerase II during transcription. O6-methylguanine DNA-methyltransferase (MGMT) (Figure 2) repaired the mutation by adding the methyl group to own sistein residue. Thus, the mutagenic effect of alkylation is eliminated (Schendel and Robins, 1978).

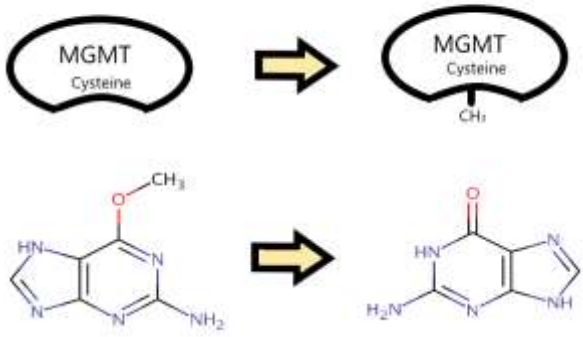

Figure 2. O6- methylguanine repair with O6- methylguainine methyl transferase

Frost and Small (1987) failed to demonstrate the presence of methyl transferase for O6-MeG in different protocols without cell extracts from the alga *Chlamydomonas reinhardtii*. They concluded that it lacks the O6-methylguanine DNA-methyltransferase enzyme and repair. Tillich et al. (2012) published a study about the lethality and rate of non-lethal point mutations for methyl methanesulphonate on the model cyanobacteria *Synechocystis* sp. PCC6803. It was found that DNA lesions could not be repaired in this algae because MGMT expression remained low in promoted applications at doses above 0.1 v%. Above this concentration, increased alkylation suppresses MGMT activity.

Base exicision repair (BER) mechanism

BER mechanisms which are one of the critical DNA repair processes, repair small DNA modifications caused by alkylation, deamination, oxidation and DNA replication errors (Krokan and Bjørås, 2013). It is based on the principle that modified bases that are damaged in the structure of DNA for various reasons are removed from where they are attached to the sugar unit and the correct base is added in its place (Fromme and Verdine, 2004). The BER mechanism consists of five stages: removal of the damaged DNA base, cutting of the abasic region, cleaning of DNA ends, placement of the correct nucleotide into the repair gap, and ligation of the remaining nick in the DNA backbone. The formation of the abasic site is carried out by DNA glycosylase; the gap formed after the restriction endonucleases cut the apurinic site is filled by a DNA polymerase. The polymeric chains are connected to each other by DNA ligase (Figure 3).

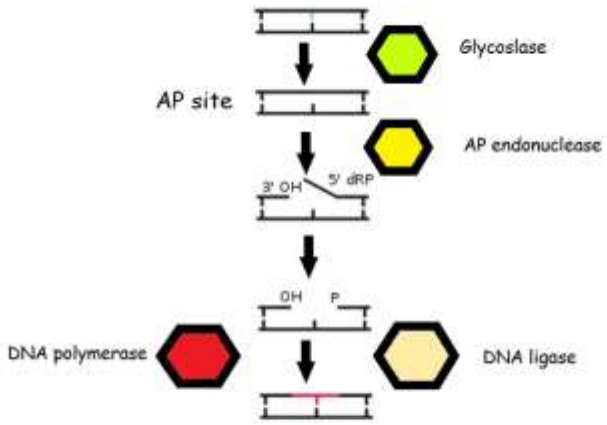

Figure 3. Base exicision repair mechanism

There are two separate sub-pathways in BER. These are short-patch BER (SP-BER) and long-patch BER (LP-BER). While a single nucleotide change occurs in the short pathway, 2-8 nucleotides are changed in the long pathway (Krokan and Bjørås, 2013). In the SP-BER subpathway, DNA polymerase β (Pol β) filled gaps by using dRPase activity, which releases the blocking 5'-dRP end and allows DNA ligation. (Srivastava et al., 1998). The LP BER subpathway involves displacement of the strand containing the 5'-dRP terminus during DNA synthesis and can be carried out by replicative DNA polymerases δ or ε, which generate a 'flap' structure that is cut by the 5'-flap endonuclease FEN1 before the ligation (Pascucci et al., 1999).

Morales-Ruiz (2018) detected base excision repair in *Chlamydomonas reinhardtii* cell extracts. Only DNA polymerase β is missing among the enzymes belonging to the BER mechanism. Homologs of glycosylase genes involved have been revealed in DNA repair of *C. renhardtii*. *C. reinhardtii* can remove mismatched uracil bases against adenine base during replication. This suggests the presence of uracil DNA glycosylase enzyme in this alga. Among the enzymes belonging to the BER mechanism, only DNA polymerase β is missing. Polymerase λ can perform the function of polymerase β. This enzyme is the only member of the X family found in higher plants. Polymerase λ has the activities of terminal deoxyribonucleotide transferase and deoxyribophosphate lyase enzymes (Morales-Ruiz 2018).

Nucleotide excision repair (NER)

Nucleotide excision repair (NER) is used to rehabilitate DNA damages that are large enough to prevent DNA replication and translation (Fışkın and Özlü, 2008, Karentz, 2015). NER mechanism repair some mutations. UV-induced pyrimidine dimers that disrupt the normal helical structure of double-stranded DNA or damage caused by DNA adducts, which are mostly formed by mutagenic and chemotherapeutic agents (Kurtoğlu and Tekedereli, 2015; Karentz, 2015). The operation of the NER mechanism begins with the recognition of the damage with Transcriptional factor IIH (TFIIH), XPC or CSA and CSB proteins. The protein complex binds (XPD, XPE, XPA, and RPA) to the damaged area. The damaged strand is cut (incision) from both sides of the lesion (XPG and XPF/ERCC1), leaving a fragment of ~24-32 nucleotides in length. The oligonucleotide containing the damage is removed (degradation). The gap formed on the DNA helix is filled by DNA polymerase (polymerization). Ligation occurs with DNA ligase I or DNA ligase IIIa (Compe and Egly, 2016; Costa et al., 2003; Kumar et al., 2020; Karentz, 2015). Upregulation of tfiih2, ccnh (a subunit of CDK activator Kinase in the TFIIH2 complex), rfc2_4, rfc3_5, pold2 and pole2 enriched in the NER pathway likely suggests an increase in replication-coupled DNA repairs treated with *R. subcapitata* (Mo et al., 2022).

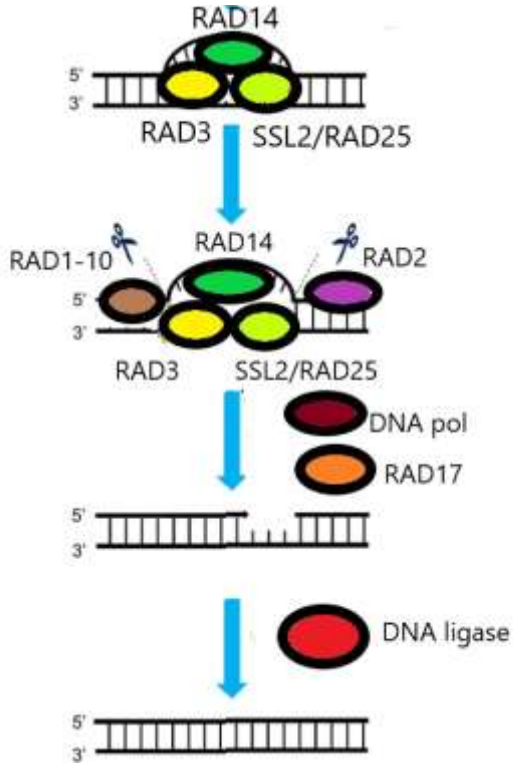

Figure 4. Nucleotide exicision repair mechanism

NER includes many enzymes that repair DNA damage induced by significant double-stranded mutations. Only the excision repair enzyme (REX1) has been described in detail in *Chlamydomonas* (Strains mutant for REX1) are unable to excise cyclobutane pyrimidine dimers and are sensitive to the alkylating agent MMS. *Chlamydomonas* contains the RAD14 gene, which is a homolog of XPA, as well as RPA. The 5' lesions include RAD1-RAD10 (XPF-ERCC-1) and the 3' lesions include RAD (2) (XPG) endonucleases RAD2 (XPG). *Chlamydomonas reinhardtii* contains homologous components of the basal transcription factor TFIIH, the 5' 3' DNA helicase RAD3 (XPD), and the 3' DNA helicase SSL2/RAD25 (XPB) genes. XPA-RPA components that open the closed DNA complex have also been identified (Vlček et al., 2008) (Figure 4).

Four insertional mutants sensitive to genotoxic insults were isolated to identify genes deficient in DDR/DNA repair in *Chlamydomonas reinhardtii*. The four mutants were found to have deletions, including known DNA repair factors. DNA Pol zeta, DNA Pol theta, SAE2/COM1 and ERCC1, and two adjacent genes encoding RAD17 were found. (Plecenikova et al., 2014).

Mismatch repair

DNA mismatch repair (MMR) is a highly conserved biological pathway that plays an vital role in maintaining genomic stability. MMR primarily corrects base-base mismatches and insertion/deletion mutations during DNA replication and recombination. MMR also suppresses homologous recombination and has recently been shown to play a role in DNA damage signaling in eukaryotic cells. *Escherichia coli* MutS and MutL and their eukaryotic homologs MutSα and MutLα, respectively, are important players in MMR-associated genome maintenance. Many other protein components involved in various DNA metabolic pathways, such as PCNA and RPA, are also required for MMR (Putnam, 2016) (Figure 5).

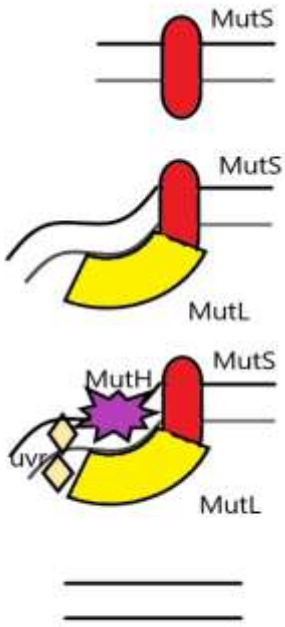

Figure 5. Mismatch repair mechanism

Mismatch repair has been described in both unicellular and multicellular eukaryotes. Eukaryotes generally carry homologs of *E. coli* mismatch repair genes. There are three essential genes in *E. coli* bacteria: mutS+, mutL+, and mutH+. MutS binds proteins to the mismatched region. MutL and MutH recognize unmethylated GATC sequences in newly synthesized DNA strands close to the mismatched region. MutH protein creates a gap in the GATC region of the methylated DNA strand. The mismatch is removed by exonucleases. The gap is repaired by DNA polymerase III and ligase. Although MutS and MutL proteins are highly conserved, MutH is found only in gram-negative bacteria. *C. reinhardtii* contains the MSH and MLH gene groups being analogs of *Echerichia coli* mismatch repair enzmye. According to mutational analyses in *C. reinhardtii* algae, uvs13 and uvs14 enzymes are involved in the mismatch repair mechanism (Vlček et al. 2008). In addition, different gene groups undertake the mismatch repair enzyme mechanism in different algae. DNA repair synthesis genes activate with 100

µg L^{-1} TCS treatment and indicates up-regulation of pms2, rfc2_4, rfc3_5 and pold2 in *Raphidiocelis subcapitata*.

Recombinational repair

The first stage of HR begins with the recognition of double-stranded breaks by the MRN complex. The MRN complex consists of Mre11, Rad50 and Nbs1 proteins. This complex acts as a break sensor. First, MRN binds to the DNA around the lesion and then cuts the DNA around the break in the 5'-3' direction. This stage acts as a signal to recruit damage recognition proteins. This cutting is stimulated as a result of the interaction between the MRN complex and CtIP in the early stage of HR. Mre11 and Exo1 cut the single-stranded DNA to provide stretched longer strand. RPA (Replication protein A), a heteromeric complex that binds single-stranded DNA, is maintained at the site of the l esion by BRCA1. The binding of RPA stabilizes the single-stranded DNA and protects it from nucleases. RPA is removed from DNA by the recombinase Rad51, which is loaded onto DNA by BRCA2. Rad51 forms a nucleoprotein filament along the single-stranded DNA, and this filament has a function that allows strand invasion of sister chromatids. The single-stranded DNA, coated by the Rad51 nucleoprotein filament, enables strand invasion of the intact homologous DNA region. Following exchange and invasion, the DNA is extended by a polymerase (usually polδ) and ligation completes the process (Figure 6), (Krejci et al., 2012).

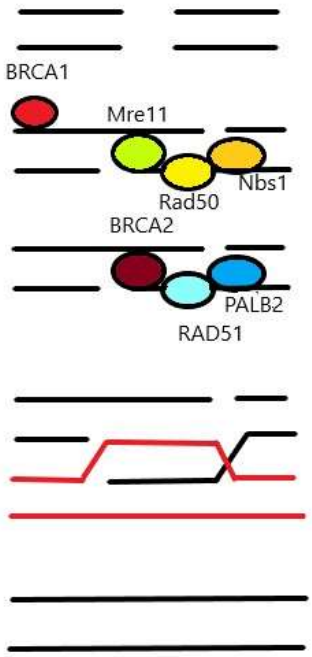

Figure 6. Homologous recombination repair mechanism

Homologous recombination events were investigated using methods for gene replacement and gene disruption on the arylsulfatase gene in the *Volvox* sp. species. For this purpose, transformants that continuously express arylsulfatase were obtained and the presence of homologous recombination was shown in this mutant. In addition, a loss of function was created with a G → A nucleotide change in a single region of the 5' region on the nuclear nitrate reductase gene (nitA) and was selected as the target region for gene replacement. According to the results obtained in these studies, it was concluded that the *Volvox* sp. organism performs strong homologous recombination (Hallmann and Rappel, 1999).

Killian et al., (2011) concluded that *Nannochloropsis* sp. is a haploid alga suitable for targeted gene manipulation by HR. The addition of flanking DNA sequences to the NT7 selectable marker cassette allowed to insert into the nuclear genome of *Nannochloropsis* sp. by HR, thus demonstrating the generation of effective HR by knocking out NR and NiR genes.

Chlamydomonas reinharditti nit1-305 strain was generated using 5' and 3' deletion derivatives and transformations in the nit1 gene. After DNA introduction into the cell, intermolecular recombination between p5' delta and p3' delta occurred at a high frequency to restore a functional

nit1 gene, indicating the existence of a homologous recombination mechanism in mitotic cells (Sodeinde and Kindle, 1993).

Rajaram et al. (2020) used the algae Anabaena/Nostoc sp. strain PCC7120 as a model organism and reassessed the recR and ndk proteins. This study identified protein-protein interactions in the DNA repair mechanism from Anabaena PCC7120 and established possible double-strand break (DSB) repair pathways.

Nonhomologous end joining (NHEJ) rejoins can be occurs in both dividing and non-dividing cells, regardless of the cell cycle. The most active phase is the G1 phase. In NHEJ, double-stranded broken ends are modified and connected to each other. With this repair system, DNA repair is carried out without the need for an undamaged DNA template, with a few nucleotide losses (Bee et al., 2013). Ku 70-Ku 80 (DNA-dependent protein kinase catalytic subunit) complexes
bind to DNA broken ends. DNA-dependent protein kinase is activated, allowing other proteins to come to the damage site. The formation of these protein complexes allows the DNA ligase IV-XRCC4 complex to ligate the broken ends (Sonoda et al., 2006) (Figure 7).

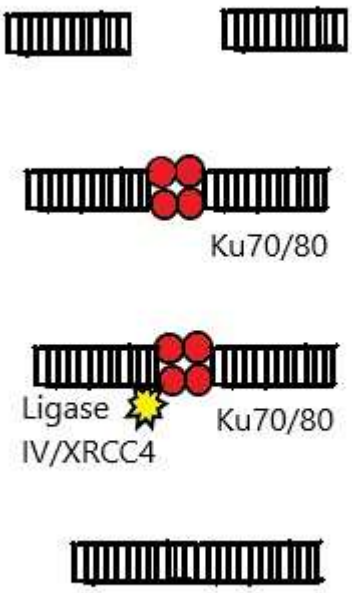

Figure 7. Nonhomologous recombination repair mechanism

Chlamydomonas reinhardtii, the wild strain used for recombinant protein expression, has a nuclear homologous recombination (HR) pathway that is inadequate for homology-mediated editing. Nonhomologous end joining (NHEJ) rejoins the ends of double-strand breaks that cause indel mutations within the target gene, while HR interact and exchange information between two similar sequences and is therefore required to insert foreign DNA into a specific sequence in the genome. Since HR rarely occurs in *C. reinhardtii*, NHEJ-mediated gene editing has been much more successful. (Sproles et al., 2022).

It has been established a protocol for directed ribonucleoprotein (RNP)-mediated genome engineering of DNA constructs in *Nannochloropsis oceanica*, demonstrating that improved RNP-targeted nonhomologous end joining (NHEJ) gene editing is possible. Poveda-Huertes et al., (2023) sequenced the HLR1 locus of 4 positive clones to determine whether HygR50bp integrates via HR or NHEJ in *Nannochloropsis oceanica*. Sequencing results supported that the cassette was inserted by NHEJ events in all four clones.

Discussion

DNA repair in algal species involves several main pathways such as photoreactivation (direct repair by photolyases), nucleotide excision repair, base excision repair, and homologous recombination. DNA repair mechanisms in algae include very different pathways and not all homologous enzymes found in the pathways have been identified yet. The best-explained pathway is the photoreactivation pathway. Although long patch BER is found in *Chlamydomonas*, there is no study on short patch in the literature. The presence of uracil DNA glycosylase and DNA polymerase λ has been proven in *Chlamydomonas* sp. The closest pathway to the mismatch repair pathway in *Chlamydomonas* sp. is

found in *E. coli*. NHEJ-related proteins are found in *Chlamydomonas*. The amount of DSB repaired by NHEJ in higher eukaryotes is higher than HR. Determination of DNA repair mechanisms in algae may provide many biotechnological benefits for other algae and higher plants.

Photoreactivation was studied in various cyanobacteria and algae species in literature. The model organism for DNA repair mechanisms in algae was frequently chosen as *Chlamydomonas reinhardtii* algae. Microalgae such as *Chlamydomonas reinhardtii* have been model organisms for studying repair mechanisms. However, macroalgal species exhibit a unique and less studied diversity in DNA repair processes, likely related to their diverse environmental adaptations. Therefore, studies on algal DNA damage and repair are limited about industrially important algae (Pescheck, 2019; Wang et al., 2022), and these pathways need to be further explored. This approach will provide a broader and more practical perspective relevant to commercial cultivation and industrial applications by better addressing repair mechanisms in species important for algal biotechnology.

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