Healthy People, Healthy Oceans

Sunscreen as a case study in unintended human and marine consequences

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The proposition

Products designed to protect people can have consequences beyond their intended use. Sunscreen offers a specific, testable example: ingredients can reach coastal waters, interact with marine organisms and coexist with nutritional consequences of reduced UV exposure. The question is whether we can retain effective human protection while reducing avoidable harm across both systems.

This paper treats sunscreen as a case study in unintended consequences, not as an explanation for all ocean degradation. Its primary focus is coral health and chemical exposure. Vitamin D, human absorption and cellular resilience supply a second assessment: a health intervention should be evaluated across relevant benefits and harms, including outcomes outside its immediate target.

The evidence establishes some individual effects and leaves other links unresolved. Multiple pressures may interact, but a multiplicative or compounding effect must be demonstrated rather than assumed. This paper proposes integrated assessment without claiming that sunscreen's overall harms exceed its benefits or that every formulation has the same risk.

1. Why coral belongs in the Blue COP conversation

Dermot O'Gorman's public call for a Blue COP connects ocean conservation with climate, biodiversity and livelihoods. His separate Blue Corridors contribution emphasises the connections between marine ecosystems, communities and economies. This paper offers one contribution within that wider agenda; it does not attribute its sunscreen conclusions to him. COP31 is scheduled for Antalya, Türkiye, on 9–20 November 2026. [1,2,24]

WWF's own explanation supplies the ecological foundation. Its coral briefing describes reefs as habitat supporting roughly a quarter of marine life despite their small area, and explains their contribution to food, livelihoods and coastal protection. Coral polyps and their symbiotic algae form a living partnership; reef-building corals also create the physical structure that provides habitat. Persistent loss of living coral can therefore affect the wider reef community and the people depending on it. [25]

UNEP likewise identifies reef biodiversity, food security and coastal protection as reasons for conservation. These are ecosystem functions, not merely visual qualities of an attractive dive site. Reefs are one important component of ocean health, not a proxy for every process in the global ocean. [26]

The logic for examining sunscreen is consequently straightforward: if reefs support those functions, avoidable chemicals that impair coral survival, reproduction or recovery deserve investigation. That inference is ours, built on WWF's reef rationale and the studies below. It is not evidence that WWF endorses a sunscreen ban. NOAA identifies warming, disease and other pressures; local chemical stewardship complements rather than replaces action on larger threats. [3]

2. The organic-filter evidence: from molecules to coral effects

Oxybenzone (benzophenone-3). Downs and colleagues tested coral larvae and cultured cells, reporting deformity, bleaching, DNA lesions and abnormal skeletal development. Their endpoints varied markedly in sensitivity. The frequently repeated 0.062 micrograms per litre result concerns a cultured-cell endpoint, not a universal concentration killing intact reefs. Vuckovic and colleagues subsequently identified phototoxic oxybenzone metabolites in anemones and a coral, linking effects to sunlight and symbiont status. These are specific mechanisms supporting hazard assessment, not a global attributable mortality estimate. [21,27]

Octocrylene. A metabolomics study exposed adult *Pocillopora damicornis* to 5–1,000 micrograms per litre. Polyp closure was prominent at 300 and above; tissue analysis identified fatty-acid conjugates and acylcarnitine changes consistent with disturbed mitochondrial metabolism. This is a direct marine connection to the cellular-energy question. It does not establish the same injury in humans or at every environmental concentration. A separate 2022 study in *Stylophora pistillata* also investigated derivatives and metabolic responses; it provides another species-specific line of inquiry, not an unrestricted replication claim. [28,29]

Octinoxate and complete products. A seven-day study compared octinoxate, octocrylene, their combination and sunscreen wash-off water in two adult coral species. In single-chemical tests, mortality and bleaching were observed only in the 1,000-microgram-per-litre octinoxate treatment of *Seriatopora caliendrum*. Product wash-off produced greater tissue accumulation and toxicity than the active ingredients alone under the tested conditions. The result points towards formulation and bioavailability, not an assumption that any two filters necessarily act synergistically. Those laboratory concentrations require comparison with measured coastal exposures. [30]

Homosalate, octisalate and avobenzone. A Hawaiian field study measured 13 filters across 19 sites and detected several in water, sediment and coral tissue. Homosalate and octisalate were prominent, demonstrating why attention cannot stop at oxybenzone. Detection establishes exposure, not injury caused by each detected compound. The search for this revision did not establish a comparable body of direct coral-outcome evidence for every filter. Uncertain ingredients should remain uncertain, rather than be designated harmless or uniformly toxic. [31]

Newer filters. Bemotrizinol, bisoctrizole, diethylamino hydroxybenzoyl hexyl benzoate and ethylhexyl triazone must not inherit the findings for oxybenzone simply because they are organic filters. The present targeted review does not certify their marine safety or demonstrate equivalent coral toxicity. Product-specific and ingredient-specific data gaps remain part of the research agenda. [22]

3. Nanoparticles are a distinct exposure question

A dissolved organic molecule is not the same material as a solid nanoparticle. Oxybenzone, octinoxate and octocrylene toxicity cannot automatically be described as nanoparticle toxicity. Some organic filters are particulate: bisoctrizole (MBBT) has a nano form assessed separately by the European scientific committee. Nanocarriers may also form part of a formulation, but their presence and properties need product-level evidence. The MBBT assessment reached a favourable dermal conclusion for specified conditions; that does not certify every particle or establish marine safety. [32]

Zinc oxide cannot be assigned blanket marine safety. A controlled study found bleaching with **uncoated zinc oxide nanoparticles** in *Acropora*, while the two modified titanium dioxide materials tested produced substantially fewer effects. This is a formulation-dependent comparison, not proof that all zinc sunscreens cause reef bleaching or all titanium products are environmentally safe. Particle coating, dissolution, concentration and exposure duration matter. [33]

4. Human effects: related questions, separate proof

The paired hypothesis is that chemical or particle exposure and reduced vitamin D synthesis may jointly affect the people using sunscreen. The two routes should be measured separately before an interaction is claimed. The human organic-filter absorption study establishes systemic exposure, not nanoparticle entry or clinical disease. The Sun-D trial establishes an effect on vitamin D status under its regimen, not a cancer outcome. The present review has not located a human study demonstrating that these two routes combine to cause greater disease than either route alone. [8,18]

The Australian TGA's 2025 ingredient review adds a relevant human assessment. Its model raised margin-of-safety concerns for homosalate and oxybenzone under maximum-concentration, whole-body exposure assumptions; five other assessed filters were considered low risk under its framework. It acknowledged incomplete data and uncertainty about human endocrine causation. This is a model-based assessment, not a clinical finding that all non-zinc sunscreens harm users, and it does not determine coral safety. [34]

Nutritional zinc must also be distinguished from zinc oxide on skin. Zinc-deficiency research and testosterone outcomes do not establish testicular-cancer prevention, prostate protection or hormonal benefits from topical zinc sunscreen. These benefits are not claimed in this paper. Human nanoparticle and zinc-ion findings are discussed below; route, chemical form and dose cannot be interchanged.

5. Exposure and ecological risk

Human absorption is measurable, but absorption alone is not a diagnosis of toxicity. In a randomised pharmacokinetic study of 48 adults, six organic sunscreen filters were assessed under single- and maximal-use conditions. Systemic absorption supported the need for further safety assessment; the study was not designed to establish cancer caused by the products. [18]

Mineral nanoparticles require separate analysis. A volunteer study found no penetration of intact zinc oxide nanoparticles into viable epidermis or local cellular toxicity under its tested conditions. A different isotope-tracing study detected small amounts of sunscreen-derived zinc in blood and urine

but could not determine whether absorbed zinc was particulate or dissolved. Neither finding should be stretched into “nothing enters the body” or “nanoparticles are proven to cause cancer”. [19,20]

The marine question changes the exposure setting and the organism. Vuckovic and colleagues demonstrated that sea anemones and a coral converted oxybenzone into phototoxic glucoside conjugates. The response involved light exposure and symbiotic algae, with greater vulnerability in anemones lacking symbionts. This supplies a mechanism and a reason to examine interacting stressors. It does not quantify global reef mortality attributable to sunscreen. [21]

The National Academies’ review identifies a need for ecological risk assessment of UV filters. This requires measured environmental concentrations, duration and frequency of exposure, species and life stage, formulation, transformation products and biologically relevant endpoints. Laboratory hazard establishes what can happen under tested conditions; field risk concerns how likely it is under actual exposure. [22]

Mineral formulations also warrant assessment: particle size, coatings, aggregation and dissolution can change environmental behaviour. A review of inorganic filters maps those issues. “Mineral”, “non-nano” and “reef-safe” should not substitute for formulation-specific evidence. [23]

6. From catchment to coral: the missing exposure connections

A coral experiences the chemical mixture delivered to its location over time, not the concentration printed on a sunscreen bottle or an isolated laboratory treatment. Riverine inputs, wastewater, coastal circulation, sediment processes and chemical transformations form a connected exposure pathway. Some processes dilute or remove a substance; others retain it, redistribute it or alter its availability. A systems assessment must allow for both directions rather than presume that complexity always increases harm.

This is not an entirely unrecognised research problem. The National Academies explicitly identifies water residence time, mixing, currents, stratification and sediment movement as determinants of UV-filter exposure. The contribution proposed here is to make the connections and validation requirements explicit when findings are translated into ecosystem claims. [35]

There are empirical and modelling examples. A Pearl River Estuary study investigated riverine organic-filter inputs to the South China Sea during the early pandemic; its particular sampling period limits extrapolation to normal activity. Another study measured filters in riverine and coastal sediments in that estuary. Neither location can stand in for all coastlines or establish damage to distant reefs without transport and biological evidence. [36,37]

A study in Spain combined UV-filter measurements in mussels with a hydrodynamic model and Lagrangian tracking of water parcels. This demonstrates an effort to connect organism measurements, circulation and possible sources. It is not a coral-outcome study, and a model successful at one estuary cannot be assumed to transfer without validation. [38]

A defensible catchment-to-reef assessment should connect five stages:

1. **Sources and timing:** distinguish swimmer wash-off, wastewater, river discharge and runoff. Measure pulses as well as seasonal conditions, and account for other uses of the same chemicals before attributing all detected material to sunscreen.

2. **Transport and retention:** represent the relevant river flow, tidal exchange, wave-driven circulation, coastal geometry and offshore currents. Establish where dilution, retention or repeated exposure is plausible instead of assuming uniform mixing.
3. **Chemical form and fate:** distinguish dissolved material, suspended particles, sediment-associated material and transformation products. Test whether settling, burial, resuspension or degradation materially changes the organism's exposure; do not assume every filter behaves identically.
4. **Biological exposure:** match the location, depth, life stage and exposure duration of the organism. A surface-water snapshot and a tissue measurement integrate different processes and time windows.
5. **Validation and uncertainty:** compare predicted concentrations and timing with independent observations in water, sediment and organisms. Carry uncertainty across the connections between models; avoid treating the output of each component as exact input to the next.

These are proposed assessment requirements, not a claim that each cited study measured all five stages. A technically sophisticated model can still omit a consequential connection. Conversely, a narrow model can be useful within its validated boundary. The test is whether the connected model predicts independent observations at the scale used for the decision.

The research hypothesis is therefore precise: **do omitted transport, transformation or biological interactions materially change estimated exposure and risk?** Evidence could reveal a local hotspot, a missed seasonal pulse or lower exposure than expected. We should report any of those outcomes. Laboratory hazard remains informative; translation to local risk requires this additional work.

7. What a Blue COP contribution could ask for

The following proposals are the author’s synthesis, not outcomes already validated by a trial or commitments attributed to WWF.

First, assess products across their use cycle. Human protection testing and marine exposure testing should be connected through realistic application, wash-off and wastewater scenarios. A filter name alone cannot represent every formulation. Comparisons should preserve equivalent human protective performance where feasible, so a lower environmental exposure is not purchased through an unmeasured loss of protection.

Second, monitor vulnerable settings. High-use, poorly flushed recreational waters are candidates for targeted monitoring. Sampling should record visitor activity, river discharge, water exchange, wave and tidal conditions, wastewater inputs and seasonal variation, with sediment sampling where relevant. Report detection limits, blanks and uncertainty. A single positive sample should not become a global pollution estimate.

Third, test multiple stressors. Candidate ecological experiments should compare product exposure with and without heat stress, using environmentally justified concentrations and suitable controls. Measure reproduction, recruitment and survival where feasible, not only short-term cellular responses. Negative findings belong in the public record alongside positive ones.

Fourth, include human nutritional outcomes. Evaluate vitamin D consequences when testing intensified sun-protection programmes, particularly sustained coverage and high-SPF use. Define whether the programme actually changes outdoor activity or only skin exposure. Record relevant

baseline characteristics and distinguish nutritional correction from a recommendation for extra UV exposure.

Fifth, examine practical combinations. Clothing can reduce treated skin area and potentially the amount of product used, but this is a hypothesis to measure rather than a guaranteed net benefit. Compare shade, garment coverage and appropriate topical protection on uncovered areas while assessing comfort, adherence, vitamin D and environmental release. Consider garment durability and shedding if making a full environmental claim.

Sixth, make claims auditable. Publish methods, funding, conflicts, adverse findings and changes to protocols. Marine safety claims should specify species, endpoints, exposure and the boundary of the conclusion. Human claims should identify whether they concern sunburn, cancer incidence, vitamin status or treatment. Product marketing should not collapse these into a single assertion of safety.

8. A bounded research agenda that could change the conclusion

An immediate feasible study would compare defined protection strategies over a seasonal cycle, with repeated measures of actual exposure, sunscreen quantity, coverage and 25-hydroxyvitamin D. Prespecified interaction analyses could examine baseline vitamin D, mineral intake, pigmentation, body composition and metabolic status. Some candidate mechanisms would require a nested tissue or cellular substudy rather than convenient but uninformative blood proxies.

Such a study could assess vitamin status and intermediate biological responses. It would generally be too short and small to establish cancer prevention. Long-term outcomes would require suitably powered follow-up, registry linkage or a separate design. The ethical comparator is usual protective practice, not deliberate harmful irradiation or untreated deficiency.

In parallel, environmental work should compare complete formulations at measured coastal concentrations, include appropriate controls and independently replicated outcomes, and connect wash-off data with field sampling. Human and ecological results should first be reported separately. A formal net-benefit analysis would need explicit assumptions and compatible measures; multiplying unrelated risk ratios would manufacture precision.

Several findings would strengthen the integrated hypothesis: reproducible interactions between nutrient state and UV-response pathways; demonstrated mediation from vitamin D changes through mitochondrial responses; and relevant clinical outcomes that survive confounding checks. Credible null results, failure to replicate, or absence of predicted mediation would weaken it. Evidence that a formulation has negligible ecological effects at realistic exposure should likewise alter its assessment.

A serious discussion leaves those possibilities open. It also recognises that a useful research programme can end with a narrower conclusion than the hypothesis that prompted it.

9. A contribution worth debating

The current evidence does not justify describing sunscreen-associated vitamin D changes as established cancer causation, mitochondrial health as immunity from UV, or sunscreen as the principal driver of reef decline. It does justify closer examination of consequences that can disappear when each discipline considers only its own endpoint.

The proposal for Blue COP is practical: protect the ocean while improving the quality of human-health and product decisions. Evaluate the whole exposure system, keep the strongest existing evidence visible and investigate the interactions that have not been adequately measured.

Responses are invited from marine scientists, photobiologists, clinicians, nutritional researchers, manufacturers and coastal communities: which evidence changes this assessment, which proposed comparisons are feasible, and which outcomes would make the greatest difference? Critique is most useful when it identifies a specific claim, supplies a source or dataset and explains the alternative interpretation.

Sources

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Method and disclosure

This is a targeted narrative evidence synthesis, not an exhaustive systematic review, new laboratory research or a new meta-analysis. Searches and source checks were conducted during preparation on 4 November 2026. Original studies were prioritised; environmental reviews map research gaps; additional regulatory assessments distinguish particle forms and human risk. Public institutional sources establish conference context and reef pressures. Some findings were checked at abstract level because complete text was not retrievable. No claim of complete risk-of-bias assessment, independent duplicate screening or peer review is made.

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