

The Elasmobranch Husbandry Manual: Captive Care of Sharks, Rays and their Relatives

Editors

Mark Smith
Doug Warmolts
Dennis Thoney
Robert Hueter



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Ohio Biological Survey
P.O. Box 21370
Columbus, OH 43221-0370
<ohiobiosurvey@sbcglobal.net>
www.ohiobiologicalsurvey.org

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Chapter 28

Goiter in Elasmobranchs

GERALD L. CROW

*Waikiki Aquarium, University of Hawaii,
2777 Kalakaua Ave.,
Honolulu, HI 96815, USA.
E-mail: crow@waquarium.org*

Abstract: Goiters are commonly observed in elasmobranch specimens from aquariums around the world. Swelling as the result of goiter can become expansive and may result in death if left untreated. Several types of goiters (i.e., diffuse hyperplastic, diffuse colloid, and multinodular colloid) can be differentiated, all of which are unlikely to result from a simple iodide deficiency. Maintaining iodide concentrations close to natural seawater levels (i.e., 0.06 mg l⁻¹), and nitrate concentrations <10 mg l⁻¹, appears to reduce the incidence of goiter. In seawater systems where iodide and nitrate concentrations cannot be controlled, an iodine derivative supplementation dosage of 10-30 mg kg body weight⁻¹ week⁻¹ PO is recommended.

Enlargement of the thyroid gland is commonly referred to as goiter. As early as the 1900's goiter has been observed in wild and captive fishes. Although goiter is often reported, the actual etiology of the condition is poorly understood. This paper reviews the current status of goiter and goiter treatment in captive elasmobranch fishes.

Goiter has been observed in both free-swimming and captive elasmobranchs since its first observation by Cameron and Vincent (1915) off Nanaimo, British Columbia. Goiter has been observed in 18 species of captive elasmobranchs (Table 28.1). Goiter is considered widespread throughout captive facilities and is particularly common in closed-system, ozonated water systems. Goiter is typically observed as a progressive swelling of the thyroid gland, which can expand to as much as 300 times its normal size. This condition, if left untreated, can result in difficulty swallowing, causing decreased food intake, starvation, and eventually, death.

The basic structure of the thyroid gland is common throughout jawed vertebrates. The thyroid gland in elasmobranchs is an encapsulated organ located in loose connective tissue between the ventral side of the coracohyal and the medial side

of the coracomandibular muscles (Honma et al., 1987); i.e., the thyroid gland is essentially located in the middle of the lower jaw muscles. The tissue of the thyroid gland is comprised of follicles with a highly vascular blood capillary system (Ferguson, 1911; Norris, 1985). Each follicle is formed of epithelial cells surrounding a fluid-filled lumen. The lumen contains a colloid suspension of an iodide-rich protein called thyroglobulin, which is engulfed by follicle cells under stimulation by the thyroid-stimulating hormone (TSH) and converted by hydrolysis into T4 (thyroxine) before being secreted into the blood stream. Studies on the release of thyroid hormone in elasmobranchs are currently being conducted at the University of Manitoba, Canada (Eales, pers. com.).

In teleost fishes only T4 is released by the thyroid gland, while in mammals both T4 and T3 (Triiodothyronine) are released. The thyroid hormones T4 and T3 are present in both the bound (total) and unbound (free) state in circulating blood; however, thyroid hormone-sensitive tissues have only T3 receptors. Thus, only T3 has biological activity and T4 acts as a prohormone available for enzymatic conversion into T3 (Leary et al., 1999). In the spiny dogfish (*Squalus acanthias*), the liver has been observed

Table 28.1. Published reports of goiter in captive elasmobranchs, showing species and reporting institution.

Species name	Common name	Institution name	Reference
<i>Carcharhinus galapagensis</i>	Galapagos shark	Ueno Zoo	Uchida and Abe, 1987
<i>Carcharhinus melanopterus</i>	blacktip reef shark	Sea Life Park Hawaii	Crow et al., 1998
<i>Carcharhinus obscurus</i>	dusky shark	Ueno Zoo	Masahito et al., 1982
<i>Carcharhinus plumbeus</i>	sandbar shark	Ueno Zoo	Masahito et al., 1982
<i>Carcharias taurus</i>	sand tiger shark	New England Aquarium	Crow et al., 1998
<i>Chiloscyllium punctatum</i>	brownbanded bambooshark	Steinhart Aquarium	Crow et al., 1998
<i>Dasyatis akajei</i>	red stingray	Ueno Zoo	Masahito et al., 1982
<i>Dasyatis lata</i>	brown stingray	Sea Life Park Hawaii	Crow et al., 1998
<i>Ginglymostoma cirratum</i>	nurse shark	Shedd Aquarium	Nigrelli and Ruggieri, 1974
<i>Hemiscyllium ocellatum</i>	epaulette shark	Steinhart Aquarium	Crow et al., 1998
<i>Heterodontus francisci</i>	horn shark	Shedd Aquarium	Nigrelli and Ruggieri, 1974
<i>Heterodontus japonicus</i>	Japanese bullhead shark	Ueno Zoo	Uchida and Abe, 1987
<i>Negaprion brevirostris</i>	lemon shark	Shedd Aquarium	Nigrelli and Ruggieri, 1974
<i>Raja eglanteria</i>	clearnose skate	Mote Marine Laboratory	Crow et al., 1998
<i>Scyliorhinus canicula</i>	smallspotted catshark	Basel Zoo	Straub, 1995
<i>Triaenodon obesus</i>	whitetip reef shark	Sea Life Park Hawaii	Crow et al., 1998
<i>Triakis scyllium</i>	banded houndshark	Ueno Zoo	Masahito et al., 1982
<i>Triakis semifasciata</i>	leopard shark	Shedd Aquarium	Nigrelli and Ruggieri, 1974

as a site for peripheral production of T3 (Leary et al., 1999). The kidney and perhaps other organs may also produce T3. Excess circulating T4 can be excreted without producing the active hormone T3.

Some preliminary information on T4 and T3 concentrations in elasmobranchs is available. Immature sharks have lower serum T4 and T3 concentrations than ovulating and pregnant females (Volkoff et al., 1999). Immature captive whitetip reef sharks (*Triaenodon obesus*) showed no significant sexual differences in serum T4 and T3 (Crow et al., 1999). Serum T4 had a significant increase during winter with a mean concentration of 6.58 ng ml⁻¹, compared to a summer mean concentration of 3.62 ng ml⁻¹ (Crow et al., 1999). Whitetip reef sharks with goiters had T4 concentrations of 0.93-0.99 ng ml⁻¹ and T3 concentrations of 0.22-0.33 ng ml⁻¹, while non-goitered whitetip reef sharks had T4 concentrations of 3.1-7.9 ng ml⁻¹ and T3 concentrations of 0.89-1.1 ng ml⁻¹ (Crow et al., 1998).

Iodine is an essential nutrient for all animal species. Although iodine occurs globally, its geographic distribution is variable. Iodine is found in organic deposits and in sedimentary phosphate rock. Iodine occurs in plant tissue and seawater, predominately as inorganic iodide, and is readily absorbed in the intestinal tract (Miller and Ammerman, 1995; Wong, 1991). The surface waters of the ocean typically contain the highest concentration of iodine (Wong, 1991).

Seawater contains two species of dissolved inorganic iodine (iodide and iodate) (Wong, 1991). Artificial, coastal, and well seawater can have variable elemental compositions and need to be monitored carefully (Atkinson and Bingham, 1997; Crow et al., 1998). Iodide is thought to be the most biologically active form of iodine and diffusion uptake of iodide occurs across the gills and stomach, with excretion primarily at the kidney and rectal gland (Shuttleworth, 1988). Water chemistry can vary between aquariums and iodine speciation needs to be monitored carefully. Facilities using saltwater wells may have different iodide and iodate speciation. Thus, total iodine alone does not give a full picture of the iodide available to elasmobranchs (Crow et al., 1998). In addition, ozone alters the speciation of iodine by reducing iodide (and dissolved organic iodide) to iodate (Sherrill et al., 2000).

The diets of captive elasmobranchs typically rely on herring (*Clupea harengus*) and smelt (*Osmerus* spp.) which are relatively low (i.e., 5-10 mg kg⁻¹) in iodine (Lall, 1989). Malnutrition can increase the likelihood and severity of goiter (Gaitan, 1990) and ascorbic acid deficiency can reduce iodide uptake (Agrawal and Mahajan, 1981). Hunt and Eales (1979) found that iodide uptake was at least 84% from surrounding water and 16% from diet in the rainbow trout. The percentage of iodide uptake in elasmobranchs is unknown.

GOITROGENIC AGENTS

A goitrogen is a chemical that interferes with the function of the thyroid gland. These chemicals cause thyroid enlargement by acting directly on the thyroid gland, altering the regulatory mechanism, affecting peripheral metabolism, or causing the excretion of thyroid hormones (Gaitan, 1990). Excess nitrogen (in the form of nitrate) may be a goitrogen. Bromide, fluoride, calcium, cobalt, manganese, and sulfides can all inhibit normal iodine uptake. Excess iodine can inhibit thyroid activity (see Miller and Ammerman, 1995).

HISTOPATHOLOGY

Enlargement of the thyroid gland can result from the following conditions (Robbins, 1994): (1) hyperthyroidism (thyrotoxicosis—elevated circulating T4 and T3 concentrations); (2) hypothyroidism (reduced concentrations of circulating T4 and T3); (3) thyroiditis (swelling caused by interstitial and infectious processes); (4) tumor (nodular or cyst formation); and (5) congenital anomalies. All of these conditions must be considered and may affect thyroid hormone concentrations. Typically, enlargement of the thyroid gland in captive elasmobranchs results in both hypertrophy (increase in size) and hyperplasia (increase in cell number) of the follicles (Crow et al., 2001). The shape of the follicles and amount of colloid present within the follicle vary widely.

Crow et al. (2001) examined goiters of captive elasmobranchs and reported the following types of goiters:

1. Diffuse hyperplastic goiter: the thyroid gland consisted of small to medium-sized follicles with little to no colloid. Follicular cells tended to be columnar.
2. Diffuse colloid goiter: the thyroid gland consisted of large rounded follicles, containing colloid, with some scattered small follicles. Follicle cell shape varied from cuboidal to columnar. A few papillary projections were present.
3. Multinodular colloid goiter: follicles varied in size from large to small, mostly with colloid. Follicular cells ranged from flattened, to cuboidal, to columnar in shape. Fibrous bands divided the thyroid gland into nodules and

fibrous scarring, with areas of hemosiderin, indicating a previous hemorrhage.

Diffuse hyperplastic goiter results from a reduction of circulating T4 and T3 with an elevation of TSH. This goiter is characterized by a loss of colloid, papillary infolding of the follicular epithelium, and prominent cellular hyperplasia and hypertrophy (Greer et al., 1967). These goiters are characteristic of iodine deficiency or goitrogenic agents blocking the uptake of iodine. If an elasmobranch has a strict iodine deficiency (i.e., insufficient iodine available in the water and food) this is the type of goiter you would expect. Low iodine in the thyroid gland results in thyroid stimulation, in an attempt to produce more circulating T4 and T3, and eventually, the thyroid gland becomes depleted of colloid as it attempts to supply this increased demand.

Diffuse colloid goiter is thought to derive from: (1) diffuse hyperplastic thyroid glands that begin to receive sufficient iodine and produce normal concentrations of thyroid hormones, resulting in iodine storage in the already enlarged follicles (Marine and Lenhart, 1909); and/or (2) slowly growing goiters in areas of moderate or intermittent iodine deficiency that may already be colloid-rich at the time of the goiter's first appearance (Gerber et al., 1981). This condition could occur if the thyroid gland becomes less responsive to TSH stimulation and the supply of iodine fluctuates (Gerber et al., 1981). Nearly all long-standing colloid goiters are transformed into multinodular colloid goiters (Robbins, 1994).

The Ueno Zoo (Tokyo, Japan) has been the most active in thyroid assessment. Interestingly, all three types of goiter have been found at this facility, suggesting that a strict iodine deficiency alone did not account for all of these goiters. It is possible that iodide deficiency and a goitrogenic agent produced a synergistic response, exacerbating the development of goiter. It is equally possible that the thyroid gland compensates for low level iodine concentrations and attempts to create some sort of equilibrium, resulting in a colloid goiter.

To sum up the challenge of goiter determination, Robbins (1994) states "...that there is no simple correlation between morphologic lesions and resultant clinical manifestations. A multinodular goiter, for example, in one instance may be associated with normal thyroid function, in another with hyperfunction, and yet another with hypofunction...". Stoskopf (1993) stated that

sharks with goiters are hypothyroid, have low circulating levels of T4, and have hyperplastic non-colloid goiters. Crow et al. (2001) found hypothyroid animals, having low circulating T4 and T3, with both hyperplastic and colloid goiters.

Available data is fragmentary and the exact etiology for the development of goiter is uncertain. A case can be made for iodine deficiency within a typical closed-system aquarium, where levels of iodide and dissolved organic iodine are nearly undetectable (Sherrill et al., 2000). Nitrate rises rapidly in closed systems (Spotte, 1992) and is purported to reduce the absorption and retention of iodide from the thyroid gland, leading to iodine deficiency and diffuse hyperplastic goiter. Mechanisms that lead to other types of goiter in elasmobranchs are unknown. Recent studies in humans found that the thyroid gland attempts to compensate for iodine deficiency by increasing the uptake of iodine and increasing the fraction of circulating T3 (Dumont et al., 1995). After a prolonged period, large goiters have a reduced efficiency for the synthesis and secretion of thyroid hormones (Dumont et al., 1995).

PROPHYLAXIS

In the early years of captive elasmobranch husbandry, goiter was thought to be related to thyroid tumors. It was believed that standard diets were naturally high in iodine, precluding the need to supplement. However, trout (*Salmo* spp.) and salmon (*Oncorhynchus* spp.) culture revealed cases of goiter that responded to treatments of

iodide added to the water or food. Thereafter, algal or iodide supplements have typically been used to treat thyroid enlargements in captive fishes. A wide variety of treatments have been used for goiter in elasmobranchs (Table 28.2). Stoskopf (1990) noted that the typical level of iodide provided to elasmobranchs is more than required to balance an iodine-deficient diet.

Dietary uptake of iodide in elasmobranchs requires detailed study. In terrestrial animals, bioavailable iodine is absorbed in the gastrointestinal tract and is usually provided as a supplement in the form of potassium iodide, sodium iodide, or calcium iodate (Miller and Ammerman, 1995). These oral supplements, at a threshold level, have reduced goiters. Synthroid (synthetic T4) has had variable results in therapy with skates and rays, requiring lower doses than carcharhinid sharks (Stoskopf, 1990). At the Ueno Aquarium, the iodine level of aquarium water was adjusted to 0.2 mg l⁻¹ (where natural seawater = 0.06 mg l⁻¹), resulting in existing goiter regression and the development of no new cases (Uchida and Abe, 1987). In another example, well-developed goiters regressed rapidly when sharks were placed in a natural seawater lagoon, where diet remained unchanged (Crow et al., 1998), suggesting that low iodide availability in seawater (and water chemistry) played a key role in goiter development. In mammals, the level of supplemented iodide is typically <0.5 mg kg body weight⁻¹ week⁻¹ (Miller and Ammerman, 1995).

In closed system aquariums it is likely that goiters will develop and supplementation will be

Table 28.2. Treatments for goiter in elasmobranchs, showing compound, dosages, and reporting institution. Both Mazuri Vita-ZU shark/ray and Sea Tabs refer to commercial supplements.

Compound	Dosage	Institution name	Reference
Calcium iodate	1087 mg kg of food ⁻¹ week ⁻¹	Mazuri Vita-ZU shark/ray	As recommended
Calcium iodine	0.03-0.05 mg kg body weight ⁻¹ week ⁻¹	Burger's Zoo	Janse, pers. com.
CLM01	1.5 ml week ⁻¹ (each specimen)	Basel Zoo	Straub, 1995
Potassium iodide	0.2 mg l ⁻¹ (constant immersion)	Ueno Zoo	Uchida and Abe, 1987
Potassium iodide	1.2 mg kg body weight ⁻¹ week ⁻¹	Blackpool Sea Life Centre	Lloyd, 1995
Potassium iodide	10 mg kg body weight ⁻¹ week ⁻¹	Acquario di Genova	Gili, pers. com.
Potassium iodide	10 mg kg body weight ⁻¹ week ⁻¹	Virginia Aquarium and Marine Science Center	Firchau, pers. com.
Potassium iodide	10-21.6 mg kg body weight ⁻¹ week ⁻¹	Aquarium of the Americas	Hewitt, pers. com.
Potassium iodide	0.89 µg kg body weight ⁻¹ week ⁻¹	Sea Tabs	As recommended
Potassium iodide	20 mg kg body weight ⁻¹ week ⁻¹	Oceanario de Lisboa	Correia, pers. com.
Thyro-block	32.5 mg kg body weight ⁻¹ week ⁻¹	Sea World Adventure Park Orlando	Davis, pers. com.
Yodolactina (iodine)	420 mg kg of food ⁻¹ week ⁻¹	Acuario de Veracruz	Marín-Osorno, pers. com.

necessary. There is no exact formula for iodide supplementation in elasmobranchs. Water chemistry, elasmobranch species, species composition, species density, age, reproductive condition, and diet (i.e., food type, fresh or frozen, etc.) may all affect thyroid health and goiter development. In facilities where goiters are expected to develop, an iodine derivative should be supplied prior to the onset of goiter and a safe dose of 10-30 mg kg body weight⁻¹ week⁻¹ is recommended. As stated by Stoskopf (1990), this dosage is more than a dietary supplement and may be high for some species. However, without thyroid assessments, hormone concentrations at known supplementation levels, and knowledge about uptake kinetics, it is best to err slightly on the high end of supplementation.

CONCLUSIONS

Although goiters are commonly observed in captive elasmobranchs around the world, few detailed studies have been conducted. A wide range of treatments has been attempted with variable results. Studies on the development of goiter and the factors that promote this enlargement are critical to successful treatment. Goiter appears to be a reaction to iodide deficiency and an attempt by the thyroid gland to compensate for prolonged deficiencies, with some goitrogenic interaction. Studies of thyroid hormone utilization and processing, and controlled experimental iodide therapy, are much needed.

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