ECKERT, S.A. & J. LIEN. 1999. Recommendations for eliminating incidental capture and mortality of leatherback turtles, *Dermochelys coriacea* by commercial fisheries in Trinidad and Tobago: A Report to the Wider Caribbean Sea Turtle Conservation Network (WIDECAST). WIDECAST Information Document 1999-2001. www.widecast.org/Resources/Docs/Eckert_and_Eckert_2005_Trinidad_Bycatch_Meeting_Proceedings. pdf

FORESTRY DIVISION (GOVERNMENT OF THE REPUBLIC OF TRINIDAD AND TOBAGO). SAVE OUR SEA TURTLES-TOBAGO AND NATURE SEEKERS. 2010. WIDECAST Sea Turtle Recovery Action Plan for Trinidad and Tobago (K.L. Eckert (Ed.). CEP Technical Report No. 49. UNEP Caribbean Environmental Programme. Kingston Jamaica.132p. www.cep.unep.org/publications-and-resources/technical-reports/technical-reports

KOCH, A.U., M.L. GUINEA & S.D. WHITING. 2007. Effects of sand erosion and current harvest practices on incubation of the flatback sea turtle, *Natator depressus*. Australian Journal of Zoology 55: 97-105.

LEE LUM, L. 2003. An assessment of incidental sea turtle catch in the artisanal gillnet fishery in Trinidad and Tobago, West Indies. Applied Herpetology 3: 357-368.

LEE LUM, L. 2005. Beach dynamics and nest distribution of leatherback at Grande Riviere, Trinidad. Revista de Biología Tropical 53: 239-248.

MORTIMER, J.A. 1999. Reducing threats to eggs and hatchlings: hatcheries. In: Eckert. K.L., K.A. Bjorndal, F.A. Abreu-Grobois & M. Donnelly (Eds.). 1999. Research and Management Techniques for the Conservation of Sea Turtles. IUCN/SCC Marine Turtle Specialist Group Publication No. 4. pp. 175-178.

SPOTILA, J.R., R.D REINA, A.C. STEYERMARK, P.T. PLOTKIN & F.V. PALADINO. 2000. Pacific leatherback turtles face extinction. Nature 405: 529-530.

TAPILATU, R.F. & M. TIWARI. 2007. Leatherback turtle, *Dermochelys coriacea*, hatching success at Jamursba-Medi and Wermon Beaches in Papua, Indonesia. Chelonian Conservation & Biology 6: 154-158.

Green Turtle (*Chelonia mydas*) Cutaneous Fibropapillomatosis Treatment by Photodynamic Therapy

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Sea turtles inhabit different regions of the world and five of the seven extant species may be found in Brazilian seas: *Caretta caretta* (loggerhead), *Chelonia mydas* (green), *Eretmochelys imbricata* (hawksbill), *Lepidochelys olivacea* (olive ridley) and *Dermochelys coriacea* (leatherback) (Marcovaldi & Marcovaldi 1999). Unfortunately, all five species are on the IUCN Red List of Threatened Species (www.iucnredlist.org). Factors such as predation, pollution, habitat degradation, fisheries bycatch and the emergence of infectious diseases may directly contribute to the population decline of these animals.

One of the major diseases observed in recent decades in sea turtles is fibropapillomatosis (Baptistotte *et al*. 2004; Brito *et al*. 2004; Herbst *et al*. 1998). Initially described during the 1930s in Florida, fibropapillomatosis is characterized by the presence of one or multiple external cutaneous tumors of varying size, which may be found on various parts of the body; occasionally there are visceral fibromas (Herbst 1994). Fibroepithelial tumors are commonly found on the turtle's eyes, including the conjunctivae and skin obstruct vision, and may interfere with feeding and locomotion, while visceral nodules may fatally disrupt normal organ function (Herbst 1994, Herbst *et al*. 1998). In advanced disease stages, affected animals may become weak, anemic, and in some cases even blind if the cornea is significantly involved.

The prevalence of this disease is associated with highly populated coastal areas polluted by large amounts of agricultural, domestic and industrial wastes or marine biotoxins (Adnyana *et al*. 1997; Aguirre & Lutz 2004; Foley *et al*. 2005; Herbst 1994). Some studies also show a correlation with tumor-associated viruses, and there is no effective treatment for this disease other than surgical removal (Ene *et al*. 2005; Herbst *et al*. 1998; Matushima *et al*. 2001; Quackenbush *et al*. 1998).

The histological characteristics of fibropapillomatosis include stromal and epidermal hyperplasic proliferation. Epithelial cells may have large nuclear pleomorphism and it is possible to observe cytoplasmic vacuolization, skin cell degeneration and fibroblast proliferation. The presence of eosinophilic intranuclear inclusions in epidermal cells may be observed microscopically (Matushima *et al*. 2001; Schumacher 1996). Dense connective tissue tumors in internal organs associated with proliferation and purulent inflammatory process was seen in a single fibropapilloma-afflicted

Animal ID	Localization	Treatment	Size (height x width)	Pigmentation	Outcomes
C. mydas 156	right back flipper	PDT	3.5 X 3.0 cm.	pigmented	responsive
	neck	PDT	2.5 X 1.0 cm.	pigmented	responsive
	neck	light + MB -	3.0 X 2.8 cm.	pigmented	non-responsive
C. mydas 183	plastron	PDT	1.3 X 0.8 cm.	non-pigmented	responsive
	left front flipper	PDT	3.4 X 2.7 cm.	pigmented	responsive
	right front flipper	PDT	0.3 X 0.2 cm.	pigmented	responsive
	right back flipper	PDT	0.7 X 0. 6 cm.	non-pigmented	responsive
	plastron	light - MB +	1.5 X 0.7 cm.	non-pigmented	non-responsive
C. mydas 187	neck	PDT	1.1 X 0.6 cm.	pigmented	responsive
	right front flipper	PDT	0.7 X 0.3 cm.	pigmented	responsive
	right front flipper	PDT	0.6 X 0.5 cm.	pigmented	responsive
	right back flipper	PDT	0.4 X 0.2 cm.	pigmented	responsive
	right back flipper	PDT	0.3 X 0.2 cm.	pigmented	responsive
	right back flipper	PDT	0.7 X 0.3 cm.	pigmented	responsive
	plastron	light + MB -	1.7 X 0.7 cm.	non-pigmented	non-responsive
C. mydas 190	plastron	PDT	1.0 X 0.8 cm.	non-pigmented	responsive
	plastron	PDT	1.0 X 0.9 cm.	non-pigmented	responsive
	left back flipper	PDT	0.5 X 0.2 cm.	pigmented	responsive
	left back flipper	light - MB +	1.4 X 0.7 cm.	pigmented	non-responsive
C. mydas 194	neck	PDT	1.0 X 0.5 cm.	pigmented	responsive
	left front flipper	PDT	2.5 X 0.8 cm.	pigmented	responsive

Table 1. Animal ID and anatomical site, size, pigmentation and outcomes of treated tumors. Lesions treated by PDT received 0.5 mL of MB at 300 µM and were irradiated by a low-intensity red diode laser emitting 100 mW at 660 nm. Each square centimeter of the lesion received 16 J of light energy, for 160 seconds. Control groups received either only light irradiation (light + MB -) or MB inoculation (light – MB +). Lesions were classified as responsive when macroscopic aspects indicating severe tumor necrosis were observed up to 30 days after the first treatment session.

turtle in Brazil (Brito *et al.* 2004). Clinical diagnosis may be based on observations of obvious external tumors, which may be classified as circumscribed, infiltrating or disseminated (Knobl *et al*. 2011). Although tumors are benign, their physical presence may cause difficulty in swimming and consequently locomotion, making food capture and breathing inefficient and leading to increased risk of predation (Baptistotte *et al.* 2001; Herbst 1994).

Photodynamic therapy (PDT) has been studied and applied to cancer and infectious disease treatments in multiple areas of human medicine (Dai *et al.* 2009; Dolmans *et al*. 2003). Currently, its use is considered one of the most investigated treatments for the control, prevention and cure of non-melanoma skin cancers (Braathen 2007; Brown 2004). PDT has also been used in humans to treat skin lesions caused by many viral infections. Examples can include lesions caused by human papilloma virus (HPV), cutaneous warts known as *Verrucae vulgaris* or *Verrucae plana* (also caused by HPV), *Molluscum contagiosum* (MC), which is a DNA poxvirus and Herpes simplex (Dai *et al*. 2009).

In the PDT procedure, the human or animal patient generally receives a systemic or local administration of a non-toxic photosensitizer (PS), which is selectively retained in tumor tissue via a few possible mechanisms. The selective drug uptake may take 10 minutes to over 24 hours according to the PS chemical structure and the manner of drug delivery. During the drug uptake time, also referred as pre-irradiation time (PIT), the photosensitized area must be kept out of direct illumination. If the PS is administered systemically, the patient should be kept in a low-luminosity room until sufficient PS elimination. Locally administered PS may require only a light-absorbing bandage to cover the photosensitive area and prevent damage to healthy tissue. After PIT, the photosensitized target-tissue is irradiated by visible or near-infrared low-intensity light at a specific wavelength and time to "activate" the PS molecules. The light absorption by the photosensitizing agent in the presence of molecular oxygen results in photochemical processes that form a variety of reactive oxygen species (ROS) and cause severe cellular damage leading to necrosis and/or apoptosis (Ribeiro 2005). Apart from cell injury by ROS, PDT may induce a local inflammatory process, reducing the ability of the tumor to evade immune system recognition and consequently enhancing the antitumor immune response (Davids 2008).

The first reports of PDT in animals were described in the early 1980s when hematoporphyrin derivative PSs were administered intravenously in dogs and cats prior to laser irradiation. Although these studies included a wide variety of tumor types, most tumors were considered responsive, demonstrating the clinical potential of PDT in the treatment of solid tumors (Lucroy 2002).

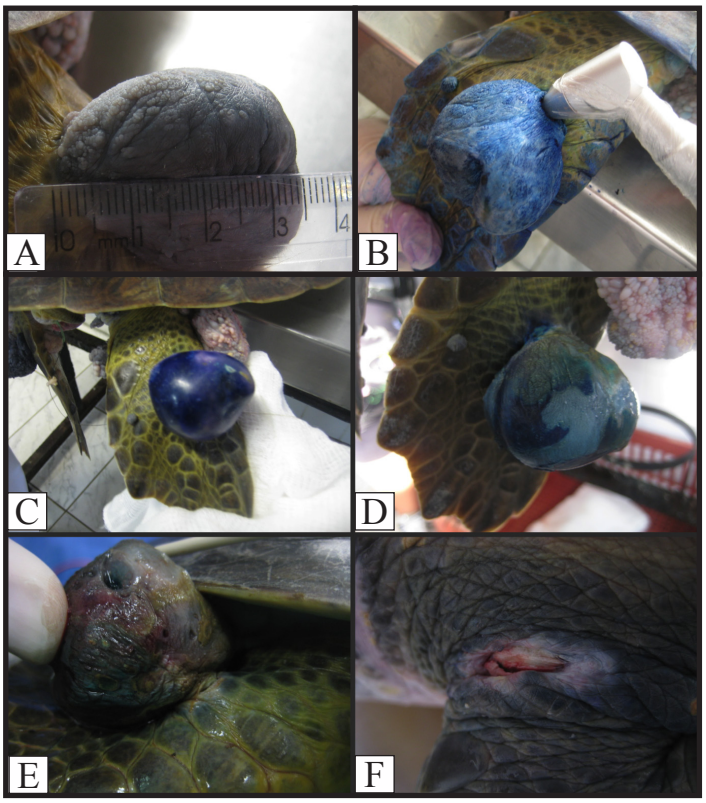

Figure 1. Aspects of one PDT-treated tumor before any intervention (A) and at the first irradiation procedure (B), after MB inoculation. One week after the first PDT session (C), tumor is highly swollen due to intense inflammatory process. Two (D) and (E) three weeks after first PDT session, macroscopic aspects indicating severe tumor necrosis can be observed. At the fourth week (F) the tumor has spontaneously detached from the turtle's tissue.

Methylene blue is a low-cost drug approved by the FDA and it has been widely used in medical practice and biomedical research involving PDT. It is a phenothiazinum salt characterized by a tricyclic heteroaromatic structure, with irrelevant toxicity in low concentrations (Lim *et al*. 2013). Its selectivity to cellular organelles such as mitochondria, lysosomes and nuclei is mainly linked to its positive charge and lipophilic nature that facilitates the crossing of cell membranes (Gabrielli *et al*. 2004; Tardivo *et al.* 2005). The photodynamic reactions, and consequently the local ROS formation, may be caused by their electronic excitation that is induced by red light (600-670 nm.) absorption provided by any light source set at the proper irradiation parameters (e.g., laser, LED, filtered broad spectrum light, etc.). In addition, the associated electromagnetic radiation at wavelengths between 600 and 1300 nm. (red and near-infrared light) exhibits privileged penetration into biological tissues, allowing effective photodynamic treatments at greater depth (Sternberg *et al*. 1998; Sternberg & Dolphin 1996). Although MB employment in oncology is relatively recent, investigations have shown positive *in vivo* activity against a wide range of tumor types including bladder cancer, inoperable esophageal tumors, adenocarcinomas and other non-melanoma skin cancers (Perussi 2007).

In this study, we attempted to treat well-developed fibropapilloma tumors employing PDT mediated by methylene blue as the PS in association with a red laser low-intensity light. We treated five green turtles (*Chelonia mydas*) that had multiple skin tumor lesions compatible with cutaneous fibropapillomatosis (Fig. 1A) in the Municipal Aquarium of Santos/SP. The lesions presented different characteristics regarding size, location, and pigmentation (Table 1). Fibropapillomatosis was diagnosed in each turtle through a clinical examination. Turtles were isolated in individual tanks for better evaluation.

Lesions were treated by two PDT applications within a 15-day interval and consisted of two injections of 0.5 ml. intralesional methylene blue (MB - concentration of 300 μM - Sigma Aldrich) at the base of all tumors, followed by 5 min of pre-irradiation time in the dark to allow for cellular uptake, and the administration of a continuous wave red diode laser irradiation operating at 100 mW of optical power, wavelength (λ) of 660 nm., for 160 seconds per point, resulting in 16 J of energy per point and an energy density of 560 J/cm^2 per point (Fig. 1B). These irradiation parameters do not generate any relevant temperature increase and all biological effects are expected to be due to products formed by MB-mediated photochemical reactions. Each square centimeter of the lesion surface was illuminated according to the parameters described above. Consequently, the number of irradiation points per lesion varied according to the lesion size. To evaluate the response to isolated MB or light interventions, two control lesions were randomly treated exclusively by intralesional MB inoculation and other two separate lesions received low-intensity laser irradiation at the same previously described parameters but free of MB inoculation (Table 1). All treated lesions were evaluated weekly by simple descriptive analysis for a time period of 30 days (Table 1).

At the seventh day post-treatment, we observed that all PDT-treated lesions were dark blue in color and swollen with a firm consistency (Fig. 1C). Lesions that were only treated with a laser did not show any noticeable macroscopic alterations. The lesions where MB was administered alone also presented with a dark blue color - similar to those treated with PDT. However, no further macroscopic alterations were observed.

At the fourteenth day, PDT-treated lesions began to take on a soft consistency, having a light blue color and with classical macroscopic characteristics of tissue necrosis (Fig. 1D). Lesions treated with laser or MB alone did not show any changes in appearance.

Twenty-one days after the first PDT session, lesions showed a partial loss of adhesion to the skin, were light brown in color, had a soft consistency and obvious tissue necrosis characteristics (Fig. 1E). On the thirtieth day, the treated lesions were totally or partially detached from the epidermis (Fig. 1F), and were easily removed using tweezers. After two PDT treatments all lesions showed local macroscopic changes consistent with cellular death. The regression time for all PDT-treated lesions was approximately the same, regardless of the tumor initial size, pigmentation, or location.

Some turtles with multiple lesions had one of the tumors randomly selected to be exclusively treated by MB or low-intensity red laser alone. Tumors not treated by the whole PDT scheme (*i.e.*, MB associated to irradiation) did not show any noticeable macroscopic changes, such as signs of inflammation or regression, during the experimental time and were classified as non-responsive. This observation strongly suggests that relevant cellular and tissue damage may only be achieved when PS was excited by light at a specific wavelength, time and intensity (Moor 2000). In addition,

it indicates that PDT does not trigger relevant systemic antitumor immunity. This conclusion agrees with the literature concerning the selectivity of PS to tumor cells and its phototoxic action being observed only in the irradiated site (Machado 2000; Luksiene 2003).

Our results indicate that MB-mediated photodynamic therapy is a potential new treatment for sea turtle fibropapillomatosis tumors. It is a low-cost ambulatory procedure that can be contemplated as a minimally invasive alternative treatment when the surgical procedure is not available. The evaluation of all mechanisms involved in the tumor regression provided by this treatment, such as the aspects of the histopathology, cellular death, molecular signaling and gene expression, and the possibility of employing PDT as post-surgical treatment to avoid recurrence deserves further investigation.

ADNYANA, W., P.W. LADDS & D. BLAIR. 1997. Observations of fibropapillomatosis in green turtles (*Chelonia mydas*) in Indonesia. Australian Veterinary Journal 10: 737-742.

AGUIRRE, A.A. & P.L. LUTZ. 2004. Marine turtles as sentinels of ecosystem health: is fibropapillomatosis an indicator? EcoHealth 1: 275-283.

BAPTISTOTTE, C., J.T. SCALFONE, B.M.G. GALLO, A.S. SANTOS, J.C. CASTILHOS, E.H.S.M. LIMA, C. BELLINI & P.C.R. BARATA. 2004. Prevalence of sea turtle fibropapillomatosis in Brazil. In: Coyne, M.S. & R.D. Clark (Comps.). Proceedings of the 21st Annual Symposium on Sea Turtle Biology and Conservation. NOAA Tech Memo NMFS-SEFSC-528. pp. 111-113.

BRAATHEN, L.R., R.M. SZEIMIES, N.B. SEGUIN, R. BISSONNETTE, P. FOLEY, D. PARISER, R. ROELANDTS, A.M. WENNBERG & C.A. MORTON. 2007. Guidelines on the use of photodynamic therapy for nonmelanoma skin cancer: An international consensus. Journal of the American Academy of Dermatology 56: 125-143.

BRITO, F.L.C., F.C.L. MAIA, L.M.O. DE FRANÇA, A.R. ALBUQUERQUE, R.A.M. SANTOS, M.A.M. CAVALCANTI & E.S.G. GUIMARÃES. 2004. Fibropapillomatosis and multiple fibromas in a green turtle from the South Cost of Pernambuco State, Brazil. Marine Turtle Newsletter 106: 12.

BROWN, S.B., E.A BROWN & L. WALKER. 2004. The present and future role of photodynamic therapy in cancer treatment. Lancet Oncology 5: 497-508.

DAI, T., Y. HUANG & M. HAMBLIN. 2009. Photodynamic therapy for localized infections - state of the art. Photodiagnosis and Photodynamic Therapy 6: 170-188.

DAVIDS, L.M., B. KLEEMANN, D. KACEROVSKÁ, K. PIZINGER & H.S. KIDSON. 2008. Hypericin phototoxicity induces different modes of cell death in melanoma and human skin cells. Journal of Photochemistry and Photobiology B: Biology 91: 67-76.

DOLMANS, D.E., D. FUKUMURA & R.K. JAIN. 2003. Photodynamic therapy for cancer. Nature Reviews Cancer 3: 380-387.

ENE, A., M. SU, S. LEMAIRE, C. ROSE, S. SCHAFF, R. MORETTI, J. LENZ & L.H. HERBST. 2005. Distribution of chelonid fibropapillomatosis-associated herpesvirus variants in Florida: molecular genetic evidence for infection of turtles following recruitment to neritic developmental habitats. Journal of Wildlife Diseases 41: 489-497.

FOLEY, A.M., B.A. SCHROEDER, A.E. REDLOW, K.J. FICK-CHILD & W.G. TEAS. 2005. Fibropapillomatosis in stranded green turtles (*Chelonia mydas*) from the eastern United States (1980-98): trends and associations with environmental factors. Journal of Wildlife Disease 41: 29-41.

GABRIELLI, D., E. BELISLE, D. SEVERINO, A.J. KOWALTOWSKI & M.S. BAPTISTA. 2004. Binding, aggregation and photochemical properties of methylene blue in mitochondrial suspensions. Photochemistry and Photobiology 79: 227–232.

HERBST, L.H. 1994. Fibropapillomatosis of marine turtles. Annual Review of Fish Diseases, 4: 389-425.

HERBST, L.H., E.C. GREINER, L.M. EHRHART, D.A. BAGLEY & P.A. KLEIN. 1998. Serological association between spirorchidiasis, herpesvirus infection, and fibropapillomatosis in green turtles from Florida. Journal of Wildlife Diseases 34: 496-507.

KNOBL, T., R. REICHE & M.C. MENÃO. 2011. Fibropapillomatosis in marine turtles. Neotropical Biology and Conservation 6: 64-69.

LIM, E.J., OAK, C.H. HEO, J. & Y.H. KIM. 2013. Methylene blue-mediated photodynamic therapy enhances apoptosis in lung cancer cells. Oncology reports 30: 856-862.

LUCROY, M.D. 2002. Photodynamic therapy for companion animals with cancer. The Veterinary Clinics of North America: Small Animal Practice 32: 693-702.

LUKSIENE, Z. 2003. Photodynamic Therapy: mechanisms of action and ways to improve the efficiency of treatment. Medicine (Kaunas, Lithuania) 39(12): 1137-1149.

MACHADO, A.E.H. 2000. Photodynamic therapy: principles, potential of application and perspectives. Quimica Nova 23(2): 237-243.

MARCOVALDI, M.A. & G.G. MARCOVALDI. 1999. Marine Turtles of Brazil: the history and structure of Projeto TAMAR-IBAMA. Biological Conservation 91: 35-41.

MATUSHIMA, E.R., A. LONGATTO FILHO, C. DI LORETTO, C.T. KANAMURA, I.L. SINHORINI, B. GALLO & C. BAPTISTOLLE. 2001. Cutaneous papillomas of green turtles: a morphological, ultra-structural and immunohistochemical study in Brazilian specimens. Brazilian Journal of Veterinary Research and Animal Science 38: 51-54.

MOOR, A.C.E. 2000. Signaling pathways in cell death and survival after photodynamic therapy. Journal of Photochemistry and Photobiology, A: Biology 57: 1-13.

PERUSSI, J.R. 2007. Photodynamic inactivation of microorganisms. Química Nova 30: 1-7.

QUACKENBUSH, S.L., T.M. WORK, G.H. BALAZS, R.N. CASEY, J. ROVNAK, A. CHAVES, L. DUTOIT, J.D. BAINES, C.R. PARRISH, P.R. BOWSER & J.W. CASEY. 1998. Three closely related herpesviruses are associated with fibropapillomatosis in marine turtles. Virology 246: 392-399.

RIBEIRO, J.N. & R.A. JORGE. 2005. Determination of the mechanism of destruction of cell mediated by meso-tetramesitylporphyrin, octaethylporphyrin, vanadyl octaethylporphyrin and visible light.

Eclética Química 30: 7-13.

SCHUMACHER, J. 1996. Viral diseases. In: MADER, D.R. (Ed.). Reptile Medicine and Surgery. 2nd Ed. London. W.B. Saunders Company. pp. 224-234.

STERNBERG, E.D., D. DOLPHIN & C. BRUCKNER. 1998. Porphyrin-based photosensitizers for use in Photodynamic Therapy. Tetrahedron 54: 4151-4202.

STERNBERG, E.D. & D. DOLPHIN. 1996. Pyrrolic photosensitizers. Current Medicinal Chemistry 3: 293-324.

TARDIVO, J.P., A.D. GIGLIO, C.S. OLIVEIRA, D.S. GABRIELLI, H.C. JUNQUEIRA, D.B. TADA, D. SEVERINO, R.F. TURCHIELLO & M.S. BAPTISTA. 2005. Methylene blue in photodynamic therapy: From basic mechanisms to clinical applications. Photodiagnosis and Photodynamic Therapy 2: 175-191.

First Use of a GPS Satellite Tag to Track a Post-Nesting Hawksbill (*Eretmochelys imbricata*) in the Hawaiian Islands With an Indication of Possible Mortality

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Hawaiian hawksbills (*Eretmochelys imbricata*) have been satellite tagged in the main Hawaiian islands (MHI) with standard Argos tags since 1997 (Parker *et al.* 2009). These turtles showed short inter-island migrations. However, the number and quality of transmissions for these tags were relatively poor with few positions with high accuracy. This poor accuracy may be a result of hawksbills having short surface periods and longer dive times (van Dam *et al.* 2008). Global Position System (GPS) tags have become increasingly popular (Jones *et al.* 2013) and obtain GPS data using a rapid "quick fix" technology producing positions that are more accurate than regular Argos data, which they also obtain (50 m. vs. 150 m. accuracy, Argos User's Manual 2011).

An adult post-nesting hawksbill was satellite tagged after the turtle's third nest was laid on 4 October 2011 during its first recorded nesting season at Makena State Park, Maui. A Telonics GPS tag (model TGM-4410; Telonics, Inc., Arizona) was attached to the turtle using modified procedures based on Balazs *et al.* (1996). We attached a GPS tag to this hawksbill turtle (the first GPS tag deployed for Hawaiian hawksbills) in order to obtain more detailed information on the movements of post-nesting hawksbills in Hawai'i. Nearly all documented hawksbills nesting on Maui have been tracked to their foraging grounds with either radio or satellite tags (Parker *et al.* 2009, NOAA unpub.). We combined the diving data received from the tag in this study with the positional data (GPS and Argos) to better understand diving and surfacing behavior. Most hawksbill turtles spend regular intervals diving and surfacing, spending more time underwater than on the surface (Blumenthal *et al.* 2009; van Dam *et al.* 2008). Hence, we interpreted our dive data to include the idea that a healthy hawksbill turtle would spend a percentage of time both above and below water over any multiple day period.

The satellite tag obtained GPS positions every 6 hr. and obtained Argos positions and dive data via a 6 hr. on/24 hr. off duty cycle. This duty cycle was used for transmitting data as well and the satellite tag transmitted GPS data preferentially, followed by Argos positions and lastly dive data. Dive data included the percent time spent underwater in 12 hr. and the number of dives in 12 hr. as well as dive durations; a dive was recorded if the turtle was underwater for more than 10 s. The GPS tag was programed so that it would transmit constantly if the salt-water contacts were continually bridged for more than 24 hr. (fail-safe), and the tag would revert to the programed duty cycle once the contacts were no longer continually bridged. Days were expressed as a linear progression rather than dates. Percent time on surface was defined as the time recorded not spent underwater. Averages of percent time on the surface, dive duration, and number of dives in a 12 hr. period were examined for differences between near shore and offshore areas, as well as compared with the corresponding track data over the course of the track. Distance of travel was calculated using the single highest accuracy position per day as close to 12:00 UTC as possible. The coastline data were Global Self-consistent Hierarchical High-resolution Shoreline (GSHHS) data (Wessel & Smith 2013) and bathymetry data were obtained from School of Ocean and Earth Science and Technology (SOEST) and General Bathymetric Chart of the Ocean (GEBCO) databases. Distances from shore were calculated from the estimated nearest coastline position, extrapolated from Google Earth (2012), and best Argos positions for that day using the great circle method of calculation (Bowditch 1995). Near shore was defined as within 2 km. of shore, while offshore was over 2 km. from shore. The islands of Hawai`i are volcanic mid-ocean mountains, so near shore areas are not as extensive as those involving a continental shelf. In addition, depths usually drop off steeply between 1-2 km. off shore, except between the islands of Moloka`i, Maui and Lana`i (Google Earth 2012; GEBCO data).

Previous published data on the movements of Hawaiian hawksbills showed only short distance movement within the main Hawaiian islands (Parker *et al.* 2009). Previous tracking has shown that Hawaiian hawksbills stay close to shore during their post-nesting movement except when moving between islands, with most turtles ending up in a foraging area near shore (Parker *et al.* 2009). The hawksbill in this study traveled from Maui along the islands of Moloka`i and Kaua`i, then far offshore with transmissions ending on Day 111 (Fig. 1). During the inter-nesting period near Maui (days 1-18), the hawksbill stayed within 2 km. of the nesting beach while staying approximately 250 m. from shore in waters around 15 m. deep. The turtle nested one last time on day 18 before moving away from the Makena State Park area (Fig. 1, 20.6°N 156.4°W).