Emergence of serranid pigment abnormality syndrome (SPAS) in wire netting cod (Epinephelus quoyanus) from Heron Island on the southern Great Barrier Reef

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Abstract. Coral reefs worldwide are under increasing stress from anthropogenic impacts, but there are relatively few reports of increased rates of disease in coral reef fish. Herein we report the emergence of abnormal skin lesions in wild-caught wire netting cod (Epinephelus quoyanus) near Heron Island in the southern Great Barrier Reef. The lesion involves conspicuous darkening and disorganisation of the brown ‘wire netting’ colouration pattern typical of this species, most commonly on the lower jaw, premaxilla and head, with occasional involvement of the flanks and dorsal fin in some fish. The lesion was not present during research conducted in the mid-1990s; however, since it was first recorded in 2012, the prevalence of grossly visible lesions has increased to 16.9% in 2017, with fish >340 mm long most affected (prevalence 64.7%). These data suggest emergence of the lesion is a recent phenomenon and that causative factors may be age related. Abnormal pigmentation lesions have only been observed to affect E. quoyanus and coral trout (Plectropomus leopardus; since 2010). Given the species affected and the currently unknown aetiology of these lesions, we name the condition serranid pigment abnormality syndrome (SPAS). Further research is required to determine its geographic distribution, establish causation and describe the course of disease in E. quoyanus.

Additional keywords: chromatophoroma, coral reefs, disease, health, Serranidae, teleost.

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Introduction

Baselines of ‘normal’ disease in wild fish populations remain elusive (Lafferty et al. 2004; Ward and Lafferty 2004), although it is often thought that emergence of new diseases may be an indicator of environmental stress (Harvell et al. 1999; Lafferty et al. 2004). It is well documented that coral reefs worldwide are under increased stress due to global warming (Couch et al. 2017; Hughes et al. 2017) and a range of anthropogenic stressors, including terrestrial run-off (McCulloch et al. 2003; Brodie et al. 2012; Wenger et al. 2016), overfishing (Cinner et al. 2009; Lamb et al. 2015) and tourism (Juhasz et al. 2010; Lamb and Willis 2011; Au et al. 2014). On the Great Barrier Reef (GBR), these stressors have mostly been documented to affect corals in the form of bleaching events (Hughes et al. 2017), outbreaks of crown of thorns starfish (Fabricius et al. 2010; Wooldridge et al. 2015) and increased prevalence of coral diseases (Chen et al. 2017; Pollock et al. 2017).

In contrast, relatively few reports of disease outbreaks in wild fish or shellfish are found in the scientific literature on coral reefs. In the Caribbean, an acute large-scale epizootic in sea urchins (Diadema antillarum; see Lessios et al. 1984; Lessios 1988) preceded phase shifts towards algal dominance in coral reefs in the early 1980s (Idjadi et al. 2010). Although a disease agent was never identified as the cause of the initial mortality event, a microbial pathogen was suspected (Bauer and Agerter 1987) and it has taken nearly 30 years before sea urchin populations (and corals) in the affected regions began to recover (Idjadi et al. 2010; Rodriguez-Barreras et al. 2015). Then, in 2000, spiny lobsters (Panulirus argus) in the Caribbean were found to be infected with a novel virus (Panulirus argus virus 1 (PaV1)) that caused disease and mortalities in juvenile lobsters (Behringer et al. 2011).

In teleosts, common naturally occurring parasites can occasionally cause disease in wild coral reef fish under certain conditions (Bunkley-Williams and Williams 1994; Landsberg 1995), whereas epizootics caused by bacterial disease agents, particularly Streptococcus iniae and Streptococcus agalactiae, have been reported in several countries (Ferguson et al. 2000; Bowater et al. 2012; Keirstead et al. 2014). Some populations of coral reef finfish near Hawaii have been described with skin diseases in the form of chromatophoromas (including melanomas) with unknown aetiology in butterflyfish (Okihiro 1988) and surgeonfish (Work and Aeby 2014), whereas bicolor damselfish (Stegastes partitus) from Florida are affected by an
infectious viral disease (neurofibromatosis) involving development of neurofibromas and chromatophoromas (Schmale et al. 2002).

Herein we report for the first time the emergence of a pigmentation abnormality in the wire netting cod or longfin grouper (*Epinephelus quoyanus*) near Heron Island on the southern GBR. We began to encounter abnormally pigmented *E. quoyanus* from 2012 onwards as an incidental finding during unrelated research that required capture and tagging of *E. quoyanus* and stripey snapper (*Lutjanus carponotatus*) following on from research conducted in the same area in the mid-1990s (Diggles and Ernst 1997). Our awareness of the historical lack of pigment lesions in *E. quoyanus* and the recent emergence (since 2010) of superficially similar pigment lesions in another member of the Family Serranidae, namely the coral trout (*Plectropomus leopardus*), from the same region (Sweet et al. 2012; Lerebours et al. 2016) prompted us to publish these observations. Due to their presently unknown aetiology, we name these abnormalities serranid pigment abnormality syndrome (SPAS). This paper details the emergence of SPAS in the *E. quoyanus* population near Heron Island (Qld, Australia), describes the prevalence and gross pathology of the lesion and outlines the research that is needed to establish a better understanding of its geographical distribution, the course of disease and the mechanism(s) responsible for its emergence.

### Materials and methods

The present study was conducted in the Scientific Research Zone and Conservation Park zones of the Great Barrier Reef Marine Park in the waters adjacent to Heron Island (23°15′54″S, 151°32′53″E) in the southern section of the GBR. Examinations of *E. quoyanus* (*n* = 189) were undertaken during five sampling trips between November 2010 and October 2017 (Table 1) and compared to historical data for *E. quoyanus* (*n* = 158) obtained in May 1996 during a previous study (Diggles and Ernst 1997). Fish were captured from both reef flat (water depth <2 m) and reef edge (water depths 2–10 m) environments using rod-and-reel methods and artificial lures with barbless hooks, as described previously (Diggles and Ernst 1997). The anatomical hooking location of each fish was noted, its total length (TL) was measured to the nearest millimetre and this was followed by examination of each fish for gross signs of SPAS lesions or other abnormalities. Each fish was then tagged with a plastic T-bar anchor tag with a unique identification number (TBA type; Hallprint, Hindmarsh Valley, SA, Australia) before being released at the site of capture. A smaller number of coral trout (*P. leopardus*; *n* = 60) were incidentally captured using similar methods in the same locations in 2016 and 2017 and were subjected to measurement and same examination before being released at the site of capture without tags. These activities were conducted under Great Barrier Reef Marine Park Authority permit numbers G08/24996.1, G011/34020.1, G13/35806.1 and G15/37589.1.

To determine whether SPAS-affected fish were significantly larger than unaffected fish, Student’s two-tailed *t*-tests were conducted on fish length using online statistical programs (http://vassarstats.net/, accessed 20 March 2018).

### Results

No pigment abnormalities were observed in any of the fish from the 1996, 2010 or 2011 studies, or in 42 of 43 fish examined in October and November 2012 (Fig. 1a). However, one fish (363 mm TL) captured on 1 November 2012 on the reef flat east of Heron Island was observed with conspicuous darkening and disorganisation of the brown ‘wire netting’ colouration pattern typical of this species over 100% of the head, 25–30% of the flanks and −5% of the dorsal fin (Fig. 1b). To the naked eye, the lesions appeared smooth without obvious hyperplasia. Therefore, the sample prevalence for SPAS lesions in October and November 2012 was 1/43 = 2.33%, and, for all *E. quoyanus* captured since 1996 until that time, 1/218 = 0.46%.

In October 2016, an additional 64 *E. quoyanus* were examined, of which 4 (prevalence 6.25%) exhibited similar abnormally darkened pigmentation lesions without obvious hyperplasia most commonly involving the lower jaw, premaxilla and head (data not shown). Then, in October 2017, a further 65 *E. quoyanus* were examined, of which 11 (prevalence 16.92%) exhibited SPAS lesions most commonly on the lower jaw (prevalence 63.63%, mean coverage 52.14%), premaxilla and head (prevalence 36.36%, mean coverage 100%) and mid-1990s (Diggles and Ernst 1997). Our awareness of the

### Table 1. Serranid pigment abnormality syndrome (SPAS) prevalence and other data from field sampling of *Epinephelus quoyanus* from Heron Island between May 1996 and October 2017

<table>
<thead>
<tr>
<th>Year</th>
<th>Month</th>
<th>Water temperature (°C)</th>
<th>Number of fish sampled</th>
<th>Mean TL (mm)</th>
<th>TL range (mm)</th>
<th>Number of fish ≥340 mm TL sampled</th>
<th>SPAS prevalence (%)</th>
<th>SPAS prevalence in fish ≥340 mm TL (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1996</td>
<td>May</td>
<td>23 ± 1</td>
<td>158</td>
<td>295.4</td>
<td>230–370</td>
<td>19</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2010</td>
<td>Nov.</td>
<td>25 ± 1</td>
<td>6</td>
<td>321.7</td>
<td>285–360</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2011</td>
<td>Oct.</td>
<td>24 ± 1</td>
<td>11</td>
<td>301.3</td>
<td>270–360</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2012</td>
<td>Oct.–Nov.</td>
<td>24 ± 1</td>
<td>43</td>
<td>306.4</td>
<td>172–370</td>
<td>11</td>
<td>2.33</td>
<td>9.1</td>
</tr>
<tr>
<td>2016</td>
<td>Oct.</td>
<td>24 ± 1</td>
<td>64</td>
<td>284.2</td>
<td>145–355</td>
<td>6</td>
<td>6.25</td>
<td>50</td>
</tr>
<tr>
<td>2017</td>
<td>Oct.</td>
<td>25 ± 1</td>
<td>65</td>
<td>306.8</td>
<td>210–380</td>
<td>17</td>
<td>16.92</td>
<td>64.7</td>
</tr>
<tr>
<td>2010–17</td>
<td>Oct.–Nov.</td>
<td>24–25</td>
<td>189</td>
<td>298.6</td>
<td>145–380</td>
<td>38</td>
<td>8.46</td>
<td>42.1</td>
</tr>
</tbody>
</table>
release, none of the SPAS-affected fish were subsequently recaptured during these sampling trips, hence information on whether the lesions observed are progressive or resolve over time is currently not available.

Only *E. quoyanus* $\geq 325$ mm TL captured on the reef flat exhibited SPAS lesions, with prevalence of lesions increasing markedly with fish size above this threshold (Table 3). Mean sample prevalence for pigmentation lesions for all *E. quoyanus* captured between November 2010 and October 2017 ($n = 189$) was 16/189 = 8.47% (Table 3), being highest in October 2017, with an overall prevalence of 16.92% and a prevalence of 64.7% in fish $\geq 340$ mm TL during that month (Table 1). The mean size of SPAS-affected *E. quoyanus* ($n = 16$; mean ± s.e.m. size 351 ± 12 mm TL, range 325–380 mm TL) was significantly greater than the mean size of unaffected fish ($n = 173$; mean ± s.e.m. size 293.8 ± 41.8 mm TL, range 145–370 mm; $P < 0.0001$, Students two-tailed $t$-test). It was not possible to weigh fish at the point of capture, but one of the SPAS-affected fish examined in October 2017 was recorded in field notes as being noticeably underweight compared with clinically normal fish of the same size (Table 2).

The only other species of fish around Heron Island observed with SPAS-like pigmentation lesions were coral trout (*P. leopardus*). In a sample of 21 *P. leopardus* captured in 2016, 2 were SPAS affected (overall prevalence 9.5%; Table 4), with both SPAS-affected fish being larger fish (>$600$ mm fork length (FL)) sampled from deeper waters ($>10$ m) from the channel between Heron and Wistari reefs ($n = 8$ fish sampled from the channel). In 2017, a sample of 39 *P. leopardus* captured from the channel, reef flat and reef edges near Heron Island revealed only 1 SPAS-affected fish (660 mm FL), which was caught and released from the reef edge (prevalence 2.56%; Fig. 4). The lesions on the coral trout were consistent in appearance with the melanised lesions reported by Sweet et al. (2012) to be present at a prevalence of 14.7% in the waters around Heron Island and nearby One Tree Island (Table 4).

Other than these two species, no abnormal pigmentation lesions were observed on any of the $>1000$ individual fish from $>17$ species captured and released by us during sampling trips.
from 2010 onwards, including Lutjanidae (e.g. *Lutjanus carponotatus* (*n* > 300) and *Lutjanus bohar* (*n* = 5)), Lethrinidae (e.g. *Lethrinus nebulosus* (*n* > 350), *Lethrinus miniatus* (*n* > 150), *Lethrinus atkinsoni* (*n* > 50) and *Gymnocranius audleyi* (*n* = 10)), Labridae (e.g. *Cheilinus trilobatus* (*n* > 20), *Choerodon cyanodus* (*n* > 20) and *Choerodon venustus* (*n* > 30)), Carangidae (e.g. *Gnathanodon speciosus* (*n* > 5) and *Caranx melampygus* (*n* = 1)) and other species of Serranidae (*Epinephelus fasciatus* (*n* > 50), *Plectropomus maculatus* (*n* > 5), *Epinephelus fusgoguttatus* (*n* = 2), *Epinephelus cyanopodus* (*n* = 2), *Cephalopholis miniata* (*n* = 5) and *Variola louti* (*n* = 3)).
Evidence from baseline studies conducted in the mid-1990s (Diggles and Ernst 1997), combined with observations of gross pathology from the fish examined here, confirm that the SPAS lesions in *E. quoyanus* at Heron Island are new, abnormal and may have emerged some time after 2010. No lesions were observed between 1996 and 2011, with the first lesion observed by us in 2012.

Abnormal skin pigmentation lesions involving melanisation were reported previously by Sweet et al. (2012) in specimens of another serranid, namely *P. leopardus*, sampled from near Heron and One Tree islands in the southern GBR since 2010. In that study, 20 fish of the 136 sampled were affected (prevalence 14.7%), with no apparent relationship between fish size and intensity of the pigmentation lesions, which appeared to have some characteristics of tumours composed of melanophore pigment cells (melanophoroma; see Sweet et al. 2012). We also observed superficially similar pigmentation lesions in *P. leopardus* (Fig. 4), but at a lower prevalence (9.5% in 2016, 2.5% in 2017). Because *P. leopardus* was not a target species for our studies, our capture methods and locations differed from those of Sweet et al. (2012) and, because we were not able to conduct pathological investigations to confirm lesion identity, our sample prevalences for *P. leopardus* for 2016 and 2017 are not directly comparable to their data from 2010 to 2012. Therefore, no conclusions should be drawn regarding possible changes in the prevalence of pigment lesions in *P. leopardus* over time based on our data.

We can confirm the pigmentation lesions we first observed in *E. quoyanus* in 2012 have increased in prevalence over time and show a strong positive relationship with fish size, with no fish <325 mm TL being affected and a high prevalence of lesions occurring in fish >340 mm TL (up to 64.7% in October 2017).

Sweet et al. (2012) reported that *P. leopardus* with skin lesions ‘struck fishing hooks as strongly as healthy individuals, appeared to have good muscle tone and were assessed by external examination as healthy aside from the skin discoloration’. The three affected *P. leopardus* examined by us in the present study also appeared, by gross observation and behaviour, to be in normal condition. Similarly, most SPAS-affected *E. quoyanus* captured in the present study appeared to have normal body condition, although one fish was in notably poor condition (based on visual assessment). It should be noted that capture methods used in the present study (line fishing with lures) could bias prevalence data (and the possible capture of any clinically affected fish) if SPAS-affected fish were more or less likely than unaffected fish to strike lures. However, because the same sampling methods were used for our original study (Diggles and Ernst 1997), their use here is entirely appropriate because it allows direct comparison of recent data with the original baseline data from 1996. Any future studies could examine the use of alternative or supplementary methods for examining lesion prevalence, such as underwater visual census.

The conditions of our research permit (for a different study) did not allow for destructive sampling of *E. quoyanus* for histopathological or other biological analysis (e.g. aging and sexing fish), hence the pathology of the lesion and risk factors remain to be determined. However, based on their darkened appearance, it is possible that increased melanin is a characteristic of the lesions in *E. quoyanus*, as was described for *P. leopardus* by Sweet et al. (2012) and Lerebours et al. (2016). Of the various pigment cell tumours in fish, melanophoromas (melanoma) are common relative to other types of tumours (Singaravel et al. 2017). Malignant neoplastic melanomas may be aggressively invasive and hyperplastic (Okihiro 1988; Okihiro et al. 1992; Raloff 2012; Work and Aeby 2014) compared with benign melanomas or preneoplastic melanosis, the latter being the most common skin pigmentation lesion recorded in redfish (*Sebastes mentella*) from the North Atlantic (Bogovski and Bakai 1989). The lesions in *E. quoyanus*, like those of *P. leopardus*, do not protrude from the skin surface and hence do not appear to be significantly invasive, hyperplastic or malignant; however, as discussed previously, further pathological characterisation is necessary. In any case, the emergence of pigment lesions in both *P. leopardus* and *E. quoyanus* (but apparently not other fish species) from the same region of the GBR at approximately the same time (2010–12), warrants further investigation to establish causation and examine the course of disease.

Because the lesions in *E. quoyanus* have only recently emerged, nothing is known in relation to risk factors that may contribute to their causation, except for fish length (and presumably age). Bogovski and Bakai (1989) found that melanosis and melanophoromas in *S. mentella* were more common in certain regions of the North Atlantic, were positively correlated with fish size (only occurring in fish >26 cm, with prevalence exceeding 50% in fish >40 cm) and exhibited variation between sexes. In that study, pigmented lesions occurred in females two-to-fivefold more often than in males, whereas in male fish 84% of pigment lesions occurred on the head and caudal fin (Bogovski and Bakai 1989). In *E. quoyanus* at Heron Island in October 2017, SPAS lesions were only observed on fish >340 mm TL, most commonly on the lower jaw (prevalence 63.63%) and head (prevalence 72.72%; Table 2). Exposure to
infectious diseases of fish, including neoplasms (Kinae et al. 1990). Recent water quality studies in Queensland have shown that biologically relevant reductions in water quality occur up to 100 km offshore during episodic flood events (Brodie et al. 2012). Heron Island is ~70 km offshore from mainland Queensland, and hence is within the range of exposure of flood plumes. However, Bogovski and Bakai (1989) sampled fish from the mid-Atlantic and suggested that melanised pigment lesions in S. mentella were not likely to be related to declines in water quality but due to aging-related factors (senescence) or reduced gene flow within certain fish populations. Reduced gene flow was also suggested by Sweet et al. (2012), although infectious aetiologies are also possible (Schmaele et al. 2002; Work and Aeby 2014).

Fish length (and possibly age) is an important risk factor for SPAS in E. quoyanus, because lesion prevalence increases markedly once total fish length exceeds 325 mm. The maximum size of E. quoyanus is between 380 and 400 mm (Fishbase 2017), but the minimum legal size of E. quoyanus in Queensland is 380 mm. Although the fish population sampled at Heron Island during the present study exists in a research zone where the taking of fish without scientific permits is prohibited, this species is effectively protected throughout Queensland by fisheries regulations, and it is possible that similar size and age structures of E. quoyanus populations will occur outside marine park no-take areas closed to fishing. The occurrence of SPAS lesions in E. quoyanus populations beyond Heron Reef would require surveys designed to elucidate the geographic extent of this syndrome.

Given that SPAS lesions are more prevalent in larger fish, it is possible that these lesions are gross signs of senescence or a progressive disease. Further research is required to describe the course of disease in E. quoyanus, to establish causation and ascertain whether the condition is infectious. This could be done in wild fish by sampling affected fish from various locations to obtain pathological samples, genetic information and age at length and sex data. Tagging and recapturing of affected fish would allow temporal monitoring of the extent of the lesions to determine whether they resolve or progress and increase in severity or the area of affected skin over time. However, if natural mortality rates or tag loss rates are high, such an approach may not yield enough information, which would necessitate bringing affected fish into captivity to monitor whether the disease progresses over time under controlled conditions.

Conflicts of interest
The authors declare that they have no conflicts of interest.

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References


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