Narrative Review Article

Polyethylene Terephthalate Induced Oxidative Stress in *Chlamydomonas reinhardtii*: Implications for Intracellular Response Pathways and Ecosystem Health

Tianhui Wang

Wellington College International Shanghai, 1500 Yaolong Road, Pudong, Shanghai, 200120, China

ABSTRACT

Polyethylene terephthalate (PET) is a cheap and versatile plastic used primarily in textiles. We dispose of it as solid waste and in wastewater. It degrades into microplastics and decomposes into small molecules. This review explores recent studies on the impact on green algae of PET microplastic pollution in freshwater and soil. Using the example of the unicellular alga *Chlamydomonas reinhardtii*, a model organism for photosynthetic eukaryotes. The analysis explores mechanisms and outcomes of reactive-oxygen-species (ROS) generation, lipid peroxidation, and disrupted electron transport leading to impaired photosynthesis. Although most prior studies have targeted PET additives or dyes, evidence increasingly suggests that the PET polymer and its monomeric fragment, terephthalic acid, are not inert but ecotoxic. Proposed pathways link PET photodegradation and metal-chelation chemistry to benzoquinone formation and chronic cellular oxidative stress. Yet existing data remain fragmented: few studies address decomposition kinetics, ion binding, or ecosystemic feedback in complex natural conditions. The article concludes that advancing quantitative, system-level models of PET impact—and implementing stricter controls on PET dispersal through wastewater and biosolids—is essential to mitigate growing biospheric and economic risk.

Keywords: Microalgae; Polyethylene terephthalate; Terephthalic acid; Microplastics; Pollution; Oxidative Stress

INTRODUCTION

Regulation of plastic waste in most countries is focused on macroplastics, particles larger than 5mm. In the US, the Microbead-Free Waters Act of 2015 has banned the addition of microplastics (less than 5mm) to cosmetics (1), and the European Union has introduced widespread controls on the use of microplastics in products (2). New EU regulations in 2025 set standards and monitoring for both industrial and urban wastewater: by 2028, polluters will be required to pay microplastics removal costs (3). Particles smaller than 1mm are termed nanoplastics and remain virtually unregulated worldwide (4).

Due to their minuscule size, microplastics have the potential to disrupt the physiology of all organisms through mechanisms such as biosynthesis disruption, DNA damage, and oxidative stress (5, 6). Research has focused on microplastics' disruption of animal physiology, additionally, finding neurological

Corresponding author: Tianhui Wang, E-mail: zahara@greener.org.

Accepted October 28, 2025
https://doi.org/10.70251/HYJR2348.36192207

degeneration, endocrine disruption, and immune system suppression (7).

The third largest non-industrial source of microplastics, after paints and tires, is textiles and geotextiles. An estimated 60% of all textile fiber globally is polyester, and of this 60% is polyethylene terephthalate (PET) (8). Annual production of PET has increased by an average of 6% over the past decade, and this growth is expected to continue (9). PET is not only representative of microplastics in general but is becoming the most abundant microplastic on account of its low cost, superior fiber properties, and short usage lifespan. As PET is so inert in normal, it has been assumed to be safe. However, research has emerged that PET microplastics are detrimental to ecosystems, and some of the mechanisms and pathways have been explored; significant gaps remain in the research, which prevents any meaningful modelling.

PET MICROPLASTICS IN THE ENVIRONMENT

The PET cycle

As we wear and wash synthetic clothing, these plastic fibers break down, through abrasion and degradation, into microplastics. Rapidly increasing quantities of PET fragments enter aquatic ecosystems through these three vectors: wastewater effluent, landfills, and biosolids land application (10, 11). Domestic washing machines emit approximately 68 mg of microplastics per cubic meter as gray water into sewage systems (12). Wastewater treatment facilities are not designed to remove microplastics: studies show primary treatment entraps 46-82%. A meta-study of 54 plants found that after all five treatment stages, only 88% of particles were removed, with a persistence of short microfibers in post-treatment water (9, 13, 14, 15). 99% removal is achievable with the latest capture and disposal technologies, but adoption requires regulatory standards. The filtrate from wastewater treatment, called gray sludge, is commonly either sent to landfill sites or applied to farmland from where soluble leachates can percolate into groundwater, and particles can escape directly into rivers via surface run-off (16, 17).

In the US, 60% of gray sludge is applied as agricultural fertilizer and is largely unregulated. Maine banned the practice in 2022 because of PFAs, and there is an ongoing campaign to establish national controls (18). Research has found that soil treated persistently with microplastic-containing wastewater or sludge experiences degradation and ultimately declining crop yields. (19, 20, 21) Alarmingly, 25 years after a single biosolids land application, a longitudinal study found a negligible decrease in soil microplastic count, only a reduction in particle size (22). This fact suggests that even banning sludge cannot remediate the harm already done and the prospect of declining agricultural productivity (23).

A bellwether species: *Chlamydomonas reinhardtii*

Although green algae differ in some regulatory mechanisms and metabolic flux patterns, they share the same fundamental photosynthetic pathways with higher plants, including the Calvin-Benson cycle and electron transport systems (24), and the speed of unicellular microalgal propagation lends them to *in vitro* study. One well-researched species is *Chlamydomonas reinhardtii* – the focus of this review. It is highly photosynthetic, found in lakes and in soil, and therefore offers insight into the mechanistic and physiological effects of both aquatic and agricultural contamination. Its genome is fully sequenced, with a rich library of mutants, so variations in biochemical pathways can easily be identified. As a consequence, sufficient studies have been conducted on the influence of PET on *C. reinhardtii* to verify that its metabolic impact is significant and to identify a range of mechanisms. It is to these that we now turn.

PET exposure impacts *C. reinhardtii* through four main modes: physically, biochemically, osmotically, and through oxidative stress. Oxidative stress has dominated research, partly because imaging techniques are well developed, and partly because reactive oxygen species (ROS) pathways in algae bear similarities to higher eukaryotes, including humans. Even from these studies, the exact causation of ROS by PET remains hypothetical and unquantified. First, therefore, we will turn to physical, biochemical, and osmotic modes of impact.

MECHANISMS OF PET IMPACT ON C. REINHARDTII

Physical impact of PET on *C. reinhardtii*

Microplastics form a physical barrier to photosynthesis, blocking or scattering light (P1). In *C. reinhardtii*, photosynthesis inhibition correlates with plastic microparticle concentration, which might suggest shading (P2) as a mechanism, but it is inversely proportional to particle size, indicating surface area as the causal factor: shading is not the only explanation

and may not even be the main mode (26). Several studies have found that PET microplastics caused damage to the cell walls of microalgae (P3), including *C. reinhardtii* (P3) (27, 28, 29).

Microplastics often accumulate a biofilm (P4) comprising leachates and excreted algal proteins and polysaccharides (30). This film can adhere to algal cell walls, potentially impeding movement and osmosis (29). Higher plant mycorrhiza are dependent on helper bacteria in biofilms on soil particles; these bacteria seem to be particularly sensitive to microplastics such as PET, and their decline influences plant nutrition both physically and chemically (31). Despite their economic importance, there is a major gap in research on modelling biofilms in mycorrhizal ecosystems. Symbiotic relationships with soil bacteria have been demonstrated for *C. reinhardtii* (32, 33), suggesting its suitability for *in vitro* studies.

Biochemical impact of PET on *C. reinhardtii*

PET fiber, like most plastics, contains additives and dyes that can also be less than benign. Many of these are common to or even originate from other plastics and need not be discussed here, such as bisphenol-A and phthalates, and inorganic fillers and pigments (34). Likewise, chain extenders (e.g., pyromellitic dianhydride, PMDA), toughening agents (e.g., styrene-ethylene-butylene-styrene terpolymer functionalized with maleic anhydride, SEBS-g-MA), and impact modifiers (e.g., ethylene-ethyl acrylate-glycidyl methacrylate terpolymer (E-EA-GMA), ethylene-ethyl-acrylate (EEA)) are widely used in PET fiber production (35). Neither are these unique to PET nor have their bioactivity or ecotoxicology been sufficiently studied (36). Much PET fiber includes recycled PET packaging material containing the additive Anthranilamide (2-aminobenzamide) (37) which is a hazard 4 oral toxin (38). This molecule has been found to inhibit ADP-ribosylation and, although unstudied in algae or plants, it is therefore unlikely to be benign to any eukaryotic lifeform (39, 40, 41, 42). However, PET itself is considered chemically inert.

This consensus is because, at ambient temperature and without a combination of sunlight and water, PET depolymerizes at a negligible rate (43). UV B, however, excites carbonyl groups and cleaves the ester bonds that bind the monomer units (44). The resulting photooxidation cascade is Bis(2-hydroxyethyl) terephthalate (BHET), then Mono(2-hydroxyethyl) terephthalate (MHET), and the end-products are PET's primary monomers, terephthalic acid (TPA) and ethylene glycol, plus carbon dioxide and oligomeric esters: all these moecules are presumed to biologically inert and readily metabolized by certain bacteria (45).

ROS, however, hydroxylate with TPA to initially form 2-hydroxyterephthalic acid (H2BDC-OH), and then 2,3-dihydroxyterephthalic acid (2,3-DHTPA). These both fluoresce detectably and are used as ROS diagnostic reagents (46). Both acids have a pH of 2 to 4 and are registered as class 4 acute oral toxicants and irritants to humans (47). Although their phytotoxicity has not been studied, both acids chelate cations (O2) and potentially disrupt phospholipid membranes, mechanisms deserving investigation. Theoretically, Zn^{2+}, Cu^{2+}, or Fe^{3+} chelated 2,3-DHTPA could self-catalyse (in the Fenton cycle) to generate ROS or to produce the potentially cytotoxic 2,3-benzoquinone-1,4-dicarboxylic acid (48).

Limited studies, some unpublished, have suggested that TPA itself, which has no toxicological registration, is not benign to animals, chelating calcium, raising the levels of two types of protein we shall introduce below, MDA and SODs, and causing hyperplasia and tumors in rats (49, 50, 51, 52). TPA's material safety was originally asserted on the basis of a lack of published experimental data and now flies in the face of the facts (53).

As PET microplastics weather, surface area increases markedly through roughening, fibrillation, fragmentation, and pitting: simulated marine weathering of polyethylene and polypropylene found a tenfold increase of surface area over 12 months (54) – and similar morphological changes in PET have been identified (55); however a formulaic relationship between surface area and aging remains to be established for PET microfibers.

Osmotic impact of PET on *C. reinhardtii*

Primary microplastics' small size and large surface area to volume ratio enable them to readily absorb ions and carry significant surface charges (56), to which unicellular organisms are particularly sensitive. Xia, Yu, *et al*. (57) found that water–solid contact electrification, a charge transfer phenomenon, spontaneously generates abiotic reactive oxygen species (ROS) even in pure water. Studies show that surface charges selectively chelate high-charge-density cations and increase ionic interactions with functional groups at the cell wall. ROS and cation absorption is increased, at the expense of anion absorption (O1). Consequences include growth inhibition and cellular damage (58, 59, 60).

Having identified PET biodegradation and electrification as plausible mechanisms of ROS generation, with particle surface area as a key catalyst, we turn to oxidative stress. (Figure 1)

OXIDATIVE STRESS PATHWAYS AND RESPONSES

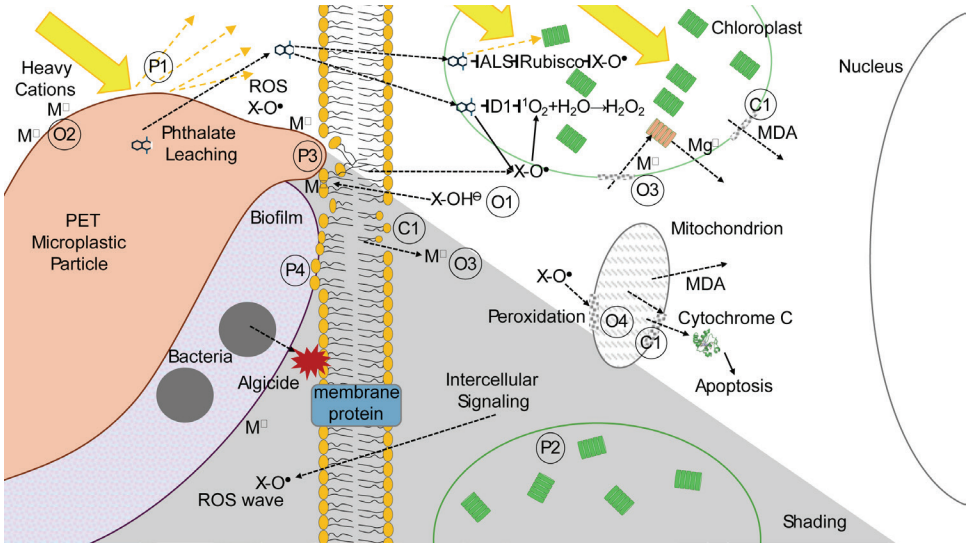
ROS and oxidative stress

Although much literature on microplastic bioactivity treats ROS, reactive oxygen species, as a homogenous class, it is important to differentiate between the main types: hydroxyl radicals, peroxides, superoxides, and singlet oxygen. Organelles vary in sensitivity to types and levels of ROS, and these relationships need to be quantified if we are to model their physiological impact.

Here we focus on three well-researched aspects – the effects of oxidative stress on cell membranes, and on photosynthesis, where there remain significant knowledge gaps, then on intra- and intercellular stress responses and signaling.

Oxidative effects of PET on membrane integrity

Cell membranes are sensitive to oxidative damage, and PET exposure of *C. reinhardtii* cells has been shown to induce extensive cell wall disruption and cell shrinkage (61). When oxygen free radicals accumulate, they interact with polyunsaturated fatty acids and produce lipid free radicals. These lipid free radicals then form peroxidized free radicals, which initiate a chain reaction among surrounding polyunsaturated fatty acids, a process known as lipid peroxidation. Lipid peroxidation at the cell membrane decreases membrane potential and may also increase membrane permeability to ions and ROS, with heavier cations directly causing ROS. Excessive intracellular lipid peroxidation causes



Key:

Symbol			
·····→	Migration	P1	Light Scattering
——→	Causation	P2	Shading, photoinhibition
→	Transformation	P3	Physical damage
⊣	Inhibition	P4	Biofilm adhesion
▶	Increase	O1	Anion loss
X-O•	Reactive oxygen species	O2	Cation chelation and concentration
C1	Lipid Peroxidation	O3	Heavy cation permeation
	Phthalate	O4	Mitochondrial membrane permeability

Figure 1. Modes and mechanisms of PET microplastic impact on *C. reinhardtii*, generated by the author.

mitochondrial membrane permeability (O4), loss of mitochondrial osmoregulation, and the release of apoptogenic proteins, such as cytochrome C, which initiate the apoptotic cascade leading to cell death (62).

Peroxidation also triggers the release of malondialdehyde (MDA), an indicator of oxidative stress (63, 64, 65, 66). Counterintuitively, studies show lipid accumulation, rather than lipid depletion, induced by PET (23). Wang Liufu, *et al* (67) explained that in *C. reinhardtii* and certain other species of microalgae, oxidative stress activates or upregulates genes responsible for lipid biosynthesis.

In *C. reinhardtii*, ROS stress tolerance varies among the cellular organelles, each requiring separate evaluation. Organelles that are most susceptible to ROS disruption are mitochondria, chloroplasts, peroxisomes, and the endoplasmic reticulum. Mitochondria contain the citric acid cycle and the electron transport chain, which are both sensitive to stress-induced intracellular signaling and to homeostatic imbalance of ions and small molecules (68).

Oxidative effects on photosynthesis

A common indicator of ROS-induced photosynthetic stress is reduced chlorophyll luminescence (69). This phenomenon has been observed in *C. reinhardtii* colonies exposed to PET, with oxidative damage to photosystems, other organelles, and cell membranes (45). Photosynthesis in *C. reinhardtii* is broadly analogous with higher plants with two exceptions: first, while the primary pathway for carbon-fixation is the Calvin-Benson Cycle ("C3"), the Hatch-Slack pathway ("C4") is absent (70); second, it has a complex CO_2-concentrating mechanism ("CCM") at pyrenoids, bodies within the chloroplast which concentrate CO_2 in response to low CO_2:O_2 ratios. The CCM is coordinated by CIA5, which activates around 600 genes, and is signaled by H_2O_2 alone, it being irresponsive to other ROS species (71, 72).

Normally, H_2O_2 is produced from superoxide radicals ($O_2^{\cdot -}$) by superoxide dismutase ("SODs"), particularly FeSOD and MnSODs (73). SODs can be inhibited by excess Cu or heavy metals (O3) and by two species of ROS, peroxynitrite ($ONOO^-$) and hydroxyl radicals ($\cdot OH$). Studies have shown total SOD levels to be elevated under PET micro- and nanoplastic exposure, with superoxide identified as the trigger (74). It is not yet known whether the balance between FeSOD (FSD1 gene) and five different MnSODs (MSD1-MSD5 genes) are affected (74). In summary, the CCM would appear relatively robust to PET-induced oxidative stress and could act as a proxy indicator of superoxide accumulation.

In the chloroplast, carotenoids transfer absorbed light energy to chlorophyll during light harvesting, while under excess light conditions, excited chlorophyll can transfer energy to carotenoids for photoprotective quenching, after which chlorophyll channels energy into the photosynthetic electron transport chain (75). Under normal photosynthetic conditions, electrons flow efficiently from photosystem II through the electron transport chain to photosystem I, maintaining optimal cellular redox balance. However, electron acceptors become disrupted during light stress (excess light or photoinhibition), particularly when the D1 protein of photosystem II is damaged or when the oxygen evolving complex (OEC) is compromised. Under such conditions, electron acceptors (QA and QB) become over-reduced, leading to charge recombination within the photosystem II reaction center that generates triplet-state chlorophyll (^3Chl). This energized triplet chlorophyll then transfers energy to stable triplet molecular oxygen ($^3O_2$), creating highly reactive singlet oxygen ($^1O_2$) through a process called triplet-triplet energy transfer (76).

PET-exposure itself has not so far been linked to these mechanisms of disruption, however other plastic leachates, bisphenol-A and the phthalate DEHP, are known to inhibit D1, binding to its QB site in photosystem II. (77, 45). This triggers a cascade resulting in "electron leakage" and superoxide production in PS I, resulting in further PS II damage (78). PS II compromise causes electrons to over-reduce the plastoquinone pool between PS II and the cytochrome b_6f complex, which becomes saturated with electrons (79). Electron flow from cytochrome b_6f to plastocyanin continues to PS I, where electron acceptors become over-reduced: A_1 phylloquinones, iron-sulfur clusters (Fx, FA/FB), and ferredoxin (80). Consequently, excess electrons transfer to molecular oxygen to generate superoxide anion radicals ($O_2^{\cdot -}$) via the Mehler reaction (81).

Singlet oxygen is primarily handled by physical or chemical quenching by carotenoids (82); a second line of defense is at the thylakoid membrane by $^1O_2$ scavengers such as tocopherol and plastoquinol (72). *C. reinhardtii* has specific responses to singlet oxygen: the SAK1 protein becomes progressively phosphorylated, upregulating further singlet oxygen-responsive genes, including GPXH and GSTS. These mechanisms mitigate the risk of positive feedback from $^1O_2$ damaging cell membranes, denaturing proteins, and further exhausting the capacity of electron acceptors (83).

Oxidative effects on C3

The outputs of the electron transport chain, ATP and NADPH, are produced at the thylakoid membrane directly into the cell stroma as inputs to the Calvin-Benson Cycle (C3) (84). NADPH acts as a reducing cofactor to fix CO_2 from the CCM into organic compounds. Rubisco binds to RuBP and incorporates CO_2 to produce 3-phosphoglycerate, which is converted to glyceraldehyde-3-phosphate (G3P), a precursor for cellular lipids, carbohydrates, and proteins (85, 86). The capacity of Rubisco and RuBP availability represents a limiting factor in photosynthetic efficiency (87). Inhibited carbon fixation causes electron leakage at PS I, generating superoxide radicals and other ROS species that eventually overwhelm cellular antioxidant defenses (64). This degrades the Rubisco large subunit (LSU), further impairing CO_2 fixation capacity (88, 89). Under sustained oxidative conditions, the amino terminus of the damaged LSU binds with RNA molecules, causing translational arrest that prevents new Rubisco assembly (88). This creates a destructive feedback loop where impaired carbon fixation generates more electron transport overload and ROS production, which causes additional Rubisco damage, progressively crippling the cell's photosynthetic capacity and leading to metabolic collapse.

C3 enzymes contain cysteine residues that are crucial for light-dependent activation and inactivation (89). Under normal conditions, glutathione reversibly attaches to these cysteine residues, preventing oxidative damage (79). Once oxidative stress exceeds the protective threshold, cysteine residues become irreversibly oxidized, causing permanent enzyme inhibition and altered protein structure (90). As cellular antioxidant capacity is overwhelmed, Glutathione is depleted, inhibiting chlorophyll synthesis and accelerating chlorophyll degradation (27, 91). This depletion deactivates acetyl-CoA carboxylase, disrupting fatty acid synthesis essential for chloroplast thylakoid membrane formation. Experimental evidence confirms that PET exposure induced oxidative stress followed by decreased chlorophyll synthesis in C. reinhardtii, creating a cascade of irreversible metabolic damage that ultimately compromises the cell's photosynthetic machinery and membrane integrity (26, 27, 92).

Oxidative stress response pathways and acclimatization

Oxidative stress is a phenomenon that occurs when ROS free radical concentration exceeds the capacity of a cell's damage-mitigating antioxidants. ROS are not necessarily detrimental and are used as signaling agents in some instances. At lower levels, 0.2-2% of electrons flowing along the transport chain react with ROS, activating multiple intracellular signaling pathways. At higher levels of ROS, oxidative stress and damage in *C. reinhardtii* are mitigated by enzymes such as superoxide dismutases (Cu-SOD and Mn-SODs), catalase (CAT), and peroxidase (Px). PET exposure has caused increases in both MDA and SOD (93, 94, 95). Alongside these indicators, other assays of oxidative stress and ROS are available, with individual strengths and weaknesses as highlighted in Table 1. Most ROS assays are either costly or do not differentiate types of ROS. This non-specificity towards exact ROS leads to vague results, as ROS are a broad category, which can lead to misleading conclusions. Additionally, many assays are susceptible to photobleaching caused by positive feedback and autocatalysis of ongoing reactions. Fluorescent imaging techniques are compromised by low sensitivity due to background noise, partly from 2,3-DHTPA (96). These limitations hinder the evidentiality of such research and will be discussed below.

Oxidative damage arises through Fenton's reaction, where H_2O_2 and Fe^{2+} ions react and create an $\cdot OH$ radical (97). In the peroxisome, the enzyme CAT mitigates oxidative damage by catabolizing H_2O into $^3O_2$, reducing ROS levels, averting both damage to electron acceptors and depletion of ROS-scavenging reducing agents (97, 64). *C. reinhardtii* possesses about 20 known ROS-activated genes. Expression of the gene GPXH, for example, is up-regulated by oxidative stress, inducing production of the enzyme GPX5, which catalyzes the reduction of H_2O_2 into H_2O (98, 99). One stress response, the PSBP2 protein, may be unique to *C. reinhardtii* (98): Semin, Boris K., and Lira N. Davletshina demonstrated that the presence of the PSBP2 protein markedly increases $^3O_2$ generation (oxygen evolution) and the substitution of Mn cations with Fe cations (100). These adaptive responses are, however, finite, as ROS-scavengers become depleted and organelle damage accumulates. Intense or prolonged oxidative stress will initially lead to impaired cell viability and eventually to cell death.

Extracellular stress response

When exposed to ROS, microalgae have been proven to signal to surrounding organisms using so-called "ROS waves". Stressed cells emit superoxide ($O_2^{\cdot -}$), which undergoes disproportionation to form H_2O_2 and

Table 1. The different types, methods, and limitations of ROS assay techniques, collated by the author

ROS Measured	Assay Technique	Process	Indicator	Limitations	References
Superoxide Radical Anion	Fluorescence	Hydroethidium reacts with superoxide to form a detectable fluorescent product	Wavelengths of fluorescence	This assay has low specificity and detects multiple ROS rather than one	131, 132
	Electron Paramagnetic Resonance and Electron Spin Resonance	Superoxide radicals are measured by spin probes, quantifying the presence of stable adducts	Electron spin resonance signals	This assay is highly specific but requires expensive and/or inaccessible equipment	133, 134
Singlet Oxygen	Near-Infrared Phosphorescence	Decaying singlet oxygen dissipates thermal and light energy through phosphorescence	Infrared wavelengths measure	This assay is highly specific towards singlet oxygen, being a standard as it is non-invasive. However, it has weak signals and only moderate sensitivity	135, 136
Hydroxyl Radical	Electrochemical sensors	The reaction of hydroxyl radicals and their products are measured using a potentiostat's anodes.	Potential difference between electrodes	This assay yields quantifiable data with high sensitivity. Although, other redox species may affect the reading, as well as the equipment being damaged through fouling	137, 138
Peroxide	Fluorescence, Chemiluminescence, Electrochemical sensors, Spectrophotometry	Peroxide reacts with probes like DCFDA to form fluorescent products, or with peroxidases to generate colorimetric or chemiluminescent signals; electrochemical sensors detect redox activity	Fluorescence intensity, chemiluminescent output, electrode potential, absorbance change	Assays may be affected by interference from other oxidants and reducing agents; sensitivity varies among methods; fluorescence can lack specificity to H_2O_2 alone; electrochemical sensors sensitive but may foul in complex samples	93, 139

is detected by neighboring cells, even of other species. This cell-to-cell communication causes a chain reaction of superoxide emission, activating acclimatization genes (101). Simultaneously, extracellular polymeric substances (EPS) are secreted into the surroundings. These polymers tend to be high-molecular-weight proteins and polysaccharides with notable adhesive properties. Functionally, they act as protection against oxidative stress and stimulate aggregation or flocculation. The result is a biofilm that buffers H_2O. Stressed cells may secrete up to 76% more EPS than normal (102). PET exposure activates both these mechanisms, causing oxidative stress to be signaled to surrounding organisms and increasing biofilm viscosity.

DISCUSSION

Despite limited ambiguities, studies clearly demonstrate that *C. reinhardtii* possesses a degree of resistance against oxidative stress but is significantly impacted by PET microplastics. As *C. reinhardtii* is a well-established model organism for photosynthetic research, these findings provide robust insights into PET microplastic effects on unicellular photosynthetic

eukaryotes and strong inferences about responses in aquatic algae, terrestrial microorganisms, and, to some extent, land plants.

Some other microorganisms may be more sensitive to microplastics than *C. reinhardtii*. Not all algae possess robust oxidative stress response mechanisms – glaucophytes, rhodophytes, and endosymbiotic algae, for example, and these together account for some 40% of global carbon fixation (72, 103). Conversely, there exist species much more resilient to prolonged microplastic pollution than *C. reinhardtii – Chlorella vulgaris* (104), for example. As aquatic microplastics levels rise, such ecological contrasts suggest that functional species like *C. reinhardtii* could be displaced by more resilient taxa such as *Chlorella* and cyanobacteria, potentially leading to eutrophication, anoxia, and system-wide impacts on aquatic communities (26, 27, 105).

Mechanistic basis of PET-induced stress

Cell membranes are particularly sensitive to multimodal contamination – biofilm, osmotic interference, ion concentration change, chemical interaction, and ROS generation. Unicellular organisms have greater surface area to cytoplasm ratios than the mycorrhizal root symbioses of higher plants. Several studies have found microplastic exposure severely depletes rhizosphere fungal growth and the soil microbiome in general (106, 107). This suggests that the mechanisms identified in *C. reinhardtii*—particularly membrane and oxidative stress responses—are extrapolatable to soil microbiomes and root symbioses, and are directly relevant to those causing crop declines. Quantitative modeling remains unfeasible, however, because the foundational research base is fragmented, simple discrete advances are clustered while empirical voids lie unaddressed: we do not need more research—our research needs different goals (108).

Limitations of current knowledge and research gaps

A limitation of this review is that *C. reinhardtii* has mainly been studied in freshwater environments. The species also occurs naturally in saltwater and soil. In saltwater, dissociated sodium and chlorine ions could affect osmolarity signaling and ionic interactions. This could be explored through knocking out specific genes in *C. reinhardtii* or repeating the same conditions on different species. Weaknesses of published research include insufficient duration, ambient concentration, comparability of results, studies on seawater, and a lack of specific ROS assays.

Whilst it is possible to conduct longer duration studies with ambient PET concentrations, it is challenging to simulate dynamic aquatic ecosystems, variation of PET and metabolite concentrations, and fluctuating environmental conditions *in vitro*. Consequently, no model capable of isolating individual variables and predicting outcomes has yet been developed. A further limitation is the dearth of systematic data linking observed biochemical responses to observed community-level phenomena.

Assumed mechanism for PET-induced ROS formation

In ecosystemic processes, microplastics usually accumulate a biofilm and collect multiple species of microalgae and bacteria, generating complex interactions and effects. These effects are mostly negative, including shading, agglomeration, competition for nutrients, heavy cation concentration, and even algicidal toxin emission (14, 105). Despite these broader impacts, the direct photochemical and redox processes leading to PET-induced oxidative stress merit particular attention.

Many effects of PET exposure on photosynthesis are also under-researched. For example, a critical component of photosystem II is the OEC, containing a Mn_4CaO_5 cluster that is potentially vulnerable to cation sequestration and disrupted cellular cation homeostasis (109). Theoretically, PET exposure might shift the $NADP^+$/NADPH equilibrium toward the oxidized state through reduced NADPH synthesis in PSII, and increased NADPH consumption by SODs and glutathione peroxidase. Real-time NADP(H) monitoring can be conducted *in vivo* but has not yet been applied to study PET exposure (110). Taken together, the mechanisms discussed thus far do not explain the multi-decade persistence of PET-induced stress responses in plants.

As discussed above, surface area increases supra-linearly with aging, but for PET, neither the age-to-area relationship, nor parametric controlling variables, nor surface morphology, nor surface electrochemistry have been quantitatively established, ruling out any systematic modeling of environmental or economic impact of PET dispersion in our biosphere. Even if surface area were correlated with impact, mitigation must be correlated with mechanism. Perhaps the largest gap in research is the mechanism by which PET, independent of leachates and ion imbalance, induces the Mehler reaction to produce superoxide radicals ($O_2^{\cdot -}$), as evidenced by elevated SODs and MDA.

Proposed mechanism for PET-induced ROS formation

Photochemical degradation of PET provides a plausible macro-link between molecular transformation and oxidative stress which consequently merits testing. Metal ions such as Zn^{2+}, Cu^{2+}, and Fe^{3+} become sorbed onto the surface of the PET. (111). Under UV-B radiation (280-315 nm), in the presence of liquid water, and possibly catalyzed by chelated metals such as Ti^{3+}, PET undergoes cleavage and hydrolysis, as the ester carbonyl groups absorb photons and undergo intersystem crossing from excited singlet state ($^1n,\pi^*$) to reactive triplet state ($^3n,\pi^*$), through Norrish type I and type II reactions to form vinyl esters, carboxylic acids, and hydroxyl radicals (·OH) (112, 113, 114, 115). The reaction now becomes autocatalytic chain scission, yielding TPA (1,4-benzenedicarboxylic acid) and ethylene glycol (116).

Hydroxyl radicals react with TPA (117), initially to form 2-hydroxyl-terephthalic acid (H2BDC-OH) (118). Further hydroxylation produces 2,3-dihydroxyterephthalic acid (2,3-DHTPA). Other byproducts may include malonic, muconic, fumaric, and oxalic acids, depending on light and catalyst concentration (119, 120). Of these, malonic acid has been shown to inhibit mitochondrial succinate dehydrogenase (SDH) in animals (121). 2,3-DHTPA is a strong chelating ligand (122, 123) and chelates metal ions at the PET surface. Chelated 2,3-DHTPA self-catalyses to form 2,3-benzoquinone-1,4-dicarboxylic acid (124, 125). So far, most of this has been known and documented in chemical industry literature since the 1970s (126). Exactly which reactions follow next is unclear, but what is known is the following.

Some benzoquinone compounds specifically inhibit inter-photosystem electron transport. 2,3-Dimethyl-5-hydroxy-6-phytyl-1,4-benzoquinone, for example, is found to inhibit the bottleneck electron-transfer step between photosystems I and II (127). A variety of *p*-benzoquinones have been found to inhibit plastoquinone both at PS II and at PS I "on the ascorbate plus N,N,N′,N′-tetramethyl-p-phenylenediamine → methylviologen pathway" (128). Superoxides can form a semi-stable radical anion with 1,4-benzoquinone-2-carboxylic acid (129).

The fully decarboxylated derivative 2,3-benzoquinone binds at quinone-binding sites, particularly the Q_p site of cytochrome b_6f complex (130). This causes the electron leakage cascade onto $O_2\text{-}^{-}$, dismutation and, via unknown H_2O_2-sensing proteins, results in CIA5 activation of the CCM. Thus, a mechanistic chain can be postulated linking PET photodegradation products to direct interference in photosynthetic electron transport and consequent ROS generation.

In summary, neither PET nor TPA is inert after all, and further research should focus on their primary degradation and decomposition cascade and its perennial and holistic metabolic impact – the effects of TPA and its byproducts H2BDC-OH, 2,3-DHTPA, and their chelates and derivatives on cell membranes and both Calvin and Fenton cycle reactions, and potentially on DNA replication too.

CONCLUSION

This review has examined four modes of PET-induced oxidative stress in *Chlamydomonas reinhardtii*. PET has been demonstrated to cause ROS and exert oxidative stress, as well as inhibit photosynthesis and biosynthesis in *C. reinhardtii*. Some of the mechanisms behind these physiological responses are already proven, while others still require further investigation. Integration of chemical evidence and physiological data suggests that PET decomposition products, particularly benzoquinone derivatives, can disrupt electron transport and drive oxidative imbalances. A unifying research priority should be to mechanistically link PET decomposition products with observed physiological and ecological stress signatures.

C. reinhardtii is not particularly important to aquatic ecosystems and, as both a convenient model and representative of terrestrial plant life, is usually assessed alone and *in vitro*. It is therefore recommended that future research should investigate microbiome impact mechanisms, not just single species, should aim to replicate environmental conditions longitudinally, should implement more precise molecular assay techniques, should investigate the bioactivity of TPA and its decomposition products, and should quantify parameters and tolerances. Given the growing environmental burden of PET fiber disposal, approaching 50 million tonnes annually, science has a duty to inform legislators, and policymakers must direct research efforts toward quantifying the biospheric and economic consequences of inaction and establishing evidence-based regulations informed by mechanistic understanding.

FUNDING SOURCES

The author received no financial support for the research, authorship, and/or publication of this article.

CONFLICT OF INTEREST

The author declares that there are no conflicts of interest regarding the publication of this article.

REFERENCES

1. Munhoz DR, Harkes P, Beriot N, Larreta J, Basurko OC. Microplastics: A Review of Policies and Responses. *Microplastics*. 2023 Mar 1; 2 (1): 1-26. https://doi.org/10.3390/microplastics2010001
2. European Chemical Agency. Implementation of the reporting requirements of the REACH restriction on microplastics. 2025.
3. Microplastics restrictions in the EU. Available from: https://www.enhesa.com/resources/article/microplastics-restrictions-in-the-eu/ (accessed on 2025-08-21)
4. Bermúdez JR, Swarzenski PW. A microplastic size classification scheme aligned with universal plankton survey methods. *MethodsX*. 2021 Jan 1; 8 (101516): 101516. https://doi.org/10.1016/j.mex.2021.101516
5. Li Y, Tao L, Wang Q, Wang F, Li G, Song M. Potential Health Impact of Microplastics: A Review of Environmental Distribution, Human Exposure, and Toxic Effects. *Environment & Health*. 2023 Aug 10; 1 (4): 249-57. https://doi.org/10.1021/envhealth.3c00052
6. Menichetti A, Mordini D, Montalti M. Penetration of Microplastics and Nanoparticles Through Skin: Effects of Size, Shape, and Surface Chemistry. *Journal of Xenobiotics*. 2024 Dec 31; 15 (1): 6-6. https://doi.org/10.3390/jox15010006
7. Jeong E, Lee JY, Redwan M. Animal exposure to microplastics and health effects: a review. *Emerging contaminants*. 2024 May 1; 10 (4): 100369-9. https://doi.org/10.1016/j.emcon.2024.100369
8. Gonzalez V, Lou X, Chi T. Evaluating Environmental Impact of Natural and Synthetic Fibers: A Life Cycle Assessment Approach. *Sustainability*. 2023 Jan 1; 15 (9): 7670. https://doi.org/10.3390/su15097670
9. Doshi Y. Polyester Fiber Market Trends, Size, Share & Forecast. Coherent Market Insights. Available from: https://www.coherentmarketinsights.com/industry-reports/polyester-fiber-market (accessed on 2025-08-21)
10. Li Y, Liu C, Yang H, He W, *et al*. Leaching of chemicals from microplastics: A review of chemical types, leaching mechanisms and influencing factors. *Science of The Total Environment*. 2024 Jan 1; 906: 167666. https://doi.org/10.1016/j.scitotenv.2023.167666
11. Li Z, Yang Y, Chen X, He Y, *et al*. A discussion of microplastics in soil and risks for ecosystems and food chains. *Chemosphere*. 2023 Feb; 313: 137637. https://doi.org/10.1016/j.chemosphere.2022.137637
12. Praveena SM, Syahira Asmawi M, Chyi JLY. Microplastic emissions from household washing machines: preliminary findings from Greater Kuala Lumpur (Malaysia). *Environmental Science and Pollution Research*. 2020 Sep 15; https://doi.org/10.1007/s11356-020-10795-z
13. Sadia M, Mahmood A, Ibrahim M, Irshad MK, *et al*. Microplastics pollution from wastewater treatment plants: A critical review on challenges, detection, sustainable removal techniques and circular economy. *Environmental Technology & Innovation*. 2022 Nov; 28: 102946. https://doi.org/10.1016/j.eti.2022.102946
14. Zoccali A, Cantoni B, Azzellino A, Malpei F. Removal of microplastics in wastewater treatment plants: insights from a literature meta-analysis. *Journal of environmental chemical engineering*. 2025 Apr 1; 116812-2. https://doi.org/10.1016/j.jece.2025.116812
15. Asadi A, Khodadoost F, Daglioglu N, Eris S. A systematic review on the occurrence and removal of microplastics during municipal wastewater treatment plants. *Environmental Engineering Research*. 2024 Sep 20; 30 (2). https://doi.org/10.4491/eer.2024.366
16. Ledford H, Chin B, Niyogi K. Acclimation to Singlet Oxygen Stress in Chlamydomonas reinhardtii. *Eukaryotic Cell*. 2007 Jun 1; 6 (6): 919-30. https://doi.org/10.1128/EC.00207-06
17. Chia WY, Ying Tang DY, Khoo KS, Kay Lup AN, Chew KW. Nature's fight against plastic pollution: Algae for plastic biodegradation and bioplastics production. *Environmental Science and Ecotechnology*. 2020 Oct; 4 (100065): 100065. https://doi.org/10.1016/j.ese.2020.100065
18. The unlikely alliance pressing Trump to regulate Pfas on US farms: "This is a basic human right". Available from: https://www.theguardian.com/environment/2025/aug/27/trump-pfas-forever-chemicals-us-farms (accessed on 2025-08-30)
19. Tiwari E, Sistla S. Agricultural plastic pollution reduces soil function even under best management practices. Whalen J, editor. *PNAS Nexus*. 2024 Oct 1; 3 (10). https://doi.org/10.1093/pnasnexus/pgae433
20. Asadi A, Khodadoost F, Daglioglu N, Eris S. A systematic review on the occurrence and removal of microplastics during municipal wastewater treatment plants. *Environmental Engineering Research*. 2024 Sep 20; 30 (2). https://doi.org/10.4491/eer.2024.366
21. Dhevagi P, Sahasa RGK, Poornima R, Ramya A. Unveiling the effect of microplastics on agricultural crops - a review. *International Journal of Phytoremediation*. 2023 Nov 8; 1-23.

22. Ramage SJFF, Coull M, Cooper P, Campbell CD, *et al*. Microplastics in agricultural soils following sewage sludge applications: Evidence from a 25-year study. *Chemosphere*. 2025 May; 376: 144277. https://doi.org/10.1016/j.chemosphere.2025.144277
23. Sutanto A, Widodo W, Rahayu ID, Sustiyana S, *et al*. The impact of microplastics on yield and economic losses in selected agricultural food commodities. *Environmental Quality Management.* 2024 Jan 30; https://doi.org/10.1002/tqem.22188
24. Salomé PA, Merchant SS. A Series of Fortunate Events: Introducing Chlamydomonas as a Reference Organism. *The Plant Cell*. 2019 Jun 12; 31 (8): 1682-707.https://doi.org/10.1105/tpc.18.00952
25. Zhang C, Chen X, Wang J, Tan L. Toxic effects of microplastic on marine microalgae Skeletonema costatum: Interactions between microplastic and algae. *Environmental Pollution*. 2017 Jan; 220: 1282-8. https://doi.org/10.1016/j.envpol.2016.11.005
26. Kabir MS, Wang H, Luster-Teasley S, Zhang L, Zhao R. Microplastics in landfill leachate: Sources, detection, occurrence, and removal. *Environmental Science and Ecotechnology*. 2023 Feb; 16: 100256. https://doi.org/10.1016/j.ese.2023.100256
27. Pencik O, Molnarova K, Durdakova M, Kolackova M, *et al*. Not so dangerous? PET microplastics toxicity on freshwater microalgae and cyanobacteria. *Environmental Pollution*. 2023 Apr 12; 121628. https://doi.org/10.1016/j.envpol.2023.121628
28. Khatiwada JR, Madsen C, Warwick C, Shrestha S, Chio C, Qin W. Interaction between polyethylene terephthalate (PET) microplastic and microalgae (Scenedesmus spp.): Effect on the growth, chlorophyll content, and hetero-aggregation. *Environmental Advances.* 2023 Oct 1; 13: 100399. https://doi.org/10.1016/j.envadv.2023.100399
29. Hadiyanto H, Khoironi A, Dianratri I, Suherman S, Muhammad F, Vaidyanathan S. Interactions between polyethylene and polypropylene microplastics and Spirulina sp. microalgae in aquatic systems. *Heliyon*. 2021 Aug; 7 (8): e07676. https://doi.org/10.1016/j.heliyon.2021.e07676
30. Sioen M, Vercauteren M, Blust R, Town RM, Janssen C, Asselman J. Impact of weathered and virgin polyethylene terephthalate (PET) micro- and nanoplastics on growth dynamics and the production of extracellular polymeric substances (EPS) of microalgae. *Science of The Total Environment.* 2024 Nov; 953: 176074. https://doi.org/10.1016/j.scitotenv.2024.176074
31. Qin Y, Tu Y, Chen C, Wang F, Yang Y, Hu Y. Biofilms on microplastic surfaces and their effect on pollutant adsorption in the aquatic environment. *Journal of Material Cycles and Waste Management*. 2024 Sep 10; https://doi.org/10.1007/s10163-024-02066-7
32. Carrasco Flores D, Hotter V, Vuong T, Hou Y, *et al*. A mutualistic bacterium rescues a green alga from an antagonist. *Proceedings of the National Academy of Sciences of the United States of America*. 2024 Apr 3; 121 (15). https://doi.org/10.1073/pnas.2401632121
33. Burgunter-Delamare B, Shetty P, Vuong T, Mittag M. Exchange or Eliminate: The Secrets of Algal-Bacterial Relationships. *Plants*. 2024 Jan 1; 13 (6): 829. https://doi.org/10.3390/plants13060829
34. Mir R, Albarqi S, Albalawi W, Alanazi G, *et al*. Molecular Mechanisms by Which Polyethylene Terephthalate (PET) Microplastic and PET Leachate Promote the Growth of Benthic Cyanobacteria. *Metabolites*. 2025 Jun 9; 15 (6): 383-3. https://doi.org/10.3390/metabo15060383
35. Murthe SS, Sreekantan S, Mydin RBSMN. Study on the Physical, Thermal and Mechanical Properties of SEBS/PP (Styrene-Ethylene-Butylene-Styrene/Polypropylene) Blend as a Medical Fluid Bag. *Polymers.* 2022 Aug 11; 14 (16): 3267. https://doi.org/10.3390/polym14163267
36. Ragoobur D, Somaroo G. Analysis and Effects of Microplastics in the Agricultural Soils. Proceedings of the 3rd World Congress on New Technologies. 2021 Aug 1; https://doi.org/10.11159/icepr21.119
37. Doungsoongnuen S, Worachartcheewan A, Pingaew R, Suksrichavalit T, *et al*. Investigation on biological activities of anthranilic acid sulfonamide analogs. *EXCLI Journal.* 2011 Oct 7; 10: 155.
38. PubChem. Anthranilamide. Nih.gov. PubChem. Available from: https://pubchem.ncbi.nlm.nih.gov/compound/Anthranilamide#section=GHS-Classification (accessed on 2025-09-19)
39. Kuykendall JR, Cox R. 2-Aminobenzamide, an inhibitor of ADP-ribosylation, antagonizes induced DNA hypomethylation during differentiation of murine Friend erythroleukemia cells by N′-methylnicotinamide. *Differentiation*. 1990 Jul; 44 (1): 69-73. https://doi.org/10.1111/j.1432-0436.1990.tb00538.x
40. Groslambert J, Prokhorova E, Ahel I. ADP-ribosylation of DNA and RNA. *DNA Repair.* 2021 Sep; 105: 103144. https://doi.org/10.1016/j.dnarep.2021.103144
41. Manco G, Lacerra G, Porzio E, Catara G. ADP-Ribosylation Post-Translational Modification: An Overview with a Focus on RNA Biology and New Pharmacological Perspectives. *Biomolecules*. 2022 Mar 13; 12 (3): 443-3. https://doi.org/10.3390/biom12030443
42. Schuller M, Ahel I. Beyond protein modification: the

rise of non-canonical ADP-ribosylation. *Biochemical Journal*. 2022 Feb 17; 479 (4): 463-77. https://doi.org/10.1042/BCJ20210280
43. Venkatachalam S, G. S, V. J, R. P, Rao K, K. A. Degradation and Recyclability of Poly (Ethylene Terephthalate). *Polyester*. 2012 Sep 26; https://doi.org/10.5772/48612
44. Mikac L, Csáki A, Zentai B, Rigó I, *et al*. UV Irradiation of Polyethylene Terephthalate and Polypropylene and Detection of Formed Microplastic Particles Down to 1 μm. *ChemPlusChem*. 2023 Nov 22; 89 (2). https://doi.org/10.1002/cplu.202300497
45. von Haugwitz G, Han X, Pfaff L, Li Q, *et al*. Structural Insights into (Tere)phthalate-Ester Hydrolysis by a Carboxylesterase and Its Role in Promoting PET Depolymerization. *ACS Catalysis*. 2022 Nov 29; 12 (24): 15259-70. https://doi.org/10.1021/acscatal.2c03772
46. Wiegand MJ, Benton TZ, Gilbert JL. A fluorescent approach for detecting and measuring reduction reaction byproducts near cathodically-biased metallic surfaces: Reactive oxygen species production and quantification. *Bioelectrochemistry*. 2019 Oct; 129: 235-41. https://doi.org/10.1016/j.bioelechem.2019.05.020
47. 2-Hydroxyterephthalic acid. Sigma Aldrich. Available from: https://www.sigmaaldrich.com/CA/en/product/aldrich/752525 (accessed on 2025-08-21)
48. Lüchtrath S, Wutke R, Held A. Secondary Formation of Light-Absorbing, Insoluble Particles from Catechol in the Presence of Fe(II) or Fe(III) and Hydrogen Peroxide at Dark and Light Conditions. *ACS ES&T Air*. 2025 Jul 27; https://doi.org/10.1021/acsestair.5c00091
49. Ball GL, McLellan CJ, Bhat VS. Toxicological review and oral risk assessment of terephthalic acid (TPA) and its esters: A category approach. *Critical Reviews in Toxicology*. 2012 Jan 1; 42 (1): 28-67. https://doi.org/10.3109/10408444.2011.623149
50. Provisional Peer-Reviewed Toxicity Values for P-Phthalic Acid (Terephthalic Acid). Available from: https://assessments.epa.gov/risk/document/&deid%3D349933
51. Hall IH, Wong OT, Reynolds DJ, Simlot R, Chang JJ. Terephthalic Acid in Sprague-Dawley Rats as a Hypolipidemic Agent. Archiv der Pharmazie. 1993 Jan; 326 (1): 5-13. https://doi.org/10.1002/ardp.19933260104
52. Dai GD, Cui LB, Song L, Liu R, *et al*. Effects of terephthalic acid on rat lipid metabolism. *Biomedical and environmental sciences: BES*. 2025; 19 (4).
53. Mastanjević K, Kartalović B, Kovačević D, Krstanović V, Habschied K. Migration of Phthalates and Bisphenol A from Polyethylene Terephthalate Bottles into Beer During Storage at Controlled Temperatures. *Foods*. 2025 Jul 30; 14 (15): 2689-9. https://doi.org/10.3390/foods14152689
54. Maddison C, Sathish CI, Lakshmi D, Wayne O, Palanisami T. An advanced analytical approach to assess the long-term degradation of microplastics in the marine environment. *npj Materials Degradation*. 2023 Jul 26; 7 (1): 1-11. https://doi.org/10.1038/s41529-023-00377-y
55. Galea J, Agius Anastasi A, Vella DA, Briffa SM. UV Aging of PET Microplastics in a Custom-Built Weathering Chamber Replicating Mediterranean Conditions. *ACS Omega*. 2025 Aug 12; 10 (33): 37486-94.https://doi.org/10.1021/acsomega.5c03491
56. Rahman AMNAA, Rusli A, Abdullah MK, Shuib RK, *et al*. A review of microplastic surface interactions in water and potential capturing methods. *Water Science and Engineering*. 2023 Dec 21; 17 (4). https://doi.org/10.1016/j.wse.2023.11.008
57. Xia Y, Chang Ming Li, Zhang Y, Yin Y, *et al*. Contact between water vapor and silicate surface causes abiotic formation of reactive oxygen species in an anoxic atmosphere. *Proceedings of the National Academy of Sciences of the United States of America*. 2023 Jul 17; 120 (30). https://doi.org/10.1073/pnas.2302014120
58. Chen Q, Zhao H, Liu Y, Jin L, Peng R. Factors Affecting the Adsorption of Heavy Metals by Microplastics and Their Toxic Effects on Fish. *Toxics*. 2023 Jun 1; 11 (6): 490. https://doi.org/10.3390/toxics11060490
59. Liu S, Shi J, Wang J, Dai Y, *et al*. Interactions Between Microplastics and Heavy Metals in Aquatic Environments: A Review. *Frontiers in Microbiology*. 2021 Apr 22;12. https://doi.org/10.3389/fmicb.2021.652520
60. Keys M, Hopkinson B, Highfield A, Chrachri A, Brownlee C, Wheeler GL. The requirement for external carbonic anhydrase in diatoms is influenced by the supply and demand for dissolved inorganic carbon. *Journal of phycology*. 2023 Dec 21; 60 (1): 29-45. https://doi.org/10.1111/jpy.13416
61. Pencik O, Molnarova K, Durdakova M, Kolackova M, *et al*. Not so dangerous? PET microplastics toxicity on freshwater microalgae and cyanobacteria. *Environmental Pollution*. 2023 Apr 12; 121628. https://doi.org/10.1016/j.envpol.2023.121628
62. Zheng Y, Sun J, Luo Z, Li Y, Huang Y. Emerging mechanisms of lipid peroxidation in regulated cell death and its physiological implications. *Cell Death and Disease*. 2024 Nov 26; 15 (11). https://doi.org/10.1038/s41419-024-07244-x
63. Hiwot Tezera Endale, Tesfaye W, Tiget Ayelgn Mengstie. ROS induced lipid peroxidation and

their role in ferroptosis. *Frontiers in Cell and Developmental Biology*. 2023 Aug 1; 11. https://doi.org/10.3389/fcell.2023.1226044

64. Dong Y, Gao M, Qiu W, Song Z. Effects of microplastic on arsenic accumulation in Chlamydomonas reinhardtii in a freshwater environment. *Journal of Hazardous Materials*. 2021 Mar; 405: 124232. https://doi.org/10.1016/j.jhazmat.2020.124232
65. Dayer R, Fischer BB, Eggen RIL, Lemaire SD. The Peroxiredoxin and Glutathione Peroxidase Families in Chlamydomonas reinhardtii. *Genetics*. 2008 May 1; 179 (1): 41-57. https://doi.org/10.1534/genetics.107.086041
66. Yang Q, Li H, Wang D, Zhang X, *et al*. Utilization of chemical wastewater for CO2 emission reduction: Purified terephthalic acid (PTA) wastewater-mediated culture of microalgae for CO2 bio-capture. *Applied Energy*. 2020 Oct 1; 276: 115502-2. https://doi.org/10.1016/j.apenergy.2020.115502
67. Wang L, Yang T, Pan Y, Shi L, Jin Y, Huang X. The Metabolism of Reactive Oxygen Species and Their Effects on Lipid Biosynthesis of Microalgae. *International Journal of Molecular Sciences*. 2023 Jul 3; 24 (13): 11041. https://doi.org/10.3390/ijms241311041
68. Tirichen H, Yaigoub H, Xu W, Wu C, Li R, Li Y. Mitochondrial Reactive Oxygen Species and Their Contribution in Chronic Kidney Disease Progression Through Oxidative Stress. *Frontiers in Physiology*. 2021 Apr 23; 12. https://doi.org/10.3389/fphys.2021.627837
69. Hurtado-Gallego J, Pulido-Reyes G, González-Pleiter M, Salas G, *et al*. Toxicity of superparamagnetic iron oxide nanoparticles to the microalga Chlamydomonas reinhardtii. *Chemosphere*. 2020 Jan 1; 238: 124562-2. https://doi.org/10.1016/j.chemosphere.2019.124562
70. Mamedov TG, Moellering ER, Chollet R. Identification and expression analysis of two inorganic C- and N-responsive genes encoding novel and distinct molecular forms of eukaryotic phosphoenolpyruvate carboxylase in the green microalga Chlamydomonas reinhardtii. *The Plant Journal*. 2005 May 5; 42 (6): 832-43. https://doi.org/10.1111/j.1365-313X.2005.02416.x
71. Moroney JV, Ynalvez RA. Proposed Carbon Dioxide Concentrating Mechanism in Chlamydomonas reinhardtii. *Eukaryotic Cell*. 2007 Aug 1; 6 (8): 1251-9. https://doi.org/10.1128/EC.00064-07
72. Wakao S, Niyogi KK. Chlamydomonas as a model for reactive oxygen species signaling and thiol redox regulation in the green lineage. *PLANT PHYSIOLOGY*. 2021 Jul 24; 187 (2): 687-98. https://doi.org/10.1093/plphys/kiab355
73. Page MD, Allen MD, Kropat J, Urzica EI, *et al*. Fe Sparing and Fe Recycling Contribute to Increased Superoxide Dismutase Capacity in Iron-Starved Chlamydomonas reinhardtii. *The Plant Cell*. 2012 Jun; 24 (6): 2649-65. https://doi.org/10.1105/tpc.112.098962
74. Yan Z, Xu L, Zhang W, Yang G, *et al*. Comparative toxic effects of microplastics and nanoplastics on Chlamydomonas reinhardtii: Growth inhibition, oxidative stress, and cell morphology. *Journal of Water Process Engineering*. 2021 Oct; 43: 102291. https://doi.org/10.1016/j.jwpe.2021.102291
75. Dogra V, Kim C. Singlet Oxygen Metabolism: From Genesis to Signaling. *Frontiers in Plant Science*. 2020 Jan 8; 10. https://doi.org/10.3389/fpls.2019.01640
76. Krieger-Liszkay A, Fufezan C, Trebst A. Singlet oxygen production in photosystem II and related protection mechanism. *Photosynthesis Research*. 2008 Sep 9; 98 (1-3): 551-64. https://doi.org/10.1007/s11120-008-9349-3
77. Li J, Wang Y, Li N, He Y, *et al*. Toxic Effects of Bisphenol A and Bisphenol S on Chlorella Pyrenoidosa under Single and Combined Action. *International Journal of Environmental Research and Public Health*. 2022 Apr 2; 19 (7): 4245. https://doi.org/10.3390/ijerph19074245
78. Tjus SE, Scheller HV, Andersson B, Møller BL. Active Oxygen Produced during Selective Excitation of Photosystem I Is Damaging Not Only to Photosystem I, But Also to Photosystem II. *Plant Physiology*. 2001 Apr 1; 125 (4): 2007-15. https://doi.org/10.1104/pp.125.4.2007
79. Baniulis D, Hasan SS, Stofleth JT, Cramer WA. Mechanism of Enhanced Superoxide Production in the Cytochrome b6f Complex of Oxygenic Photosynthesis. *Biochemistry*. 2013 Dec 6; 52 (50): 8975-83. https://doi.org/10.1021/bi4013534
80. Krieger-Liszkay A, Kós PB, Hideg É. Superoxide anion radicals generated by methylviologen in photosystem I damage photosystem II. *Physiologia Plantarum*. 2010 Oct 22; 142 (1): 17-25. https://doi.org/10.1111/j.1399-3054.2010.01416.x
81. Foyer CH, Hanke G. ROS production and signalling in chloroplasts: cornerstones and evolving concepts. *The Plant Journal*. 2022 Jun 28; 111 (3): 642-61. https://doi.org/10.1111/tpj.15856
82. Ramel F, Birtic S, Cuiné S, Triantaphylidès C, Ravanat JL, Havaux M. Chemical Quenching of Singlet Oxygen by Carotenoids in Plants. *Plant Physiology*. 2012 Jan 10; 158 (3): 1267-78. https://doi.org/10.1104/pp.111.182394
83. Triantaphylidès C, Havaux M. Singlet oxygen in

plants: production, detoxification and signaling. *Trends in Plant Science*. 2009 Apr; 14 (4): 219-28. https://doi.org/10.1016/j.tplants.2009.01.008

84. Burlacot A. Quantifying the roles of algal photosynthetic electron pathways: a milestone towards photosynthetic robustness. *New Phytologist*. 2023 Oct 23; 240 (6): 2197-203. https://doi.org/10.1111/nph.19328
85. Meloni M, Gurrieri L, Fermani S, Velie L, *et al*. Ribulose-1,5-bisphosphate regeneration in the Calvin-Benson cycle: Focus on the last three enzymatic steps that allow the formation of Rubisco substrate. *Frontiers in Plant Science*. 2023; 14: 1130430. https://doi.org/10.3389/fpls.2023.1130430
86. Ferro L. Wastewater treatment and biomass generation by Nordic microalgae: growth in subarctic climate and microbial interactions. 2019 Jan 1;
87. Yang B, Liu J, Ma X, Guo B, *et al*. Genetic engineering of the Calvin cycle toward enhanced photosynthetic CO2 fixation in microalgae. *Biotechnology for Biofuels*. 2017 Oct 5; 10 (1). https://doi.org/10.1186/s13068-017-0916-8
88. Desimone M, Henke A, Wagner E. Oxidative Stress Induces Partial Degradation of the Large Subunit of Ribulose-1,5-Bisphosphate Carboxylase/Oxygenase in Isolated Chloroplasts of Barley. *Plant Physiology*. 1996 Jul 1; 111 (3): 789-96. https://doi.org/10.1104/pp.111.3.789
89. Cohen I, Knopf JA, Irihimovitch V, Shapira M. A Proposed Mechanism for the Inhibitory Effects of Oxidative Stress on Rubisco Assembly and Its Subunit Expression. *Plant Physiology*. 2005 Feb 1; 137 (2): 738-46. https://doi.org/10.1104/pp.104.056341
90. Michelet L, Zaffagnini M, Morisse S, Sparla F, *et al*. Redox regulation of the Calvin-Benson cycle: something old, something new. *Frontiers in Plant Science*. 2013; 4. https://doi.org/10.3389/fpls.2013.00470
91. Ramazi S, Zahiri J. Post-translational Modifications in proteins: resources, Tools and Prediction Methods. *Database*. 2021 Apr 7; 2021 (2021). https://doi.org/10.1093/database/baab012
92. Jiang J, Cai X, Ren H, Cao G, *et al*. Effects of polyethylene terephthalate microplastics on cell growth, intracellular products and oxidative stress of Scenedesmus sp. *Chemosphere*. 2024 Jan 1; 348: 140760-0. https://doi.org/10.1016/j.chemosphere.2023.140760
93. Murphy MP, Bayir H, Belousov V, Chang CJ, *et al*. Guidelines for measuring reactive oxygen species and oxidative damage in cells and *in vivo*. *Nature Metabolism*. 2022 Jun 1; 4 (6): 651-62. https://doi.org/10.1038/s42255-022-00591-z
94. Zhao R, Jiang S, Zhang L, Yu Z. Mitochondrial electron transport chain, ROS generation and uncoupling (Review). *International Journal of Molecular Medicine*. 2019 May 8; 44 (1): 3-15. https://doi.org/10.3892/ijmm.2019.4188
95. Aikaterini Koletti, Dimitrios Skliros, Dervisi I, Roussis A, Emmanouil Flemetakis. Oxidative Stress Responses in Microalgae: Modern Insights into an Old Topic. *Applied Microbiology*. 2025 Apr 8; 5 (2): 37-7. https://doi.org/10.3390/applmicrobiol5020037
96. Brandes RP, Rezende F, Schröder K. Redox Regulation Beyond ROS. *Circulation Research*. 2018 Jul 20; 123 (3): 326-8. https://doi.org/10.1161/CIRCRESAHA.118.313146
97. Ransy C, Vaz C, Lombès A, Bouillaud F. Use of H2O2 to Cause Oxidative Stress, the Catalase Issue. *International Journal of Molecular Sciences*. 2020 Nov 30; 21 (23). https://doi.org/10.3390/ijms21239149
98. Brzezowski P, Wilson KE, Gray GR. The PSBP2 protein of Chlamydomonas reinhardtii is required for singlet oxygen-dependent signaling. *Planta*. 2012 Jun 21; 236 (4): 1289-303. https://doi.org/10.1007/s00425-012-1683-1
99. Fischer BB, Krieger-Liszkay A, Rik I.L. Eggen. Photosensitizers neutral red (type I) and rose bengal (type II) cause light-dependent toxicity in Chlamydomonas reinhardtii and induce the Gpxh gene via increased singlet oxygen formation. *Environmental Science & Technology*. 2004 Dec 1; 38 (23): 6307-13. https://doi.org/10.1021/es049673y
100. Semin BK, Davletshina LN. High-efficiency oxygen evolution by photosystem II oxygen-evolving complex containing 3Mn per reaction center. *JBIC Journal of Biological Inorganic Chemistry*. 2023 Apr 12; 28 (4): 393-401. https://doi.org/10.1007/s00775-023-01987-2
101. Fichman Y, Rowland L, Oliver MJ, Mittler R. ROS are evolutionary conserved cell-to-cell stress signals. *Proceedings of the National Academy of Sciences*. 2023 Jul 26; 120 (31). https://doi.org/10.1073/pnas.2305496120
102. Sioen M, Vercauteren M, Blust R, Town RM, Janssen C, Asselman J. Impact of weathered and virgin polyethylene terephthalate (PET) micro- and nanoplastics on growth dynamics and the production of extracellular polymeric substances (EPS) of microalgae. *Science of The Total Environment*. 2024 Nov; 953: 176074. https://doi.org/10.1016/j.scitotenv.2024.176074
103. Wang M, Ye X, Bi H, Shen Z. Microalgae biofuels: illuminating the path to a sustainable future amidst challenges and opportunities. *Biotechnology for Biofuels and Bioproducts*. 2024 Jan 23; 17 (1). https://

doi.org/10.1186/s13068-024-02461-0
104. Dey TK, Uddin ME, Jamal M. Detection and removal of microplastics in wastewater: evolution and impact. *Environmental Science and Pollution Research International*. 2021 Apr 1; 28 (14): 16925-47. https://doi.org/10.1007/s11356-021-12943-5
105. Huertas MJ, Mallén-Ponce MJ. Dark side of cyanobacteria: searching for strategies to control blooms. *Microbial Biotechnology*. 2021 Nov 29; 15 (5): 1321-3. https://doi.org/10.1111/1751-7915.13982
106. Gamage M, Mudannayake NH, Premaratne M, Madawala HMSP. Effect of microplastics on rhizosphere and arbuscular mycorrhizal fungi of Zea mays. *Ceylon Journal of Science*. 2025 Aug 5; 54 (3): 855-64. https://doi.org/10.4038/cjs.v54i3.8944
107. Lozano YM, Dueñas JF, Zordick C, Rillig MC. Microplastic fibres affect soil fungal communities depending on drought conditions with consequences for ecosystem functions. *Environmental microbiology.* 2024 Jan 10; 26 (2). https://doi.org/10.1111/1462-2920.16549
108. Lonardo SD, Diamantidou D, Chatzitaki A, Fatouros D, Theodoridis G. Determination of plastic polyester oligomers in real samples and their bioeffects. *EFSA Journal*. 2023 Nov 1; 21. https://doi.org/10.2903/j.efsa.2023.e211008
109. Zhang Z, Fu D, Sun Z, Ju C, *et al*. Tonoplast-associated calcium signaling regulates manganese homeostasis in Arabidopsis. *Molecular Plant.* 2021 Mar 7; 14 (5): 805-19. https://doi.org/10.1016/j.molp.2021.03.003
110. Kadac-Czapska K, Ośko J, Knez E, Grembecka M. Microplastics and Oxidative Stress-Current Problems and Prospects. *Antioxidants*. 2024 May 8; 13 (5): 579-9. https://doi.org/10.3390/antiox13050579
111. Frost H, Bond T, Sizmur T, Felipe-Sotelo M. Sorption of metal ions onto PET-derived microplastic fibres. *Environmental Science: Processes & Impacts*. 2024; https://doi.org/10.1039/D4EM00373J
112. Rostampour S, Cook R, Jhang SS, Li Y, Fan C, Sung LP. Changes in the Chemical Composition of Polyethylene Terephthalate Under UV Radiation in Various Environmental Conditions. *Research Square (Research Square).* 2024 May 22; https://doi.org/10.21203/rs.3.rs-4402725/v1
113. Wikipedia Contributors. Norrish reaction. Wikipedia. Wikimedia Foundation. Available from: https://en.wikipedia.org/wiki/Norrish_reaction (accessed on 2025-09-17)
114. Ding L, Yu X, Guo X, Zhang Y, *et al*. The photodegradation processes and mechanisms of polyvinyl chloride and polyethylene terephthalate microplastic in aquatic environments: Important role of clay minerals. *Water Research*. 2022 Jan; 208: 117879. https://doi.org/10.1016/j.watres.2021.117879
115. Gewert B, Plassmann MM, MacLeod M. Pathways for degradation of plastic polymers floating in the marine environment. *Environmental Science: Processes & Impacts.* 2015; 17 (9): 1513-21. https://doi.org/10.1039/C5EM00207A
116. Page SE, Arnold WA, McNeill K. Terephthalate as a probe for photochemically generated hydroxyl radical. *Journal of Environmental Monitoring.* 2010; 12 (9): 1658. https://doi.org/10.1039/c0em00160k
117. Linxiang L, Abe Y, Nagasawa Y, Kudo R, *et al*. An HPLC assay of hydroxyl radicals by the hydroxylation reaction of terephthalic acid. *Biomedical Chromatography*. 2004 Aug 26; 18 (7): 470-4. https://doi.org/10.1002/bmc.339
118. Fuentes I, Rodríguez JL, Poznyak T, Chairez I. Photocatalytic ozonation of terephthalic acid: a by-product-oriented decomposition study. *Environmental science and pollution research international.* 2014 Nov; 21 (21): 12241-8. https://doi.org/10.1007/s11356-014-3176-1
119. Qutob M, Hussein MA, Alamry KA, Rafatullah M. A review on the degradation of acetaminophen by advanced oxidation process: pathway, by-products, biotoxicity, and density functional theory calculation. *RSC Advances*. 2022; 12 (29): 18373-96. https://doi.org/10.1039/D2RA02469A
120. Beevers H. MALONIC ACID AS AN INHIBITOR OF MAIZE ROOT RESPIRATION. *Plant Physiology.* 1952 Oct 1; 27 (4): 725-35. https://doi.org/10.1104/pp.27.4.725
121. Kurisaki T, Etou M, Okaue Y, Wakita H, Yokoyama T. Acid-base behavior and Al3+ complex formation of synthesized 2,3-dihydroxyterephthalic acid (DHTPA) at pH 3 as a model compound of Inogashira fulvic acid (IFA). *Polyhedron.* 2014 Apr; 72: 135-9. https://doi.org/10.1016/j.poly.2014.02.006
122. Kiss T, Micera G, Sanna D, Kozlowski H. Copper(II) and oxovanadium(IV) complexes of 2,3-dihydroxy-terephthalic and 2,3,4-trihydroxybenzoic acids. *Journal of the Chemical Society Dalton transactions*. 1994 Jan 1; (3): 347-7. https://doi.org/10.1039/dt9940000347
123. Comba P, Martin B, Muruganantham A, Straub J. Structure, Bonding, and Catecholase Mechanism of Copper Bispidine Complexes. *Inorganic Chemistry.* 2012 Aug 21; 51 (17): 9214-25. https://doi.org/10.1021/ic3004917
124. DuVall SH, McCreery RL. Self-catalysis by Catechols and Quinones during Heterogeneous Electron Transfer at Carbon Electrodes. *Journal of the American Chemical Society.* 2000 Jul 1; 122

(28):6759-64. https://doi.org/10.1021/ja000227u
125. Gómez-Herrero AC, Sánchez-Sánchez C, Chérioux F, Martínez JI, *et al*. Copper-assisted oxidation of catechols into quinone derivatives. *Chemical Science.* 2021 Feb 18; 12 (6): 2257-67. https://doi.org/10.1039/D0SC04883F
126. Pearce E. Photodegradation, photo-oxidation and photostabilization of polymers, B. Ranby and J. F. Rabek, Wiley-Interscience, New York, 1975, 573 pp. $47.50. *Journal of Polymer Science: Polymer Letters Edition*. 1975 Oct; 13 (10): 621-2. https://doi.org/10.1002/pol.1975.130131008
127. Bøler J, Pardini R, Mustafa HT, Folkers K, Dilley RA, Crane FL. Synthesis of Plastoquinone Analogs and Inhibition of Photosynthetic and Mammalian Enzyme Systems. *Proceedings of the National Academy of Sciences.* 1972 Dec 1; 69 (12): 3713-7. https://doi.org/10.1073/pnas.69.12.3713
128. BARR R, CRANE FL, BEYER G, MAXWELL LA, FOLKERS K. The Effect of Various Substituted p-Benzoquinones on Electron Transport in Spinach Chloroplasts. *European Journal of Biochemistry.* 1977 Oct 1; 80 (1): 51-4. https://doi.org/10.1111/j.1432-1033.1977.tb11854.x
129. Nakayama T, Honda R. Electrochemical and Mechanistic Study of Superoxide Elimination by Mesalazine through Proton-Coupled Electron Transfer. *Pharmaceuticals*. 2021 Feb 4; 14 (2): 120. https://doi.org/10.3390/ph14020120
130. Cramer WA. Structure-function of the cytochrome b6f lipoprotein complex: a scientific odyssey and personal perspective. *Photosynthesis Research*. 2018 Oct 11; 139 (1-3): 53-65. https://doi.org/10.1007/s11120-018-0585-x
131. Podsiadły R. Superoxide radical anion reacts with hydroethidine to produce 2-hydroxyethidium but not ethidium. Available from: https://www.researchgate.net/publication/307599891_Recent_developments_in_detection_of_superoxide_radical_anion_and_hydrogen_peroxide_Opportunities_challenges_and_implications_in_redox_signaling (accessed on 2025-08-31)
132. Nazarewicz RR, Bikineyeva A, Dikalov SI. Rapid and Specific Measurements of Superoxide Using Fluorescence Spectroscopy. *Journal of Biomolecular Screening*. 2012 Nov 27; 18 (4): 498-503. https://doi.org/10.1177/1087057112468765
133. Scheinok S, Leveque P, Sonveaux P, Driesschaert B, Gallez B. Comparison of different methods for measuring the superoxide radical by EPR spectroscopy in buffer, cell lysates and cells. *Free Radical Research*. 2018 Oct 3; 52 (10): 1182-96. https://doi.org/10.1080/10715762.2018.1541321
134. Bonke SA, Risse T, Schnegg A, Brückner A. In situ electron paramagnetic resonance spectroscopy for catalysis. *Nature Reviews Methods Primers.* 2021 May 6; 1 (1). https://doi.org/10.1038/s43586-021-00031-4
135. JASCO. EEM and emission spectra (λEx: 525 nm) of singlet oxygen. Available from: https://jascoinc.com/applications/detection-of-phosphorescence-from-singlet-oxygen-using-nir-spectrofluorometer. (accessed on 2025-09-03)
136. Koh E, Fluhr R. Singlet oxygen detection in biological systems: Uses and limitations. *Plant Signaling & Behavior.* 2016 May 27; 11 (7): e1192742-2. https://doi.org/10.1080/15592324.2016.1192742
137. Cui H, Ma J, Liu Y, Wang C, Song Q. Dimethyl Sulfoxide: An Ideal Electrochemical Probe for Hydroxyl Radical Detection. *ACS Sensors.* 2024 Feb 22; 9 (3): 1508-14. https://doi.org/10.1021/acssensors.3c02644
138. Jafari B, Muthuvel M, Botte GG. Nickel-based electrochemical sensor with a wide detection range for measuring hydroxyl ions and pH sensing. *Journal of Electroanalytical Chemistry.* 2021 Jul 21; 895: 115547-7. https://doi.org/10.1016/j.jelechem.2021.115547
139. Gaikwad R, Thangaraj PR, Sen AK. Direct and rapid measurement of hydrogen peroxide in human blood using a microfluidic device. Scientific Reports. 2021 Feb 3; 11 (1). https://doi.org/10.1038/s41598-021-82623-4