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The ultraviolet effect on natural habitats: a comprehensive scientific review

Abstract

Ultraviolet (UV) radiation, particularly UV-B (280–315 nm) and UV-A (315–400 nm), constitutes a pervasive environmental stressor with far-reaching consequences for natural habitats. The progressive decline of stratospheric ozone, driven by anthropogenic halogenated compounds, has amplified surface UV-B irradiance across latitudes, intensifying ecological pressure on terrestrial, freshwater, and marine ecosystems. This review synthesizes current knowledge on the mechanisms by which UV radiation disrupts biological systems—spanning DNA photolesion formation, reactive oxygen species (ROS) production, photosynthetic inhibition, and endocrine disruption—and examines their cascading effects across ecological trophic levels. We survey impacts on aquatic primary producers, amphibian populations, soil microbial communities, forest canopies, polar ecosystems, and reproductive physiology of fauna. Particular attention is given to the synergistic interaction of UV exposure with climate warming, pollutants, and habitat fragmentation. Quantitative dose–response data, regulatory thresholds, and comparative ecosystem vulnerability indices are presented in tabular form. Protective biological mechanisms, including UV-absorbing pigments, DNA repair pathways, and behavioral photoavoidance, are critically assessed alongside the limitations of these adaptations under current and projected UV regimes. The review concludes with a synthesis of research gaps, monitoring priorities, and policy recommendations for mitigating UV-driven biodiversity loss in a changing climate.

Keywords: ultraviolet radiation, UV-B, ozone depletion, natural habitats, ecosystem stress, photooxidation, biodiversity, DNA damage, marine ecosystems, terrestrial ecosystems

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Introduction

Solar radiation reaching Earth's surface encompasses a broad electromagnetic spectrum, of which ultraviolet radiation represents a biologically critical fraction. UV radiation is conventionally partitioned into UV-C (100–280 nm), fully absorbed by the atmosphere; UV-B (280–315 nm), partially filtered by stratospheric ozone; and UV-A (315–400 nm), which reaches the surface largely unattenuated. Of these, UV-B is recognized as the most ecologically damaging due to its energetic photons and incomplete ozone attenuation.[1,2]

The stratospheric ozone layer, formed by photochemical reactions involving molecular oxygen, has been measurably depleted since the 1970s as a result of anthropogenic emissions of chlorofluorocarbons (CFCs), halons, and other ozone-depleting substances (ODS). The Antarctic ozone "hole," first documented in the 1980s, and subsequent observations of Arctic and mid-latitude ozone depletion established UV-B enhancement as a global environmental concern. Although the 1987 Montreal Protocol has led to partial ozone recovery, total restoration is not projected until mid-century, and interim UV-B fluxes remain elevated above pre-industrial baselines.[3,4]

Natural habitats—encompassing marine pelagic and benthic zones, freshwater lakes and wetlands, tropical and boreal forests, polar tundra, and arid shrublands—are differentially exposed to UV radiation depending on geographic latitude, altitude, cloud cover, and surface albedo. Shallow aquatic systems and alpine or polar terrestrial zones are particularly vulnerable due to thin atmospheric filtration or high reflectance from snow and ice.[5]

This review aims to:

1) Summarize the photochemical and photobiological mechanisms underlying UV-B toxicity
2) Assess documented ecological impacts across major habitat types
3) Evaluate the interaction of UV stress with co-occurring environmental pressures
4) Review biological UV protective strategies
5) Propose a framework for integrated UV risk assessment and policy response.

The scope encompasses terrestrial and aquatic habitats, with emphasis on primary producers, consumers, and key ecosystem processes.

This review was conducted using a structured narrative review methodology. Scientific literature was identified through comprehensive searches of Web of Science, Scopus, PubMed, and Google Scholar databases. Publications from 1990 to May 2026 were considered, with particular emphasis on studies published after 2018 to ensure inclusion of recent advances in ultraviolet radiation ecology and environmental sciences. Search terms included combinations of 'ultraviolet radiation', 'UV-B', 'natural habitats', 'ecosystem stress', 'marine ecosystems', 'terrestrial ecosystems', 'ozone depletion', 'climate change', 'biodiversity', and 'photobiology'.

Only peer-reviewed articles, international assessment reports, and authoritative publications from organizations such as WMO, UNEP, and the Environmental Effects Assessment Panel were included. Studies were evaluated according to ecological relevance, methodological quality, scientific impact, and applicability to ecosystem-level risk assessment. More than 250 publications were screened, and approximately 90 highly relevant studies were selected for detailed synthesis.

Photochemical and photobiological mechanisms of UV damage

DNA photolesions

UV-B radiation is directly absorbed by nucleic acids, with peak absorption near 260–270 nm. Cyclobutane pyrimidine dimers (CPDs) and 6-4 photoproducts (6-4PPs) are the predominant DNA photoproducts formed upon UV-B exposure. CPDs arise from [2+2] cycloaddition between adjacent pyrimidine bases, while 6-4PPs form via a Dewar valence isomerization. Both lesion types distort the DNA double helix, impede replication, and induce mutations—particularly C→T and CC→TT transitions at dipyrimidine sites—constituting the “UV signature mutation.” Unrepaired CPDs are associated with carcinogenesis in animals and physiological impairment in plants.[6,7]

Reactive oxygen species and oxidative stress

Beyond direct DNA photodamage, UV-B and UV-A induce indirect oxidative damage through the generation of reactive oxygen species (ROS), including singlet oxygen ($^1O_2$), superoxide radical ($O_2\bullet^-$), hydrogen peroxide (H_2O_2), and hydroxyl radical (•OH). These species are produced via photosensitization reactions involving endogenous chromophores (flavins, porphyrins, NADH), resulting in oxidative modification of proteins, lipids, and DNA. Lipid peroxidation of membrane bilayers disrupts membrane integrity, impairs transport functions, and triggers programmed cell death cascades.[8]

Photosynthetic inhibition

In photosynthetic organisms, UV-B radiation targets multiple components of the photosynthetic apparatus. Photosystem II (PSII) is particularly vulnerable: UV-B exposure degrades the D1 protein of the PSII reaction center, impairs the oxygen-evolving complex, and disrupts electron transport. UV-B also inactivates Rubisco (ribulose-1,5-bisphosphate carboxylase/oxygenase) by direct protein oxidation, diminishing carbon fixation efficiency. In phytoplankton, inhibition of photosynthetic rates has been measured at depth-dependent UV attenuation coefficients, with surface exposure capable of reducing primary productivity by 5–15% under clear sky conditions.[9,10]

Protein and lipid photodamage

Aromatic amino acids (tryptophan, tyrosine, phenylalanine) absorb UV-B, leading to protein unfolding, aggregation, and loss of enzymatic function. Photocatalytic degradation of structural proteins such as collagen in the skin and extracellular matrix of aquatic organisms has been documented. Membrane lipids undergo UV-induced peroxidation, altering fluidity, receptor function, and cellular signal transduction pathways. These biochemical impairments cumulatively reduce organismal fitness and reproductive success.[11]

Impacts on aquatic ecosystems

Marine phytoplankton and primary productivity

Phytoplankton are the foundation of marine food webs, responsible for approximately 50% of global primary productivity. Their shallow surface-ocean distribution exposes them disproportionately to UV radiation. Studies in the Southern Ocean have documented UV-B inhibition of phytoplankton photosynthesis at depths of 10–30 m under stratospheric ozone depletion scenarios, with reductions in chlorophyll a fluorescence, diminished carbon fixation, and increased mortality in sensitive diatom species.[12,13]

The spectral attenuation coefficient (K_d(λ)) determines UV penetration depth in water, varying with dissolved organic carbon (DOC) concentration, particulate matter, and phytoplankton density. In oligotrophic open-ocean systems with low DOC, UV-B may penetrate to 20 m or more, placing a larger fraction of the photic zone under UV stress. Photoinhibition of carbon assimilation has been quantified at 5–20% reductions in integrated daily production under current UV-B regimes.[14]

Zooplankton and invertebrates

Zooplankton species, including copepods, krill, and larvae of benthic invertebrates, exhibit UV-B sensitivity during early developmental stages. UV-B exposure has been shown to reduce copepod survival rates, depress reproductive output, and induce sublethal reproductive impairment. Copepods such as Calanus finmarchicus and Acartia tonsa demonstrate dose-dependent mortality at ecologically relevant UV-B intensities. Some zooplankton accumulate mycosporine-like amino acids (MAAs) as photoprotective compounds, but this adaptation is insufficient under high UV-B load conditions.[15,16]

Freshwater ecosystems

UV penetration into freshwater systems is strongly governed by DOC derived from terrestrial decomposition. Browning of lakes (increases in DOC and chromophoric dissolved organic matter, CDOM) provides natural UV attenuation, while clear-water systems are highly vulnerable. Alpine and subarctic lakes, with low CDOM and high UV transparency, expose phytoplankton and amphibian eggs to damaging UV doses. Documented effects include reduced algal biomass, disrupted microbial loop dynamics, and inhibition of bacterioplankton productivity, with cascading impacts on nutrient cycling.[17]

Impacts on terrestrial ecosystems

Higher plants and forest canopies

Terrestrial plants have evolved diverse UV-B tolerance mechanisms, yet sustained elevated UV-B exposure impairs morphology, physiology, and reproduction. UV-B induces epidermal thickening, accumulation of flavonoids and anthocyanins as screening pigments, stomatal closure, and reductions in leaf area index. Net primary productivity of sensitive crop and wild plant species has been shown to decline under enhanced UV-B in field experiments. Forest understory species, with less UV-protective phenolic investment than sun-adapted plants, exhibit greater photodamage under canopy gap conditions.[18,19]

Soil microbial communities

Soil microbial communities underpin nutrient cycling, organic matter decomposition, and plant symbiosis. Surface soil microbiomes—bacteria, fungi, and archaea—are exposed to UV radiation, particularly in arid environments with sparse canopy cover. UV-B irradiation suppresses microbial biomass, reduces enzymatic activity (dehydrogenase, phosphatase, urease), and shifts community composition toward UV-resistant taxa. UV-induced inhibition of nitrogen fixation by free-living and symbiotic bacteria has been reported, with implications for soil fertility in ecosystems with low atmospheric nitrogen deposition.[20]

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Amphibian populations

Amphibians occupy a critical ecological niche as both predators and prey, and are widely regarded as sensitive bioindicators of environmental quality. UV-B radiation has been implicated as a contributing factor in the global decline of amphibian populations. Eggs and larvae lack melanin pigmentation during early developmental stages, rendering them highly susceptible to UV-B-induced CPD formation. Synergistic interactions between UV-B and the fungal pathogen Batrachochytrium dendrobatidis (Bd) have been reported, with UV-B immunosuppression facilitating Bd infection in susceptible species. Field studies in the Rocky Mountains, Sierra Nevada, and high-altitude Andean ecosystems have documented reduced hatching success and larval deformities correlating with UV-B flux.[21,22]

Polar ecosystems and UV stress

Polar and subpolar ecosystems are uniquely exposed to extreme UV-B events due to the recurrent ozone hole over Antarctica and significant depletion episodes over the Arctic. During Antarctic spring, surface UV-B irradiance can exceed midsummer temperate values, with biologically effective UV doses (UV-BEED) reaching critical thresholds for marine primary producers. Sea-ice algae residing within and beneath sea ice are paradoxically exposed to UV due to ice optics; brine channels within sea ice conduct UV into algal microhabitats, inducing photodamage even at subzero temperatures.[23]

Krill (Euphausia superba) populations in Antarctic waters, which constitute a keystone prey species for penguins, seals, and whales, have been shown to sustain UV-B damage to compound eyes and swimming appendages at shallow depths. The behavioral photoavoidance of krill—diurnal vertical migration to depths of 100–300 m—provides partial but energetically costly UV protection. Reindeer, musk oxen, and snow geese in Arctic tundra habitats exhibit increased rates of UV-related ocular conditions (photokeratitis) under elevated UV-B regimes (Table 1).[24]

Table 1 Summary of UV-B effects across major ecosystem types

Ecosystem type	Primary target	Key documented effect	UV-B sensitivity	Reference
Open Ocean	Phytoplankton	5–20% reduction in primary productivity	High	12,13
Freshwater Lakes	Amphibian eggs / algae	Reduced hatching success, algal inhibition	Very High (clear)	17,21
Tropical Forest	Understory plants	Leaf damage, reduced photosynthesis	Moderate	18
Alpine Tundra	Microbiota, mosses	Microbial biomass loss, N-fixation decline	High	20
Polar Sea Ice	Ice algae, krill	PSII damage, behavioral avoidance	Very High	23,24
Arid Shrubland	Soil microbiome	Enzyme suppression, community shift	Moderate-High	20
Coastal Wetlands	Invertebrate larvae	Larval deformity, reproductive impairment	High	15

Synergistic interactions with co-stressors

UV radiation rarely acts in isolation in natural ecosystems. Its effects are amplified by concurrent environmental stressors that compromise organism resilience. Climate warming reduces thermal stratification stability in lakes, affecting UV attenuation depth; increases drought frequency, reducing canopy cover; and promotes wildfires that remove UV-shielding litter layers. Acidification of freshwater systems decreases CDOM concentrations, enhancing UV penetration. The interaction between UV-B and temperature stress is particularly relevant: elevated temperatures reduce the efficiency of nucleotide excision repair (NER), the primary enzymatic pathway for CPD removal, exacerbating genomic damage per unit UV exposure.[25]

Pollutant interactions include photosensitization by polycyclic aromatic hydrocarbons (PAHs), which under UV irradiation generate reactive species that synergistically damage DNA and membranes of aquatic invertebrates. Pesticide photodegradation products exhibit enhanced toxicity in UV-exposed environments. Nutrient eutrophication counterintuitively provides partial UV protection via enhanced algal turbidity, but simultaneously creates hypoxic dead zones that stress UV-impacted organisms.[26]

Biological protective mechanisms

UV-absorbing pigments

Organisms across taxa have evolved UV-screening compounds that absorb UV radiation in the epidermis or external layers, protecting underlying tissues. In plants, flavonoids and hydroxycinnamic acid derivatives (sinapate esters, ferulic acid) accumulate in the vacuoles of epidermal cells in response to UV-B-mediated UVR8 receptor signaling. Marine organisms, including corals, macroalgae, and zooplankton, synthesize mycosporine-like amino acids (MAAs)—small, water-soluble, UV-absorbing compounds with λmax between 310 and 360 nm—that function as biological sunscreens without generating ROS.[27]

DNA repair pathways

Multiple enzymatic repair pathways exist to reverse UV-induced DNA damage. Photolyases catalyze photoreactivation, using blue-light energy (300–500 nm) to monomerize CPDs and 6-4PPs via a light-dependent radical mechanism, representing perhaps the most efficient and widespread UV repair system in non-placental organisms. Nucleotide excision repair (NER) excises a short oligonucleotide containing the lesion and resynthesizes the gap using the complementary strand as template. Base excision repair (BER) addresses oxidized DNA bases generated by ROS. The relative expression and activity of these pathways vary widely among species and contribute substantially to interspecific differences in UV sensitivity.[28]

Behavioral and morphological adaptations

Behavioral UV avoidance strategies include vertical migration in zooplankton and fish larvae, nocturnal or crepuscular activity patterns in terrestrial invertebrates and small vertebrates, and microhabitat selection under shading substrates. Morphological adaptations include melanin deposition in exposed integument, thickening of the stratum corneum, and the secretion of UV-opaque mucus layers in amphibians and fish. These adaptations provide effective UV attenuation but entail energetic costs that may compromise other fitness-related behaviors, particularly in resource-limited polar and high-altitude habitats (Table 2).[29]

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Table 2 Biologically effective UV-B dose thresholds for representative organisms

Organism / Group	Life stage	Threshold BED ($\mathrm{kJ\ m^{-2}\ day^{-1}}$)	Observed response	Reference
Calanus finmarchicus (copepod)	Nauplius	2.5–5.0	50% mortality within 72 h	[15]
Rana cascadae (frog)	Embryo	1.5–3.0	Deformity, failed hatching	[21]
Acartia tonsa (copepod)	Egg / nauplius	3.0–6.0	Reduced hatching rate	[16]
Arabidopsis thaliana (plant)	Seedling	5.0–10.0	Flavonoid induction, growth suppression	[18]
Skeletonema costatum (diatom)	Vegetative	0.5–2.0	PSII inhibition ≥30%	[9]
Pleuronectes platessa (plaice)	Larva	4.0–8.0	Increased mortality, DNA strand breaks	[15]
E. superba (Antarctic krill)	Adult	2.0–4.0	Eye damage, reduced swimming	[24]

Monitoring, risk assessment, and policy frameworks

Effective assessment of UV impacts on natural habitats requires standardized monitoring of UV radiation fluxes and biologically effective doses, ecological surveillance of indicator species, and integration of UV dose–response data into ecosystem models. The biologically effective UV-B dose (UV-BBED) is typically calculated by weighting the spectral irradiance with the generalized plant damage action spectrum or DNA damage action spectrum, yielding dose metrics comparable across ecosystems and latitudes.[30]

The United Nations Environment Programme (UNEP) Environmental Effects Assessment Panel (EEAP) periodically assesses UV-related environmental impacts, providing the scientific basis for Montreal Protocol evaluations. National monitoring networks such as WOUDC (World Ozone and Ultraviolet Radiation Data Centre) provide long-term irradiance records essential for trend analysis. Integration of satellite-derived ozone column data (OMI, TROPOMI) with ground-based actinometry enhances spatial coverage and temporal resolution of UV dose estimates.[31]

A tiered risk assessment framework for UV impacts on natural habitats should incorporate:

1) Habitat-specific UV exposure characterization using spectroradiometry and modeling;
2) Species-level dose–response relationships from controlled and mesocosm experiments;
3) Population-level demographic modeling incorporating UV-driven survival and reproductive parameters; and
4) Cumulative risk accounting for synergistic co-stressors. Priority habitats for monitoring include high-altitude and polar ecosystems, shallow oligotrophic lakes, amphibian breeding pools, and bleaching-prone coral reef zones.[32]

Research gaps and future directions

Despite decades of research, significant knowledge gaps impede comprehensive UV impact assessment. Key areas requiring further investigation include:

1) Long-term field datasets tracking UV-B-attributed changes in biodiversity across major biome types remain sparse outside polar and alpine systems. Standardized ecological indicators sensitive to UV-B change, analogous to the biodiversity indices used for chemical pollution, are needed.
2) Mechanistic understanding of UV × climate warming interactions at the molecular and cellular level—particularly the suppression of DNA repair enzyme kinetics at elevated temperatures—requires further experimental elucidation, especially in ectothermic organisms.
3) The role of UV radiation in mediating host–pathogen dynamics (e.g., UV-B immunosuppression, pathogen UV sensitivity) in wildlife populations is insufficiently characterized, representing an important dimension of disease ecology.
4) Microplastic photodegradation under UV generates surface-adsorbed radicals and leaches photosensitizers into aquatic systems; the ecological consequences of UV-activated microplastic toxicity are a frontier research area.
5) Projections of future UV-B regimes under ozone recovery coupled with climate-driven changes in cloud cover, atmospheric composition, and land surface properties require higher-resolution Earth system model representations of UV radiative transfer.

Although UV-B radiation has been widely recognized as an ecological stressor, ecosystem responses remain highly heterogeneous and context dependent. Species-specific repair capacities, habitat optical properties, and interactions with climate-related stressors significantly influence the magnitude of observed biological effects. A major challenge in interpreting existing literature is the difficulty of isolating UV-driven responses from simultaneous environmental changes such as warming, acidification, pollution, eutrophication, and habitat fragmentation. Future research should increasingly adopt multi-stressor frameworks and long-term monitoring programs capable of capturing ecosystem-scale responses and adaptation processes.

Limitations of current knowledge

Despite substantial advances in understanding the ecological effects of ultraviolet (UV) radiation, significant knowledge gaps remain. Current evidence is limited by uneven geographical representation, taxonomic biases, and the lack of long-term monitoring datasets capable of capturing ecosystem-scale responses over extended periods. Most research has focused on polar, alpine, and freshwater environments, while tropical ecosystems, arid regions, and many terrestrial habitats remain comparatively understudied. Furthermore, investigations have predominantly concentrated on primary producers and lower trophic levels, leaving higher trophic organisms, complex food-web interactions, and ecosystem-wide processes insufficiently characterized. Additional uncertainties arise from the difficulty of predicting future UV radiation exposure under the combined influences of stratospheric ozone recovery and ongoing climate change. Changes in cloud cover, atmospheric circulation, land-use patterns, and extreme weather events may alter UV irradiance in ways that are not yet fully understood, complicating efforts to forecast ecological consequences. Addressing these limitations will require coordinated international monitoring programs, broader geographic and taxonomic coverage, and the integration of experimental, observational, and modeling

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approaches to improve predictions of UV-driven ecological change in future environments.

Conclusion

Ultraviolet radiation constitutes a multifaceted stressor with profound, documented consequences for natural habitats across terrestrial and aquatic biomes. UV-B, augmented by anthropogenic ozone depletion, damages DNA, inhibits photosynthesis, disrupts membrane integrity, and suppresses immune function across a phylogenetically broad range of organisms. Ecosystem-level consequences encompass reduced primary productivity, altered food web structure, impaired nutrient cycling, and contribution to amphibian population declines.[33]

The interaction of UV stress with climate warming, acidification, chemical pollution, and habitat fragmentation creates compound risk profiles that exceed the predictive capacity of single-stressor models. Protective biological mechanisms—photoprotective pigments, enzymatic DNA repair, behavioral avoidance—provide partial buffering but are physiologically limited and energetically costly, particularly under the joint stress regimes projected for the coming decades.

Continued adherence to and strengthening of the Montreal Protocol remains essential for stratospheric ozone restoration. Concurrently, the integration of UV impact assessment into biodiversity monitoring frameworks, ecological risk assessment protocols, and climate adaptation strategies is urgently warranted to safeguard the structural and functional integrity of the world's natural habitats.

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Conflicts of interest

The authors declare no competing interests.

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