# Sunscreens Cause Coral Bleaching by Promoting Viral Infections

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BACKGROUND: Coral bleaching (i.e., the release of coral symbiotic zooxanthellae) has negative impacts on biodiversity and functioning of reef ecosystems and their production of goods and services. This increasing world-wide phenomenon is associated with temperature anomalies, high irradiance, pollution, and bacterial diseases. Recently, it has been demonstrated that personal care products, including sunscreens, have an impact on aquatic organisms similar to that of other contaminants.

OBJECTIVES: Our goal was to evaluate the potential impact of sunscreen ingredients on hard corals and their symbiotic algae.

METHODS: *In situ* and laboratory experiments were conducted in several tropical regions (the Atlantic, Indian, and Pacific Oceans, and the Red Sea) by supplementing coral branches with aliquots of sunscreens and common ultraviolet filters contained in sunscreen formula. Zooxanthellae were checked for viral infection by epifluorescence and transmission electron microscopy analyses.

RESULTS: Sunscreens cause the rapid and complete bleaching of hard corals, even at extremely low concentrations. The effect of sunscreens is due to organic ultraviolet filters, which are able to induce the lytic viral cycle in symbiotic zooxanthellae with latent infections.

CONCLUSIONS: We conclude that sunscreens, by promoting viral infection, potentially play an important role in coral bleaching in areas prone to high levels of recreational use by humans.

KEY WORDS: bleaching, corals, sunscreens, UV filters, viruses. *Environ Health Perspect* 116:441–447 (2008). doi:10.1289/ehp.10966 available via *http://dx.doi.org/* [Online 3 January 2008]

Coral reefs are among the most biologically productive and diverse ecosystems in the world, representing hot spots of marine biodiversity, and directly sustaining half a billion people (Moberg and Folke 1999; Wilkinson 2004). Approximately 60% of coral reefs are currently threatened by several natural and anthropogenic impacts (Hughes et al. 2003; Pandolfi et al. 2003). Over the last 20 years, massive coral bleaching (i.e., loss of symbiotic zooxanthellae hosted within scleractinian corals) has increased dramatically, both in frequency and spatial extent (Hoegh-Guldberg 1999; Hughes et al. 2003; Knowlton 2001). This phenomenon has been associated with positive temperature anomalies, excess ultraviolet (UV) radiation or altered available photosynthetic radiation, and presence of bacterial pathogens and pollutants (Brown et al. 2000; Bruno et al. 2007; Douglas 2003; Glynn 1996; Jones 2004).

Production and consumption of personal care and cosmetic sun products are increasing worldwide, reaching unexpected levels, with potentially important consequences on environmental contamination. The release of these products is also linked with the rapid expansion of tourism in marine coastal areas (Wilkinson 2004). Chemical compounds contained in sunscreens and other personal care products have been demonstrated to reach detectable levels in both fresh and seawater systems (Daughton and Ternes 1999; Giokas et al. 2007). These compounds are expected to be potentially harmful for the environment; hence, the use of sunscreen products is now banned in a few popular tourist destinations, for example, in marine ecoparks in Mexico, and in some semi-enclosed transitional systems (Xcaret 2007; Xel-ha 2007). Because sunscreens are lipophilic, their UV filters can bioaccumulate in aquatic animals (Giokas et al. 2007) and cause effects similar to those reported for other xenobiotic compounds (Balmer et al. 2005; Daughton and Ternes 1999). Paraben preservatives and some UV absorbers contained in sunscreens have estrogenic activity (Daughton and Ternes 1999; Schlumpf et al. 2004). In addition it has been demonstrated that several sunscreen agents may undergo photodegradation, resulting in the transformation of these agents into toxic by-products (Giokas et al. 2007, and literature therein).

Recently, it has also been demonstrated that sunscreens have an impact on marine bacterioplankton (Danovaro and Corinaldesi 2003), but there is no scientific evidence for their impact on coral reefs.

To evaluate the potential impact of sunscreen ingredients on hard corals and their symbiotic algae, we conducted several independent *in situ* studies with the addition of different concentrations of sunscreens to different species of *Acropora* (one of the most common hard-coral genus), *Stylophora pistillata*, and *Millepora complanata*. These studies were performed from 2003 to 2007 in different areas of the world, including the Celebes Sea (Pacific Ocean), the Caribbean Sea (Atlantic Ocean), and the Andaman Sea and the Red Sea (Indian Ocean).

## Materials and Methods

*Study areas and experimental design.* *In situ* experiments were conducted in four coral reef areas: Siladen, Celebes Sea (Indonesia, Pacific Ocean); Akumal, Caribbean Sea (Mexico, Atlantic Ocean); Phuket, Andaman Sea (Thailand, Indian Ocean), and Ras Mohammed, Red Sea (Egypt, Indian Ocean). Nubbins of *Acropora* spp. (~ 3–6 cm) were collected, washed with virus-free seawater filtered onto 0.02-μm membranes (Anotop syringe filters; Whatman, Springfield Mill, UK), immersed in polyethylene Whirl-pack bags (Nasco, Fort Atkinson, WI, USA) filled with 2 L virus-free seawater, and incubated *in situ*. Additional experiments were also performed with other hard coral genera: *S. pistillata* and *M. complanata*. Replicate sets containing nubbins from different colonies ($n = 3$, including more than 300 polyps each) were supplemented with aliquots of sunscreens (at final quantities of 10, 33, 50, and 100 μL/L seawater) and compared with untreated systems (used as controls). Corals were incubated at the same depth of donor colonies at *in situ* temperature (Table 1). During two experiments conducted in the Red Sea and in the Andaman Sea, we tested the effects on coral bleaching of the same chemical filters and preservatives contained in the sunscreen formula of different brands (Tables 1 and 2). Subsamples (50 mL) of seawater surrounding coral nubbins were collected at 12-hr intervals and fixed in 3% glutaraldehyde for subsequent

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# Active ingredients in sunscreens act as topical penetration enhancers for the herbicide 2,4-dichlorophenoxyacetic acid.

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## Author information

## Abstract

Agricultural workers are encouraged to use sunscreen to decrease the risk of UV-related skin cancer. Our previous studies have shown certain commercial sunscreens to be penetration enhancers. The focus of this project is to determine whether active ingredients in sunscreen formulations (i.e., the UV absorbing components and insect repellants for the sunscreen/bug repellant combinations) also act as dermal penetration enhancers for herbicides in vitro. The total percentages of 2,4-dichlorophenoxyacetic acid (2,4-D) penetrating through hairless mouse skin in 24 h ranged from 54.9 +/- 4.7 for the no sunscreen control to 86.9 +/- 2.5 for padimate-o. Of the active ingredients tested (7.5% octyl methoxycinnamate, 7% octocrylene, 0.6% oxybenzone, 5% homosalate, 5% octyl salicylate, 8% padimate-o, 10% sulisobenzone, and 9.5% and 19% N,N-diethyl-m-toluamide [DEET]), all but octocrylene led to a significant increase in total 2,4-D penetration as compared to the control ($P < 0.05$), and only octocrylene and oxybenzone did not significantly decrease the corresponding lag time. Octyl salicylate ($P < 0.01$) and octyl methoxycinnimate ($P < 0.05$) significantly increased the 3H2O penetration across mouse skin, indicating physical damage to the stratum corneum. Additional studies demonstrated that the penetration enhancement seen across hairless mouse skin also occurred with human skin. Thus, the active ingredients of sunscreen formulations enhance dermal penetration of the moderately lipophilic herbicide 2,4-D.

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## Publication types, MeSH terms, Substances

## LinkOut - more resources

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# Transdermal absorption of the herbicide 2,4-dichlorophenoxyacetic acid is enhanced by both ethanol consumption and sunscreen application.

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## Abstract

Xenobiotics absorption is a health concern and skin is a major exposure site for many of these chemicals. Both alcohol consumption and topical sunscreen application act as transdermal penetration enhancers for model xenobiotics. The effect of combining these two treatments on transdermal absorption of the herbicide 2,4-dichlorophenoxyacetic acid (2,4-D) was therefore examined. Skin from rats ingesting low (1.5 g/kg) medium (4.3 g/kg) or high (6 g/kg) ethanol doses or saline control was treated with a commercially available sunscreen containing titanium dioxide and octyl methoxycinnimate and transdermal absorption of 2,4-D was monitored. Ethanol increased penetration by a factor of 1.9, 2.0 and 2.5 for animals treated with 1.5, 4.3 and 6 g/kg respectively, demonstrating an ethanol-induced dose response. Sunscreen application to skin from ethanol gavaged rats caused 2,4-D absorption above that induced by ethanol alone by an additional factor of 1.3, 2.1 and 2.9 for 1.5, 4.3 and 6 g/kg respectively. Comparing 2,4-D transdermal absorption after exposure to both ethanol and sunscreen with a theoretical value (sum of penetration after ethanol or sunscreen treatment) demonstrates that these two treatments enhance additively at the higher doses tested. Results of this study emphasize the importance of limiting excessive alcohol consumption in individuals with potential herbicide exposure rather than discouraging the use of sunscreens, since the consequences of UV-induced skin cancer are far more series than the risks that would be associated with observed increases in chemical exposure.

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## Publication type, MeSH terms, Substances, Grant support

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# *CDC: Americans Carry Body Burden of Toxic Sunscreen Chemical*

*TUESDAY, MARCH 25, 2008* 25 MAR 2008 A new study by the U.S. Centers for Disease Control (CDC) reveals that 97% of Americans are contaminated with a widely-used sunscreen ingredient called oxybenzone that has been linked to allergies, hormone disruption, and cell damage. A companion study published just one day earlier revealed that this chemical is linked to low birth weight in baby girls whose mothers are exposed during pregnancy. Oxybenzone is also a penetration enhancer, a chemical that helps other chemicals penetrate the skin.

**VIEW NAME-BRAND PRODUCTS THAT CONTAIN OXYBENZONE:**

**Sunscreens (588 products)**
**facial moisturizer/treatment**
**other products with SPF**
**lip balm**
**lipstick**
**moisturizer**
**anti-aging creams**
**conditioner**
**fragrance for women**

Although oxybenzone is most common in sunscreen, companies also use the chemical in at least 567 other personal care products.

Environmental Working Group identified nearly 600 sunscreens sold in the U.S. that contain oxybenzone, including products by Hawaiian Tropic, Coppertone, and Banana Boat (**see the full list of 588 sunscreens here**) as well as 172 facial moisturizers, 111 lip balms, and 81 different types of lipstick.

The Food and Drug Administration has failed miserably in its duty to protect the public from toxic chemicals like oxybenzone in personal care products. At the request of industry lobbyists, including Supreme Court Chief Justice John Roberts, who represented the Cosmetic Toiletry and Fragrance Association, the agency has delayed final sunscreen safety standards for nearly 30 years. FDA issued a new draft of the standards last October under pressure from EWG, but continues to delay finalizing them at the behest of the regulated industry.

EWG research shows that 84% of 910 name-brand sunscreen products offer inadequate protection from the sun, or contain ingredients, like oxybenzone, with significant safety concerns.

The last safety review for oxybenzone was done in the 1970s, and does not reflect a wealth of information developed since that time indicating increased toxicity concerns and widespread human exposure. A recent review in the European Union found that sufficient data were not available to assess if oxybenzone in sunscreen was safe

Environmental Working Group again calls on FDA to review the safety of oxybenzone, given this new data on widespread contamination of the U.S. population, and to finalize its sunscreen safety standards so that consumers

Top scientists from CDC published results March 21, 2008 from a national survey of 2,500 Americans, age 6 and up, showing that oxybenzone readily absorbs into the body and is present in 97% of Americans tested (Calafat 2008). Oxybenzone, also known as benzophenone-3, was detected in the urine of nearly every study participant. Typically, women and girls had higher levels of oxybenzone in their bodies than men and boys, likely a result of differences in use of body care products including sunscreens.

A companion study released a day earlier revealed that mothers with high levels of oxybenzone in their bodies were more likely to give birth to underweight baby girls (Wolff 2008). Low birth weight is a critical risk factor linked to coronary heart disease, hypertension, type 2 diabetes, and other diseases in adulthood (Lau 2004).

## Oxybenzone damages and penetrates the skin

Among common sunscreen chemicals, oxybenzone is most likely to be associated with allergic reactions triggered by sun exposure. In a study of 82 patients with photoallergic contact dermatitis, over one quarter showed photoallergic reactions to oxybenzone (Rodriguez 2006); another study reported 1 in 5 allergic reactions to photopatch tests resulted from exposure to oxybenzone (Bryden 2006).

Sunlight also causes oxybenzone to form free radical chemicals that may be linked to cell damage, according to 2 of 3 studies (Allen 1996; Serpone 2002; Hanson 2006).

A less visible but more alarming concern, this chemical absorbs through the skin in significant amounts, as indicated by the CDC study. A previous biomonitoring study reported that 96% of 6 to 8 year old girls had detectable amounts of oxybenzone in their urine (Wolff 2007). An earlier study detected oxybenzone in the urine of all 30 adult participants (Ye 2005).

Studies on human volunteers indicate a wide variation in the level of oxybenzone absorbed into the body, with some individuals absorbing at least 9% of the applied dose, as measured in excretions in urine (Hayden 1997; Janjua 2004; Sarveiya 2004; Gonzalez 2006). Volunteers continued to excrete oxybenzone many days after the last application of the chemical, an indication of its tendency to accumulate in fatty tissues in the body (Gonzalez 2006).

In addition to its ability to absorb into the body, oxybenzone is also a penetration enhancer, a chemical that helps other chemicals penetrate the skin (Pont 2004).

## Oxybenzone may disrupt the human hormone system

Studies on cells and laboratory animals indicate that oxybenzone and its metabolites, the chemicals the body make from oxybenzone in an attempt to detoxify and excrete it, may disrupt the hormone system. Under study condition oxybenzone and its metabolites cause weak estrogenic (Nakagawa 2002; Schlumpf 2001, 2004; Kunz 2006; van Liempd 2007) and anti-androgenic (Ma 2003) effects. Oxybenzone displays additive hormonal effects when teste with other sunscreen chemicals (Heneweer 2005). Laboratory study also suggests that oxybenzone may affect the adrenal hormone system (Ziolkowska 2006).

One human study coapplying 3 sunscreen active ingredients (oxybenzone, 4-MBC, and octinoxate) suggested a minor, intermittent, but statistically significant drop in testosterone levels in men during a one-week application



before and during treatment.

2006 European Union review concluded that a rigorous exposure assessment of oxybenzone was impossible, due to lack of information about the levels of absorption into the body (SCCP 2006). The levels of contamination reported in this latest CDC study indicate that absorption may be significant, consistent with previous, small-scale biomonitoring reports. A decades-old evaluation by FDA, as well as more recent review by the cosmetics industry's own safety panel, do not consider concerns regarding hormone disruption, nor the implications of the ability of oxybenzone to penetrate the skin (USPC 1975; FDA 1978; CIR 1983, 2002). At present, no health-based standards exist for safe levels of oxybenzone in the body.

Additional cautions must be employed when considering the effects of oxybenzone on children. The surface area of a child's skin relative to body weight is greater than adults. As a result, the potential dose of a chemical following dermal exposure is likely to be about 1.4 times greater in children than in adults (SCCNFP 2001). In addition, children are less able than adults to detoxify and excrete chemicals, and children's developing organ systems are more vulnerable to damage from chemical exposures, and more sensitive to low levels of hormonally active compounds (NAS 1993; Janjua 2004). Children also have more years of future life in which to develop disease triggered by early exposure to chemicals (NAS 1993). Despite these well-documented concerns regarding children's sensitivity to harmful substances, no special protections exist regarding ingredients in personal care products marketed for babies and children. (Babies? Not for children 1 & 2 yrs)

The fraction of oxybenzone that is not absorbed into the human body often contaminates water, washed from the skin during swimming and water play or while bathing (Lambropolou 2002; Danovaro 2008). Wastewater treatment removes only a fraction of this sunscreen chemical (Li 2007), resulting in detection of oxybenzone in treated wastewater, in lake and sea waters due to recreational use or to discharges from water treatment facilities, and even in fish (Balmer 2005; Cuderman 2007; Li 2007). Studies show oxybenzone can trigger outbreaks of viral infection in coral reefs (Danovaro 2008), and can cause feminization of male fish (Kunz 2006). Despite significant ecological concerns, there are no measures in place to protect sensitive ecosystems from damage caused by this contaminant.

## EWG to FDA: Oxybenzone investigation is long overdue

FDA last reviewed the safety of oxybenzone in the 1970s (USPC 1975), republishing its evaluation in 1978, at the same time it announced plans to develop comprehensive standards for sunscreen safety and effectiveness (FDA 1978). 30 years later, the Agency has yet to issue final regulations. Instead, it encourages manufacturers to follow draft guidelines that the Agency has delayed finalizing at the behest of the sunscreen industry. As a result, sunscreen manufacturers in the U.S. are free to market products containing ingredients like oxybenzone that have not been proven safe for people.

Found in over half of the 910 name-brand sunscreen products we reviewed, oxybenzone is tied to significant health concerns that must be scrutinized. Instead, FDA's refusal to re-examine this ingredient keeps sunscreens containing oxybenzone on the market. Petitions for review of newly developed sunscreen ingredients approved for use in other countries, and with far fewer health concerns, have been met with similar inattention, blocking Americans' access to better products.

FDA foot-dragging has left the U.S. without enforceable standards for sunscreen safety and effectiveness for decades. EWG demands that FDA finalize the latest version of its monograph on sunscreen products immediately, and launch an investigation into the safety of the sunscreen ingredient oxybenzone.



**More EWG comments on FDA's sunscreen monograph**

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# Land Use, Macroalgae, and a Tumor-Forming Disease in Marine Turtles

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## Abstract

Go to:

Wildlife diseases are an increasing concern for endangered species conservation, but their occurrence, causes, and human influences are often unknown. We analyzed 3,939 records of stranded Hawaiian green sea turtles (*Chelonia mydas*) over 28 years to understand fibropapillomatosis, a tumor-forming disease linked to a herpesvirus. Turtle size is a consistent risk factor and size-standardized models revealed considerable spatial and temporal variability. The disease peaked in some areas in the 1990s, in some regions rates remained constant, and elsewhere rates increased. Land use, onshore of where the turtles feed, may play a role. Elevated disease rates were clustered in watersheds with high nitrogen-footprints; an index of natural and anthropogenic factors that affect coastal eutrophication. Further analysis shows strong epidemiological links between disease rates, nitrogen-footprints, and invasive macroalgae and points to foraging ecology. These turtles now forage on invasive macroalgae, which can dominate nutrient rich waters and sequester environmental N in the amino acid arginine. Arginine is known to regulate immune activity, promote herpesviruses, and contribute to tumor formation. Our results have implications for understanding diseases in aquatic organisms, eutrophication, herpesviruses, and tumor formation.

## Introduction

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Combined with overexploitation, habitat loss, and climate change, emerging diseases pose major impacts to biodiversity worldwide [1], [2]. Marine turtles suffer numerous population threats [3] with green sea turtles (*Chelonia mydas*) afflicted by fibropapillomatosis (FP) a debilitating tumor-forming disease [4]. While surveys show key green turtle populations are steadily growing [5], [6], FP remains widespread and its origins are unknown. Here we present a spatial epidemiology from 28 years of

disease records from the Hawaiian population of green turtles. We construct time series of disease rates, address the spatial scale of variability, and examine the role of land use and invasive macroalgae.

Early hypotheses of causal factors of the disease examined vascular trematodes and toxins but results were inconclusive [7], [8]. A viral origin for FP became apparent after experiments successfully transmitted the disease using cell-free tumor extracts [9]. Later studies identified $\alpha$-herpesviruses as the leading candidate after their DNA fragments were discovered in turtle tumors, but were absent in tumor-free turtles [10], [11]. Subsequent results also showed sampled herpesviruses had low genetic variability [11], [12] implying contact transmission, perhaps via ectoparasites [13].

Further advances to understanding this disease have been limited by the inherent complexities of epidemics and their ecosystems [14]. Infectious diseases involve individual susceptibility, exposure, infection, and immune response. These phases often operate independently; interact in nonlinear ways; and vary demographically, geographically and through time. Mass-action models [15], for example, can predict the course of many diseases by their host population density. These models are intuitive, as communicable diseases often spread rapidly in dense populations. Understanding the variability of FP, however, is likely more complicated than transmission dynamics alone. In Hawaiian green turtles, for example, FP became prevalent in the 1980s, and apparently peaked in the 1990s [16], [17] though the turtle population has grown continually [5]. Furthermore, recent phylogenetic analyses of the implicated herpesviruses show low mutability and coevolution with their turtle hosts over millions of years [12]. Investigating factors that can promote disease, such as environmental [18] or dietary conditions [19], may therefore provide insights.

Green turtles develop FP (Fig. 1) only after recruiting to nearshore habitat [17], [20] indicating these environments are influential. Most Hawaiian green turtles hatch in the Northwestern Hawaiian Islands (NWHI, 900 km from Honolulu) and spend up to a decade in pelagic waters [21]. Juveniles recruit to nearshore waters at around 35 cm straight carapace length (SCL). Here turtles maintain spatiotemporal fidelity to specific macroalgae beds in shallow, nearshore sites [16], [22]. After reaching ~80 cm SCL, individuals seasonally migrate to the NWHI to breed. There they spend months, afterwards return to their foraging sites in the Main Hawaiian Islands (MHI), and subsequently breed every 3–4 or more years [23]. Therefore all neritic green turtles are chronically and locally influenced by their local nearshore habitat in the MHI.

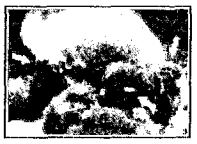

Figure 1
**Hawaiian green turtle (*Chelonia mydas*) with fibropapillomatosis (FP) on the seafloor in Pupukea Marine Life Conservation District, North Shore, Oahu.**

We examine FP records of green turtles stranded on Hawaiian beaches from 1982–2009 considering the uniqueness of the archipelago. Unlike stranding investigations in the southeastern USA where turtles drift considerable distances after offshore mortality [24], we assume most turtles and most population threats are proximate to coasts. The Hawaiian islands are oceanic pinnacles with no continental shelf and local fisheries bycatch is not a major threat [17]. Population and ecosystem changes are likely important considerations, however. Conservation efforts established in the 1970s preceded a dramatic population recovery, in spite of the widespread occurrence of FP [5]. Additionally during this period, invasive macroalgae bloomed across the MHI reportedly spurred by nutrient enrichment from agriculture runoff and discharged sewage [25], [26], [27]. We therefore examine the following questions. Is turtle size a risk factor for this disease? At what scales do disease rates vary in space and time? Are disease rates spatially clustered? Do epidemiological links to land use or macroalgae exist?

## Materials and Methods

Go to:

## Turtle strandings data

We compiled strandings data from dead or moribund turtles reported to the National Marine Fisheries Service, Pacific Islands Fisheries Science Center [17]. These data span the entire archipelago, but we restricted the analysis to Oahu, Maui, and Hawaii due to observer coverage. We documented stranding locations from locality descriptions from 1982–1999, afterwards using global positioning system coordinates. We considered turtles FP positive when external exams identified tumors (Fig. 1) as no turtles with internal tumors lacked them externally. Demographic data were limited to size measurements. We used SCL for size and calculated it from curved carapace length (using $SCL = 0.93*CCL$, $r^2=0.99$), when only the latter was available. This yielded 3,939 records spanning 28 years containing location, disease, and turtle size data.

## Standardizing disease rates

As size is a known risk factor for FP [17], [28] we calculated the stranding frequencies of size classes through time and determined their size-specific disease rates. Understanding these relationships is essential for accurate comparisons, especially to avoid reporting differences that are merely demographic artifacts [29]. To describe changes in the strandings during the study, we grouped strandings into five equal time periods and six size classes and fit probability models to the size frequency data. We used the log-normal, gamma, and log-hyperbolic secant functions as they typify population data [30], [31]. A maximum likelihood estimator chose model parameters and an Akaike Information Criterion (AIC) ranked models [32]. To describe the relationship between size and disease rate, we retained the above time and size bins and calculated the simple disease incidence proportion in each group. We plotted disease rates against size, fit quadratic models to the data and differentiated the predicted expression to determine where rates peaked.

Next we explored the spatiotemporal variability of FP by standardizing disease rates to account for the risk factor of turtle size. Standardized disease rates for subsets of the database are local incidence proportions, corrected to the size structure of a "standard" population, we defined as the most recent decade of data. We calculated them using:

$$E^{(S)} = \frac{\sum_{j=1}^{J} (y_j/n_j) n_j^{(s)}}{n_+^{(s)}} \quad (1)$$

where $y_j$ and $n_j$ are the FP positive and the total individuals, respectively, in each size bin (i.e., at risk) in the locally-observed population; and $n_j^{(s)}$ and $n_+^{(s)}$ are the number of individuals at risk and the total number of individuals in the standard population, respectively [29]. Essentially, this metric weights local, size-specific disease rates according to each size class's occurrence in the standard population.

Having a comparable measure of local FP rates, $E^{(s)}$, we calculated their annual time series at three spatial scales: with all islands grouped, by island, and by within-island regions. Not all locations were well sampled (especially pre-1988) so we combined adjacent years with <5 records and plotted the resulting rates as the mean time. We distinguished island regions by terrestrial hydrology, identifying seven regions on Oahu (North Shore, Kahuku, Kaneohe, Waianae, South Shore, Maunalua, and Waimanalo), three on Maui (West, North, and South Maui), and two on Hawaii (Kona and Hilo). We then compared the statistical variability of the time series between spatial scales (see Table S1) ranking models using the corrected AIC ($AIC_c$) [33]. This treats scale as a model factor to identify the appropriate scale for understanding disease variability.

## Characterizing land use

To understand the influence of spatial scale more acutely, we calculated disease rates in individual watersheds and examined the influence of land use. We obtained GIS coverages of land features and land use from the State of Hawaii Office of Planning [34] and the Hawaii Department of Health [35]. We combined adjacent watersheds if they shared water courses, if stranding beaches crossed boundaries, or if <5 stranding events occurred within a single area. Isolated watersheds with <5 observations were excluded. Watersheds accumulated strandings if they occurred within the boundary or <1km from shore. This provided 82 watersheds on Oahu (n=55), Maui (n=16), and Hawaii (n=11).

As individual green turtles in Hawaii are repeatedly captured in the same nearshore sites [16], [22] the local ecosystem influences are likely important. We developed a nitrogen-footprint to capture the combination of factors that generate, deliver, and retain N in nearshore waters [36]. Spatially-explicit footprint statistics summarize human influences across large geographic areas [37] when other empirical records are lacking. We chose ten factors for the Nitrogen-footprint based on their known effect to nearshore ecosystems [26], [36], [38], [39]: sewage injection wells, urbanization, sugar and pineapple agriculture, intensive poultry and hog farms, cattle grazing and dairy production, aquaculture and fishponds, perennial streams and rivers, estuaries and wetlands, boat harbors, and coastal lagoons created by fringing barrier reefs. (We excluded golf courses as their major nutrient contribution is phosphorus [40] which is less important than N for ecosystem changes [36], [41] or for macroalgae [38], [42], [43].) Each watershed accumulated a nitrogen-footprint score where each contributing factor is measured, equally weighted, summed, and rescaled.

For urbanization, sugar/pineapple, cattle grazing, and poultry/hog production, the Nitrogen-footprint score is the average of the % area coverage and the % drainage coverage. We preferred this to area coverage alone as human activity tends to be clustered along coastal waters and may this may skew its impact. Perennial streams, rivers, and canals accumulate within each watershed, receiving a value of 0.5 for each contribution. We scored aquaculture/fishponds and estuaries/wetlands as the % coastline coverage of their maximum width. We scored sewage injection wells by their permitted flow rates: "major" wells are municipal facilities or wells pumping 50,000–3,000,000 gallons per day (gpd), "significant" wells pump 10,000–49,999 gpd, and "minor" wells pump 1,000–9,999 gpd. We only used wells located in "Underground Injection Control Areas," or immediately proximate to coastal waters [35]. We scored major wells=1, significant wells=0.25, and minor wells=0.025. Watersheds within an embayment or bordered by a fringing reef received a score of 1. Harbors are considered "major" if they contain >100 boat docks or accommodate large ocean going vessels (military ships, commercial cruise liners, container ships), and "minor" if not: major harbors=1, minor harbors=0.1.

## Geographically weighted regression models

We calculated standardized disease rates for watersheds with (1) and tested for spatial autocorrelation with Moran's Index. We built geographically weighted regression (GWR) models to compare the variable relationships within watersheds, considering that parameters themselves are influenced by surrounding areas [29], [44]. The GWR models compared disease rates in each watershed to Nitrogen-footprint values, locating parameters with a Monte Carlo search using both fixed and adaptive bandwidths [44]. Because the highest-ranked time series model grouped observations at island regions we capped neighbor influences to 10 km distance and to <15 watersheds. We ran GWR models in ArcGIS [45] and ranked models using $AIC_C$.

We then examined the spatial structure of the highest-ranked model's residuals, testing for autocorrelation and potential differences between islands or from macroalgae distribution. We described macroalgal history from the known occurrence of three nonnative invasives that comprise the majority of Hawaiian green turtle diets [46], [47], [48]: *Hypnea musciformis*, *Gracilaria salicornia*, and *Acanthophora spicifera*. We documented occurrence using the definitive authority on Hawaiian rhodophytes [25] and field surveys [27]. We considered occurrence "major" if it chronically exceeded

>1 km of coastline and "minor" if it did not (Celia M. Smith, personal communication). If we lacked records of these species at a location, we considered them absent. We used Moran's Index to examine residual autocorrelation and we plotted the predicted $E^{(s)}$ values from the GWR, coding them for island and macroalgal distribution.

## Results

Go to:

### Establishing risk factors

Fig. 2 plots the demographic proportions of stranded green turtles through time from the islands of Oahu, Maui, and Hawaii and describes the relationship between turtle size and disease incidence. Bar plots show the demographic proportions through time fitted to a log-normal distribution, the highest-ranked model in all time steps. The second time step shows a pulse of juveniles in comparison to the previous period, and later periods show a shift towards a population skewed in favor of juveniles. This is demonstrated in that the standard deviation of the log-normal model decreases through time (see Table S2).

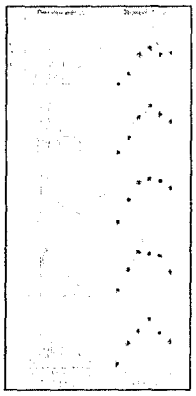

Figure 2

**Turtle size is a consistent risk factor through time.**

Simple incidence proportions of FP show disease increases with turtle size, peaks, and then declines ( Fig. 2). Fitted models are first-order polynomials for all time periods, corroborating earlier results [17], [28]. All models (red lines) fit the data well ($r^2$=0.94–0.99) and as a result, all further comparisons of disease rates are standardized according to turtle size [29]. Fitted models further indicate that size at peak incidence decreases ~10cm over the study period.

### Disease variability in space and time

Fig. 3 plots time series of standardized disease rates at varying spatial scales. Regional time series reveal dramatic local differences (Fig. 3*A–C*). The Oahu plot (Fig. 3*A*) peaks in the mid-1990s and gradually declines after, and seems to drive the signal when all islands are grouped. The Oahu trend however is quite different from regions within. North Shore, Kaneohe, and Waimanalo all peak in the 1990s and then decline; Kahuku and Maunalua gradually asymptote; and Waianae and South Shore increase. Fig. 3*B* shows a similar result for Maui where the overall Maui trend masks the recent declines of West and South Maui. The Kona region of Hawaii is nearly disease free (Fig. 3*C*). The appropriate spatial scale, therefore, seems relevant to understanding FP. Considering spatial scale as a variable, the highest-ranked model is a curvilinear fit when regions within islands are considered separately (Table S1 provides $\delta AIC_c$ values). This indicates that FP varies locally, which when considered in conjunction with spatiotemporal fidelity, encourages investigation into local causes.

Figure 3

**Time series of standardized disease rates show significant regional variability and suggest a local cause.**

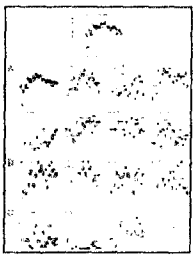

Fig. 4 maps standardized disease rates and Nitrogen-footprints for local watersheds. The left series maps elevated disease rates as warm colors, with cool colors indicating low rates. High rates are clustered in all Oahu regions (save Waianae and Waimanalo) as well all three Maui regions. Four of the five highest disease rates are in Oahu watersheds - Maleakahana, Kahuku ($E^{(s)}$=0.91); Kualoa, Kaneohe ($E^{(s)}$=0.90); Kamiloiki, Maunalua ($E^{(s)}$=0.89); and Waikele, South Shore ($E^{(s)}$=0.88) – with the highest disease rate found on Maui - Hapapa, South Maui ($E^{(s)}$=0.93). By comparison, Hawaii has relatively low disease rates - with the exception of Wailuku, $E^{(s)}$=0.77. In general, the disease rate maps in Fig. 4 correspond well to the time series in Fig. 3.

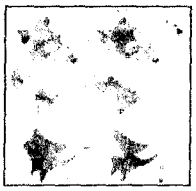

Figure 4

**Spatial analyses reveal that disease rates are highest in watersheds where human land use impacts are greatest.**

The right series in Fig. 4 maps Nitrogen-footprints with warm colors symbolizing high values and cool colors, low values. Watersheds in orange and red therefore indicate the combined presence of multiple factors that generate, deliver, and retain N in coastal waters. The watersheds of central Oahu for example contained pineapple and sugar agriculture, cattle grazing, sewage injection wells, urbanization, perennial water courses, and coastal estuaries. As a result, three of the top five Nitrogen-footprint values are in this area: Paukauila, North Shore ($N_i$=1.0); Waikele, South Shore ($N_i$=0.97); and Halawa, South Shore ($N_i$=0.93). Table S3 provides values for all watersheds.

Watershed disease rates are spatially clustered (Moran's $I$=0.14, $z$=3.4, $p$<0.01) indicating spatial statistics are required. The GWR examines how Nitrogen-footprint influences disease rates within watersheds; comparing the two map series in Fig. 4. The highest-ranked model used an adaptive bandwidth kernel featuring the influence of <15 neighbor watersheds (Table S4). The Nitrogen-footprint values therefore account for much of the spatial variation ($r^2$=0.72) in observed disease rates. Importantly, the model produces randomly arrayed residuals (Moran's $I$=−0.03, $p$=0.65) indicating no systemic model deficiencies.

Fig. 5 plots the GWR predicted disease rates for each watershed according to island and macroalgae records. Maui has the highest average disease rates with nearly 94% (15/16) of Maui watersheds clustered in quadrants I and II. Oahu watersheds are well-distributed with 87% (48/55) of points in quadrants II and III. On Hawaii, 82% (9/11) of watersheds are clustered in quadrant III. Again, Hawaii is relatively disease free with the lone triangle in quadrant II being Wailuku - the same watershed that appears reddish in both plots in Fig. 4. Fig. 5*B* shows a strong association between disease rates, Nitrogen-footprints, and macroalgae consumed by turtles. Almost 93% (37/40) of watersheds where macroalgae occurred are clustered in quadrant II where both disease rates and Nitrogen-footprint values are high. Negative correlations are also prominent. Almost 85% (17/21) of the watersheds with no such history are clustered in quadrant III. Disease rates are highest in watersheds with high Nitrogen-footprints and where nonnative algae have been chronically significant.

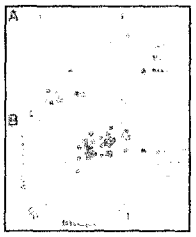

Figure 5

**Invasive macroalgae are chronically widespread in watersheds · rates and Nitrogen-footprint values are elevated.**

## Discussion

Go to:

Our spatial epidemiology of FP provides four significant results: (i) turtle size is a consistent disease risk factor, (ii) disease variability is at the local scale, (iii) disease rates and land use are correlated, and (iv) the disease is linked to macroalgae. We discuss these results and potential mechanisms below.

### Variation in disease risk and rates

The observed demographic patterns of stranded turtles are likely influenced by factors besides disease. As a result, it is uncertain how these patterns relate to the population's actual population demographics. For example, the first size class never outnumbers the second (Fig. 2) which is impossible in a closed population. The pattern surely reflects the juvenile pelagic phase of the population [23] and indicates juveniles recruit to nearshore habitats in both of the first two size classes. Conservation efforts may affect stranding demographics also. The moratorium on turtle harvests since the 1970s likely contributed to the spike in juveniles in the second time step that seems to subsequently bolster larger size classes (Fig. 2).

Despite any demographic changes through the study, however, the relationship between turtle size and disease rate is consistent. The highest-ranked models show subadults are always the most affected group, but through time the size at peak disease rate decreases. This could reveal a variety of dynamics. Adults, for example, may have developed greater immunity or the disease may have become increasingly virulent, killing off younger turtles. In essence, opposite factors could produce similar patterns. The result could have little to do with epidemiology, on the other hand, and simply reflect density-dependent factors slowing somatic growth rates [22]. Future studies might examine these interactions and how risk factors themselves vary geographically.

Size-standardized disease rates reveal considerable spatiotemporal variation (Figs. 3–4) and focus attention on local disease dynamics. Though local time-series models are ranked highest, neighboring areas theoretically should be similar [29]. On Oahu - the island with the greatest coverage - the four regions on the northern half of the island have similar time series (Fig. 3*A*). The North Shore, Kahuku, Kaneohe, and Waimanalo all show peak values in the 1990s. The three southern regions of Oahu - Waianae, South Shore, and Maunalua - all peak near 2005. When disease rates are calculated by watershed, FP rates remain spatially clustered (Figs. 4, 5*A*). The Waianae, Waimanalo, and Kona regions all have low FP rates. Conversely, watersheds on Maui typically have elevated FP rates; true for several Oahu regions as well. The time series and the watershed-based analysis lead to similar conclusions: describing FP rates at large spatial scales masks important local differences.

### Limits to land use maps

The disease and Nitrogen-footprint maps have compelling similarities (Fig. 4) which the GWR test confirms. Watersheds with high disease rates tend to also have high Nitrogen-footprint values. Disease rates for Maui are relatively high across a range of Nitrogen-footprint values (Fig 5*A*). Maui is also the only island-level time series where annual disease rates surpass 90% (Fig. 3*B*). The Kona (Hawaii) and Waianae (Oahu) regions have Nitrogen-footprint values slightly above their disease rates (Fig. 4). These results may suggest variables other than those the Nitrogen-footprint accounts for factor in FP dynamics; either additional N sources or other factors entirely. Oceanographic currents, for example,

could increase dilution of nutrient runoff and mitigate land use influences. However these currents are stochastic in nearshore waters and not easily characterized, especially historically. Irrigation using treated sewage might also add nutrients to ecosystems, but its use is not documented. The GWR model explains much of the variability in the data ($r^2=0.72$) and as its residuals have no spatial structure, the model does not appear to have systemic deficiencies.

The high ranking of the local time series model (Table S1) encouraged us to increase the spatial resolution to individual watersheds. This had three effects. The first is that there were not sufficient data in each watershed to calculate annual disease rates. So though we produced a fine-scale map of disease rates to individual watersheds (Fig. 4), we could not resolve the maps in time. Secondly, this naturally impacted our environmental descriptions. The Nitrogen-footprint is only a snapshot of environmental variables that vary through time. Any limitations this might impose are limited as only three of the ten components used in the Nitrogen-footprint varied considerably. These are the agricultural coverages (e.g. sugar/pineapple, cattle, poultry/hog, etc.) which actually may help explain some of the time series variability. Sugar cane and pineapple agriculture declined across the MHI during the 1990s, which broadly parallels the declines in FP rates in North Shore, North Maui, South Maui, and Hilo where these crops were formerly dominant (Fig. 3). Thirdly, the watershed maps and time-series analysis provided two sets of independent results, reinforcing their conclusions. The absence of the disease in both Kona series (Fig. 3C, Fig. 4) for example is also interesting. Nonnative macroalgae records on the Kona coast are few [25], [27] and land use influences there are slight (Fig. 4 ).

## Epidemiological Links

One explanation for our results is the dietary promotion of FP in eutrophic habitats. After 1950, native Hawaiian algae and sea grasses were displaced by nonnative species, especially in locations with elevated nutrient loads [25], [26], [27]. Nonnative macroalgae have become so dominant, that in some locations they compose >90% of green turtle diets [47], [48]. The implications of this dietary shift may be profound. When and where N is abundant, plants store excess environmental N in arginine (Arg), the only tetra-amine amino acid [49]. One study in Hawaii [50] identified two invasive algae consumed by turtles, *Hypnea musciformis* and *Ulva fasciata*, as having elevated Arg. Later isotope analysis revealed up to 43% of stored N in these species originated from discharged sewage [26]. Nonnative algae thus appear to sequester anthropogenic N, store it as Arg, and pass it on as turtle forage. This is significant as various lines of evidence implicate Arg in herpesvirus promotion and tumor growth.

Immunology and virology studies are particularly revealing. In many chronic diseases, Arg is involved in cell inflammation and immune dysfunction [51] and in promoting viral tumors [52]. But Arg is specifically important for herpesviruses which are linked to FP tumors. Experiments show that herpes does not grow without Arg [53], [54], [55], as Arg is a key building block of the viral envelope that facilitates localization, fusion, and entrance to host cell nuclei [56], [57]. Arg also seems to promote herpes-associated corneal tumors [58] and was highly concentrated in tears of rabbits with corneal herpes [59]. This is particularly relevant, as 93% of Hawaiian green turtles with FP have ocular tumors [60] (Fig. 1). How herpesviruses may promote tumor growth is uncertain, but studies show herpes may inhibit apoptosis and manipulate cell growth [61], [62]. Beyond its demonstrated role in herpesviruses, Arg is also common in a tornovirus recently found in Florida turtles with FP [63]. Histopathology studies also support an Arg-FP link. Blood assays show Hawaiian turtles with FP have elevated blood urea nitrogen compared to disease free turtles [64] which in the absence of gastrointestinal pathology [60] can indicate enhanced dietary intake of N [65]. Considered with the results of the current study, this evidence suggests nonnative macroalgae play a significant dietary role in promoting FP in marine turtles.

Fig. 5*B* clearly summarizes the links between disease rates, land use, and invasive macroalgae, yet we urge interpretative caution. Many factors contribute to the course of an infectious disease. Here we addressed the spatiotemporal variability of FP, and the environmental factors associated with promoting infections. Understanding this disease will be further advanced by examining nearshore nutrient cycling, herpesviruses, and tumor formation more acutely. Our results show that environmental factors are significant in promoting FP and suggest that eutrophic coastal ecosystems may promote herpesvirus infections among herbivores. Given the broad role of Arg in viral promotion and immune regulation our results may be significant for viral oncology more generally.

## Supporting Information

Go to:

### Table S1

Model results comparing temporal demographics of stranded Hawaiian green turtles, 1982–2009. Times are divided into five equal 55-month periods. N represents the strandings sample size during the period. The log-normal model is always the highest-ranked model evidence by the δAICc value is always zero. We provide log-normal parameters as a result. All models have two parameters.

(0.07 MB PDF)

Click here for additional data file.(72K, pdf)

### Table S2

Model structure and correlates used to examine disease rate time series (Fig. 3). D is the root mean square deviation of the model from the data. N is the number of points in the analysis. The error term is assumed to be Gaussian. The highest ranking model considers disease at the regional level, within islands, and allows curvilinear variability.

(0.08 MB PDF)

Click here for additional data file.(76K, pdf)

### Table S3

Complete data table for watersheds used in the geographically weighted regression and seen in Figs. 4–5. Data table is included as a .txt file.

(0.01 MB TXT)

Click here for additional data file.(4.9K, txt)

### Table S4

Full model results from the geographically weighted regression that allows model coefficients to vary in space. The null model is the "global" or traditional linear regression, using ordinary least squares methods. But even though this model has the lowest AICc value, it is inappropriate

because the variables are spatially autocorrelated (see Results). The highest ranked model considers how a watershed's N Footprint affects disease rates within, and also factors the N Footprint of the nearest 15 watersheds. N is the number of points in the analysis, σ is the standard deviation of the model residuals.

(0.08 MB PDF)

Click here for additional data file.(79K, pdf)

## Acknowledgments

Go to:

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## Footnotes

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DISPATCH

# Sea Turtles: Old Viruses and New Tricks

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## Abstract

Recent years have seen an inexplicable increase in the frequency of an appalling disease in sea turtles: fibropapillomatosis, which is likely caused by a herpesvirus and causes tumors to grow throughout the turtle's body. New research has led to the disturbing conclusion that recent, human-induced environmental changes are responsible.

Emerging diseases seemed to enter the nightmares of mainstream culture with the ebola and hantavirus scares of the 1990s. Since then, emerging diseases have become a major health concern in human populations, with such diseases as severe acute respiratory syndrome (SARS), avian influenza and West Nile virus disease sickening and scaring people around the globe.

Most people do not realize that emerging diseases are also a problem for wildlife and may be a major threat to endangered species [1]. In the last decade, there has been an increase in the number of cases of a wide spectrum of diseases in populations of diverse species of plants and animals [1., 2.]. Emerging diseases in wildlife are important for the obvious reason that they can cause population declines in the susceptible species. But these diseases in wildlife are important from a human health standpoint too, because many of the emerging diseases in humans have been linked to wildlife species that serve as reservoirs of the pathogen. Furthermore, the study of emerging diseases in wildlife may well provide general insights that help us to understand the dynamics of emerging diseases in human populations.

As reported in a paper just published in *Current Biology*, Herbst *et al.*[3] investigated the cause of the recent outbreak of marine turtle fibropapillomatosis by examining the evolution of the virus that causes the disease. This disease affects mainly the green sea turtle (Figure 1), but cases have also been documented in loggerhead, olive ridley and now Kemp's ridley sea turtles. The fibrous growths typical of fibropapillomatosis were first described in 1938 and reports of the disease were relatively rare until after 1980 [4]. Now, fibropapillomatosis occurs around the globe and in one recent sample from the Hawaiian Islands more than 90% of green turtles showed symptoms of the illness [4].

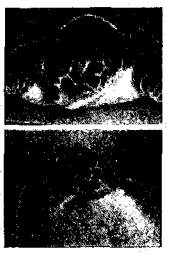

Figure 1
The green turtle, *Chelonia mydas*.

Images of uninfected green turtles (courtesy of Jack Lenz).

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From the standpoint of a wildlife enthusiast, fibropapillomatosis is a heinous disease, marring the usually noble appearance of the beloved sea turtles. The growths associated with the disease occur mainly on the soft skin of the turtle, but they can appear internally as well. The growths can be so large that they interfere with normal mobility, vision, feeding and organ function. In addition to these gross mechanical effects, the disease appears to result in suppression of the immune system and a susceptibility to bacteremia [5., 6.]. Consequently, death is the ultimate outcome for many of the turtles affected by the disease.

All the current evidence suggests that marine turtle fibropapillomatosis is caused by a herpesvirus that has been shown to be associated with the growths [7] (although definitive experiments involving cultured virus particles have not yet been possible). But why does the virus causes

so much harm now compared to 50 years ago? Two non-exclusive hypotheses can explain the sudden increase in frequency of fibropapillomatosis (and most other emerging diseases in wildlife for that matter). One possibility is that a change in the environment caused the host species to become extremely susceptible to a previously harmless strain of virus, for example, as a consequence of immune suppression, a new vector and so on. The other possibility is that the disease is caused by a virulent mutant form of a previously harmless virus.

Herbst *et al.*[3] took advantage of the fact that these two hypotheses make distinct predictions about the molecular phylogeny of the virus, allowing a test of whether or not the emergence of this disease was due to a new form of the virus. If the disease is a consequence of a new mutation that arose in the last 50 years, then all of the new, virulent viruses should be close relatives of one another, members of a lineage that has swept through the turtle populations in the last few decades. In fact, the time frame is so short that we would expect virus particles isolated from distinct turtles to have virtually identical sequences over large parts of their genome.

The phylogeny of viruses isolated from 25 individual turtles of three distinct species – green, loggerhead and Kemp's ridley – clearly shows that the now virulent form of the virus has been around a very long time. The viral DNA isolated from some of the tumors of individual turtles exhibited so much sequence divergence among turtles that the lineages had likely been distinct for millions of years, assuming 'clock-like' molecular evolution. So a recent origin of a virulent form of the virus is not consistent with the phylogenetic data.

The data also show that viruses isolated from distinct sea turtle species sometimes have essentially identical DNA, indicating that the virus can be passed across species boundaries. This observation may be somewhat troubling, as the virus does cause disease in these other species, and even though instances of the disease are rare in sea turtles other than greens now, we must remember that 30 years ago the disease was rare in green sea turtles as well.

The only reasonable conclusion from the analysis of Herbst *et al.*[3] appears to be that some change in the environment of sea turtles has rendered them more vulnerable to fibropapillomatosis than they have been in the past. Unfortunately, the environmental cause is not known, and may be very difficult to diagnose. This sea turtle example can be added to a fairly long list of species that have become susceptible to various types of pathogen as a consequence of environmental change – almost all of which are human-induced [2]. If biologists didn't have enough to worry about with the relatively obvious (but difficult to solve) conservation problems imposed by human population growth, habitat destruction, overharvesting and invasive species, this and other recent studies [1., 2.] remind us that we can now add the relatively subtle but potentially devastating effects of increased susceptibility to pathogens to our growing list of threats to the biosphere.

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# Fibropapillomas in the Hawaiian Green Sea Turtle

***Ed. note:*** *This essay was originally posted on May 21, 1995. Although the format of this page has been modified since then, the content of the essay has remained untouched.* ***Some of the material in this essay has become outdated with progress in the research concerning fibropapilloma, and also with our own continuing education about the disease.*** *The essay remains here for context.*

## What Are Fibropapilloma Tumors?

In humans, a papilloma tumor is a benign growth that is spread by a virus. One example is the ordinary wart. Other mammals also develop papilloma tumors, and scientists have noted many similarities and varieties.

When papilloma tumors develop predominant fibrous tissue, they are called fibropapillomas. Green sea turtles develop fibropapillomas that appear as lobe-shaped tumors. These tumors can infect all soft portions of a turtle's body. Tumors grow primarily on the skin, but they can also appear between scales and scutes, in the mouth, on e eyes, and on internal organs.

The lesions are small at first, but they can grow to 10 cm or more in diameter. In the turtles we have observed since 1989, the early stages of the disease have followed a predictable pattern. First, the turtle developed suspicious white spots on its body, most often around the neck and shoulders. We refer to this as "salt and pepper" because the appearance is not unlike what you would get if you took a salt shaker and salted a turtle.

Within a year, these white spots usually developed into full blown tumors. The disease frequently affects the eyes first, but we have seen tumored turtles with clean, healthy eyes, so this is by no means a rule.

The course of the tumors can vary greatly among individuals. We have seen turtles develop a mild case that changes little from year to year. We have even seen what appears to be regression of tumors--but this is not the norm.

Most of the turtles we have observed with the disease steadily worsen. Tumors increase in both number and size. Particularly gruesome are tumors that develop along the neck of turtles. Picture roughly two dozen tumors the appearance and texture of "horse apples" stuck to a turtle's neck, and you have an excellent idea of what a stricken turtle looks like.

For whatever reason, the disease runs rampant in the juvenile turtles we observe. From "salt and pepper" to emaciation can be as little as two years. (Noke is a graphic example.) In adults, the disease is less predictable. We have seen everything from one turtle (Tutu) who had a mild case and recovered completely to another (1991 Turtle 1) who was weak and emaciated within four years.

## What Causes the Tumors? How are They Spread?

The sad truth of the matter is that scientists aren't yet certain what causes fibropapilloma tumors or how they are spread.

There is a lot of research being conducted to answer these questions, and while progress is being made, so far ere are no definitive answers. Because papilloma tumors are spread by a virus in other animals, it is quite likely nat a virus is the culprit in green turtles. Recent research has strongly indicated that a herpesvirus might be the culprit, but more work is needed to decide the issue. Unfortunately, money is hard to get, making it difficult to fund the kind of sustained project that would have the best chance of succeeding.

It is also not clear how the virus, if a virus is indeed the cause, infects the turtles. Infected turtles have remained in close contact with clean turtles in captivity for years without spreading the disease to them. This suggests a carrier that is present only in the wild, but attempts to find such a carrier have not yet been successful.

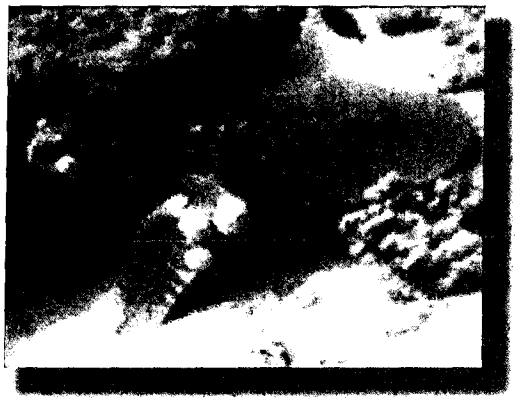
Another unexplained mystery is why the disease has reached epidemic proportions in Hawaii, Florida, and Barbados simultaneously. Since green turtles have been known to range quite far, it is possible that the outbreaks in Florida and Barbados are somehow related, but this cannot explain the problem in Hawaii.

There have also been attempts to discover whether pollutants could be a factor. So far, studies have been unable to find any unusual concentrations of pollutants in the afflicted populations.

It is possible that the disease is the result of a combination of factors. For example, turtles under stress from pollution could be more vulnerable to a virus that would otherwise be relatively harmless. Again, thus far research has not been able to find such a relationship.

While considerable effort has been expended in understanding the cause and spread of fibropapilloma, it is none the less quite frustrating that there is still so much we do not know.

## The Impact



*1991 Turtle 9*

The disease is as tragic as any cancer. Turtle scientists are working hard to determine how the disease is transmitted, and they hope to find a cure as well.

For us, however, it has become personal. We have dived with the same turtles for years and we have come to love them and regard them as friends. Each summer we return to see yet another friend develop "salt and pepper" and then the tumors. Even though the "salt and pepper" gives us a year to steel ourselves, seeing the actual tumors show up on a friend still makes us cry at the moment of sighting.

For the turtles, tumors, even small ones, mean they become the target of saddleback wrasses and whitespotted tobies at the cleaning station. These fish inflict painful bites directly on the fibropapilloma, apparently eating parasites that infest the tumors. Afflicted turtles are often forced to leave a cleaning site prematurely. Over time, their tumors worsen and their time at cleaning stations becomes shorter and shorter.

A thick coat of algae develops on their shells, and even on their skin. Tumors increase in number and size, resulting in more drag and making swimming more difficult.

Tumors often grow to cover both eyes. In 1994, we saw a turtle with an eye tumor so large that it obscured half , left profile. Tumors growing in the corners of the mouth make breathing and eating difficult. One turtle we saw had a tumor growing out of its anus. No soft part of a turtle is spared.

There comes a point in the disease when you think the turtle is really just a way for the tumors to get around. There is little turtle left, the way there is little grasshopper left once a spider is finished sucking it dry. Our

friends, whom we have seen daily for years, develop tumors, worsen, and then one day are nowhere to be found. Animals that have had routines like clockwork are just--gone.

# Scientific Description

*1991 Turtle 13*

The previous description is fibropapilloma as we have come to know it. It is by no means scientific and if you are interested in a more scientific and technical account, we offer it here with permission from the National Marine Fisheries Service. It is extracted verbatim from NOAA Technical Memorandum NOAA-TM-NMFS-SWFSC-156, Research Plan for Marine Turtle Fibropapilloma, March 1991.

## Current Status of Fibropapillomas In the Hawaiian Green Sea Turtle, Chelonia Mydas

The growing incidence of a debilitating and disfiguring disease known as fibropapilloma in the Hawaiian green turtle (or honu), *Chelonia mydas*, formed the subject of a recent review calling attention to this potentially serious phenomenon. Since that time the occurrence of the disease in the Hawaiian Islands has continued to increase in both geographic range and magnitude. This assessment is based on the capture, examination, and tagging of live turtles; records of strandings; and verifiable reports received from divers and other ocean users in Hawaii. The principal concern among the public and scientific sectors for this worsening situation centers on the well-being and survival outlook of the green turtle, a protected species under the U.S. Endangered Species Act and wildlife laws of the state of Hawaii. Other important concerns include the negative visual impacts related to marine tourism and underwater photography by Hawaii's substantial skin and scuba diving industry; the perception that toxic pollutants of some unknown nature and origin may be contaminating certain nearshore marine habitats, thereby causing the disease; and possible human health hazards related to exposure to live afflicted turtles and stranded carcasses.

In reponse to the above issues, the draft Hawaiian Sea Turtle Recovery Plan designated the fibropapilloma problem as a high priority research need. The green turtle population in Hawaii has shown a gradual increase in numbers since full legal protection was afforded in 1978. However, members of the population are known to exhibit slow rates of growth, with the average age of sexual maturity estimated at 25 years old. Consequently, the full impact to the population in terms of recruitment of adult nesting turtles, if the disease continues at the present pace, may not be manifested for decades.

The earliest confirmed case of green turtle fibropapilloma (GTFP) in Hawaii dates back to January 1958. The 1958 case GTFP case involved an immature turtle captured alive by fishermen in Kaneohe Bay on the Island of Oahu. Kaneohe Bay is the largest bay in the Hawaiian Islands, comprising substantial foraging and resting habitat for green turtles. Numerous other reports involving the capture, handling, and sighting of hundreds, if not thousands, of turtles by former turtle fishermen and other reliable informants indicate that GTFP was virtually nonexistent prior to and during the 1950's and early 1960's. Furthermore, no evidence has bene found that live green turtles were ever imported alive into Hawaii from Florida or elsewhere in the Caribbean, where GTFP was first described in the scientific literature in the 1930's as an occasional occurence.

From 31% to 53% of the stranded turtles examined each year since 1983 have had GTFP. During 1990, 154 green turtle stranding cases occurred throughout the 8 main (inhabited) Hawaiian Islands, the highest number since the stranding network was established in 1983. During 1989 and 1990, GTFP was present in 77% and 85% of the turtles stranded on the Island of Maui, mainly in the Kahalui Bay area.

In Kaneohe Bay the live capture by hand of 121 turtles at 4 discrete habitat sites since February 1989 has shown GTFP rates of 49-92%. Turtles with GTFP have been coded by their degree of tumor severity on a scale of 1-4 (stage 4 being the most severe). This evaluation is based on the size, number, and location of the tumors present. Some turtles with codes as high as 3 and 4 have shown substantial vigor, fleeing with force and trying to ;gressively bite the persons restraining them. Other turtles with similar tumor severity are lethargic, emaciated, and easily captured for examination. Cardiovascular parasites are commonly found in these turtles when they strand ashore near death.

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For the remainder of this article and other information concerning marine turtle fibropapillomas, contact the U.S. National Marine Fisheries Service.

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Sickbay

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