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Histopathology of the Internal Anchor Tag in Spot and Spotted Seatrout

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Abstract.—Pathological tissue changes elicited by an internal anchor tag in spot *Leiostomus xanthurus* and spotted seatrout *Cynoscion nebulosus* were evaluated histologically. Forty-five spot (32 tagged fish and 13 untagged controls) and 13 tagged spotted seatrout were sampled periodically from 4 h to 4 months posttagging. Early tissue changes (4 h–4 d posttagging) in both species were characterized by acute inflammation and irritation. Changes included hemorrhage, fibrin exudation, extravasation of leukocytes, epidermal hyperplasia and spongiosis, and necrosis in exposed myotomes. Tags were progressively encapsulated and sequestered by fibrogranulation tissue in a similar sequence in both species, commencing with deposition and organization of fibrin at the tag surface. This early fibrinous capsule (at day 2) was infiltrated by leukocytes and fibroblasts, and by day 4–7 it was thickened, highly cellular, and well vascularized. Deposition of collagen within the granulation tissue capsule increased progressively for at least 8 weeks. Complications of this process occurred frequently in tagged spot, whereas the incision in control spot receiving no tag healed quickly and without complications. Almost all tagged spot showed secondary mycotic and bacterial infections, and several spot exhibited gross peritonitis. Other frequently observed complications included dislocation of scales, damage to peritoneum and ovaries by tag penetration, and displacement of tags into swimbladder or intestine. Several spot appeared actively to expel the internal anchor tag. Severe pathological changes in tagged spot suggest that both mortality and tag expulsion may have contributed to the relatively low (0.2%) tag return rate observed during the past 8 years for this sciaenid. We have discontinued tagging spot with the internal anchor tag and do not recommend its use. However, complications in spotted seatrout were minor, and tag expulsion was not observed. We have observed an 8.8% tag return rate and continue to tag this sciaenid with the internal anchor tag.

Fishery biologists conduct tagging studies to assess growth rates, population sizes, and migratory behavior of fish stocks. Several different marking devices have been developed, primarily because no single tag serves all purposes (e.g., Jakobsson 1970). Most tags are designed to be inserted into a fish's dorsum (Dell 1968), and many have been tested in a variety of fishes (Latapie 1968; Saunders 1968; Butler and Loeffel 1972; Rawstrom 1973; Wilbur and Duchrow 1973; Sackett and Hein 1979; Bulak 1983). Most authors have reported only minimal tag-induced mortalities. However, expulsion of tags by the fish and frequent breakage of certain tag types have resulted in tag losses ranging from 2 to 78%. Some studies have reported secondary infection of the tagging lesions (Roberts et al. 1973c; Sackett and Hein 1979).

The Gulf Coast Research Laboratory has conducted a fish-tagging program since 1975. A dart tag, inserted into the fish's dorsal musculature, was used in a small-scale program until 1979. Its use was discontinued because many of the tags came apart and others were apparently expelled by the fish (Overstreet 1983). An internal anchor tag is now used exclusively. The decision to switch to this tag was based partly on a preliminary study in which small numbers of striped bass *Morone saxatilis* and Atlantic croaker *Micropogonias undulatus,* tagged and held in raceways, did not shed the internal anchor tag after several months in captivity. Incisions healed rapidly, and the fish showed no gross complications from the tagging procedure (Overstreet 1983). During the past 8 years, however, few tags have been recovered for the frequently tagged sciaenid spot *Leiostomus xanthurus,* whereas tag recovery rates for the spotted seatrout *Cynoscion nebulosus* are significantly higher (Overstreet 1983, unpublished observations). Our objective, therefore, was to compare the tissue responses elicited by the internal anchor tag in spot and spotted seatrout. We undertook

this histopathological assessment to establish whether tags were expelled or whether tagging could have caused disease or mortality in spot.

Methods

Forty-five spot (mean standard length SL, 141 mm; mean total length TL, 173 mm) and 13 spotted seatrout (mean SL, 322 mm; mean TL, 368 mm; mean weight, 466 g) were captured in Mississippi Sound by trawl and by hook and line, respectively. Fish were transported by boat to the laboratory in a 400-L aerated trough, tagged, and placed in a 3,000-L outdoor raceway with filtered, recirculating water. Average water temperature was 24°C (19–29°C) and the salinity was a constant 18‰.

Tagging procedure.—Each fish to be tagged was placed on a flat surface and immobilized with slight pressure of the hand. An 8–10-mm-long ventrolateral incision was made through the body wall to accept the internal anchor tag, a procedure that has been described in detail and illustrated by Overstreet (1983). This incision paralleled the fish's vertebral column and was slightly off midline a short distance anterior to the anus. The anchor portion of the tag was then inserted into the body cavity of the fish and twisted until the anchor lay perpendicular to the incision without binding any internal organs against the wall of the peritoneal cavity. We did not anesthetize experimental fish because they are not anesthetized during normal tagging operations in the field.

Sampling and histopathology.—Twenty-seven spot received internal anchor tags, seven received only the abdominal incision (positive controls), and six fish received neither the incision nor the tag (negative controls). Three tagged spot were sampled at 4 h, 10 h, 24 h, 2 d, 4 d, 7 d, 2 weeks, 4 weeks, and 8 weeks posttagging, and two tagged spot were sampled 16 weeks posttagging. Two negative control fish were sampled at 4 h, 4 weeks, and 8 weeks posttagging. One positive control fish was sampled at 4 h, 10 h, 24 h, 2 d, 4 d, and 7 d posttagging. Twelve spotted seatrout received internal anchor tags. One fish sampled 4 weeks posttagging served as a negative control. One tagged seatrout was sampled at 24 h and another at 48 h posttagging, and two tagged fish were sampled at each of the following posttagging intervals: 4 and 7 d and 2, 4, and 8 weeks.

Individual fish were removed from the raceway by dip net and were killed by overdose with tricaine (MS-222). Each fish was then measured (SL and TL) and transferred whole to Lillie's fixative (85 mL 2% picric acid, 10 mL formaldehyde, and 5 mL 90% aqueous formic acid) for approximately 5 d to assure decalcification of bone. A slit in the body cavity anterior to the tag assured proper penetration of the fixative. After this period, a block of tissue containing the tag was excised, and the tag was removed carefully with minimal disturbance to surrounding tissues. The resulting tissue slabs then were transferred to fresh Lillie's solution for three more days. Fixative was washed from the slabs in running tap water overnight. The tissue blocks were dehydrated in a graded ethanol series, embedded in paraffin, and oriented so that the entire peritoneal cavity was visible in transverse section and the space created by the removed tag was clearly exposed. Tissues were sectioned to 6-μm thicknesses and stained with hematoxylin and eosin.

Results

Normal Histology of Spot

Figure 1a illustrates the placement of the internal anchor tag in spot. The body wall at the tag's placement site consisted of integument, hypodermis, muscle, and peritoneum. In transverse section (Figure 1b), the left and right hypaxial myotome segments tapered ventrally and became discontinuous within the ventral aspect of the body wall. A thickening of the stratum compactum and hypodermis occupied the gap between left and right myotome segments.

Normal integument (Figure 1c) consisted of a thin epidermis, a basement membrane, and a well-developed dermis comprised of a loosely organized stratum spongiosum containing the scales and a densely collagenous stratum compactum. A thin layer of connective tissue fibers separated the hypodermis, comprised chiefly of adipose tissue with few connective tissue fibers, from the stratum compactum. The amount of adipose tissue varied among individual fish.

Subcutaneous muscles of the body wall lay below the integument. Histologically, they appeared identical to the skeletal muscles of other vertebrates. A thick epimysium bordered the musculature. The peritoneum lay below the epimysium and lined the entire abdominal cavity. Its appearance varied from a simple squamous mesothelium to a simple columnar epithelium containing rodlet cells (Figure 1d). Between this cellular layer and the musculature lay a thin, heavily melanized connective tissue layer.

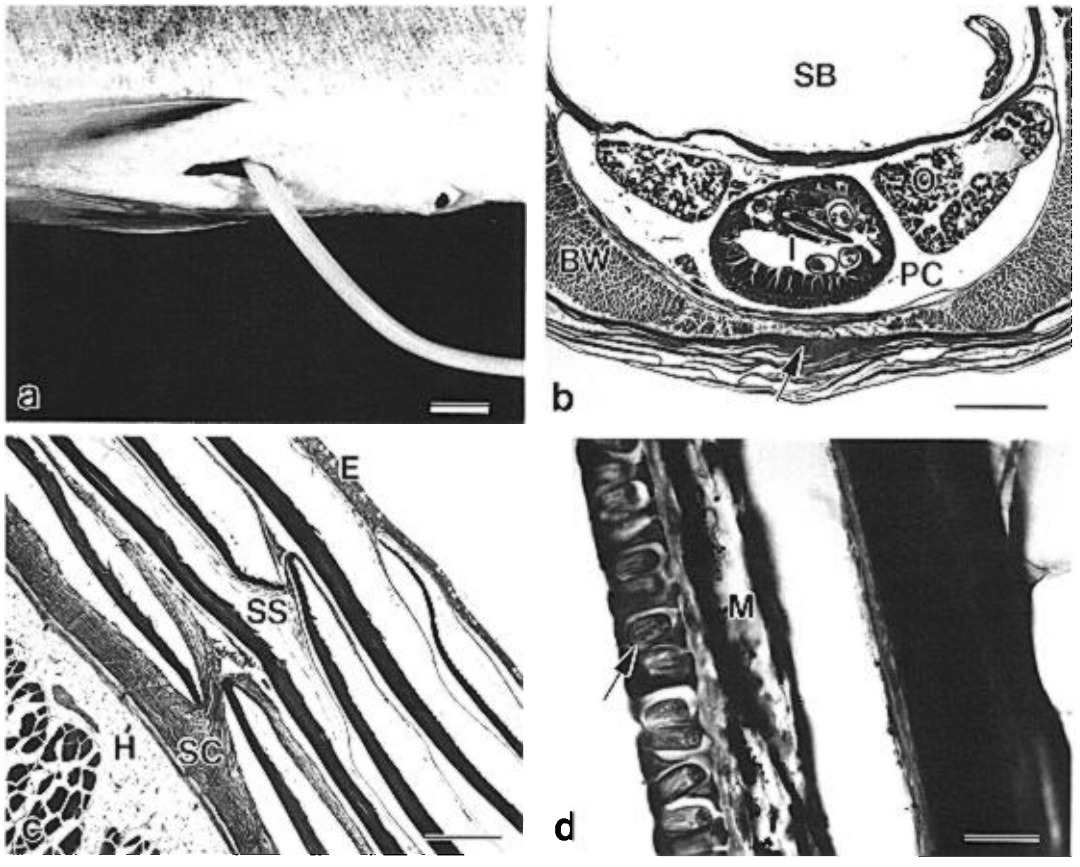

FIGURE 1.—Placement of the internal anchor tag in, and normal histology of, spot. **(a)** Position of the ventral incision. Bar = 5 mm. **(b)** Transverse section through abdominal cavity of negative control at same level where tagging incision occurred in other individuals. BW = body wall; I = intestine; O = ovary; PC = peritoneal cavity; SB = swim bladder; arrow indicates thickening of stratum compactum at the ventral surface. Bar = 1 mm. **(c)** Normal integument. E = epidermis; SS = stratum spongiosum; SC = stratum compactum; H = hypodermis. Bar = 250 μm. **(d)** Peritoneal lining of negative control fish. M = melanized connective tissue layer; arrow indicates a rodlet cell. Bar = 15 μm.

Incision in Control Spot

Healing in positive control spot was rapid and without complication. Epidermal proliferation had sealed the incision by 24 h. Hemorrhage occurred within the incision before day 2, and necrosis of myotomes bordering the incision was evident in the early samples. Inflammation was minimal, although some leukocytes were present prior to 4 d. Fibrosis within the incision first occurred at 2 d. By day 7, inflammatory and degenerative changes were much reduced, and the incision was being filled with granulation tissue. Repair was essentially completed at 2 weeks when sections were last examined, but a small amount of fibrotic tissue still marked the site of the original incision. The 4-d sample exhibited extensive myoregeneration; however, this was not a consistent feature of healing.

Acute Inflammation in Tagged Spot

Initial tissue changes resulting from the tagging operation were characteristic of acute inflammation and occurred within the peritoneal cavity and within tissues of the body wall (Figure 2a). These alterations included hemorrhage, fibrin exudation, and influx of inflammatory cells. Both proliferative and degenerative changes occurred within the incision.

Several, but not all, of the fish sampled 4 d posttagging and earlier bled slightly from their incision. Intraperitoneal bleeding was extensive in some of these fish (Figure 2b), but it was generally

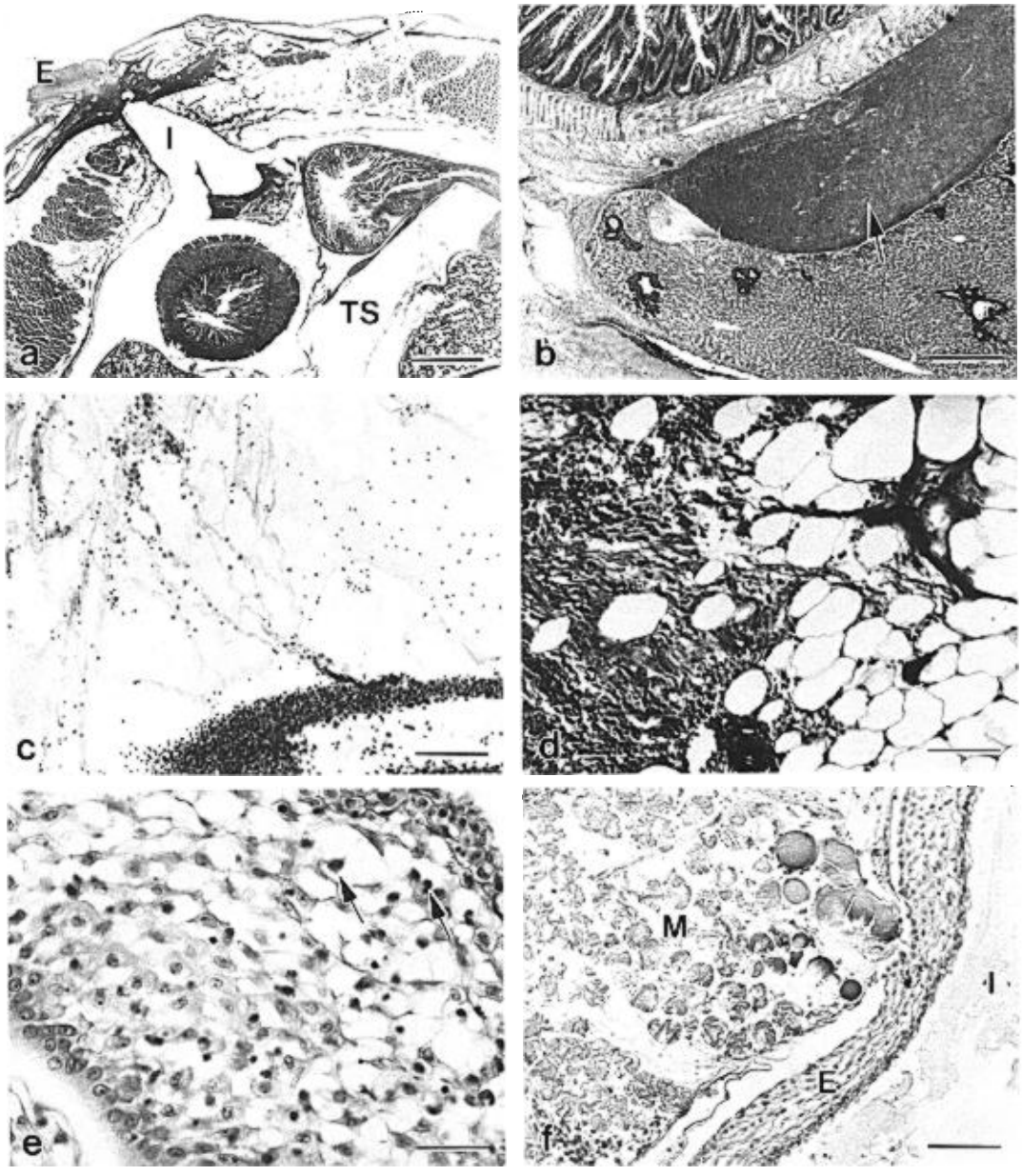

FIGURE 2.—Acute, tag-induced tissue changes in spot. **(a)** Survey micrograph of tagging lesions 24 h posttagging. I = incision; E = hyperplastic epidermis; TS = tag sinus (space originally occupied by tag anchor). Bar = 1.4 mm. **(b)** Intraperitoneal hemorrhage (arrow). Bar = 150 μm. **(c)** Intraperitoneal fibrin deposition and hemorrhage at 4 h posttagging. Bar = 65 μm. **(d)** Tracking of leukocytes in hypodermis at the edge of incision at 2 d posttagging. Bar = 70 μm. **(e)** Hyperplasia and spongiosis of the epidermis 24 h posttagging. Leukocytes (arrows) infiltrating thickened epidermis. Bar = 25 μm. **(f)** Early myodegeneration (M), and epidermal ingrowth (E) along incision edge (I) at 2 d posttagging. Bar = 55 μm.

not observed after 4 d posttagging. Pooling of blood in the abdominal cavity was associated with irritation and mechanical damage by the tag anchor to the peritoneal lining or other visceral organs. Intraperitoneal fibrin deposition was first observed at 4 h and peaked by 2–4 d. Histologically, it appeared as a fine, eosinophilic fibrous meshwork containing erythrocytes and inflammatory cells (Figure 2c).

Three leukocyte types predominated in early tagging lesions. In several fish, an eosinophilic granulocyte was the most commonly observed leukocyte before day 2, but its numbers were greatly reduced by day 7. This observation, however, was not consistent in all fish. Two other types of leukocytes emigrated from the intestinal, mesenteric, and hypodermal blood vessels by 10–24 h posttagging and remained prominent throughout the experimental period in all tagged fish. One was a mononuclear phagocyte (monocyte–macrophage) with agranular, basophilic cytoplasm and deeply basophilic nuclear chromatin. Many of these cells accumulated intraperitoneally beginning at hours 10–24. The other cell, a heterophil also present in large numbers by 24 h, had a pale cytoplasm containing a small number of lightly eosinophilic granules. Its eccentric oval (rarely bilobate) nucleus stained more purplish than that of the mononuclear phagocyte. Small mononuclear cells (probably lymphocytes) were present as well. Inflammatory cells infiltrated the integument bordering the incision. This tracking of leukocytes from the incision outward was especially evident within hypodermis (Figure 2d).

Hyperplasia (excessive proliferation of normal cells) and spongiosis (intercellular edema of the malpighian layer) of the epidermis occurred consistently in all tagging lesions. By 4 h, the epidermis around the incision became slightly thickened and edematous, and, by 24 h, it had become greatly thickened and spongiotic (Figure 2e). It remained that way for the duration of the experiment. In many fish sampled on day 4 and later, inflammatory cells had infiltrated the epidermis (Figure 2e). By day 2, the epidermis had grown into and lined the incision (Figure 2f).

Myotomes exposed by the incision exhibited early signs of degeneration at 4 h posttagging (Figure 2f). By 2–4 d, heterophils and mononuclear cells (large and small) infiltrated necrotic myofibers, and macrophages actively phagocytosed tissue debris.

The peritoneum appeared to be secretory in early tagging lesions. A flocculent basophilic precipitate coated the peritoneal lining of some fish sampled 10, 24, and 48 h posttagging. In several of these early lesions, some areas of peritoneum damaged by the tag appeared to be proliferating, with cells being liberated into the peritoneal cavity.

Chronic Changes in Tagged Spot

The tag anchor elicited a chronic inflammatory response that progressively encapsulated and sequestered the tag. By 24 and 48 h posttagging, a thin capsule surrounded the anchor (Figure 3a). This capsule consisted of a loosely organized meshwork of fibrin (Figure 3b) infiltrated by leukocytes and fibroblasts. Its cellularity increased progressively, and, by day 4, it had become considerably thickened and had formed adhesions with adjacent visceral organs (Figure 3c). By day 4, the capsule wall consisted of organizing fibrogranulation tissue rich in fibroblasts and inflammatory cells (Figure 3d). Multinucleate giant cells occurred in inflammatory tissue by this period and persisted during later periods. Vascularization of the developing granulation tissue was first observed at this time (Figure 3d). By day 7, the tag capsule had thickened further (Figure 3e), was highly cellular, and exhibited considerable organization. Vascularization of the capsule wall was advanced (Figure 3f) and some collagen was deposited in the outer wall layers. Collagen deposition increased progressively over the remaining sampling periods, whereas the capsule's cellularity decreased somewhat. In many of the later samples, portions of the tagging sinus were lined by an ingrowth of epidermis that was often disrupted when the tag was removed during tissue processing. This layer lacked the goblet and eosinophilic granular cells present in typical epidermis. Considerable amounts of necrotic tissue debris occurred within the tag sinus. The fibrogranulation tissue that filled in the healing incision appeared in several instances to be qualitatively different from that tissue comprising the tag capsule and was composed primarily of fibroblastic elements and fine connective tissue fibers with few leukocytes. Myoregeneration, although evident to some degree in several incisions, was not an important component of the healing process in spot.

Tagging Complications in Spot

Two tagged spot died during the first week of the experiment. They had sustained more trauma

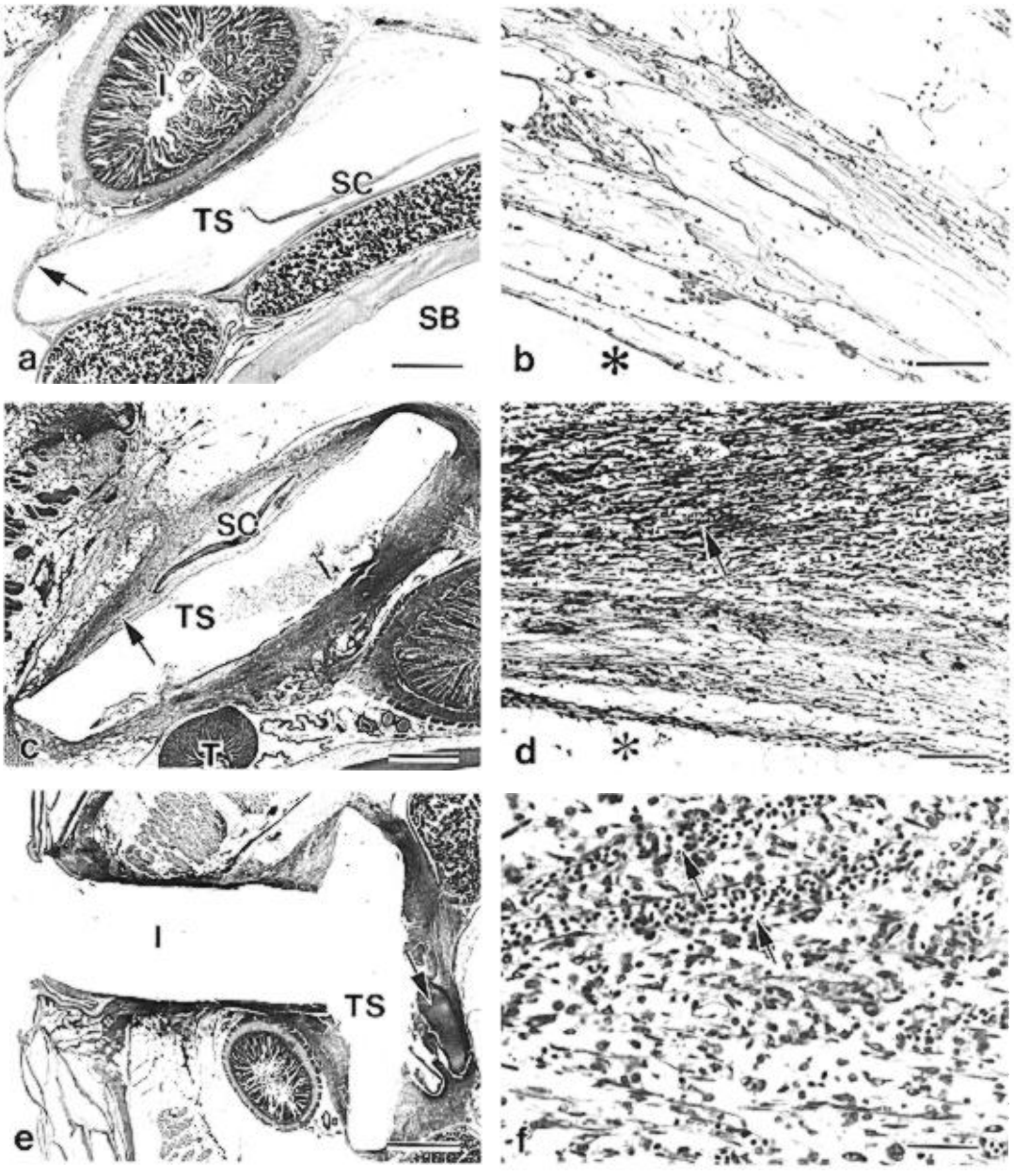

FIGURE 3.—Encapsulation of internal anchor tag by spot. **(a)** Host tissue capsule (arrow) at 2 d posttagging consisting of a thin layer of fibrin. TS = tag sinus (space originally occupied by tag anchor); SC = dislocated scale; SB = swim bladder; I = intestine. Bar = 750 μm. **(b)** Tag capsule wall at 2 d posttagging consisting of fibrinous meshwork infiltrated by leukocytes. * = tag sinus. Bar = 75 μm. **(c)** Tag capsule (arrow) 4 d posttagging, considerably thickened. TS = tag sinus; SC = scale; T = testis. Bar = 800 μm. **(d)** Tag capsule wall 4 d posttagging, composed of organizing fibrogranulation tissue rich in fibroblasts and inflammatory cells. Initial vascularization (arrow); * = tag sinus. Bar = 75 μm. **(e)** Tag capsule and incision (I) at 7 d posttagging. TS = tag sinus; arrow indicates necrotic focus containing bacterial colonies. Bar = 1.2 mm. **(f)** Tag capsule wall at 7 d posttagging consisting of fibroblasts and leukocytes and exhibiting abundant vascularization (arrows indicate venules). Bar = 25 μm.

during capture than their cohorts and developed large hemorrhaging epidermal ulcerations within several days after being tagged; consequently, they were not examined histologically.

Secondary infections with opportunist pathogens masked the normal healing and encapsulation process in many tagged individuals, especially those sampled after 2 weeks. We frequently observed granulomas and melanomacrophage centers (also termed macrophage aggregates) within tagging lesions (Figure 4a). Often the granulomas encapsulated fungal hyphae or bacteria, and frequently those agents became incorporated into the granulation tissue that encapsulated the tag anchor. In some fish, the tagging sinus harbored bacterial colonies (Figure 3e) as well as considerable necrotic tissue debris. Two fish sampled at 4 and 8 weeks posttagging exhibited severe peritonitis (Figure 4b). The swimbladder wall was destroyed in both fish, and most of the peritoneal cavity was occupied by fungal hyphae (Figure 4c) and granulation tissue. The 8-week sample also had developed a bacterial infection of the ovary. Neither of these fish presented any clinical signs of disease at the time they were sampled.

In three fish, the tag appeared to be in the process of expulsion. In one fish sampled 4 weeks posttagging, a portion of the anchor protruded through the body wall (Figure 4d). In two others sampled 8 weeks posttagging, the tag anchor was displaced from its original site within the peritoneal cavity and resided within the myotomes of the body wall (Figure 4e). The disrupted peritoneum appeared to be growing over the displaced encapsulated anchors. In neither of these lesions, however, was the damaged peritoneum completely reestablished. The tissue encapsulating these tag anchors was highly cellular and consisted of tightly compacted cords of fibroblastic cells.

Both fish sampled 16 weeks posttagging had lost their tag, and neither retained its fibrogranulation tissue capsule. The incisions had healed without complications; however, large melanomacrophage centers and granulomas occurred in the mesentery and peritoneum of both fish.

Displacement of the tag anchor constituted an additional serious complication. In several gravid females, the displaced tag had disrupted the ovary and caused the release of oocytes into the peritoneal cavity. Extensive hemorrhage often accompanied this condition. Granulomas also formed in response to liberated ova. In five fish (10-h, 24-h, 4-d, 1-week, and 8-week samples), the tag resided partially or entirely within the swim bladder (Figure 4f), and, in one of the 8-week samples exhibiting gross peritonitis, the tag anchor had partially penetrated the intestine as well (Figure 4b).

Response in Tagged Spotted Seatrout

Only minor structural differences distinguished the normal body wall and peritoneal cavity of spotted seatrout from that described above for spot. The layer of hypodermal fat present in many spot was not observed in any spotted seatrout. Melanin, which was abundant in spot peritoneum, was only rarely observed in spotted seatrout. Rodlet cells were never observed in spotted seatrout peritoneums.

The progression and timing of events leading to the encapsulation and sequestering of the tag by spotted seatrout was similar to that described for spot, with only minor variations. Initial tissue changes were characteristic of acute inflammation and included hemorrhage, intraperitoneal deposition of fibrin, and extravasation (escape from blood vessel into tissues) of inflammatory cells. Degenerative and necrotic changes in myotomes exposed by the incision occurred during the first week but were not observed after that. Epidermal hyperplasia and spongiosis surrounded the incision during the entire experimental period, but these changes were not as pronounced as in spot.

The early tag capsule was composed of an organizing, loose fibrinous meshwork infiltrated by inflammatory cells. This capsule progressively thickened and on day 4 consisted of highly cellular fibrogranulation tissue. The tag capsule appeared to form, in part, by serosal proliferation at sites where the tag anchor abutted visceral organs (Figure 5a). Such proliferation and thickening was not observed in tagged spot.

A major difference in the host response to the anchor tag between the two sciaenids was the apparent lack of complications in tagged spotted seatrout. Evidence of secondary infection in spotted seatrout was limited to small capsular granulomas and melanomacrophage centers in several fish. One fish sampled 4 weeks posttagging had a bacterial infection with associated necrotic tissue debris within the tag sinus. There was no evidence of tag dislocation or expulsion in our samples. Additionally, the tag capsules of fish sampled 8 weeks posttagging were thinner than in earlier samples, had fewer cells, and consisted mainly of fibrous connective tissue (Figure 5b).

Discussion

Tag returns for spot have been considerably lower (0.2%) than returns for spotted seatrout (8.8%) during the past 8 years (unpublished re-

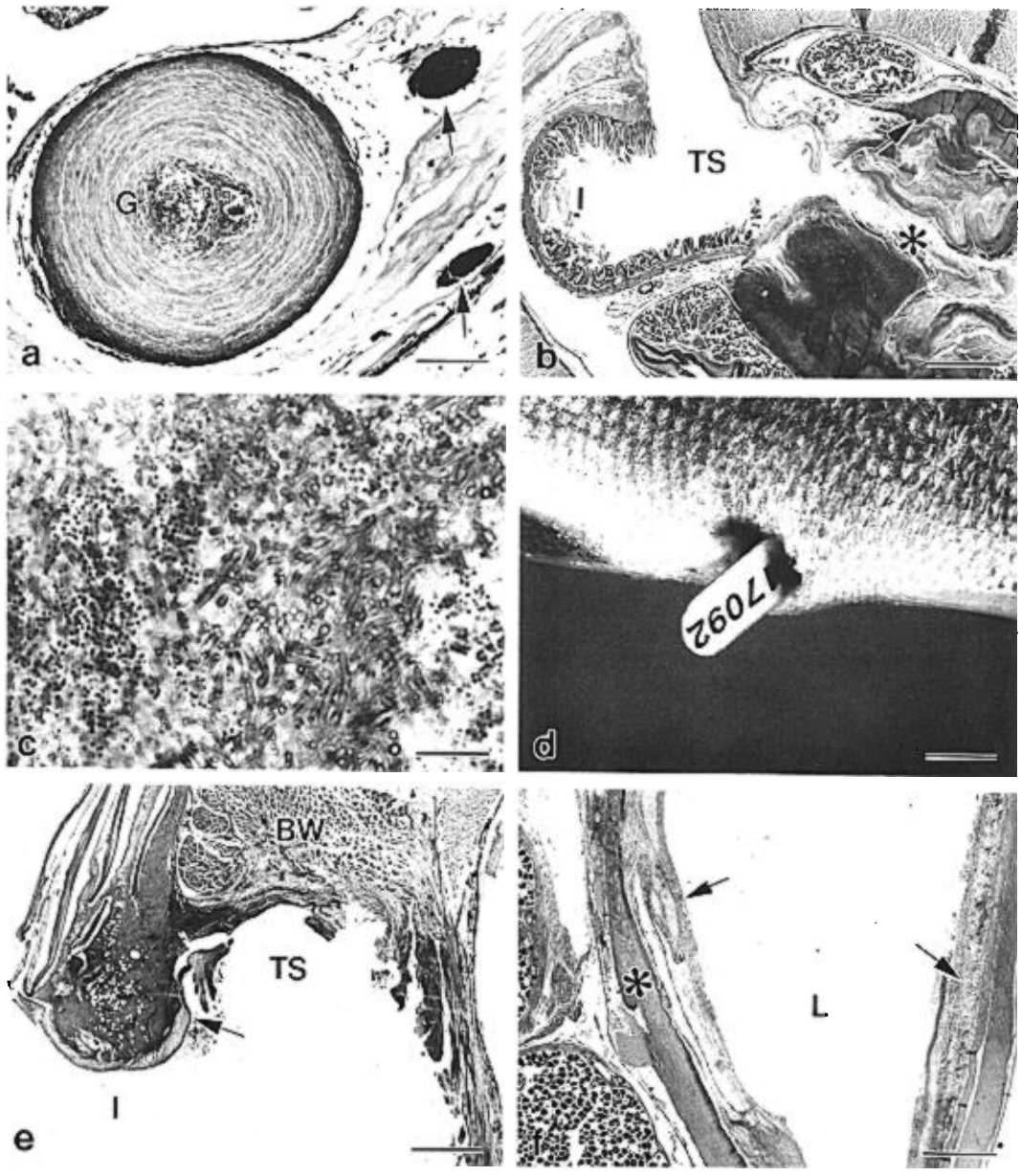

FIGURE 4.—Complications of the tagging process in spot. **(a)** Granuloma (G) and melanomacrophage centers (arrows) occurring in almost all tagged fish sampled 4 d posttagging and later. The presence of this and other granulomas suggested that secondary infection occurred frequently. Bar = 80 μm. **(b)** Severe peritonitis 8 weeks posttagging, indicating that the tag had penetrated the intestine (I). The swim bladder wall was disrupted (arrow), and much of the abdominal cavity was filled with fungal hyphae and reactive tissue (*). TS = tag sinus. Bar = 1.7 mm. **(c)** High magnification at * of panel **(b)** illustrating fungal hyphae. Bar = 27 μm. **(d)** Tag anchor protruding through body wall of 4-week sample at site other than where inserted. Bar = 8.1 mm. **(e)** Tagging lesion of an 8-week sample in which tag anchor was situated within the ventral body wall rather than within the peritoneal cavity. BW = body wall; I = incision; TS = tag sinus; arrow marks epidermis. Bar = 650 μm. **(f)** Displacement of tag anchor into the swimbladder. * = swimbladder wall; arrows indicate fibrogranulation tissue capsule; L = swimbladder lumen (= tag sinus). Bar = 640 μm.

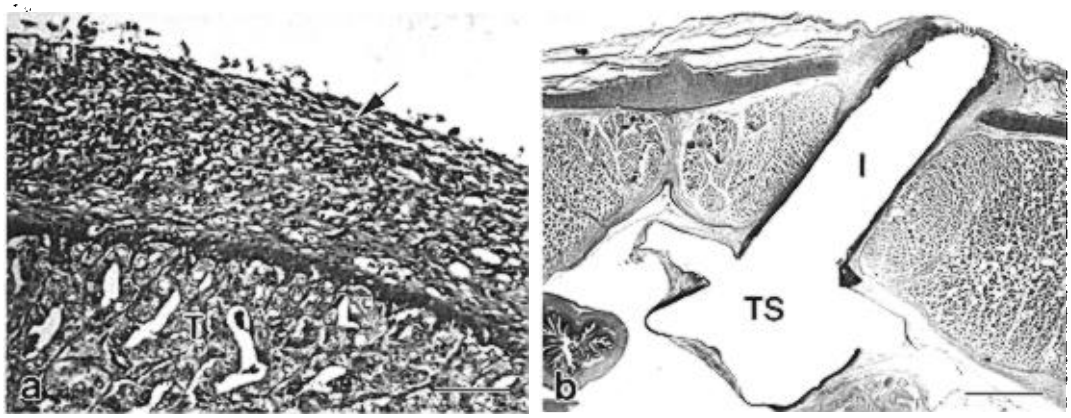

FIGURE 5.—Tissue response to internal anchor tag by spotted seatrout. **(a)** Thickening of the testicular serosa where tag anchor abuts testis (arrow). T = testis. Bar = 50 μm. **(b)** Well-healed incision and tag capsule in the 8-week sample. I = incision; TS = tag sinus. Bar = 1.9 mm.

sults). Local fishermen generally do not catch spot by hook and line (McIlwain 1978); however, the species is trawled in large numbers, both inshore and offshore, by commercial and recreational shrimpers who regularly return tags to us that have been taken from other species. Expected returns for tagged spot have also been estimated as low by others (Green et al. 1979); however, the reasons for the low recovery rate are not clear.

Repair in positive control spot (with incision, but without tag) maintained under experimental conditions was similar to first-intention healing in mammals as described by Slausen and Cooper (1982). Epithelialization of the fish incision was complete by 24 h, and fibroplasia occurred by day 2. Repair of the incision in this type of healing was uncomplicated by secondary microbial infections and was slightly quicker than that observed in mammals (Slausen and Cooper 1982).

Tissue responses to the internal anchor tag in both spot and spotted seatrout resemble the foreign-body reaction observed in mammals. This reaction was considered by Coleman et al. (1974) to be a special type of chronic inflammation consisting of an early acute exudative phase followed by histiocytic infiltration, fibroplasia, and eventual encapsulation of the foreign body by fibrogranulation tissue. This general sequence of events occurred in both spot and spotted seatrout but was modified, especially in the spot, by secondary infection and other complications. Roberts et al. (1973a, 1973b) observed a similar response in tagged parr of Atlantic salmon *Salmo salar*. In recently tagged Atlantic salmon, myodegenerative, exudative, and inflammatory changes preceded fibrosis. Severely affected muscles were subsequently replaced by fibro-granulation tissue. Myoregeneration was not a major component of the healing process in Atlantic salmon. Tagged fish were considerably more susceptible than control counterparts to infection by opportunist pathogens including species of *Saprolegnia* and *Myxobacter* (Roberts et al. 1973c), and we observed the same relationship for spot. Those authors also observed several outbreaks of furunculosis; associated mortality was almost 40% higher among tagged Atlantic salmon than among untagged fish. This finding suggested either that the stress of tagging the fish produced greater predisposition to furunculosis, or that infecting organisms could more easily invade the traumatized skin of the tagged fish. The attachment portion of the tag used in those studies extended entirely through the dorsal myotomes of the fish.

The point of insertion of the internal anchor tag may provide opportunist pathogens with continual or periodic access to the peritoneal cavity, especially in the bottom-feeding spot. Invasion by pathogens caused gross peritonitis in several spot and some evidence of secondary infection in almost all tagged spot. Nevertheless, we have no direct evidence that tagging causes mortality of spot. Although spot with peritonitis exhibited no external signs of disease at the time they were sampled (4 and 8 weeks posttagging), they might not have survived in the wild with such severe infections. On the other hand, they might be less stressed in the wild than in the raceway, and might recover more readily from infections. Considerably less histological evidence of microbial contamination

occurred in the spotted seatrout lesions than in those of the spot. In the spotted seatrout, granulomas and melanomacrophage centers occurred rarely, and the granulation tissue encapsulating the tag anchor consisted principally of connective tissue fibers by 8 weeks. Capsule cellularity was considerably decreased by this time, suggesting a healing process uncomplicated by secondary infections.

Tag-related complications other than those caused by microbial infections occurred more commonly and were more severe in spot than in spotted seatrout. Evidence of tag expulsion, entirely lacking in spotted seatrout, was notable in spot, suggesting specific differences in the ability to retain tags. Variations in tissue responses have also been described in fishes equipped with intraperitoneal telemetry transmitters. Channel catfish *Ictalurus punctatus* expel dummy transmitter implants transintestinally, through the sutured incision, and directly through the body wall (Summerfelt and Mosier 1984; Marty and Summerfelt 1986). Rainbow trout *Salmo gairdneri* also expel transmitters across the intestine (Chisholm and Hubert 1985); however, the published telemetry studies reporting migration of various species suggest that expulsion is not a common problem (Marty and Summerfelt 1986).

Marty and Summerfelt (1986) postulated that the mechanism of transmitter expulsion in channel catfish involved interactions between the maturing fibro-granulation tissue capsule and the transmitter. They suggested that channel catfish granulation tissues have contractile properties that provide the force necessary to push the transmitter through the intestinal wall. Contractility, a well-documented property of certain mammalian granulation tissues (Gabbiani et al. 1972; Montandon et al. 1977), has been ascribed to specialized contractile cells called myofibroblasts (Guber and Rudolph 1978). Marty and Summerfelt (unpublished) have ultrastructural evidence that channel catfish granulation tissue fibroblasts contain intermediate filaments, which have contractile properties in mammals and other animals. Demonstration of the ability of these elements to contract would suggest that similar mechanisms operate in both mammalian and piscine wound contraction.

Tag expulsion in spot may also be an active process. In two 8-week samples, the tag anchor had become dislocated from its normal intraperitoneal position and resided within the body wall. Reactive tissue encapsulating these anchors was highly cellular and consisted mainly of fibroblastic cells. Whether or not these cells were myofibroblasts, capable of contraction and responsible for tag expulsion, requires functional and ultrastructural confirmation. However, both spot sampled 16 weeks posttagging had shed their tags, suggesting that tag expulsion may account, at least partially, for the low tag return rate observed for this species.

Tags were observed in the swim bladder of five spot and in the alimentary tract of one. Three of these fish were recently tagged (10 h, 24 h, and 4 d posttagging), suggesting that tag displacement occurred during the initial tagging operation of some individuals and was not an active attempt at tag expulsion involving the formation and contraction of a granulation tissue capsule as described by Marty and Summerfelt (1986). Tag displacement may have resulted from poor technique during the tagging operation. However, this complication did not occur in spotted seatrout, nor did we observe it in other species (unpublished observations). A more plausible explanation for these observations is that the spot, a relatively small sciaenid with a laterally compressed body, has considerably less free abdominal space than does the spotted seatrout. It is probable that in the smaller and narrower peritoneal cavity of spot, tag-induced irritation would be greater than in the larger cavity of spotted seatrout. Tag displacement, expulsion, and damage to visceral organs would, therefore, be more likely to occur in spot.

Meaningful comparisons of the cellular responses in spot with those reported for other species require that quantitative studies be conducted. Variations in host responses depend on the species, individuals within a sample, and other factors such as initial extent of damage, degree and type of infection, stress, and nutrition (e.g., Mawdesley-Thomas and Bucke 1973). Temperature has been shown to affect piscine inflammatory responses quantitatively (Finn and Nielson 1971). Moreover, difficulty in comparing cases results because of nomenclatural problems. The terminology presently employed for fish leukocytes is based on their morphological resemblances to the leukocytes of mammals (see review by Ellis 1977), usually without regard to functional or ontogenetic relationships. This is true especially for piscine granulocytes, which show interspecific variation in morphology and staining affinities.

We do not recommend use of the internal anchor tag with spot, although the tag appears to be suitable for spotted seatrout. Other types of

tags that may be more appropriate, but probably still are not adequate for spot, include Petersen discs (Ingle et al. 1962) and monofilament nylon streamer tags (Pacheco 1962).

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