

Synopsis of infections in sea turtles caused by virus, bacteria and parasites: an ecological review

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Abstract

During the last centuries sea turtle populations worldwide have been declining or have been driven nearly to extinction due to human activities. According to IUCN, all of the world's seven sea turtle species have become threatened, five of them are endangered and two vulnerable. This precipitous decline in sea turtles numbers has awakened an interest in the use of classical anatomic pathology to describe their infectious diseases their prevalence and to determine cause of death. Lists of parasites species of sea turtles exist and new species are described continually, but few data are available on parasite life histories; how infestation affects an individual turtle's health, growth, and reproductive output; or effects on population structure and dynamics in both the pelagic and terrestrial environments (Zug *et al.* 2001). However, very little is known about sea turtles in their wild environment. Our understanding of sea turtle biology, ecology and pathology is obtained almost entirely through the short phase in their lives when they come to ashore to lay their eggs or by incidental catch at sea. From these periods, pathogens and parasites such as virus, bacteria, protozoa, worms, leeches and insects have been found and described. This paper examines the known infections caused by virus, bacteria and parasites of sea turtles, and groups them using classic systematic taxonomy. Two families of viruses (possibly six), 56 species of bacteria, 15 fungi, 6 protozoa, 87 plathelminthes (mainly trematodes), 6 nematodes, 4 annelids (leeches), 17 arthropods were found to be the cause or related to infections in sea turtles. The roll of epibionts in sea turtles is also mentioned briefly. A taxonomic summary of infectious virus, bacteria and parasites are presented by sea turtle host species at the end of this synopsis.

Keywords: sea turtles, infections, diseases, pathogens, parasites, life cycle, epibionts.

Introduction

Sea turtles have graced the seas for hundreds of millions of years, and even in more recent times, millions of sea turtles filled the oceans. Scientists conservatively estimate that there were 100 million green turtles in the Caribbean Sea alone before the arrival of Europeans to the region. They ate 6.09 trillion kg of seagrass a year, or about half the Caribbean's annual production of seagrass biomass (Spotila, 2004). As the seagrass was consumed, nutrients were recycled and the seagrass beds were fertilized, resulting in a smoothly working ecosystem. Turtles seemed to have complete control of the diversity of plants and animals found there (Spotila, 2004). At high population levels, sea turtles play major roles in marine systems as consumers, prey, and competitors; as host for pathogens and parasites; as substrates for epibionts; as nutrient transporters; and as modifiers of the landscape.

In the last centuries, however, things have radically changed for sea turtles. Today, almost all species of sea turtles are under threat and some of them are nearly extinct due to human activities. For instance, during the last 25 years the Pacific leatherback sea turtle population has been reduced in 95% and nearly depleted (Reina *et al.* 2002).

There are numerous threats to marine turtle populations, many of which are caused by man. Nevertheless, there are also natural threats such as predation and infections caused by pathogens

and parasites. These factors must be studied in an effort to assess their impact on these endangered species (Broderick *et al.* 1997).

In the first part of this paper, a general description and identification of all sea turtles species will be presented, as well as their habit and geographical distribution. Even though there are just seven species of marine turtles, their evolutionary process, morphology and identification is not simple. Without a general overview of the sea turtle's world the understanding of their pathology and parasitology will be limited.

Infections diseases, as a group, are one of the largest causes of morbidity and mortality in all reptiles. Infections in sea turtles are almost always the result of immunosuppression. Infections are often associated with the stress of captivity (Mader, 2006). The compromise of the immune system in the host in many of the infections caused by microorganisms and virus will be mentioned and discussed on the following pages.

Most of these pathogens and parasites have not yet been described properly and there is a definite lack of adequate information. Furthermore, our knowledge regarding infections in these animals is extremely limited, hindered even more so by the limited literature containing almost no description of the parasites life cycle. Despite these limitations, the cause and origin, mode of proliferation, effects and diseases for some of the most common pathogens and parasites will be presented and discussed.

The references and sources used in this report show where the latest available information was obtained; and it is extremely important to clarify that most of these references do NOT necessarily indicate which author(s) described the infection, disease, pathogen or parasite for first time.

The main purpose of this paper is to produce a general index listing all known infections in sea turtles that may be caused by virus, bacteria and parasites. The report can be used as a source for persons who are interested in sea turtles and their diseases. A large number of articles related to parasites and pathogens in sea turtles will be discussed as well as some of the most important diseases resulting from these infestations.

Part One. General introduction

1. Evolution of marine turtles and geographic distribution

Seven species of sea turtles are found worldwide. Of these the following five are considered endangered by the International Union for the Conservation of Nature and Natural Resources (IUNC) red list of threatened species; Leatherback (*Dermochelys coriacea*), Kemp's Ridley (*Lepidochelys kempii*), Olive Ridley (*Lepidochelys olivacea*), Hawksbill (*Eretmochelys imbricata*) and Green (*Chelonia mydas*). The remaining two species are classified as vulnerable; Flatback (*Natator depressa*) and Loggerhead (*Caretta caretta*).

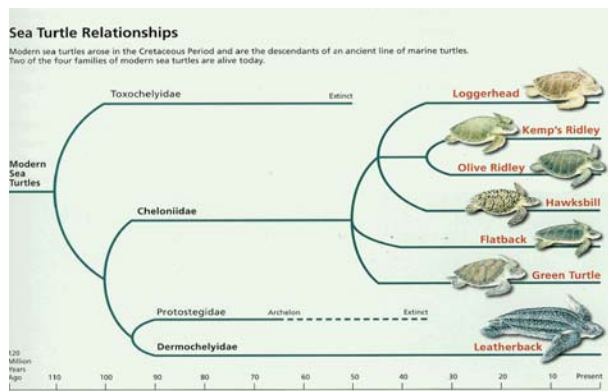


Figure 1. Sea turtle evolutionary tree (relationships) in million of years. Only two of the four families of modern sea turtles are alive today (picture taken from Spotila, 2004. P. 59).

The oldest marine turtle fossils found date from the Jurassic period (208 to 145 million years ago). By that time, the main lineage of turtles had split into two branches: the side-neck turtles (pleurodires), which protect the head by folding the neck and head over to one side, and the hidden-neck or arch-neck turtles (cryptodires), which pull the neck into a vertical S-curve and retract the head straight back between the shoulders. The side-necked turtles produced many sea-going species during the Cretaceous period, but all of these died out. Modern pleurodires live

in freshwater. Jurassic sea turtles belonged to the hidden-neck group, the group to which more turtles belong today. Many families once made up the hidden-neck group (Cryptodira), but most had become extinct by the early part of the Cretaceous period (Spotila, 2004).

Four important families of hidden-neck sea turtles did survive into the mid-Cretaceous period. Two of these families, the Dermochelyidae and the Cheloniidae, have modern descendants. The leatherback sea turtle is the only surviving member of the Dermochelyidae family. All other modern sea turtles belong to the Cheloniidae (Perrine, 2003).

The six (or seven)¹ living species of sea turtles from the Cheloniidae family all have a well-developed, bony skull with a secondary palate and their carapace and plastron are fully formed of bone. They occupy a variety of ecological roles or niches in the oceans by living in a variety of habitats and eating many different types of food such as seagrass, crabs, clams, and sponges (Spotila, 2004). Only one species, the green turtle (*Chelonia mydas*), is largely herbivorous and grazes on seagrasses; the other sea turtles are mainly carnivorous, feeding on jellyfish, crabs, barnacles, sea urchins, fish, and sponges (Mader, 1996).

Their ancestors arose in the early Cretaceous in parallel with the extinct family Toxochelyidae. As many as twenty different types plied the oceans over time, but only five of these survive today as distinct genera, *Chelonia*, *Eretmochelys*, *Lepidochelys*, *Natator* and *Caretta* (see Figure 1 above). The chelonids were more generalized species than the toxochelids and thus were able to out-complete them for resources and were more adaptable to changing conditions in the oceans (Spotila, 2004).

Finally there is the family Dermochelyidae, which has a sole extant species, the leatherback turtle. Splitting off from the Protostegidae (the fourth extinct family) in the late Cretaceous period, about 100 million years ago, the leatherback family reached a peak of diversity in the Eocene Epoch (50 million years ago) after the Protostegids had disappeared. It was as if new niches opened in the seas and leatherbacks rushed to fill them. At least six different species of leatherbacks lived in the oceans worldwide, their fossils found in Africa, Europe, North America, and New Zealand. These turtles were very much like the modern leatherback, though carapace structure varied from fairly thin, flat shells to thicker, more robust shells with pointed ridges, although all of them made up of small bony plates. As time progressed, food source became more limited and leatherbacks had to specialize as

¹ Valid arguments can be presented both in favour and against the designation of Black turtle as a full species within the genus *Chelonia*; namely, *Chelonia agassizii*. The systematic status and nomenclature of Black turtle or east Pacific green turtle sometimes referred to *Chelonia agassizii* or *Chelonia mydas agassizii* remains uncertain.

food types diminished. Leatherback diversity decreased to the extent that only two species survived at the end of the Miocene Period (around 25 million years ago), and by two million years ago only *Dermochelys coriacea*, the modern leatherback, survived. It appears that the secret to our leatherback's success was its adaptation into a jellyfish predator (Spotila, 2004).

Sea turtles inhabit all tropical oceans, with several species ranging into temperate water (see Figure 2 below).

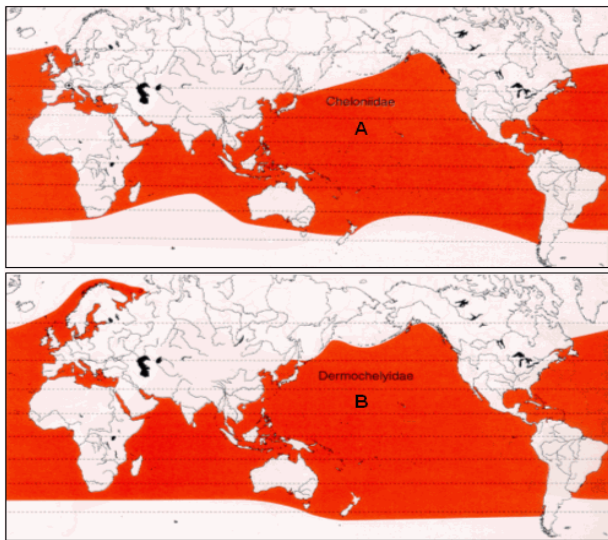


Figure 2. (A) Geographic distribution of the extant Cheloniidae, including the five different genera. Worldwide in tropical and some species as well in temperate seas. (B) Geographic distribution of the extant Dermochelyidae, representing by the unique *Dermochelys* genera species. Worldwide in tropical to cold temperate seas (pictures taken from Zug *et al.* 2001. P. 446-447).

Almost all seven sea turtles species have a worldwide distribution. Only two species show a geographically restricted distribution; the flatback (*Natator depressa*) is endemic to Australia and the kemp's ridley (*Lepidochelys kempii*) is restricted to the North Atlantic.

Leatherbacks are highly specialized, pelagic sea turtles. They are unique among the living reptiles because they are inertial endotherms. Leatherback turtles maintain body temperature above ambient temperatures and do so even in the cooler waters of the north and south temperate zones (Zug *et al.* 2001).

2. Anatomy, identification and ecology of sea turtles

Turtles are immediately recognizable because of their shell. The shell consists of an upper carapace and lower plastron connected laterally by bony bridges. The carapace consists of some 50 bones derived from ribs, vertebrae, and dermal elements of skin (Mader, 1996).

The carapace, or dorsal (top) shell, forms while the turtle is still in the egg. As the embryo develops, the ribs expand and then fuse to the

vertebral column. The ribs then grow connections to one another, forming a hard, shield-like structure (Spotila, 2004). The plastron evolved from the clavicles, interclavicles, and gastralia (abdominal ribs).

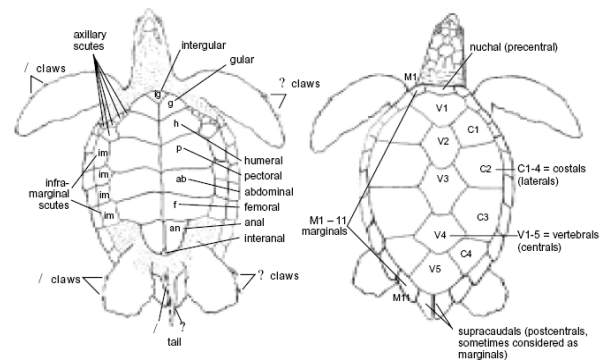


Figure 3. An illustrated guide to external morphological features of sea turtles, including scutes of the plastron (lower shell) and carapace (upper shell). Where scutes have more than one name, alternative names are provided in parentheses. The secondary sexual characteristics indicated are visible only in adult turtles. Note that inframarginal scutes span the distance between the marginal and large plastron scales (h, p, ab, f)- (picture taken from Pritchard and Mortimer, 1999. P. 2).

The bony shell is covered by a superficial layer of keratin shields called scutes. Scutes do not precisely overlap the underlying bones of the shell. Instead they are staggered so that the seams between scutes with each major growth sutures. Turtles produce new scutes with each major growth period and retain or shed the scutes from the preceding growth period. In some species scute growth zones or rings can be used to estimate age. This technique requires considerable expertise and is reliable only when a distinct growth period is present, as in wild temperate turtles. The difficulty of estimating age from scute growth zones is further appreciated when one considers that some species shed scutes (particularly temperate aquatic turtles) and that multiple growth zones can be produced within a single year.

Continuous growth is common in captivity, and growth zones can smooth with age. Therefore, contrary to popular belief, the age of most turtles cannot be determined accurately by counting so called "growth rings" on the scutes. Nevertheless, scales and scutes help to the identification and recognition of the different species (see Figure 4).

In all species except the leatherback the shell is made of scutes. And as mentioned before scutes are pieces of keratin covering the bone. Keratin is a hard protein substance that is remarkably similar to the protein that forms human fingernails and hair.

Unlike the hard shells of other sea turtle species, leatherback shells have no keratinized scutes but rather a leathery skin that lies over a loose mixture of thin bony plates connected by soft cartilage. Under the leathery shell lies a thick layer

of oily fat and fibrous tissue. Several bony keels, or ridges, run along the back of the turtle from the neck area to the tail. These keels appear to help in swimming, perhaps one of the factors contributing to the fact that the leatherback is the fastest swimming sea turtle (Spotila, 2004).

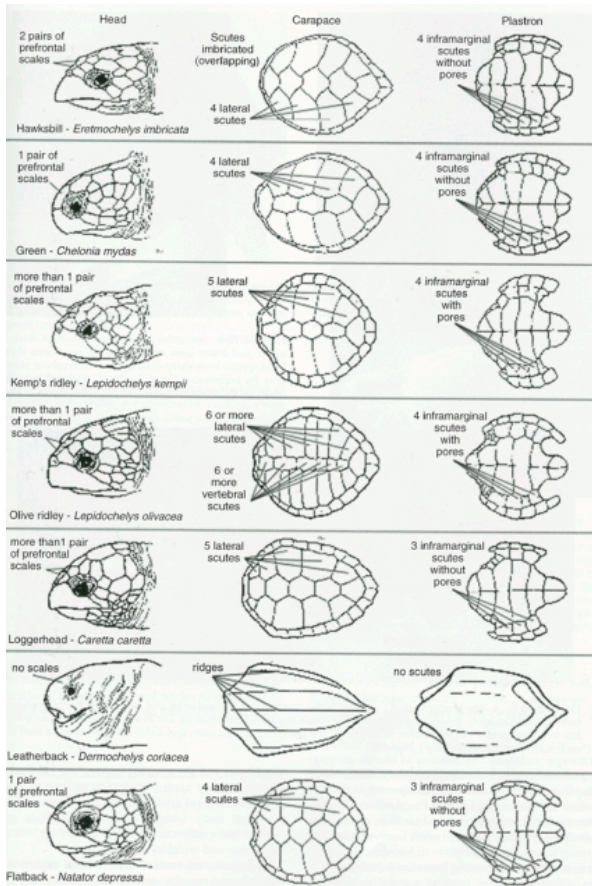


Figure 4. Key to sea turtles species showing the main external morphological features of sea turtles including scutes, scales and ridges used for identification of species (picture taken from Mader, 2006. P. 975).

Leatherbacks lack distinct head scales as adults, have no claws, and have a thin keratin covering on the jaws. The key characteristics include the presence or absence of complete longitudinal ridges along the carapace versus a hard keratinous shell (Dermochelyidae versus Cheloniidae, respectively). As mentioned before, the family Dermochelyidae is represented by just one extant species. Within the Cheloniidae, the six species are characterized and distinguished from one another by the number and patterns of particular scales on the head, the form of the jaws, the number of claws, and the numbers and pattern of the plates or scutes on the shell. The numbers and patterns of carapacial scutes are key characteristics (Mader, 2006).

In the sea age determination can be a difficult dilemma. Sometimes due to the effect of epibionts on the shell or other circumstances, scutes can

wear out after long periods in the sea water, making species determination very difficult and age estimation nearly impossible. Even in this age of technology, there is no precise technique or method to determine the exact age of these marine reptiles. The tagging and recapture methods remain the most efficient ways of age determination.

3. Pathogens, parasites and parasitism

A pathogen can be defined as anything that causes a disease or interferes with the normal cell functions by producing toxins or enzymes that may disrupt or destroy cells in the host. While, a parasite is specifically defined as an organism that lives on or inside another organism and eats the food of the host or the host itself. However, a disease is the result of a dynamic, detrimental relationship between an organism that parasitizes or interferes with the normal processes of cells and/or tissue of the host (Trigiano *et al.* 2004). A pathogen may or may not be a parasite. Put in other words, not all parasites are pathogens, but some parasites are more pathogenic than others. In fact, some organisms associated with a host do little or no harm. The term "commensalism" is used for describing this type of association (Buchmann and Bresciani, 2001). For example, a well known commensalism relationship in the sea is the case of epibionts. An epibiont is an organism which attaches itself to a host without either benefit or harm to the host, such as most barnacles, algae, and even sucker fish (remora).

Moreover, this relationship or association mentioned above between parasite and a host is defined as parasitism. The term parasitism emphasizes four important features of the host-parasite relationship:

1. Parasitism always involves at least two species, the parasite and the host (in some cases many other species can be involved as intermediate hosts).
2. Many of these parasitic associations produce pathological changes in the host that may result in disease.
3. Successful treatment and control of parasitic diseases requires not only comprehensive information about the parasite itself, but also a good understanding of the nature of a parasite's interactions with its hosts.
4. The parasite is always the beneficiary and the host is always the provider in any host-parasite relationship (Johnstone, 1998).

The following pages provide a general description of those interactions between pathogen-host mentioned above.

The long-term effects of pathogens on reptiles are relatively poorly known. Parasites can have a

nearly undetectable impact on their host or, in some instances, can kill their host. If the fitness of host (sea turtles) is negatively affected by parasitism, then parasites are effectively another kind of predators because the likelihood of an infected individual's genes being represented in future generations is reduced (Zug *et al.* 2001). Potential negative effects of parasitism in sea turtles include anaemia and reduced performance followed by reduced survival, competitiveness, social status, ability to sequester mates, and, for females, reduced fecundity (Bull and Burzacott, 1993).

Parasitism is so widespread and common among sea turtles that nearly every scenario is possible. Some attempts to list most of the parasitic species in sea turtles exist, and new species are described continually from reptiles. However, few data are available on pathogen life histories; how infestation affects an individual turtle's health, growth, and reproductive output; or effects on population structure and dynamics (Zug *et al.* 2001).

Sea turtles as the rest of the reptiles are ectotherms, the core body temperature of reptiles is more prone to fluctuate than that of mammals or birds. This range in body temperatures is more likely to allow pathogens to grow and proliferate. For instance mesophilic fungi are unable to grow at 37°C and so they can easily infect reptiles, especially if the animal is at suboptimal temperatures. The immune system, coupled with mechanical barriers such as the integument, protects the reptile from pathogen or parasite invasion. Sea turtles have a functional and complex immune system that is comparable with that of other vertebrates. The efficiency of the reptilian immune system is influenced by a combination of factors that include health and nutritional status, environmental temperature, seasonal changes, age and stress (Mader, 2006). Any change in the status of these factors would compromise the performance of the immune system in the turtle, allowing the breakdown of this barrier to the external environment full of opportunistic organisms ready to attack.

4. Location of pathogens in the host

Scientists have long been aware that large animals such as marine turtles can serve as a living substrate for various organisms. Practically any anatomical structure of the turtle, both external and internal represents a habitat and can host pathogens in the sea. Figures 5 and 6 below, show the external parts of a normal hawksbill turtle (*Eretmochelys imbricata*) and the internal organs in a leatherback (*Dermochelys coriacea*), respectively.

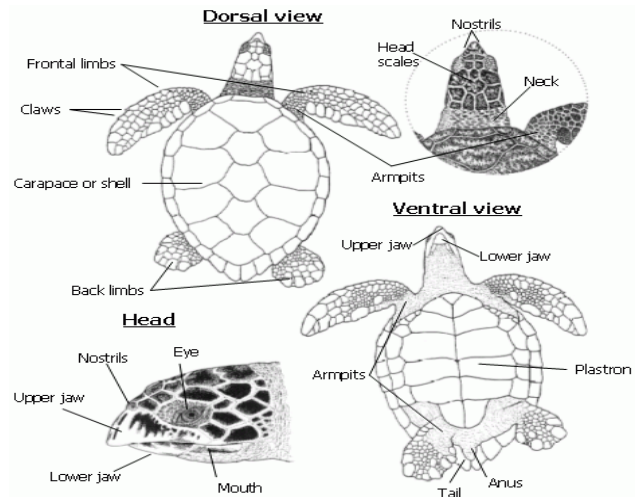


Figure 5. Dorsal and ventral external view of anatomic structures of an adult hawksbill turtle. External habitats and location of pathogens in the host (background picture taken from Pritchard and Mortimer, 1999 P. 8; picture modified and labelled by Alonzo Alfaro, 2006).

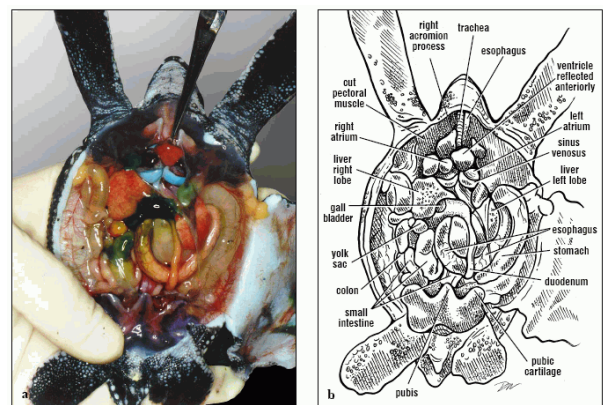


Figure 6a and 6b. Ventral view of a leatherback hatchling's viscera and heart. Internal habitats and location of pathogens in the host (picture taken from Wyneken, 2001 pag. 111).

There follows a description of the most common groups of pathogens and parasites which have been arranged according to classic systematic taxonomic. This is the most convenient way to explain them in terms of origin, biological description and mode of infection in the cases where that information was available.

Part Two. Systematic descriptions

5. Virus

Viruses are obligate intracellular pathogens; they can reproduce only by invading and taking over other cells as they lack the cellular machinery for self reproduction. They infect a wide variety of organisms: both eukaryotes (animals, fungi and plants) and prokaryotes (bacteria). It has been argued extensively whether viruses are living

organisms. Most virologists consider them non-living, as they do not meet all the criteria of the generally accepted definition of life. They are similar to obligate intracellular parasites as they lack the means for self-reproduction outside a host cell (Madigan and Martinko, 2006), but unlike parasites, viruses are generally not considered to be true living organisms as already mentioned before.

Twenty-four families of animal viruses have been described; of these, only 6 families occur in chelonians (Mader, 2006). Two of them are well documented in sea turtles (Herpesviridae and Papillomaviridae) and it is suspected that the other four families (Iridoviridae, Reoviridae, Retroviridae and Togaviridae) which infect other chelonians could possibly also infect marine chelonians. Within reptiles, sea turtle as hosts of virus are the least well studied of the major classes of vertebrates (Clark and Lunger, 1983).

In sea turtles, a virus may cause cellular damage that allows other pathogens (especially bacteria and fungi) to colonize unhealthy tissues. The reptile may clear the viral infection that initiated the disease process, and bacteria or fungus is often recovered from clinically ill animals. The relative lack of experimental infection studies in reptiles makes determination of which pathogens cause which problem difficult (Mader, 2006). In sea turtles experimental studies are even more problematic since virtually all the species are threatened and research and experimental are extremely restricted and limited by international laws.

Herpesviruses of undetermined relationship have been associated with three distinct syndromes in marine turtles: circular popular skin lesions (grey-patch disease); ulcerative lung, eye and tracheal (LETD) lesions; and external and internal fibropapillomas (Mader, 2006).

Nowadays, green turtle fibropapillomatosis is probably the most studied infectious disease in these reptiles. Fibropapillomatosis was first described in the late 1930s as an isolated case in the Florida Keys by Smith and Coates (1939). More recently, the prevalence has dramatically increased, and it has been estimated that more than 50% of the world's green sea turtle (*Chelonia mydas*) populations are now affected (Cray *et al.* 2001).

Some biologists consider fibropapillomatosis (FP) the most serious epidemic now raging in the natural (nonhuman) world, and there are several reasons for this distinction. Most epidemics are localized, striking animals in a single population; FP, however, has been found in turtles from every ocean basin in the world. In a surprisingly short period, FP has changed from a rare disease to an epidemic and from epidemic to a global pandemic. The high incidence of the disease also sets it apart from many other epidemics. In some populations more than 90% of turtles are affected. And somewhere along the line, the disease jumped species. Until recently, FP had been restricted to green turtles but Mader (2006) mentioned in his

book that the disease is now found in all species of sea turtles except for the leatherback. However, Huertas *et al.* (2003) published the first official record of a leatherback infected by FB. Now FP tumours have been reported in all seven species of sea turtles.

The fibropapilloma tumours are most often seen on front flippers and neck, periocular tissues, plastron, carapace, and the cornea (occasionally) and in the lung, liver, kidneys, and gastrointestinal tract (Campbell, 1996) (See Figure 7). FP is a severe debilitating disease with the tumours occluding vision, impeding locomotion, obstructing the gastrointestinal tract, and reducing breathing capacity. The understanding and recognizing the importance of this disease has generated considerable effort to find its cause (Cray *et al.* 2001).



Figure 7. Picture showing a green turtle (*C. mydas*) infected by FP. Tumours grow all around the neck, eyes and limbs of the animal (picture taken from Perrine, 2003).

The aetiology of FP is unknown, although a variety of hypotheses concerning that pathogenesis of FP or more specifically green turtle fibropapillomatosis (GTFP) have been proposed and discussed by scientists without any real conclusions being reached. For years scientists have been trying to determine all the possible transmitter agents that have spread the disease during the last 60 years. Possible aetiologies include viruses, metazoan parasites, ultraviolet radiation, and chemical carcinogens (Herbst, 1994). Another group of scientists have added trematode ova to this list (Dailey and Morris, 1995), while pollutants, temperature, and genetic factors have been suspected as causal agents (Balazs and Pooley, 1991). Currently the strongest evidence indicates the involvement of a herpesvirus (Herbst *et al.* 1998 and Quakenbush *et al.* 1998). This involvement of a virus will undoubtedly compromise the sea turtle immune system at the time it first becomes afflicted with fibropapilloma disease. This is an important consideration because many of the papilloma-associated lesions could be secondary to a compromised immune system (Duncan *et al.* 1975 and Sundberg *et al.* 1994).

Dr. Cray and his research group at the University of Florida in 2001, compared blood samples from two groups of turtles, one containing turtles infected with fibropapilloma, the other comprised of healthy individuals. They revealed a significant difference in the number of lymphocytes² between the two groups. The research showed a lower number of lymphocytes and altered white blood cell composition for the diseased turtle group. It may be a relation between altered immune function related to the development of the papillomas, but unfortunately very little is still known about the green sea turtle immune system and more research is needed to understand completely the FP disease.

FP and other isolated cases of disease outbreak in some wild turtle populations suggest the possibility of immunosuppression as a result of environmental contaminants. In higher vertebrates, DNA adduct formation (binding of pollutants to DNA) is thought to be the early event leading to neoplasia and malignancy; in general, neoplasia or malignant tumours are uncommon in lower vertebrates. However, the incidence of FP, characterized by single to multiple histologically benign fibroepithelial tumours, has been reported in several marine turtle species (Gardner and Oberdörster, 2006).

Although FP is not cancerous, its excessive growth internally and externally is life threatening to turtles. As mentioned before, experimental results suggest a filterable infectious agent, probably a virus, as the primary cause. The papillomas have never been reported in the youngest juveniles of the pelagic phase but increase in populations in the larger size class living in near-shore waters (Gardner and Oberdörster, 2006). Several field studies also suggest a prevalence of FP in green sea turtles that reside in marine habitats that have been affected by agricultural, industrial, or urban development. This has led to speculation that environmental contaminants may play a role in FP pathogenesis.

Grey-patch disease primarily affects 56-day old to 1-year old captive-raised green turtles (Jacobson, 1991). In the first reported outbreak, most affected turtles were 8 to 10 weeks old. Skin lesions are characterized by small nonspreading papules or, more typically, raised coalescing areas of raised grey patches that frequently ulcerate. Overcrowding and high temperatures are considered precipitating factors in the development and severity of lesions in captive turtles (Rebell *et al.* 1975 and Mader, 2006).

Besides the papilloma virus as a potential infective agent in the FP disease, there are also other recorded infections caused by herpesvirus. A herpesvirus-associated disease characterized by ulceration and accumulation of caseous debris

over the globe and the oropharynx³, lungs, and trachea has been described in marine turtles. Affected turtles typically died weeks to several months after the onset of clinical signs. More than a decade of studies has concluded that most epizootics were found to occur between January to August. Most affected turtles had secondary gram-negative bacterial infections of the virus-damaged epithelial tissues. Typically, herpesviruses have a restricted host range (Mader, 2006). Nevertheless, the relationship of the herpesviruses recovered from sea turtles has been insufficiently studied and herpesviruses may infect any genera and individuals of any age. Studies have mainly been conducted in the one single commercial species, the green turtle (*Chelonia mydas*) which has a very high value from the aquaculture point of view in some parts of the world. Very little is known about virology in the remaining 5 sea turtles families and non literature was found.

6. Bacteria

Bacteria are microscopic, unicellular organisms. All bacteria reproduce through asexual reproduction (one parent) binary fission, which results in cell division. Two identical clone daughter cells are produced. Bacteria, as asexual organisms, inherit an identical copy of their parent's genes (i.e. are clonal). All bacteria, however, have the ability to evolve through selection on changes to their genetic material (DNA) which arise either through mutation or genetic recombination. Most bacteria are either spherical, called coccus (pl. cocci, from Greek kókkos, grain, seed) or rod-shaped, called bacillus (pl. bacilli, from Latin baculus, stick) (Madigan and Martinko, 2006).

Bacteria appear to play a very important role in marine turtle diseases, both as primary pathogens and as secondary invaders when host's immune system has been compromised. Those species of bacteria that have been found in turtles are commonly isolated in localized infections but are also involved in epizootics characterized by bacteraemia (presence of bacteria in the blood) and septicaemia⁴ (Cooper, 1983).

Gram-negative bacteria are the most common bacterial pathogens. This fact is not surprising because gram-negative bacteria are common isolates in healthy reptiles. Most gram-positive bacteria are not considered pathogenic in reptiles; they are actually common inhabitants, especially of the skin. However, some gram-positive bacteria can cause disease (Mader, 2006).

Most of the studies conducted on identification of bacteria species in sea turtles have been focused in the search for a potential pathogen epidemiologically linked to the etiologic agent of

² Lymphocytes are one of the five kinds of white blood cells or leukocytes

³ Oropharynx is the part of the throat at the back of the mouth. It includes the soft palate, the base of the tongue, and the tonsils.

⁴ Septicaemia is defined as a systemic disease associated with the presence and persistence of pathogenic microorganisms or their toxins in the blood.

Fibropapillomatosis. So far, no study has proved any potential relation between bacterial pathogenic infections with the already mentioned FP. From the nasopharyngeal and cloacal swabs taken from green turtles researchers managed to isolate 28 Gram negative bacteria, five Gram positive cocci, *Bacillus* sp., and diphtheroids⁵. *Pseudomonas fluorescens* was the most frequent bacterium isolated in both locations, followed by *P. putrefaciens*, *Vidrio alginolyticus*, non-hemolytic *Streptococcus*, *V. damsela* and *V. fluvialis* (Aguirre *et al.* 1994). Due to the nature of this research, no conclusion regarding the infection mode or life circle of the pathogens was studied.

From 1993 to 2001, a group of researchers observed digestive lesions in 84 of 136 sea turtles (128 *Caretta caretta*, four *Chelonia mydas* and four *Dermochelys coriacea*) stranded in the Canary Islands. A wide range of Gram-negative and Gram-positive bacteria, including *Bacillus* sp., *Escherichia coli*, *Pasteurella* sp., *Proteus* sp., *Staphylococcus* sp., *Streptococcus* sp. and *Vibrio alginolyticus*, were isolated from these lesions. During the necropsy, lesions were also commonly observed in the liver; they affected 29 of the turtles and were associated with *Aeromonas hydrophila*, *Citrobacter* sp., *E. coli*, *Proteus* sp., *Staphylococcus* sp. and *V. alginolyticus* infections (Orós¹ *et al.* 2004).

However, marine bacteria of the genus *Vibrio* are commonly suspected in mortality of crustaceans, teleost fish and shellfish, particularly when adverse environmental conditions, nutritional deprivations or overcrowding are expected (Ghittino *et al.* 1984). Obendorf *et al.* (1987) reported a unique case of a leatherback turtle (*Dermochelys coriacea*) found dead in the eastern coast of Tasmania. Necropsy determined that the animal died by drowning as a result of a pre-existing valvular endocarditis and septicaemia. In other words, a systematic disease caused by a pathogenic organism, in this case *Vidrio damsela*, which was isolated from the endocardial thrombus. The route of infection in this stranded turtle may have been through the intestinal lesions with haematogenous spread to liver and heart that were also reported in the study. The importance of vibriosis as a mortality factor of leatherback turtles still remains unknown.

In a human health case, *Vidrio mimicus* was isolated and determined to be the agent causing chronic diarrhoea in 33 hospitalized patients referred in different geographic regions of Costa Rica between 1991 and 1994. The consumption of sea turtle eggs from olive ridley turtle (*Lepidochelys olivacea*) is still allowed in this country. The common factor in this case was that 19 patients were interviewed and all recalled the ingestion of raw turtle eggs within a week of the first symptoms of diarrhoea appearing (Campos *et al.* 1996). A complete investigation was done by

the health authorities and they managed to isolate the same pathogen from eggs in the market. This was the first time *V. mimicus* was isolated from a sea turtle (*L. olivacea*). The aetiology of the pathogen was not even mentioned in the article and still remains unknown.

Moreover, the association of sea turtles with bacteria has attracted particular attention because of the potential danger of such infections to humans. *Salmonella* sp. in particular is one of the most studied groups due to the salmonellosis disease in humans. Remarkably, only one single record made by Keymer *et al.* (1968) was found in the literature. They described very briefly a catarrhal colitis in the hawksbill sea turtle *Eretmochelys imbricata* infected by what was a new species at that time, *Salmonella regent*. Furthermore, Marcus (1971) reported enteritis and septicaemia as possible manifestations of reptilian salmonellosis.

Mycobacterium sp. are everywhere in the environment. Potentially pathogenic species in sea turtles include *M. chelonae*, *M. avium*. Although commonly isolated from cutaneous lesions, mycobacteria can also cause systematic illness accompanied by non-specific signs such as anorexia, lethargy and wasting. *Mycobacterium chelonae* has been shown to cause osteoarthritis and systematic disease in sea turtles, and the bacteria has recently been purely isolated in a Kemp's Ridley sea turtle (*Lepidochelys kempi*) recently (Greer *et al.* 2003 and Mader, 2006).

The Gram-positive, non-motile microaerophilic bacteria *Aerococcus viridans* is a pathogen of crustaceans, causing gaffkemia⁶ in marine lobsters (*Homarus americanus*), and has also been isolated from rainbow wrasse (*Coris julis*) (Torrent *et al.* 2002). In December 1998 a female loggerhead sea turtle was found still alive floating off the coast of Gran Canaria; the animal died some hours after. From the necropsy it was concluded that the cause of death, was an oesophageal diverticulum, which means an outpocketing of the wall of the oesophagus or of the pharynx just above the opening to the oesophagus. This was the first time that this disease was reported in a sea turtle, and *A. viridans* was isolated in the same tissue of the animal. No other microorganism was isolated. The researchers concluded that for this turtle *A. viridans* may have entered by ingestion of one infested lobster and from there proliferated in the diverticulum, resulting in the death of the animal (Torrent *et al.* 2002).

Most of the bacteria that have been reported in sea turtles are non-specific pathogens and previously they have been found in fish, crustaceans or other marine animals.

⁵ Diphtheroids are species of the genus *Corynebacterium* which are non-pathogenic. They are normally commensals of the skin and upper respiratory tract.

⁶ Gaffkemia is a bacterial disease of lobsters mainly associated with the pathogen *Homarus americanus* and *H. gammarus* and often causes fatal septicemic disease in lobsters. The mean time to death for infected lobsters increases as temperature decreases.

7. Fungi

Fungi (plural of fungus) are saprophytic and parasitic organisms that lack chlorophyll and therefore no photosynthetic. They were once considered part of the plant kingdom but are now recognised as a separate kingdom. Along with bacteria, fungi are the principal organisms responsible for the decomposition of carbon in the biosphere. Fungi have two ecological advantages over bacteria: (1) they can grow in low moisture areas, and (2) they can grow in low pH environments (Austwick and Baxter, 1983). Most fungi are saprophytes, obtaining their nourishment from dead organic matter. But there are many others which are extremely parasitic to almost any kind of living organism and they cannot live without taking energy and/or nutrients from a host. Fungi exist in an anamorph (asexual, mitotic, and imperfect) or a teleomorph (sexual, meiotic and perfect) stage and reproduce by means of asexual spores (e.g. ascospores, zygospores) (Deacon, 1997).

There are relatively few publications and reports about mycotic diseases in sea turtles compared with other reptiles, birds, and mammals, although infections have been described in both captive (Jacobson *et al.* 1979; Wiles and Rand, 1987; Leong *et al.* 1989; Glazebrook and Campbell, 1990; Glazebrook *et al.* 1993) and wild sea turtles (Lewbart and Medway, 1993; Cabañes *et al.* 1997).

A number of fungi are true pathogens of mammals but to date none have been clearly identified as a true pathogen of reptiles. Infection in sea turtles by fungi has been regarded as opportunistic, caused by normally saprophytic organisms that invade living tissue strictly under favourable circumstances (Mader, 2006).

Fungal skin diseases in captive marine turtles are believed to be more frequent than in wild sea turtles (Wiles and Rand, 1987). For instance a dermatomycosis, a normal fungal infection, affects mostly the shell and less often the feet and skin. All marine turtles are susceptible (Mader, 2006). The main pathogenic agent of dermatomycosis infection in sea turtles has been identified as being caused mainly by *Fusarium* species.

Fusarium solani (Austwick and Baxter, 1983; Cabañes *et al.* 1997), *Aspergillus* sp. (George, 1997), *Geotrichum* sp., *Penicillium* sp., *Scolecobasidium* sp., *Drechslera* sp. (Leong *et al.* 1989; Sison *et al.* 1990) and several unidentified fungi (Wiles and Rand, 1987) have been isolated from skin lesions in sea turtles. *Fusarium* species are common soil saprobes and plant pathogens. Although *Fusarium* species are not considered part of the normal epidermal microbiota of marine animals (Frasca *et al.* 1996) they have occasionally been reported in marine environments (Rebell, 1981). However, in recent years they have been reported with increasing frequency as causes of opportunistic infections in humans and animals, including sea turtles. These infections have usually been limited to superficial mycoses, but recently

the number of deep tissue and disseminated infections has greatly increased, particularly in captivity sea turtle patients with underlying immunosuppressive conditions (Castellá *et al.* 1999).

Cabañes *et al.* (1997) reported a massive cutaneous infection in a sea turtle (*Caretta caretta*) caused by *Fusarium solani*. When the turtle was collected, although it had suffered traumatic injuries, it has no visible signs of fungal infection. Cutaneous fungal lesions appeared during the second month of the rehabilitation period and so it was thought that the source of infection was related to the tank where the turtle was kept. Cultures of the sand from the tank yielded growth of fungal colonies belonging to *F. solani* (see Figure 8). In spite of the fact that numerous species of *Fusarium* are considered to be cosmopolitan (Nelson *et al.* 1994), in the mycological survey of the sand from the tank *F. solani* was the only *Fusarium* species isolated (Cabañes *et al.* 1997).

Fusarium solani was also reported as the causative agent of an infection of the shells and skin of baby loggerhead turtles in the Bahamas (Rebell, 1981). On this occasion the source of the infection was traced to *Fusarium*-infected rubberized horse hair matting used as a filter in an onshore marine well, dug into the sand, which supplied water to a wet lab facility.

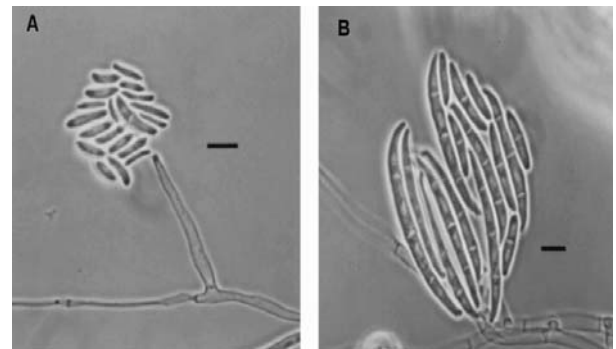


Figure 8. (A) Characteristic conidiogenous cell and microconidia in false heads of *Fusarium solani*. (B) Characteristic macroconidia of *F. solani*. Both isolated from a culture of loggerhead (*Caretta caretta*) mycotic infection. Bar, 8 mm (pictures taken from Cabañes *et al.* 1997. P. 3344-3345).

As well as *Fusarium* species, *Colletotrichum acutatum* is a cosmopolitan plant pathogen with a wide host range. Before 2000, there were not reports in the veterinary literature of any species of *Colletotrichum* naturally infecting animals. But on 14 March 2000 a juvenile Kemp's ridley died and *Colletotrichum* sp. was identified as the cause of death. Four months earlier, the sea turtle was admitted to a Sea Turtle Rehabilitation Hospital in Florida, with a severe cold stunning (extended hypothermia). Gross necropsy findings included abnormalities in the lungs, kidneys, and liver. Histopathological findings included severe chronic granulomatous mycotic nephritis and pneumonia and a chronic moderate granulomatous hepatitis

as well as a granulocytic hyperplasia of the bone marrow of the carapace. Cultures were taken from the fungal hyphae, and subculture of the isolate was identified as *Colletotrichum acutatum* (Manire *et al.* 2002).

In both cases (*Fusarium* sp. and *Colletotrichum* sp.), it is important to stress that the fungal infections were not wholly responsible for the death of the animals and it is possible to speculate and argue that the immune systems of the sea turtles were already compromised. Although it is almost impossible to determinate in sea turtles at present, it is suspected that the turtles in these cases were immunocompromised. It is unlikely that a plant pathogen would cause primary disease in an immunocompetent animal, and one can only speculate where the turtle acquired the fungus.

Coelomycetes are known to survive in salt water (Sutton, 1980) and may have been acquired there, but it is also possible that it was acquired as an airborne inhalant during rehabilitation.

Systemic mycotic infections occur primarily in the lungs, even though granulomas can be found in the liver of wild and captive sea turtles (George, 1997). Pulmonary mycoses infections were described in Kemp's ridley turtles (*Lepidochelys kempi*) due to *Scolecobasidium constrictum* and *Paecilomyces* sp. by Leong *et al.* (1989) and Lewbart and Medway (1993). In the same article, Leong *et al.* (1989) described a variety of necrotic lesions on the head, neck, and shell of Kemp's ridley turtles, associated with different fungal pathogens including *S. constrictum*, *Paecilomyces* sp., *Penicillium* sp. and *Cephalosporium* sp. From a necrotic skin lesion of a Philippine hawksbill turtle (*Eretmochelys imbricata*) Sison *et al.* (1990) isolated *Geotrichum* sp., *Penicillium* sp., *Scolecobasidium* sp., *Drechslera* sp. and *Fusarium* sp. as already mentioned above.

Jacobson *et al.* (1979) isolated *Sporotrichium* sp., *Cladosporium* sp., and *Paecilomyces* sp. from pneumonic lesions in captive-reared green turtles (*Chelonia mydas*). *Paecilomyces* sp., *Fusarium scirpi* and *Penicillium* sp. were isolated from pneumonic lesions in farmed green turtles in Australia by Glazebrook and Campbell (1990).

More recently *Candida albicans* was also isolated for first time from a systemic mycotic infection in a loggerhead sea turtle in the Canary Islands (Orós *et al.* 2004), hence increasing the number of fungi species that can be pathogenic to sea turtles.

Unfortunately, there is usually little or no hematologic evidence when mycotic infections develop in sea turtles.

8. Protozoa

A protozoan is a complete organism in which all life activities occur within the limits of a single plasma membrane. Protozoa was for many years the name of a phylum. Evidence from electron microscopy, life cycle studies, genetics, biochemistry, and molecular biology has shown that this group include at least seven phyla (and

according to some authors, up to 30) (Hickman *et al.* 2001).

The eimeriid coccidian are single-celled intracellular eukaryotic parasites that pass an oocyst⁷ in the faeces of their definitive host. They parasitize all classes of reptiles, including turtles. Most of these parasites develop in the epithelial cells of the gut, but a few species develop in other organs (Greiner, 2003).

Documented cases of protozoan parasites in sea turtles exist for numerous populations throughout the world. For instance *Entamoeba invadens* is a protozoan that was described as causing mortality in green turtle hatchlings in captive situations (Aguirre *et al.* 2006). *Crystosporidium parvum* is considered an emerging foodborne pathogen in humans (Schlundt *et al.* 2004) with symptoms of infection that include diarrhea, nausea, abdominal cramps, vomiting, and fever. *Crystosporidium* sp. has been observed in fecal and intestinal samples from free-ranging green turtles in the Hawaiian Islands as a potential source of marine waterborne oocyst. Raw sewage disposal into marine waters is a common practice in many coastal areas and enhances the risk of pathogen pollution and potential transmission to both humans and sea turtles (Graczyk *et al.* 1997).

In 1973, an epidemic disease in hatched green turtles was found to be associated with what was at that time a new coccidial intestinal parasite. Coccidiosis appeared sequentially in two groups of turtles hatched in the spring of 1973. The disease appeared in the second and larger group of hatchlings after they had already reached a maximum of 15 days old. Characteristic ellipsoidal oocysts of the parasite were abundant in the intestinal tract. The severity of coccidial infections depends ultimately on the number of oocysts which are ingested and the resistance state of the host animal. In the article published by Rebell *et al.* in 1974, two genera of coccidial parasites are mentioned, *Eimerie* sp. and *Caryospora* sp. but without any proper description of the parasite which causes the Coccidiosis. It is also mentioned that the taxonomic description will be published separately. With little reference to the previous study, in 1978 Leibovitz *et al.* published a paper with the description of a completely different species of coccidial pathogen, *Caryospora cheloniae*. Tissue sections of the internal organs and intestinal contents were collected during necropsy from dead, moribund and clinically normal turtles; the greatest concentrations of developmental stages of *C. cheloniae* were found in the hindgut.

Unsporulated oocysts were elongate, ellipsoidal in shape, and with smooth walls. Sporulated⁸ oocysts (see Figure 9) were monosporocystic and octozoic; but during the process of sporulation

⁷ Oocyst = a thick-walled structure in which sporozoan zygotes develop and that serves to transfer them to new hosts.

⁸ Sporulate = to produce or release spores.

(releasing of the spores), the fragile double layered oocyst wall usually disintegrated. The article discussed the economical importance of *C. cheloniae* protozoan in mariculture-reared green sea turtles, and also reported the first *Caryospora* record for sea turtles.

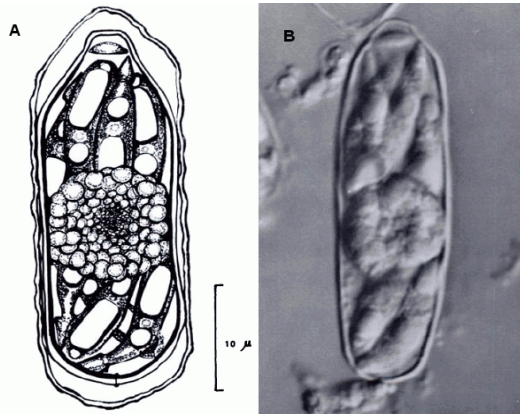


Figure 9. (A) Illustration of a sporulated monosporocystic octazoic oocyst of *Caryospora cheloniae*. (B) A single sporulated sporocystic of *Caryospora cheloniae* showing the central residuum, sporozoites with refractile and pointed apical ends (pictures taken from Leibovitz *et al.* 1978. P. 271 and 273).

Even though there are several documented cases of protozoan in sea turtles, the paper of Leibovitz *et al.* (1978) is probably the only case where a complete pathologic description was given.

In a more recent publication in Portugal, Eiras *et al.* (2000) described small intraerythrocytic (elongate organisms which divide by binary fission) inclusion bodies from the blood cells of twenty juvenile loggerhead sea turtles (*C. caretta*). Unfortunately, the authors of this paper did not manage to identify whether those structures were viral, rickettsial (a type of bacteria), or have another origin.

9. Metazoa

Multicellular organisms (metazoa) are typically divided into three main groups: 1- Mesozoa (a single phylum), 2- Parazoa (phylum Porifera, sponges; and phylum Placozoa), and 3- Eumetazoa (all other phyla) (Hickman *et al.* 2001). Using modern ecologic terminology, macroparasite are metazoan organisms that survive on or within another animal (Mader, 2006). Thus by convention and definition, Fungi and Protozoa together with Metazoa represent all that we can usually call as parasites (micro and macro).

There is no real consensus on the taxonomy of the helminths, differing textbooks showing different groupings, particularly with the nematodes. The term 'Helminth' itself contains a number of phyla, many of which are completely unrelated (for example the Annelids are much more closely related to the Arthropods than the Platyhelminthes, and the Nematodes appear to be

even more distantly related). However, for convenience the term is still used to describe four groups with superficial similarities, the phyla Annelida, Platyhelminthes, Nematoda and Acanthocephala.

Sea turtles seem to exhibit an evolutionary association with their parasites largely independent from that of other marine hosts. Therefore, the composition of helminth communities of marine turtles may be predictably restricted to plathelminthes, nematodes and annelids specific to these reptiles. It is believe that gastrointestinal helminths in sea turtles exchange at 2 host taxonomic scales: exchange between marine turtles and other marine host and exchange within turtles (Aznar *et al.* 1998). From this, the complete life cycle of these parasites have been established. They mainly affect loggerhead sea turtle (*Caretta caretta*) since they feed on the mollusc which is the second indeterminate host of gastrointestinal helminth parasites (e.g. anisakid nematode *Sulcascaaris sulcata* infects bivalves and snails as second host; see Figure 12 in pag. 26). *E. imbricata* consumes, primarily, poriferans that are toxic to most vertebrates; while *C. mydas* is primarily a grazer on sea grass and algae. Finally and as mentioned before, *C. caretta* which feeds on crustaceans and molluscs (both snails and bivalves) and this may not be coincidental that is the species with most non-turtle helminth records (Aznar *et al.* 1998).

9.1 Phylum Platyhelminthes (flatworms)

Platyhelminthes (or commonly called flatworms) were derived from an ancestor that probably had many cnidarian-like characteristics, but evolving in a bilaterally symmetrical animal in which the body can be divided along only one plane of symmetry to yield two halves that are mirror images of each other. Flatworms range in size from a millimetre or less to some of the tapeworms that are many meters in length. Their flattened bodies may be slender, broadly leaf-like, or long and ribbon-like.

Flatworms include both free-living and parasitic forms, but free-living members are found exclusively in the class Turbellaria. A few turbellarians are symbiotic or parasitic, but the majority are adapted as bottom dwellers in marine or fresh water, or live in moist places on land (Hickman *et al.* 2001).

Intravascular trematodes (blood flukes) are pathogens of mammals, birds, reptiles and fish. The flukes or trematodes of traditional veterinary and medical significance are almost all digenetic flukes (i.e., Subclass: Digenea) that require a mollusc or snail as the first intermediate host. Some of these flukes are severe pathogens and inflict great damage to the host and cause severe economic loss to the producer in those areas of the world where they are found. Those affecting homeotherms or endotherms include the families Schistosomatidae and Ornithobilharziidae while

those affecting poikilotherms⁹ are found in the families Sanguinicolidae and Spirorchidae (Wolke *et al.* 1982). There are 16 genera within the family Spirorchidae, all of which infect turtles. Of those genera, eight are found in sea turtles of the family Cheloniidae.

Of all parasitic organisms in sea turtles, spirorchid blood flukes are probably the most well studied and are where the largest amount of investigative effort has been focussed. Amazingly, the life cycle of these marine spirorchids remains unknown but cercariae probably develop in molluscs, particularly gastropods. Smith (1972) reported that very little was known of the pathological effects of spirorchid blood fluke infections in marine turtles, most of the reports available at that time being concerned only with pathology in fresh-water turtle species. However, since then several relevant studies of pathology caused by spirorchids in both wild and cultured marine turtles have been conducted, mostly in the green turtle *Chelonia mydas* (see Smith, 1997).

Species representing three genera, *Caretta*, *Haplotrema* and *Neosporichis* have been reported in the heart or blood vessels of the loggerhead turtle *Caretta caretta* by Wolke *et al.* (1982). In this study, spirorchid eggs were recovered from scrapings of fixed intestinal mucosa, walls of the colon, and liver. The eggs were of three types and fit the description of eggs from spirorchids (see Figure 10) known to infect marine turtles, including the loggerhead. Blood fluke eggs pass from their origin intravascularly through the endothelium and vessel wall into the surrounding tissue. During this passage, vessel wall integrity is violated and localized haemorrhage is common. The eggs then begin a migration that will result in their reaching the lumen of the intestine. This in turn results in a severe and acute inflammatory response (Wolke *et al.* 1982).

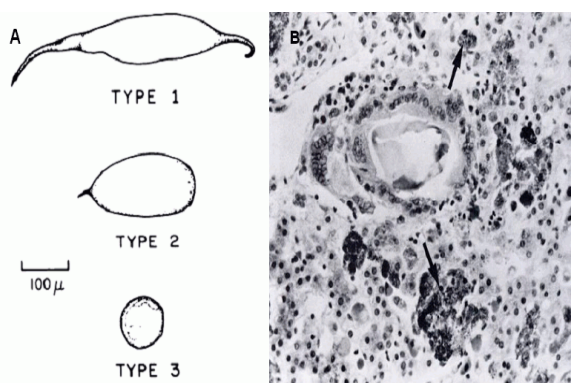


Figure 10. (A) Drawing of fecal spirorchid eggs, Type 1: *Learedius*, *Monticellius*; Type 2: *Carettacola*, *Haemoxenicon*; Type 3: *Neosporichis*. (B) Spirorchid egg in liver (pictures taken from Wolke *et al.* 1982. P. 177 and 179).

No adult spirorchids were recovered from the mesenteric vessels; however, an adult blood fluke was found from a section of artery of the muscularis of the colon, in close proximity to masses of eggs. The adult worm is less pathogenic, but has been reported responsible for the formation of thrombi¹⁰ and infarction. Wolke and colleagues concluded in their study that effects of blood flukes on the population dynamics of wild turtles was not investigated, nor has the incidence or prevalence of this disease been studied in free-living sea turtles.

Most of the description and records on platyhelminthes have been mainly focused on morphological descriptions. Blair (1987) published a full article describing the morphology of three parasitic fluke species in sea turtles; *Octangium sagitta*, *Octagium hyphalum* and *Neoctangium travassosi*. The parasites were mainly obtained from green turtles (*C. mydas*) in Queensland and yielded species in the microscaphid families. Blair discussed mainly the subfamily Octangiinae in the paper. Moreover, Glazebrook *et al.* (1989) from Australia published a vast article in which two species of cardiovascular flukes, *Haplotrema* sp. and *Learedius* sp. were fully described to infect green (*C. mydas*) and hawksbill (*E. imbricata*) turtles. Flukes were recovered from the three chambers of the heart and major vessels (right aortic arch and brachiocephalic artery), where they were attached to the walls or free in the lumen. In the same paper, the authors made a review of spirorchiid in sea turtles. Nine genera and 17 species of spirorchiid parasite have been recorded from hearts and blood vessels of the green turtles (see table in Part 3 of this report).

Of 11 wild green turtles from Bermudan waters examined by Rand & Wiles (1985), three to 53 adult specimens of *Learedius learedi* were recovered from six harboured turtles and two turtles harboured from one to 23 adult specimens of *Neosporichis schistosomatoides*. Adults of both spirorchid species were recovered from only one turtle.

Investigations on bacteria and other disease agents in turtles were conducted by Aguirre *et al.* (1994); these authors found eight green turtles containing granulomata associated with spirorchid eggs identified as *Haplotrema* sp.

A moribund green turtle from off Hawaii examined by Graczyk *et al.* (1995) harboured various spirorchids, 78 specimens of *Haplotrema dorsopora* and 34 specimens of *Learedius learedi* in the heart chambers and the major blood vessels, and 17 specimens of *Carettacola hawaiiensis* in the hepatic vessels. Gross lesions included variously sized, lobulated tumours, serous atrophy of fat, and oedema in the subcutaneous tissues and in the pectoral and coracoid muscles.

⁹ Poikilotherms = an organism, such as a fish or reptiles, having a body temperature that varies with the temperature of its surroundings; an ectotherm.

¹⁰ Thrombi= A fibrinous thick, viscous, or coagulated mass formed in a blood vessel or in a chamber of the heart.

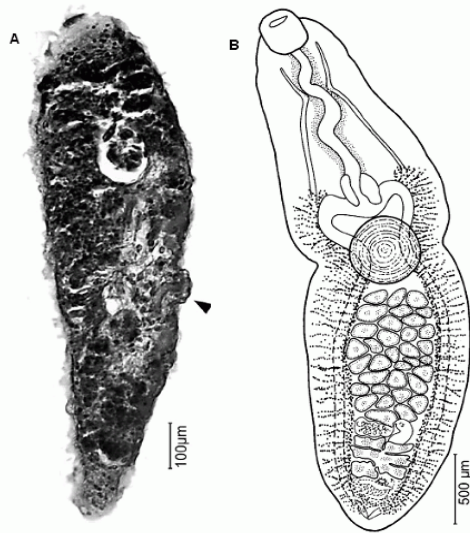


Figure 11. (A) *Learedius learedi*, transversal histological section at Laurer's canal pore level, showing the distinctive papilla on dorsal surface (arrow). (B) Drawing of *Learedius learedi*, adult, ventral view (pictures taken from Inohuye-Rivera *et al.* 2004. P. 38-39).

Learedius learedi (see Figure 11) was redescribed in Baja California South, Mexico in 2004 by Inohuye-Rivera *et al.* by using specimens obtained from the hearts of 3 *Chelonia mydas agassizii*¹¹, the black turtle or Eastern Pacific green turtle. In all, 128 parasites were found, a mean intensity of 43 worms per heart. This re-description was based on the original for this parasite but provided new range of variation in testes shape and position, and adds new information about the reproductive systems. However, the report is again deficient in terms of the life cycle description of the *L. learedi*.

Most of the descriptions of spirorchiid infections have been done by identifying and quantifying the eggs of those parasites in the different tissues in sea turtles. In very few occasions adults have been recovered from the host.

Recently, Work *et al.* (2005) described the epizootiology of spirorchiid trematode infections in Hawaiian green turtles by quantifying tissue egg burdens in turtles. Based on tissue egg burdens and antibody status, the authors of this study hypothesized that immature turtles become infected with spirorchiids shortly after recruiting into coastal foraging pastures from the pelagic environment; that infection level decrease with age; and that spirorchiids detrimentally affect the body condition of sea turtles independent of the tumour burden. Three species of spirorchiids were described in this paper; the endemic trematode *Carettacola hawaiiensis*, *Laeredius* sp. and *Hapalotrema* sp.

¹¹ As mentioned previously, some biologist considered that there is a number eight species of sea turtles, the *Chelonia mydas agassizii* or the black turtle from Mexico. Since there is not yet a consensus about its official recognition, in this paper we keep the seven species terminology.

Finally, the unpublished article from a research study at Tortuguero National Park, Costa Rica made by Santoro *et al.* (2006) recorded the presence of 29 different species of trematodes from 40 green sea turtles (*C. mydas*) that were investigated. Organs, including the heart, great vessels, lungs, oesophagus, stomach, upper and lower intestine, spleen, liver, gall bladder, kidneys and urinary bladder, were carefully examined for parasites. Only 3 species, i.e., *Learedius learedi*, *Microscaphidium reticulare*, and *Pyelosomum cochlear*, infected more than 50% of the hosts sampled. *Learedius learedi* was the most prevalent (97.5%) and the second most abundant species. However, more parasites such as *Microscaphidium reticulare*, *Deuterobaris intestinalis*, *Schizamphistomoides erratum*, *Pyelosomum cochlear*, *Cricocephalus resectus* and *Desmogonius desmogonius* were also recorded with prevalence higher than 45%. It is very important to mention that species richness was the highest recorded from a sea turtle species. All digenetic species were recorded from Costa Rica for the first time, and this study by Santoro *et al.* (2006) represents the first report on the helminth community of the green turtle.

9.2 Phylum Nematoda (roundworms)

Nematoda phylum bellows into the group of animals called Pseudocoelomates. A pseudocoel is a space between the gut and the mesodermal and ectodermal components of the body wall, and it is not lined with peritoneum. This inter-space gives to those organisms greater freedom of movement and space for development of digestive, excretory and reproductive systems and many other features that make nematodes more efficient and competitive. Approximately 12,000 species of Nematoda have been named, but it has been estimated that if all species were known, the number would be nearer 500,000. Nematodes also parasitize virtually every type of animal and many plants and it is believed that every species of vertebrate and many invertebrate serve as hosts for one or more types of parasitic nematodes (Hickman *et al.* 2001).

Remarkably there is only one single species of nematode that has been properly studied and described for sea turtles. A few other species have been found and identified as endoparasites in sea turtles, but without any extant description of life cycle, importance or infection frequencies.

Larval anisakid nematodes identified as *Sulcascaaris sulcata*, which matures in sea turtles (*Caretta caretta* and *Chelonia mydas*), have been reported previously from various marine mollusc of the Atlantic continental shelf of North America, and the Western and Queensland coasts of Australia (Lichtenfels *et al.* 1978; Lichtenfels *et al.* 1980; Lester *et al.* 1980; and Sawyer *et al.* 1980). There is also a long list of second intermediate hosts for *S. sulcata*. This parasite infects many different species of scallops and commercial

shellfish species causing a large economic loss every year.

As an adult, the only known species of this nematode genus (*Sulcascaris sulcata*) parasitizes the stomach and intestine of green and loggerhead sea turtles. Parasitized turtles occur in areas where the larvae parasitize the molluscs (Lichtenfels *et al.*, 1978).

In a report made by Lester *et al.* in 1980 at Shark Bay in Australia, *S. sulcata* was shown to infect saucer scallops, *Amusium balloti*. Five loggerhead turtles, *C. caretta*, from the same area were also found to contain adult *S. sulcata*; four of the turtles had been feeding chiefly on bivalves, one on crabs, and one on gastropods. Ulcers, probably associated with *S. sulcata* infection, occurred in the stomach. The same report also mentioned the presence in the turtles of the cucullianid *Cucullanus cauettiae*, the oxyuroid *Kathlania leptura* and the larval gnathostome *Echinocephalus* sp. all which are also nematodes. No further research has been done besides the mention of those three species of nematodes in sea turtles.

Because of the occurrence of *Sulcascaris sulcata* nematode in commercial shellfish that may be consumed uncooked (calico scallop and Atlantic bay scallop), attempts were undertaken in the middle 70's to determinate the possible infectivity of the nematode for mammals. This effort was complicated by the presence of a protozoan hyperparasite in most of the nematodes from surf clams collected from winter to spring 1974-1975. The incidence of this hyperparasite was low in a one-year survey initiated in October 1976 by Payne and colleagues in the U.S. (published in 1977). The hyperparasite protozoon, *Urosporidium spisuli*, debilitates the nematodes and, therefore, rendered inconclusive the negative results of feeding and culturing tests (Lichtenfels *et al.* 1978). Researchers could not manage to prove either the infectivity of *S. sulcata* larvae for mammals. Other investigators tried unsuccessfully to infect chickens and cats. Although the worm causes losses to the scallop fishery due to the need to remove the unsightly worm from scallops, it did not develop in chickens or cats suggesting that there is little likelihood of danger to man (Berry and Cannon, 1981).

Sprent (1977), Cannon (1978) and Lichtenfels *et al.* (1978) assigned the "molluscan" larvae and those from the stomach of turtles to the fourth stage by reverse designation from the adult since earlier stages were not available for study. Although most nematodes are infective for the definitive host as third stage larvae, in some anisakines (marine ascaridoids), both third and fourth stages may be found in the tissues of the intermediate host (Berry and Cannon, 1981).

It was not until 1981 that Berry and Cannon finally described the entire life cycle of *Sulcascaris sulcata*. Experimentally, laboratory-reared loggerhead turtles (*C. caretta*) were fed fourth stage larvae which first attached at the oesophagus-gastric junction where they moulted

to adults in 7-21 days. Subsequent growth to mature adults was obtained by at least 5 months after infection. The authors suggested that under natural conditions the life history may take up to 2 years to complete. Clearly turtles become infected by ingesting scallops (and presumably other molluscs) and at the same time defaecate providing eggs from which larvae hatch and infect more scallops.

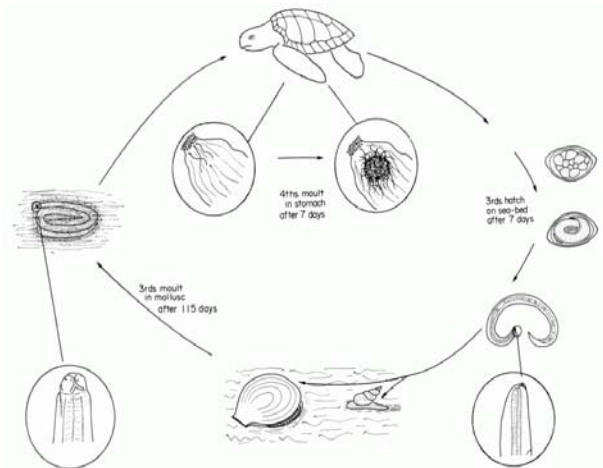


Figure 12. Life cycle of *Sulcascaris sulcata* in the loggerhead turtle (*Caretta caretta*). Time intervals were found experimentally at 25°C (picture taken from Berry and Cannon, 1981. P. 53).

The above figure illustrates the entire life cycle of *S. sulcata*; eggs pass out in the feces and fall to the sea floor. The larva develops in the egg and undergoes 2 moults to the L3 stage. The L3 hatch from the eggs beginning at 7 days after the egg was laid. L3 are taken up by molluscs (scallops and possibly others) and go in to the tissues. The L3 will moult to the L4 in the scallop in 3 to 4 months. When the infected mollusc is eaten by the loggerhead turtle the L4 attach to the stomach wall (at the esophago-gastric junction) and will moult to the adult in 7 days. Adults will become gravid in 5 to 6 months (Berry and Cannon, 1981).

9.3 Phylum Annelida (segmented worms)

Annelida are worms whose bodies are divided into segments, arranged in linear series and externally marked by circular grooves called "annuli". Phylum Annelida consists of approximately 15,000 species. Annelids have a worldwide distribution, and few species are cosmopolitan. Oligochaetes and leeches (Class Hirudinea) occur predominantly in fresh water or terrestrial soils. Although there are few leech species, some are predators, and many are specialized for piercing their prey and feeding on blood or soft tissue. Leech species parasitic to marine turtles are adapted for forcing their pharynx or proboscis into the soft tissue. Some are temporary parasites and some are permanent parasites, never leaving their host (Hickman *et al.* 2001).

There are only four species of annelids recorded to occur in sea turtles, two of them polychaetes and two leeches or hirudineans. Annelids have been reported as associated or mentioned as simple epibionts living on sea turtles and not as real parasites. No research has been done into the infections that those parasites could cause to their host. However, Frick *et al.* (2003) documented the presence of *Hydroides* sp. and *Loimia* sp. (both members of the Class Polychaeta) in a study of the epibiont community in the hawksbill turtle (*E. imbricata*) off the coast of Brazil. Besides mentioning these two species, no more information was available in the article.

The *Ozobranchus* sp. leech was later proposed as a candidate for vector for the fibropapilloma-associated green turtle (*C. mydas*) herpesvirus by Greenblatt *et al.* (2004). This theory was established since marine leeches (*Ozobranchus* sp.) were found to carry high viral DNA loads; some samples approached 10 million copies per leech. Unlike most leeches, which must leave the host to reproduce, *Ozobranchus branchiatus* and *O. margoi* can complete their entire reproductive cycle on the turtle hosts (Choy *et al.* 1989). Thus, they are not necessarily limited to coastal areas, as stated by Greenblatt *et al.* (2004). Greenblatt and colleagues somehow managed to publish an entire paper on these leeches without ever explaining exactly which species they were studying, nor they did state the species (*Ozobranchus* sp.) in reference (Williams & Bunkley-Williams, 2006). Both *Ozobranchus branchiatus* and *O. margoi* occur on Hawaiian green sea turtles (Choy *et al.* 1989).



Figure 13. *Ozobranchus branchiatus* annelid, ectoparasite found in the skin of Olive Ridley turtles (*Lepidochelys olivacea*) in Mexico (picture taken from Gámez *et al.* 2006. P. 434).

A recent study of the identification of parasites and epibionts in Mexico by Gámez *et al.* (2006) recorded the presence of *Ozobranchus branchiatus* infecting 28% of the Olive Ridley turtle (*Lepidochelys olivacea*) analyzed in this report (see Figure 13).

9.4 Phylum Arthropoda (arthropods)

Arthropoda is the most extensive phylum in the animal kingdom, composed of more than three-quarters of all known species. Approximately 900,000 species of arthropods have been recorded, and probably at least as many more remain to be classified. Arthropods possess an exoskeleton containing chitin that is highly protective without sacrificing mobility. Arthropods include, but are not limited to spiders, scorpions, ticks, mites, crustaceans, millipedes, centipedes and insects. They utilize all modes of feeding (carnivorous, herbivorous and omnivorous) although most are herbivorous. However, some are parasitic to almost any other kind of organism including plants, vertebrates and even other insects. Larvae of many varieties of insects live inside the bodies of other arthropods and small vertebrates or their youngest, consuming their hosts and eventually killing them. Because they always destroy their hosts, they are known as **parasitoids** (considered a particular type of parasite); typical parasites normally do not kill their hosts (Hickman *et al.* 2001).

The egg is one of the most vulnerable stages in the life cycle of turtles. There have been several studies conducted on the effect of insect larvae infestation of marine turtle eggs. Thus, the larvae of two families of diptera (Phoridae and Sarcophagidae) are most commonly found to infest clutches of both freshwater (Iverson and Perry, 1994), and marine turtles (Lopes, 1982; Acuña-Mesen and Hanson, 1990; Andrade *et al.* 1992; Vázquez, 1994; Broderick and Hancock, 1997; McGowan *et al.* 1997; and McGowan *et al.* 2001). It has been suggested that these larvae feed on weakened or already dead hatchlings and therefore pose no real threat to the reproductive success of turtles. However, larvae have been reported to attack viable hatchlings and reduce hatching success by at least 30% (Lopes, 1982). In the same report, Lopes described the morphology of the diptera, *Eumacronychia sternalis* with larvae infesting eggs and hatchlings of the green turtle.

Two species of phorid flies, *Megaselia scalaris* and *Puliciphora borinque* have been found infesting the eggs of the Atlantic green turtle (*C. mydas*) and the hawksbill (*E. imbricata*) at Tortuguero, Costa Rica perhaps for first time by Bjorndal *et al.* (1985) and Fowler (1979), respectively. In the study done by Andrade *et al.* (1992) in Mexico, the researchers found fly larvae infestations in sea turtle nests caused by two genera of the Sarcophagidae Family, *Phrosinella* sp. and *Eusenotainia* sp. They recorded also an infestation in some of the nest caused by acaroids, *Rizoglyphus* sp. which belongs to the Acaridae. They concluded that fly larvae did not seriously affect the survival of leatherback (*Dermodochelys coriacea*) or olive ridley (*Lepidochelys olivacea*) sea turtles at Michoacán, Mexico. The life cycle of *Eusenotainia rufiventris* was studied by Vázquez (1994), again in Mexico and she reported the

effect of this diptera larvae species in the eggs and hatchlings of the leatherback turtle (*D. coriacea*).

- And finally, the hatchling emergence duration was also found to be significant, with the number of infested eggs increasing as hatchlings emergence duration increased.

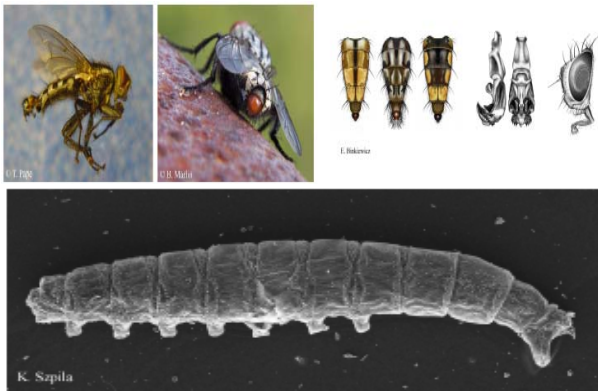


Figure 14. Adult morphology with abdominal sternites without sensilla trichodea (alpha setae) and head and mouth parts of Sarcophagidae diptera fly. Larval and puparial morphology of Sarcophagidae diptera fly (picture obtained from <http://www.sel.barc.usda.gov/Diptera>, March 2006).

In the Mediterranean, coleopteran larvae have been reported infesting eggs of the loggerhead turtle (*Caretta caretta*) in Turkey (Baran and Türkozan, 1996). The two genera of coleopteran were *Cardiophorine* sp. and *Agriotine* sp. Moreover, McGowan *et al.* (1997) reported all data regarding turtle nesting and hatching insect larvae infestations for loggerhead (*C. caretta*) and green (*C. mydas*) turtles in the Northern Cyprus. Five species of Sarcophagidae diptera (see Figure 14) larvae were reported with *Sarcotachina aegyptiaca* being the most commonly occurring in the infested turtle nests. Furthermore, another four species of diptera from different families were also recorded in addition to the two species of coleopteran mentioned before.

A common feature of all the studies that have done in this field is that most have failed to ascertain why only certain nests become infested while others remain free of larvae.

A recent study made by McGowan *et al.* (2001) on Northern Cyprus found a number of possible relationships between different factors that could explain what makes a nest more sensitive to infestation. They concluded that there are three main factors:

- The number of infested eggs in a nest decreases with increasing nest depth, and in shallow nest the number of infested eggs increases with increasing number of dead hatchlings.
- The distance from the high water mark to the nest was also found to have a significant effect on the number of infested eggs in a nest, with infestation decreasing further above the high water mark.

From this first interaction between nest depth and total number of dead, it can be conclude that there may possibly be differences in the ability of dipteran species to detect decaying tissue matter within the sand column. Furthermore, this interaction may suggest that chemical odours from decaying tissue may lose potency as they permeate up through the sand column. Nevertheless, this does not rule out variation in larval burrowing ability as a possible explanation as to why nest depth plays such an important role in the turtle clutch infestation (McGowan *et al.* 2001) but could possibly justify why leatherback nest are less affected by dipteran larvae. In average, leatherback nests are more than 20cm deeper than other marine turtle species.

The distance to the high water mark was also found to have a significant effect on the number of eggs in clutch that were infested, with nest further from the high water mark less prone to infestation (McGowan *et al.* 2001). However, some researches before could not obtain the same satisfactory conclusion. All those studies were carried out in different latitudes than McGowan's study in Northern Cyprus, which suggest the possibly association of the behaviour of different fly species and the area in which they are occur. Another conclusion could be the moisture concentration.

As hatchling emergence duration increased, the number of infested eggs in a clutch also increased (McGowan *et al.* 2001). This is a quite logic conclusion and coincides with another very famous study made in Mexico by Vásquez (1994) who reported that the longer the delay between hatchling emergence and excavation of a leatherback turtle nest the higher the likelihood of clutches being infested.

10. Epibionts (e.g. barnacles)

The term "epibiont" is not a real taxonomic group and actually contains many different organisms that come from many different phyla and even kingdoms. An epibiont could be defined as an organism that lives attached to another organism (i.e. barnacles) but without benefit or detriment to the host. According to the last definition, epibionts can not be considered as a pathogen nor as a parasite in sea turtles and therefore will not be included in this synopsis. Thus, epibionts species are not listed in the summary tables of this report. Nevertheless, one question being studied is the effects of epibionts on turtle health. Most epibionts are harmless as they reside on the turtle's shell and live off algae. Though these organisms do not feed parasitically off the turtle's resources, however, their effects may not be

completely neutral, especially when they reside in the skin of the turtle.

Barnacles attach to sea turtles and although they do not feed on the turtle as parasites and disease organisms do, they can impact the turtle's health by increasing water resistance when it swims, requiring the turtle to expend more energy (Perrine, 2003). They attach to any hard surface, including ships, piers, and the hard shell of a turtle. Using their wispy legs, barnacles grab at planktonic organisms from water flowing by.

Barnacles are atypical among maxillopod crustaceans in that they are hermaphroditic, and they are unusual among sessile animals in being copulatory. Most barnacle species are not self-fertile, because they are either sequential hermaphrodites or they alternate between male and female modalities during breeding (Anderson, 1994). Therefore, barnacles generally cross-fertilize with receptive neighbours and thus need to settle in aggregations, the difficulty of which is compounded by their dispersion in the plankton during larval development. Barnacles that specialize as commensals have the additional complication of needing to locate a suitable host that may be scarce and is often mobile.

That some barnacles are obligate commensals of sea turtles has long been known; however, little attention has focused on understanding the details of this association. In particular, early life-history traits of turtle barnacles, which may be the key to establishing the association, have not been well studied (Zardus *et al.* 2004).

Much of the information available on sea turtle epibionts concerns the organism associated with loggerhead sea turtles (Frick *et al.* 2003). For instance, loggerheads apparently support the largest and most diverse set of epibionts. Loggerheads nesting in the south-eastern United States host 100 species from thirteen phyla, and loggerheads nesting at Xcatel, Mexico, carry thirty-seven species of algae with up to twelve species on an individual turtle. The ramifications of these epibiont loads for the turtles are not clear. Sea turtles may benefit from being camouflaged by their epibionts, but the drag resulting from epibionts increases the energetic cost of swimming (Perrine, 2003). Thus, it is typically believed that loggerheads support larger and more diverse epibiotic communities than any other sea turtle species (Frick *et al.* 2000), although this could be explained because the largest effort on identification of epibionts has been conducted for this species. For instance, before 2003, there were no published studies that monitored the epibiont community in hawksbill turtles (*Eretmochelys imbricata*) in the Caribbean. The only record is from Carr *et al.* in Costa Rica in 1966; describing large white barnacles (apparently *Chelonibia* sp.) on the carapace and plastron of a hawksbill turtle.

There are no published studies that have monitored the epibiont community composition of population of hawksbill turtles continuously throughout the course of a nesting season. Today, there are only official studies concerning barnacle

communities for *Chelonia mydas*, *Caretta caretta* and recently for *Eretmochelys imbricata* species; for all other species (leatherback, kemp's ridley, olive ridley and flatback) studies remain undone.



Figure 15. These invertebrate epibionts were collected primarily from Olive Ridley turtles (*Lepidochelys olivacea*) coming ashore to nest at Teopa Beach near Chamela, Mexico. One of the most common barnacle species in sea turtles is the *Chelonibia testudinaria*. (picture obtained from <http://www.yale.edu/inverts>, March 2006).

Nevertheless, in a study made by Bugoni and colleagues from Brazil in 2001 it was shown that the number of barnacle specimens per host turtle (green and loggerhead) ranged from 1 to 70. Individual turtles hosted up to three different barnacle species. Approximately the same amount of number and species of barnacles has also been reported from the Mediterranean Sea and the turtle populations along the Florida coast.

A quick foray through the literature in loggerhead barnacles reveals that loggerhead turtles host more than 125 epibiotic species worldwide (Bugoni *et al.* 2001 and Frick *et al.* 1998; Frick *et al.* 2003 and 2004). Just by speculating from these numbers it could be concluded that the same amount of barnacles may be found in all other sea turtle species. Presumably fewer barnacles would be expected in the *D. coriacea* (leatherback) due to its physiological differences (carapace morphology and design), behaviour, deep swimming and long migrating pattern.

More than 80 percent of juvenile loggerheads in the North Atlantic carry "hitchhikers" in the form of small Columbus crabs. Most of these crabs travel in male/female pairs and live at the base of the turtle's tail. In return for a secure mobile home, the crabs provide a valuable service; they eat small barnacles and parasites from the turtle's shell and skin. If left to grow, gooseneck barnacles can cover a turtle's shell like a mop and greatly increase the energy required for swimming. The crabs also cannibalize additional members of their

own species that try to colonize the turtle (Perrine, 2003).

Discussion and Conclusion

In terms of biology, evolution and conservation turtles as a group of marine vertebrates are quite well studied animals. However, regarding to pathology and infectious diseases there is still a long way to go; more research and studies must be done due to fully understand the interactions between turtles and other organisms in the marine environment.

Contemporary information on pathogens and parasites from sea turtles is incomplete because no data exist for *Natator depressa*, and information from *Dermochelys coriacea*, *Lepidochelys kempii* and *L. olivacea* are still limited. However, published data include a century of worldwide reports focussing in three turtle species that have high mariculture economical importance *Chelonia mydas*, *Caretta caretta* and *Eretmochelys imbricata*. Some previous reports have mentioned the lack of information for *L. kempii* and *D. coriacea* species', however this study was able to find and discuss a large number of publications related to infections for these two turtle species.

There have been remarkably few attempts to pull together the available information regarding infections in sea turtles to date. Glazebrook and Campbell (1990) published one of the only surveys collecting information about diseases of farmed turtles in Queensland, Australia. Perhaps the most outstanding book regarding medicine in reptiles is that edited by Mader (2006), which comprises most of the known diseases for marine turtles. There are also several other reports that deal with specific groups of parasites, such as the article made by Aznar *et al.* (1998) about gastrointestinal helminths in loggerheads; and two publications made by Smith (1972 and 1997) about blood flukes of cold-blooded vertebrates. Besides the latest publications, most of the information about pathogens and parasites in sea turtles has mainly been recorded as anecdotal findings.

The relative lack of experimental studies on reptilian infections makes determination of which pathogens causes which problem extremely difficult. This point was raised by Mader (2006) and it is one of the salient conclusions from this report. As mentioned previously, experimental studies of sea turtles are even more problematic since all the species are threatened and, therefore, research and experimentation are extremely restricted and limited by international laws.

It seems that most of the infections caused by viruses and bacteria are due to opportunistic circumstances where the pathogenic organism attacks when the host immune system is already compromised; while for protozoa and macro-parasites (i.e. helminths) a high level of specialization seems to rule the mode of infections.

Sea turtles as for most higher vertebrates seem to show a similar pattern of infections levels decreasing with age. Antibody levels and resistance appear to increase considerably with age. Nevertheless, this pattern does not apply to fibropapillomatosis where as mentioned before, the disease increases in populations within larger size class excluding almost entirely the youngest juveniles (Gardner and Oberdörster, 2006).

Some reports fail to mention the species of sea turtle affected by a particular infection, pathogen or parasite. In some cases even the nomenclature used to classify reptiles by earlier authors who are cited in more recent review articles is outdated; a problem also cited by Mader (2006).

Paramphistomum papillostomum, *Schizamphistomoides chelonei*, *Schizamphistomum scleroporium* and *S. taiwanense* are four different platyhelminthes species, or at least seem to be. Nevertheless, Blair (1983) listed them all as synonymous which have been described by different authors in different places and different times, all showing the same species. Unfortunately, no further information was found to clarify this subject. However, this over replication of a parasite singular name could not be unique. For instance, Aznar *et al.* (1998) mentioned in their article the existence of 88 different species of platyhelminthes worldwide that were found in sea turtles. However, this report consulted the majority of the same sources as Aznar *et al.* and identified fewer species of platyhelminthes. As well as the platyhelminthes, many other pathogen and parasite species have been renamed and it is, therefore, possible that some authors may mistakenly have included the same species twice under two different names.

Every single sea turtle species represents a completely different environment from the taxonomic scale of pathogens and parasites. Therefore, it has been concluded that the main idea of trying to list all infections in sea turtles was probably too ambitious. Regardless it is hoped that this report may be a source of information for persons interested in the subject.

Part Three. Taxonomic summary per host

As mentioned in the introduction of this report it is important to emphasis that most of the references or sources do NOT indicate which author(s) initially described the infection, disease, pathogen or parasite; whereas the references used in this report show where the latest available information was obtained.

Number of pathogens and parasites species per host

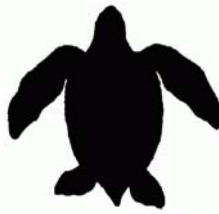
***Caretta caretta*. Loggerhead sea turtle**



Caretta

Virus	1
Bacteria	14
Fungi	4
Protozoa	2
Plathelminthes	28
Nematodes	5
Annelids	1
Arthropods	11

***Dermochelys coriacea*. Leatherback sea turtle**



Dermochelys

Virus	1
Bacteria	11
Fungi	1
Protozoa	1
Plathelminthes	4
Nematodes	-
Annelids	-
Arthropods	4

***Chelonia mydas*. Green sea turtle**



Chelonia mydas

Virus	2
Bacteria	48
Fungi	6
Protozoa	4
Plathelminthes	53
Nematodes	1
Annelids	2
Arthropods	7

***Lepidochelys kempii*. Kemp's Ridley sea turtle**



Lepidochelys kempii

Virus	1
Bacteria	1
Fungi	6
Protozoa	-
Plathelminthes	-
Nematodes	-
Annelids	1
Arthropods	-

***Eretmochelys imbricata*. Hawksbill sea turtle**



Eretmochelys

Virus	1
Bacteria	3
Fungi	5
Protozoa	-
Plathelminthes	17
Nematodes	-
Annelids	3
Arthropods	2

***Lepidochelys olivacea*. Olive Ridley sea turtle**



Lepidochelys olivacea

Virus	1
Bacteria	1
Fungi	1
Protozoa	-
Plathelminthes	3
Nematodes	-
Annelids	1
Arthropods	3

***Natator depressa*. Flatback sea turtle**



Natator

Virus	1
Bacteria	-
Fungi	1
Protozoa	-
Plathelminthes	-
Nematodes	-
Annelids	-
Arthropods	-

Caretta caretta (Linnaeus, 1758). Loggerhead sea turtle



Caretta

Taxon	Sub-Taxon	Pathogen or parasite	Location in the host	Disease	Reference or source	
Virus	Family Papillomaviridae	Papilloma virus	skin	fibropapillomatosis	Mader, 2006	
Bacteria	Bacillus (variable) Gram-negative	<i>Bacillus sp.</i>	intestine	gastritis	Orós ¹ et al., 2004	
		<i>Achromobacter sp.</i>	oral cavity	oral cavity ulcerative	Orós ¹ et al., 2004	
		<i>Aeromonas hydrophila</i>	oral cavity and oesophagus	oral cavity ulcerative and ulcerative oesophagitis	Orós ¹ et al., 2004	
		<i>Burkholderia cepacia</i>	oral cavity	oral cavity ulcerative	Orós ¹ et al., 2004	
		<i>Citrobacter sp.</i>	oesophagus and liver	ulcerative oesophagitis and hepatitis	Orós ¹ et al., 2004	
		<i>Escherichia coli</i>	oesophagus, intestine and liver	ulcerative oesophagitis, gastritis and hepatitis	Orós ¹ et al., 2004	
		<i>Proteus sp.</i>	stomach, intestine and liver	ulcerative stomatitis, gastritis and hepatitis	Orós ¹ et al., 2004	
		<i>Pseudomonas sp.</i>	oesophagus	ulcerative oesophagitis	Orós ¹ et al., 2004	
	Gram-positive	<i>Serratia marcescens</i>	liver	hepatitis	Orós ¹ et al., 2004	
		<i>Vibrio alginolyticus</i>	oral cavity, oesophagus, stomach, intestine and liver	ulcerative in oral cavity, oesophagus and stomach; gastritis and hepatitis	Orós ¹ et al., 2004	
		<i>Aerococcus viridans</i>	oesophagus	ulcerative oesophagitis and oesophageal diverticulum	Orós ¹ et al., 2004 and Torrent et al., 2002	
		<i>Pasteurella sp.</i>	intestine	gastritis	Orós ¹ et al., 2004	
		<i>Staphylococcus sp.</i>	oesophagus, stomach, intestine and liver	ulcerative oesophagitis and stomatitis; gastritis and hepatitis	Orós ¹ et al., 2004	
		<i>Streptococcus sp.</i>	intestine and liver	gastritis and hepatitis	Orós ¹ et al., 2004	
Fungi	Class Ascomycetes	<i>Aspergillus sp.</i>	skin lesions	-	George, 1997	
		<i>Candida albicans</i>	oral cavity and cloaca	systematic mycotic infection	Orós et al., 2004	
		<i>Fusarium solani</i>	Skin, eyes, shell, gastro-intestine	dermatomycosis	Rebell, 1981	
	Class Euscomycetes	<i>Paecilomyces lilacinus</i>	lungs	-	George, 1997	
Protozoa	Class Sporozoea/ Subclass Coccidia or bacteria ?	<i>Eimeria caretta</i>	-	-	Mader, 2006	
		<i>Grahamella thalassochelys</i>	-	rickettsias ??	Barnard & Upton, 1994	
Platyhelminthes	? Class Cestoda	<i>Diastoma constrictum</i>	heart	-	Glazebrook et al., 1989	
		<i>Ancistrocephalus imbricatus</i>	stomach and intestine	-	Aznar et al., 1998	
		<i>Tentacularia coryphaenae</i>	stomach and intestine	-	Aznar et al., 1998	
	Class Trematoda/ Subclass Aspidogastrea	<i>Trypanorhynchon sp.</i>	stomach and intestine	-	Aznar et al., 1998	
		<i>Lophotaspis vallei</i>	stomach and intestine	-	Mader, 2006	
		Class Trematoda/ Subclass Digenea	<i>Calycodes anthos</i> *	stomach and intestine	-	Manfredi et al., 1998
			<i>Calycodes anthos</i> *	stomach and intestine	-	Aznar et al., 1998
			<i>Carettacola bipora</i>	-	-	Smith, 1997
			<i>Carettacola sp.</i>	heart or blood vessels	spirorchidiasis	Wolke et al., 1982
			<i>Cricocephalus albus</i>	-	-	Aznar et al., 1998
			<i>Cymatocarpus solearis</i>	intestine	-	Blair and Limpus, 1982
			<i>Enodiotrema megachondrus</i>	stomach and intestine	-	Manfredi et al., 1998
			<i>Haemoxenicon sp.</i>	heart or blood vessels	spirorchidiasis	Wolke et al., 1982
			<i>Hapalotrema loossi</i>	-	-	Smith, 1997
			<i>Hapalotrema mistroides</i>	-	-	Smith, 1997
			<i>Hapalotrema synorchis</i>	heart	-	Glazebrook et al., 1989
			<i>Hemiuroidea sp.</i>	stomach and intestine	-	Aznar et al., 1998
			<i>Learedius sp.</i>	heart or blood vessels	spirorchidiasis	Wolke et al., 1982
			<i>Monticellius sp.</i>	heart or blood vessels	spirorchidiasis	Wolke et al., 1982
			<i>Neocaballerotrema caballeroi</i>	-	-	Smith, 1997
	<i>Neospororchis pricei</i>		heart	-	Glazebrook et al., 1989	
	<i>Neospororchis sp.</i>	heart or blood vessels	spirorchidiasis	Wolke et al., 1982		
	<i>Orchidasma amphiorchis</i>	stomach and intestine	-	Manfredi et al., 1998		
	<i>Pachypsolus irroratus</i>	stomach and intestine	-	Manfredi et al., 1998		
	<i>Plesiochorus cymbiformis</i>	stomach and intestine	-	Manfredi et al., 1998		
	<i>Pleurogonius trigonocephalus</i>	stomach and intestine	-	Manfredi et al., 1998		
	<i>Rhytidodes gelatinosus</i>	stomach and intestine	-	Manfredi et al., 1998		
<i>Shobanatrema shobanae</i>	-	-	Smith, 1997			
Nemathelminthes Class Nematoda	<i>Cucullanus cauttiae</i>	stomach	-	Lester et al., 1980		
	<i>Echinocephalus sp.</i>	stomach and intestine	-	Lester et al., 1980; Aznar et al., 1998		
	<i>Kathlania leptura</i>	stomach	-	Lester et al., 1980		
	<i>Oxyuroidea sp.</i>	stomach and intestine	-	Aznar et al., 1998		
	<i>Sulcascaaris sulcata</i>	stomach and intestine	-	Lichtenfels et al., 1978		

Annelida	Class Hirudinea	<i>Ozobranchus margoi</i>	skin	-	Mader, 2006
Arthropoda	Class Insecta/Family Chloropidae	<i>Eutropha fulvifrons</i>	eggs and hatchlings	-	McGowan et al., 1997
	Class Insecta/Family Ephydriidae	<i>Hecamede albicans</i>	eggs and hatchlings	-	McGowan et al., 1997
	Class Insecta/Family Muscidae	<i>Atherigona orientalis</i>	eggs and hatchlings	-	McGowan et al., 1997
	Class Insecta/Family Phoridae	<i>Megaselia scalaris</i>	eggs and hatchlings	-	McGowan et al., 1997
	Class Insecta/Family Sarcophagidae	<i>Parasarcophaga argyrostoma</i>	eggs and hatchlings	-	McGowan et al., 1997
		<i>Parasarcophaga tibialis</i>	eggs and hatchlings	-	McGowan et al., 1997
		<i>Phyllotelles pictipennes</i>	eggs and hatchlings	-	McGowan et al., 1997
		<i>Sarcotachina aegyptiaca</i>	eggs and hatchlings	-	McGowan et al., 1997
		<i>Wohlfahrtia nuba</i>	eggs and hatchlings	-	McGowan et al., 1997
	Class Insecta/Order Coleoptera	<i>Agriotine sp.</i>	eggs and hatchlings	-	Baran and Türkozan, 1996
	<i>Cardiophorine sp.</i>	eggs and hatchlings	-	Baran and Türkozan, 1996	

Lepidochelys olivacea (Eschscholtz, 1829). Olive Ridley sea turtle



Lepidochelys olivacea

Taxon	Sub-Taxon	Pathogen or parasite	Location in the host	Disease	Reference or source
Virus	Family Papillomaviridae	Papilloma virus	skin	fibropapillomatosis	Mader, 2006
Bacteria	Gram-negative	<i>Vibrio mimicus</i>	eggs	-	Campos et al., 1996
Fungi	Class Ascomycetes	<i>Fusarium solani</i>	skin lesions	dermatomycosis	Mader, 2006
Platyhelminthes	?	<i>Pronopsis psenopsis</i>	-	-	Aznar et al., 1998
	Class Trematoda/ Subclass Digenea	<i>Adenogaster serialis</i>	intestine	-	Gómez et al., 2006
		<i>Cricocephalus albus</i>	-	-	Aznar et al., 1998
Annelida	Class Hirudinea	<i>Ozobranchus branchiatus</i>	soft skin, fins, eyes and cloaca	-	Lazo-Wasen, 2002
Arthropoda	Class Arachnida/Family Acaridae	<i>Rizoglyphus sp.</i>	eggs and hatchlings	-	Andrade et al., 1992
	Class Insecta/Family Sarcophagidae	<i>Eusenotainia sp.</i>	eggs and hatchlings	-	Andrade et al., 1992
		<i>Phrosinella sp.</i>	eggs and hatchlings	-	Andrade et al., 1992

Lepidochelys kempii (Bauer, 1890). Kemp's Ridley sea turtle



Lepidochelys kempii

Taxon	Sub-Taxon	Pathogen or parasite	Location in the host	Disease	Reference or source
Virus	Family Papillomaviridae	Papilloma virus	skin	fibropapillomatosis	Mader, 2006
Bacteria	Gram-positive	<i>Mycobacterium chelonae</i>	skin, elbow, lungs, liver, spleen, kidney and pericardium	osteoarthritis	Greer et al., 2003
Fungi	?	<i>Colletotrichum acutatum</i>	lungs and kidney	-	Manire, 2002
	Class Actinomycetes	<i>Cephalosporium sp.</i>	lungs, head, neck and shell	pneumonia, ?	Leong et al., 1989
	Class Ascomycetes	<i>Fusarium solani</i>	skin lesions	dermatomycosis	Mader, 2006
		<i>Scolecobasidium constrictum</i>	lungs, head, neck and shell	pneumonia	Leong et al., 1989; Lewbart and Medway, 1993
	Class Euscomycetes	<i>Paecilomyces sp.</i>	lungs, head, neck and shell	pneumonia	Leong et al., 1989; Lewbart and Medway, 1993
	<i>Penicillium sp.</i>	lungs, head, neck and shell	pneumonia	Leong et al., 1989	
Annelida	Class Hirudinea	<i>Ozobranchus margoi</i>	skin	-	Mader, 2006

Chelonia mydas (Linnaeus, 1758). Green sea turtle



*Chelonia
mydas*

Taxon	Sub-Taxon	Pathogen or parasite	Location in the host	Disease	Reference or source	
Virus	Family Herpesviridae	Herpes virus	skin	herpes infection	Mader, 2006	
	Family Papillomaviridae	Papilloma virus	eyes, skin, flippers	fibropapillomatosis	Mader, 2006	
Bacteria	?	<i>Chlamydomphila psittaci</i>	-	-	Aguirre et al., 2006	
	Bacillus (variable)	<i>Bacillus</i> sp.	oesophagus, intestine and cloaca	fibropapillomatosis and gastritis	Aguirre et al., 1994 and Orós ¹ et al., 2004	
	Diphtheroids	<i>Corynebacterium</i> sp.	oesophagus and cloaca	-	Aguirre et al., 1994	
	Gram-negative		<i>Aeromonas hydrophila</i>	oral cavity and oesophagus	oral cavity ulcerative and ulcerative oesophagitis related with coccidiosis pneumonia	Orós ¹ et al., 2004
			<i>Arizona</i> sp.	intestinal mucosa	-	Rebell et al., 1974
			<i>Arizona hinshairi</i>	lungs	-	Glazebrook and Campbell, 1990
			<i>Citrobacter diversus</i>	cloaca	-	Aguirre et al., 1994
			<i>Citrobacter freundii</i>	cloaca	fibropapillomatosis	Aguirre et al., 1994
			<i>Citrobacter</i> sp.	oesophagus and liver	ulcerative oesophagitis and hepatitis	Orós ¹ et al., 2004
			<i>Enterobacter aerogenes</i>	cloaca	fibropapillomatosis	Aguirre et al., 1994
			<i>Enterobacter agglomerans</i>	oesophagus and cloaca	-	Aguirre et al., 1994
			<i>Enterobacter cloacae</i>	oesophagus and cloaca	fibropapillomatosis	Aguirre et al., 1994
			<i>Escherichia coli</i>	oesophagus, intestine, liver and cloaca	ulcerative oesophagitis, gastritis and hepatitis	Aguirre et al., 1994 and Orós ¹ et al., 2004
			<i>Flavobacterium</i> sp.	lungs	pneumonia	Glazebrook and Campbell, 1990
			<i>Hafnia alvei</i>	cloaca	fibropapillomatosis	Aguirre et al., 1994
			<i>Klebsiella oxytoca</i>	cloaca	fibropapillomatosis	Aguirre et al., 1994
			<i>Klebsiella pneumoniae</i>	oesophagus	-	Aguirre et al., 1994
			<i>Morganella morganii</i>	cloaca	-	Aguirre et al., 1994
			<i>Proteus mirabilis</i>	cloaca	fibropapillomatosis	Aguirre et al., 1994
			<i>Proteus penneri</i>	cloaca	-	Aguirre et al., 1994
			<i>Proteus</i> sp.	stomach, intestine and liver	ulcerative stomatitis, gastritis and hepatitis	Orós ¹ et al., 2004
	<i>Proteus vulgaris</i>	cloaca	-	Aguirre et al., 1994		
	<i>Providencia alcalifaciens</i>	cloaca	-	Aguirre et al., 1994		
	<i>Pseudomonas aeruginosa</i>	oesophagus and cloaca	fibropapillomatosis	Aguirre et al., 1994		
	<i>Pseudomonas fluorescens</i>	oesophagus and cloaca	fibropapillomatosis	Aguirre et al., 1994		
	<i>Pseudomonas maltophilia</i>	oesophagus and cloaca	fibropapillomatosis	Aguirre et al., 1994		
	<i>Pseudomonas putida</i>	oesophagus and cloaca	fibropapillomatosis	Aguirre et al., 1994		
	<i>Pseudomonas putrefaciens</i>	oesophagus and cloaca	fibropapillomatosis	Aguirre et al., 1994		
	<i>Pseudomonas</i> sp.	oesophagus	ulcerative oesophagitis	Orós ¹ et al., 2004		
	<i>Pseudomonas stutzeri</i>	oesophagus and cloaca	fibropapillomatosis	Aguirre et al., 1994		
	<i>Pseudomonas visicularis</i>	oesophagus	-	Aguirre et al., 1994		
	<i>Salmonella chester</i>	turtle meat(consumption)	-	Aguirre et al., 2006		
	<i>Vibrio alginolyticus</i>	oesophagus and cloaca	fibropapillomatosis, and ulcerative in oral cavity, oesophagus and stomach; gastritis and hepatitis	Aguirre et al., 1994 and Orós ¹ et al., 2004		
<i>Vibrio damsela</i>	oesophagus and cloaca	fibropapillomatosis	Aguirre et al., 1994			
<i>Vibrio fluvialis</i>	oesophagus and cloaca	fibropapillomatosis	Aguirre et al., 1994			
<i>Vibrio</i> sp.	oesophagus	-	Aguirre et al., 1994			
Gram-negative bacillus		<i>Acinetobacter anitratus</i>	oesophagus and cloaca	fibropapillomatosis	Aguirre et al., 1994	
		<i>Acinetobacter lwoffii</i>	cloaca	fibropapillomatosis	Aguirre et al., 1994	
		<i>Clostridium fallax</i>	intestinal mucosa	related with coccidiosis	Rebell et al., 1974	
		<i>Lactobacillus</i> sp.	oesophagus and cloaca	fibropapillomatosis	Aguirre et al., 1994	
		<i>Micrococcus</i> sp.	oesophagus and cloaca	fibropapillomatosis	Aguirre et al., 1994	
		<i>Mycobacterium avium</i>	skin	mycobacteriosis	Greer et al., 2003	
		<i>Staphylococcus aureus</i>	oesophagus	-	Aguirre et al., 1994	
		<i>Staphylococcus epidermidis</i>	oesophagus	fibropapillomatosis	Aguirre et al., 1994	
		<i>Staphylococcus</i> sp.	oesophagus, stomach, intestine and liver	ulcerative oesophagitis and stomatitis; gastritis and hepatitis	Orós ¹ et al., 2004	
		<i>Streptococcus alpha</i>	oesophagus and cloaca	fibropapillomatosis	Aguirre et al., 1994	
<i>Streptococcus</i> non-hemolytic	oesophagus and cloaca	fibropapillomatosis	Aguirre et al., 1994			
<i>Streptococcus</i> sp.	intestine and liver	gastritis and hepatitis	Orós ¹ et al., 2004			
Fungi	Class Ascomycetes	<i>Fusarium scirpi</i>	skin lesions	-	Glazebrook and Campbell, 1990	
		<i>Fusarium solani</i>	Flipper	dermatomycosis	Cabañes et al., 1997	
	Class Ascomycetes (Hyphomycetes?)	<i>Cladosporium</i> sp.	lungs	pneumonia	Jacobson et al., 1979	
		<i>Sporotrichium</i> sp.	lungs	pneumonia	Jacobson et al., 1979	
	Class Basidiomycetes	Class Euscomycetes	<i>Paecilomyces</i> sp.	lungs	pneumonia	Jacobson et al., 1979; Glazebrook and Campbell, 1990
			<i>Penicillium</i> sp.	skin lesions	-	Glazebrook and Campbell, 1990

Protozoa	Class Sporozoea/ Subclass Coccidia	<i>Caryospora cheloniae</i>	intestines (hindgut)	-	Leibovitz et al., 1978
		<i>Eimerie sp. or Caryosporis sp.</i>	intestine of hatchlings	coccidiosis	Rebell, 1974
	Superclass Rhizopoda/ Class Lobosea	<i>Entamoeba invadens</i>	hatchlings	-	Aguirre et al., 2006
		<i>Cryptosporidium sp.</i>	-	-	Aguirre et al., 2006
Platyhelminthes	?	<i>Squarocetabulum solus</i>	heart	-	Glazebrook et al., 1989
	Class Cestoda	<i>Tentacularia coryphaenae</i>	stomach and intestine	-	Aznar et al., 1998
	Class Trematoda/ Family Paramphistomidae	<i>Schizamphistomoides erratum</i>	lower intestine	-	Santoro et al., 2006
		<i>Schizamphistomoides scleroporium</i> (*)	intestine and stomach	-	Santoro et al., 2006
	Class Trematoda/ Subclass Digenea	<i>Amphiorchis amphiorchis</i>	visceral blood vessels	-	Glazebrook et al., 1989
		<i>Amphiorchis coborojoensis</i>	blood vessels of lungs	-	Glazebrook et al., 1989
		<i>Amphiorchis indicus</i>	-	-	Smith, 1997
		<i>Amphiorchis solus</i>	intestine	-	Santoro et al., 2006
		<i>Carettacola bipora</i>	abdominal blood vessels	-	Glazebrook et al., 1989
		<i>Carettacola hawaiiensis</i>	vessels in bladder and ventriculus	-	Work et al., 2005
		<i>Carettacola stunkardi</i>	mesenteric veins	-	Glazebrook et al., 1989
		<i>Charaxicephaloides sp.</i>	stomach	-	Santoro et al., 2006
		<i>Charaxicephalus robustus</i>	oesophagus and stomach	-	Santoro et al., 2006
		<i>Cheloneotrema testicaudata</i>	-	-	Smith, 1997
		<i>Clinostomum complanatum</i>	oesophagus	-	Santoro et al., 2006
		<i>Cricocephalus albus</i>	-	-	Aznar et al., 1998
		<i>Cricocephalus megastomus</i>	oesophagus and stomach	-	Santoro et al., 2006
		<i>Cricocephalus resectus</i>	oesophagus and stomach	-	Santoro et al., 2006
		<i>Desmogonius desmogonius</i>	oesophagus and stomach	-	Santoro et al., 2006
		<i>Deuterobaris intestinalis</i>	intestine	-	Santoro et al., 2006
		<i>Haemoxenicon chelonenecon</i>	mesenteric veins	-	Glazebrook et al., 1989
		<i>Hapalotrema dorsopora</i>	-	-	Smith, 1997
		<i>Hapalotrema loossi</i>	-	-	Smith, 1997
		<i>Hapalotrema mehrai</i>	heart	-	Glazebrook et al., 1989
		<i>Hapalotrema pambanensis</i>	-	-	Smith, 1997
		<i>Hapalotrema postorchis</i>	heart	-	Glazebrook et al., 1989
		<i>Hapalotrema sp.</i>	-	-	Smith, 1997
		<i>Himasomum lobatus</i>	intestine	-	Santoro et al., 2006
		<i>Learedius europaeus</i>	-	-	Smith, 1997
		<i>Learedius learedi</i>	heart	-	Inohuye-Rivera et al., 2004
		<i>Learedius loochooensis</i>	-	-	Smith, 1997
		<i>Learedius orientalis</i>	heart	-	Glazebrook et al., 1989
		<i>Learedius similis</i>	circulatory system	-	Glazebrook et al., 1989
<i>Learedius sp.</i>		-	-	Smith, 1997	
<i>Microscaphidium reticulare</i>		intestine	-	Santoro et al., 2006	
<i>Microscaphidium warui</i>		urinary bladder	-	Santoro et al., 2006	
<i>Monticellius indicum</i>		heart	-	Glazebrook et al., 1989	
<i>Neoctangium travassosi</i>	stomach, small and large intestines	-	Blair, 1987		
<i>Neosporichis schistosomatoides</i>	visceral blood vessels	-	Glazebrook et al., 1989		
<i>Octangium hyphalum</i>	stomach, small and large intestines	-	Blair, 1987		
<i>Octangium sagitta</i>	stomach, small and large intestines	-	Blair, 1987		
<i>Pleurogonius linearis</i>	intestine	-	Santoro et al., 2006		
<i>Pleurogonius longiusculus</i>	intestine	-	Santoro et al., 2006		
<i>Pleurogonius sindhii</i>	intestine	-	Santoro et al., 2006		
<i>Pleurogonius solidus</i>	intestine	-	Santoro et al., 2006		
<i>Pleurogonius sp.</i>	lower intestine	-	Santoro et al., 2006		
<i>Polyangium linguatula</i>	intestine	-	Santoro et al., 2006		
<i>Pronocephalus obliquus</i>	high intestine	-	Santoro et al., 2006		
<i>Pyelosomum cochlear</i>	urinary bladder	-	Santoro et al., 2006		
<i>Rameshwarotrema uterocrescens</i>	oesophagus	-	Santoro et al., 2006		
<i>Rhytidodoides intestinalis</i>	gall bladder	-	Santoro et al., 2006		
<i>Rhytidodoides similis</i>	gall bladder and liver	-	Santoro et al., 2006		
<i>Satyanarayanotrema satyanarayani</i>	-	-	Smith, 1997		
Nemathelminthes	Class Nematoda	<i>Anisakis sp.</i>	gut and pleuroperitoneal cavity	-	Glazebrook and Campbell, 1990
Annelida	Class Hirudinea	<i>Ozobranchnus branchiatus</i>	soft skin	fibropapillomas	Greenblatt et al., 2004
		<i>Ozobranchnus margoi</i>	soft skin	fibropapillomas	Greenblatt et al., 2004
Arthropoda	Class Insecta/Family Phoridae	<i>Megaselia scalaris</i>	eggs and hatchlings	-	Bjorndal et al., 1985
		<i>Puliciphora borinque</i>	eggs and hatchlings	-	Bjorndal et al., 1985
	Class Insecta/Family Sarcophagidae	<i>Eumacronychia sternalis</i>	eggs and hatchlings	-	Lopes, 1982
		<i>Parasarcophaga argyrostoma</i>	eggs and hatchlings	-	McGowan et al., 1997
		<i>Phyllotelles pictipennes</i>	eggs and hatchlings	-	McGowan et al., 1997
		<i>Sarcotachina aegyptiaca</i>	eggs and hatchlings	-	McGowan et al., 1997
<i>Wohlfahrtia nuba</i>	eggs and hatchlings	-	McGowan et al., 1997		

Natator depressa ((Garman, 1880 (Cogger, Cameron & Cogger 1983)). Flatback sea turtle



Natator

Taxon	Sub-Taxon	Pathogen or parasite	Location in the host	Disease	Reference or source
Virus	Family Papillomaviridae	Papilloma virus	skin	fibropapillomatosis	Mader, 2006
Fungi	Class Ascomycetes	<i>Fusarium solani</i>	skin lesions	dermatomycosis	Mader, 2006

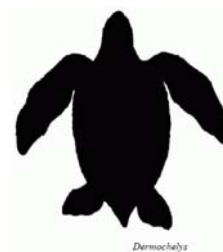
Eretmochelys imbricata (Linnaeus, 1766). Hawksbill sea turtle



Eretmochelys

Taxon	Sub-Taxon	Pathogen or parasite	Location in the host	Disease	Reference or source	
Virus	Family Papillomaviridae	Papilloma virus	skin	fibropapillomatosis	Mader, 2006	
Bacteria	Gram-negative	<i>Arizona hinshairi</i>	lungs	pneumonia	Glazebrook and Campbell, 1990	
		<i>Flavobacterium sp.</i>	lungs	pneumonia	Glazebrook and Campbell, 1990	
		<i>Salmonella regent</i>	-	-	Keymer et al., 1968	
Fungi	Class Ascomycetes	<i>Drechslera sp.</i>	necrotic skin lesions	-	Sison et al., 1990	
		<i>Fusarium solani</i>	necrotic skin lesions	dermatomycosis	Sison et al., 1990; Mader, 2006	
	Class Euscomycetes	<i>Geotrichum sp.</i>	necrotic skin lesions	-	Sison et al., 1990	
		<i>Scolecobasidium sp.</i>	necrotic skin lesions	-	Sison et al., 1990	
		<i>Penicillium sp.</i>	necrotic skin lesions	-	Sison et al., 1990	
Platyhelminthes	Class Trematoda/ Family Paramphistomidae	<i>Paramphistomum papillostomum</i> (*)	-	-	Blair, 1983	
		<i>Schizamphistomoides spinulosum</i>	-	-	Blair, 1983	
		<i>Schizamphistomoides chelonei</i> (*)	-	-	Blair, 1983	
		<i>Schizamphistomoides taiwanense</i> (*)	-	-	Blair, 1983	
	Class Trematoda/ Subclass Digenea		<i>Amphiorchis amphiorchis</i>	-	-	Smith, 1997
			<i>Amphiorchis caborojoensis</i>	-	-	Smith, 1997
			<i>Amphiorchis indicus</i>	-	-	Smith, 1997
			<i>Amphiorchis lateralis</i>	-	-	Smith, 1997
			<i>Cricocephalus albus</i>	-	-	Aznar et al., 1998
			<i>Hapalotrema orientale</i>	-	-	Smith, 1997
			<i>Hapalotrema sp.</i>	-	-	Smith, 1997
			<i>Hapalotrema synorchis</i>	-	-	Smith, 1997
			<i>Learedius learedi</i>	-	-	Smith, 1997
<i>Learedius orientalis</i>	-	-	Smith, 1997			
<i>Learedius sp.</i>	-	-	Smith, 1997			
Annelida	Class Hirudinea Class Polychaeta	<i>Ozobranchus margo</i>	skin	-	Mader, 2006	
		<i>Hydroides sp.</i>	skin and shell	-	Frick et al., 2003	
		<i>Loimia sp.</i>	skin and shell	-	Frick et al., 2003	
Arthropoda	Class Insecta/Family Phoridae	<i>Megaselia scalaris</i>	eggs and hatchlings	-	Fowler, 1979	
		<i>Puliciphora borinque</i>	eggs and hatchlings	-	Fowler, 1979	

Dermochelys coriacea (Vandelli, 1761). Leatherback sea turtle



Taxon	Sub-Taxon	Pathogen or parasite	Location in the host	Disease	Reference or source	
Virus	Family Papillomaviridae	Papilloma virus	skin	fibropapillomatosis	Huertas et al., 2003	
Bacteria	Bacillus (variable)	<i>Bacillus sp.</i>	intestine	gastritis	Orós ¹ et al., 2004	
		Gram-negative	<i>Aeromonas hydrophila</i>	oral cavity and oesophagus	oral cavity ulcerative and ulcerative oesophagitis	Orós ¹ et al., 2004
		<i>Citrobacter sp.</i>	oesophagus and liver	ulcerative oesophagitis and hepatitis	Orós ¹ et al., 2004	
		<i>Escherichia coli</i>	oesophagus, intestine and liver	ulcerative oesophagitis, gastritis and hepatitis	Orós ¹ et al., 2004	
		<i>Proteus sp.</i>	stomach, intestine and liver	ulcerative stomatitis, gastritis and hepatitis	Orós ¹ et al., 2004	
		<i>Pseudomonas sp.</i>	oesophagus	ulcerative oesophagitis	Orós ¹ et al., 2004	
		<i>Vibrio alginolyticus</i>	oral cavity, oesophagus, stomach, intestine and liver	ulcerative in oral cavity, oesophagus and stomach; gastritis and hepatitis	Orós ¹ et al., 2004	
		<i>Vibrio damsela</i>	heart	valvular endocarditis and septicemia	Ghittino et al., 1984	
		Gram-positive	<i>Mycobacterium chelonae</i>	skin	mycobacteriosis	Greer et al., 2003
			<i>Staphylococcus sp.</i>	oesophagus, stomach, intestine and liver	ulcerative oesophagitis and stomatitis; gastritis and hepatitis	Orós ¹ et al., 2004
		<i>Streptococcus sp.</i>	intestine and liver	gastritis and hepatitis	Orós ¹ et al., 2004	
Fungi	Class Ascomycetes	<i>Fusarium solani</i>	skin lesions	dermatomycosis	Mader, 2006	
Protozoa	Superclass Rhizopoda/ Class Lobosea	<i>Entamoeba histolitica</i>	-	-	Aguirre et al., 2006	
Platyhelminthes	Class Trematoda/ Subclass Digenea	<i>Cricocephalus albus</i>	-	-	Aznar et al., 1998	
		<i>Enodiotrema carettae</i>	liver	-	Manfredi et al., 1996	
		<i>Enodiotrema instar</i>	intestine	-	Manfredi et al., 1996	
		<i>Pyelosomum renicapite</i>	intestine	-	Manfredi et al., 1996	
Arthropoda	Class Arachnida/Family Acaridae	<i>Rizoglyphus sp.</i>	eggs and hatchlings	-	Andrade et al., 1992	
	Class Insecta/Family Sarcophagidae	<i>Eusenotainia rufiventris</i>	eggs and hatchlings	-	Vázquez, 1994	
		<i>Eusenotainia sp.</i>	eggs and hatchlings	-	Andrade et al., 1992	
		<i>Phrosinella sp.</i>	eggs and hatchlings	-	Andrade et al., 1992	

Notes:

- * It is not clear whether *Calicodes anthos* and *Calycodes anthos* are two different species or just a spelling mistake, since some authors keep using both scientific names
- (*) The four different platyhelminthes names seem to be the same species which has been described by different authors in different places and different times, all showing the same species. Further information provided by Blair, 1983.

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