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Prostaglandin A_2: an agent of chemical defense in the Caribbean gorgonian *Plexaura homomalla*

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ABSTRACT: The Caribbean gorgonian *Plexaura homomalla* contains levels of prostaglandin A_2 (PGA_2) which are 1 million times higher than those of most other organisms. In many sections of the Caribbean, *P. homomalla* produces 15(S)-PGA_2, which has the same configuration at carbon 15 as mammalian prostaglandins. 15(S)-PGA_2 possesses potent biological activity. In Florida and other areas, however, *P. homomalla* synthesizes large amounts of 15(R)-PGA_2, which has a different configuration at carbon 15. 15(R)-PGA_2 is inactive in several prostaglandin bioassays. The function of these compounds in the gorgonian is unknown. Both 15(R) and 15(S)-PGA_2 may serve, however, as a chemical defense against predatory fish. Laboratory studies demonstrate that oral doses of 15(R)-PGA_2 produce vomiting in killifish *Fundulus heteroclitus*. Killifish quickly learn to avoid food items containing 15(R)-PGA_2. Experiments performed underwater at Curaçao show that both 15(R) and 15(S)-PGA_2 produce vomiting in yellowhead wrasses *Halichoeres garnoti*. Wrasses also learn to avoid food which contains either 15(R) or 15(S)-PGA_2. Oral doses of prostaglandins produce nausea and vomiting in a wide range of organisms. Thus, the PGA_2 of *P. homomalla* probably can serve as an effective defense against most predatory reef fish.

INTRODUCTION

The Caribbean sea whip *Plexaura homomalla* (Coelenterata: Octocorallia: Gorgonacea) contains levels of prostaglandin A_2 (PGA_2) which are 1 million times higher than those of most other organisms. Prostaglandins – fatty-acid derivatives with highly potent, hormone-like activity – have been found in nanogram amounts in a great variety of animal tissues (Christ and van Dorp, 1972; Nomura and Ogata, 1976). In *P. homomalla*, however, PGA_2 (Fig. 1a) and its 15-acetate, methyl ester comprise 1 to 2 % of the gorgonian's wet tissue weight (Schneider et al., 1977). Levels as high as 5 to 8 % have been reported (Dominguez et al., 1980). Other Caribbean gorgonians do not contain large amounts of prostaglandins (Schneider et al., 1977). The function of these massive amounts of PGA_2 in *P. homomalla* is unknown.

Some colonial reef invertebrates may be chemically defended against predators (Vermeij, 1978; Bakus, 1981; Coll et al., 1982). Predation by fish in coral reef environments is intense (Bakus, 1981 and references therein). Gorgonians and other colonial marine organisms are common members of coral reef communities, but they are rarely eaten by fish (Randall, 1967; Bakus, 1981). Many gorgonians contain large amounts of chemical compounds which have no obvious function in the organisms which produce them (Ciereszko and Karns, 1973; Tursch et al., 1978; Fenical, 1982). Tissue extracts or purified compounds from gorgonians often

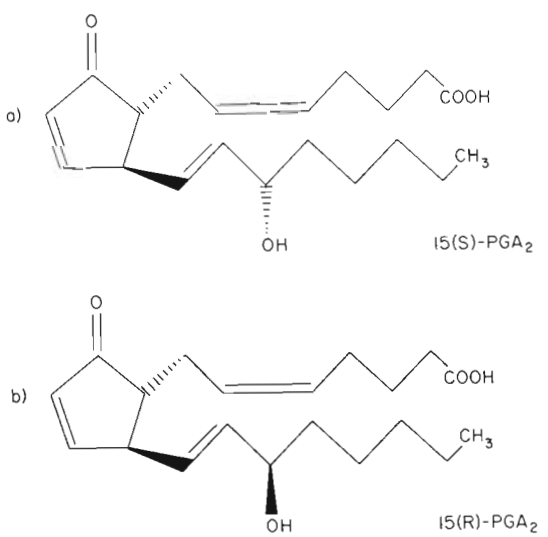

Fig. 1. (a) 15(S)-PGA_2; (b) 15(R)-PGA_2, which has a different configuration at carbon 15

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can kill or stun fish when applied externally in large amounts (Tursch et al., 1978; Bakus, 1981; Coll et al., 1982). There have been very few studies, however, on the effects of orally-delivered marine secondary compounds, even though the defensive chemicals of a gorgonian or other colonial marine invertebrate would presumably be delivered orally to predators. Lucas et al. (1979) noted that oral doses of starfish saponins, which are very toxic when added to water surrounding fish (Mackie et al., 1975), had no apparent effect when eaten, although they seemed to be distasteful to fish. Green (1977) force-fed small pieces of sponges to the labrid fish *Halichoeres bivittatus* and found that some sponge species were harmless, while others quickly killed the fish. Other studies have demonstrated that some marine natural products are distasteful, but have not reported adverse physiological effects produced by ingestion of the compounds (Tursch et al., 1978; Paul et al., 1980; Ayer and Anderson, 1982).

Prostaglandins can produce nausea, diarrhea, and vomiting when given orally to mammals (Lee et al., 1973; Smith and Mason, 1974). If oral doses of PGA_2 are also toxic to fish, then this compound could deter potential predators of *Plexaura homomalla*. In Florida and several other areas of the Caribbean, however, colonies of *P. homomalla* contain large amounts of the 15(R) isomer of PGA_2, and only traces of the more typical 15(S)-PGA_2 (Weinheimer and Spraggins, 1969; Light and Samuelsson, 1972; Schneider et al., 1977). 15(R)-PGA_2 has a different configuration at carbon 15 than 'mammalian' 15(S)-PGA_2 (Fig. 1), and is inactive in several prostaglandin bioassays. 15(S)-PGA_2 is highly active in the same assays (Nakano, 1969; Nakano and Kessinger, 1970; Spraggins, 1972). Both '15(R)' and '15(S)' variants of *P. homomalla,* however, appear to be equally immune to fish predation (Gerhart, own obs.). This implies that both PGA_2 isomers effectively reduce fish predation, or that the prostaglandins of *P. homomalla* are not important chemical defense agents.

To test the hypothesis that prostaglandin A_2 is used as a chemical defense in *Plexaura homomalla,* I performed experiments to determine the effects of orally-delivered 15(R) and 15(S)-PGA_2 on fish.

MATERIALS AND METHODS

Preliminary toxicology. I tested an aqueous extract of *Plexaura homomalla* tissue for toxicity, using a method employed by Bakus (1974, 1981). Colonies of *P. homomalla* were collected in the Florida Keys and kept frozen until use. A crude aqueous extract was prepared by homogenizing 5 g of frozen tissue in 20 ml of distilled water, and then centrifuging the homogenate for 15 min at 10,000 × g to remove spicules and fragments of tissue. The supernatant was decanted into a beaker containing 100 ml of fresh water. A goldfish was then added to the beaker, and the time until death of the fish was recorded. As a control, other goldfish were placed in 100 ml of fresh water containing no octocoral extract. In this experiment I made no effort to distinguish which toxins in the extract were responsible for the death of the fish.

Oral doses of gorgonian lipid extract to killifish: Pellet choice experiment. I performed this experiment to determine whether a lipid extract of *Plexaura homomalla* tissue had any adverse effects when delivered orally to fish. I collected colonies of *P. homomalla* from the Florida Keys and kept the tissue frozen until its use. The frozen tissue was extracted with 95 % ethanol, the ethanol was removed *in vacuo,* and the remaining aqueous slurry extracted with ethyl acetate. The organic phase was partitioned against 1N HCl and NaCl, and then dried over Na_2SO_4 and filtered. Removal of the solvent *in vacuo* gave a dark brown, viscous oil, which contained approximately 30 % 15(R)-PGA_2 by weight. This procedure was used by Schneider et al. (1977) as a preliminary step in the purification of PGA_2 from *P. homomalla* tissue.

I dissolved enough unflavored gelatin in distilled water to give a 6 % solution, added 0.10 g of the lipid extract to 10 ml of the gel, and stirred the mixture thoroughly to disperse the extract through the gel. I also added 0.25 g of commercial flaked fish food to the mixture to make it more appealing to fish. I pipetted 50 µl aliquots of the mixture into the wells of a plastic microtiter plate, and chilled the plate to set the gelatin. The aliquots were removed from the plate and quartered just before feeding them to fish. Each quarter of an aliquot was referred to as a 'pellet'. Thin-layer chromatography, using the A-IX solvent system of Hamberg and Samuelsson (1966), demonstrated that the pellet-making process did not cause conversion of the 15(R)-PGA_2 to the 15(S) form. The levels of lipid in these pellets were approximately 5 times lower, on a percentage weight basis, than the levels found in *Plexaura homomalla* tissue. I also made control pellets which contained no gorgonian lipid extract.

I presented a series of 12 control or 12 gorgonian extract pellets to killifish *Fundulus heteroclitus* which had been acclimated to 30 ‰ artificial seawater for at least 2 wk. Ten fish were used in each treatment. To reduce potential variation between replicates, only male fish (sexed by color pattern) collected from a single location in Stony Brook Harbor, New York, were used as test organisms. Fish were fed commercial flaked fish food until their use in the experiment. At the start of each trial of the experiment, a killifish was isolated in a 1 l glass aquarium and allowed to adjust

to its new surroundings for several minutes. Each fish was trained to eat gelatin by offering it 4 control pellets. The fish was then offered a series of 12 pellets containing lipid extract of *Plexaura homomalla*, or 12 control pellets. The fish was given 1 min to accept or reject each pellet. I recorded whether the fish ate the pellet, mouthed and rejected it, or ignored it. Rejected pellets were removed as quickly as possible to reduce any diffusion of octocoral toxins into the water. After offering each fish 12 gelatin pellets, I presented it with two small pieces of white shrimp to determine whether the fish had become satiated.

Oral doses of 15(R)-PGA_2 to killifish: (a) Pellet choice experiment. In this experiment I purified PGA_2 from the gorgonian lipid extract described above. (It should be noted that *P. homomalla* stores its prostaglandins as 15-acetate, methyl diesters, which may have different activity than the free acid. The gorgonian tissue contains an esterase, however, which hydrolyzes the esters when the tissue is disturbed [Schneider et al., 1977]. For this reason, I used the free acid in these experiments.) The purification was performed by 'flash chromatography' on a silica gel column (Still et al., 1978), employing the solvent conditions outlined by Schneider et al. (1977). Thin-layer chromatography (TLC) in the A-IX solvent system (Hamberg and Samuelsson, 1965) indicated that the isolated 15(R)-PGA_2 was very pure. The purified material was identified as 15(R)-PGA_2 on the basis of its physical characteristics, its TLC behavior relative to authentic 15(S)-PGA_2, and its IR and NMR spectral properties. No 15(S)-PGA_2 could by detected in the purified 15(R)-PGA_2 by TLC.

I added 20 mg of purified 15(R)-PGA_2 (delivered in 100 µl of ethanol) to 10 ml of 6 % gelatin (as prepared above), thus giving 0.1 mg of 15(R)-PGA_2 per 50 µl aliquot. I fed the fish a series of 12 pellets, using the procedure described in the preceding paragraph, to determine whether 15(R)-PGA_2 affected the acceptance rate of the pellets by fish. I then repeated the experiment, using control pellets which contained ethanol but no 15(R)-PGA_2.

Oral doses of 15(R)-PGA_2 to killifish: (b) Vomiting experiment. During the pellet choice experiment, I observed vomiting in one third of the fish which had ingested octocoral compounds. To pursue this observation further, I fed killifish doses of 0.1 to 0.2 mg of 15(R)-PGA_2 in gelatin, or an equivalent amount of control gelatin. Each fish was closely watched for 25 min after it swallowed the gelatin. I noted any unusual behavior of the fish during this time.

To determine whether any adverse reactions of the fish could be caused by chemicals leaking out of the pellets and into the water, I placed 8 killifish into individual beakers, each holding 200 ml of seawater containing 7.5 mg of 15(R)-PGA_2. This level of PGA_2 was much higher than could have possibly been reached during the vomiting experiment.

In situ feeding experiment in Curaçao. To determine whether coral reef fish react to oral doses of prostaglandins in the same way as killifish, I fed doses of 15(R) or 15(S)-PGA_2 to yellowhead wrasses *Halichoeres garnoti*. This experiment was performed underwater, using SCUBA, on a coral reef near the Caribbean Marine Biological Institute at Piscaderabaai, Curaçao. I isolated 15(S) and 15(R)-PGA_2 from colonies of *Plexaura homomalla* collected in the Cayman Islands and the Florida Keys, respectively, using the procedures described above; 10.0 mg of 15(S)-PGA_2 or 9.1 mg of 15(R)-PGA_2, delivered in 50 µl of methyl alcohol, was applied to pieces of fish. (The fish had been cooked thoroughly to denature any enzymes which could inactivate or modify the prostaglandins.) After application, I allowed the alcohol to evaporate from the pellet. Each 'pellet' was kept sealed in plastic wrap until it was offered to a wrasse. The dose of PGA_2 ingested by each fish was equivalent to consuming 0.5 to 1.0 g (wet weight) of *P. homomalla* tissue, since PGA_2 comprises at least 1 to 2 % of the wet weight of this gorgonian (Schneider et al., 1977; Dominguez et al., 1980). Since methyl alcohol has toxic properties, I also fed wrasses 'control pellets', which were made by applying 50 µl of methanol to cooked fish and allowing the alcohol to evaporate. I recorded whether each pellet was eaten, mouthed and rejected, or ignored by the yellowhead wrasses. If a pellet was eaten, I observed the wrasse constantly for 10 min to determine if it behaved unusually.

RESULTS

Preliminary toxicology. The aqueous homogenate of gorgonian tissue was toxic to goldfish. Fish exposed to the homogenate died in an average time of 95.9 ± 6.45 min (n = 10). All control fish (9 of 9) survived at least 46 h, at which time the experiment was terminated.

Oral doses of gorgonian lipid extract to killifish: Pellet choice experiment. Killifish showed a strong aversion to gelatin pellets containing gorgonian lipid extract, but readily ate control pellets which contained no octocoral compounds (Table 1a). This difference was highly significant (G test, $p < 0.005$).

The lipid extract of *Plexaura homomalla* seemed to be distasteful to fish, for the number of fish which ate the first lipid extract pellet offered to them (6 of 10) was lower than the number of fish ingesting the first control pellet (10 of 10). This difference was significant at $p < 0.025$ (G test).

The proportion of killifish eating the gorgonian lipid

Table 1. (a) Acceptance and rejection of gelatin food pellets containing a lipid extract of *Plexaura homomalla*. (b) Acceptance and rejection of gelatin food pellets containing purified 15 (R)–PGA_2. Ten killifish were used in each treatment. In both experiments, the pellets containing octocoral compounds were eaten less frequently than the control pellets (G test, $p < 0.005$)

	Eaten	Rejected
(a)		
Gorgonian lipid extract pellets	16	104
Control pellets	118	2
(b)		
15 (R)–PGA_2 pellets	32	88
Control pellets	119	1

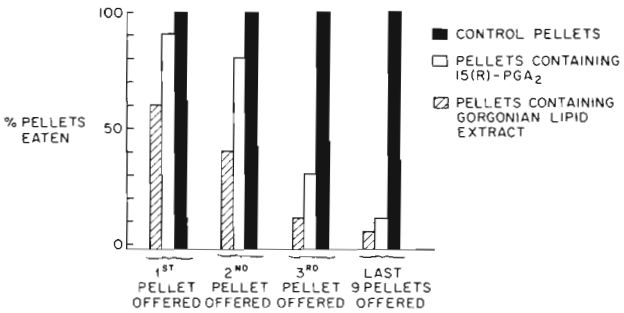

Fig. 2. Percentage of pellets eaten by fish as a function of time. Pellets were delivered in 1 min intervals

extract pellets decreased significantly as subsequent pellets were offered (G test, $0.05 > p > 0.025$; Fig. 2). This increased rate of rejection was not due to satiation of the fish, since rejection did not occur in fish presented with control pellets, and since fish which rejected gelatin pellets containing gorgonian compounds readily ate white shrimp at the end of the experiment (Table 2).

Oral doses of 15(R)-PGA_2 to killifish: (a) Pellet choice experiment. The results of these experiments were similar to those from experiments performed with gorgonian lipid extract. Killifish avoided pellets containing purified 15(R)-PGA_2 (Table 1b). The proportion of fish eating these pellets decreased significantly as subsequent pellets were offered (Fig. 2). The proportion of killifish which ate the first 15(R)-PGA_2 pellet offered to them (9 of 10), however, was not significantly different from the proportion of fish eating the first control pellet offered to them (10 of 10; G test, $p > 0.1$). Fish which rejected 15(R)-PGA_2 pellets still accepted white shrimp at the end of the experiment (Table 2).

Oral doses of 15(R)-PGA_2 to killifish: (b) Vomiting experiment. Killifish fed doses of 0.1 to 0.2 mg of 15(R)-PGA_2 behaved normally for 1 to 2 min, but then became hyperactive. Shortly thereafter, 80 % (8 of 10)

Table 2. (a) Numbers of white shrimp accepted and rejected by killifish at the end of the gorgonian extract pellet choice experiments. (b) Acceptance and rejection of white shrimp at the end of the 15 (R)–PGA_2 pellet choice experiments. In both experiments, the frequencies for the treatments are not significantly different from the control frequencies (G test, $p > 0.1$)

	# eaten	# mouthed and rejected	# ignored
(a)			
Fish offered:			
Control pellets	17	1	2
Extract pellets	16	0	4
(b)			
Fish offered:			
Control pellets	18	1	1
15 (R)–PGA_2 pellets	19	1	0

of the fish vomited a mass of gelatin fragments and stomach contents into the water. The average time to vomiting was 7.25 ± 1.47 min (n = 8). Fish fed an equivalent amount of control gelatin did not vomit (Table 3). In the control experiment, killifish placed in seawater containing 15(R)-PGA_2 did not vomit.

Table 3. Vomiting induced in killifish by oral doses of 15 (R)–PGA_2. Frequencies for each treatment are significantly different at $p < 0.005$ (G test)

	Number of fish vomiting	Not vomiting
Fish fed:		
15 (R)–PGA_2	8	2
Control gelatin	0	10

In situ feeding experiments in Curaçao. The results of these experiments paralleled the results obtained with killifish. Yellowhead wrasses which ingested 10 mg of 15(S)-PGA_2 all vomited within 10 min. Average time to vomiting was 1.52 ± 0.35 min (n = 9). None of the wrasses which ate control fish pellets vomited (Table 4). All wrasses which ingested 9.1 mg

Table 4. Vomiting induced in yellowhead wrasses by oral doses of 15 (R) and 15 (S)–PGA_2

	# wrasses vomiting	# not vomiting
Fish fed:		
15 (S)–PGA_2 (10.0 mg)	9	0
15 (R)–PGA_2 (9.1 mg)	4	0
Control pellets	0	9

of 15(R)-PGA_2 (4 of 4) also vomited, with an average time to vomiting of 3.55 ± 2.66 min (Table 4). Times to vomiting for the 15(R)-PGA_2 and the 15(S)-PGA_2 treatments were not significantly different (Student's *t*-test, $0.1 > p > 0.05$).

Wrasses showed a significant aversion to food pellets treated with 15(S) or 15(R)-PGA_2 (Table 5a). This was due to fish rejecting PGA_2 pellets offered later in the trial. In all feeding trials, the wrasses ate the first 3 PGA_2-containing pellets offered to them. Subsequent PGA_2 pellets, however, were almost always avoided (Table 5b). Control pellets were always eaten by the wrasses.

Table 5. (a) Acceptance and rejection of pellets offered to yellowhead wrasses. Frequencies for both treatments are significantly different from the control frequencies (G test, $p < 0.01$). (b) Acceptance and rejection of food pellets by yellowhead wrasses. Numbers are broken down to show the percent acceptance of the first 3 pellets offered in a trial, and to show the percent acceptance of pellets offered subsequent to the first 3. In parentheses: number of pellets eaten over the total number of pellets offered

	Eaten	Not Eaten
(a)		
Pellets containing:		
15 (S)–PGA_2	9	5
15 (R)–PGA_2	4	3
Control pellets	11	0
	% eaten, 1st 3 pellets in a trial	% eaten, subsequent pellets
(b)		
Pellets containing:		
15 (S)–PGA_2	100 % (8/8)	16 % (1/6)
15 (R)–PGA_2	100 % (3/3)	25 % (1/4)
Control pellets	100 % (9/9)	100 % (2/2)

The numbers of 15(S)-PGA_2 fish pellets which were eaten, mouthed and rejected, or ignored, were not significantly different from the numbers for 15(R)-PGA_2 fish pellets (G test, $p > 0.5$) (Table 5a).

DISCUSSION

The results presented in this paper demonstrate that oral doses of 15(R)-PGA_2, at levels which could easily be delivered to fish feeding on *Plexaura homomalla*, induce vomiting in killifish (Family Cyprinodontidae). Oral doses of both 15(R) and 15(S)-PGA_2, again at levels which would be ingested by potential predators, cause vomiting in yellowhead wrasses (Family Labridae). Killifish and yellowhead wrasses are not closely related, nor are they found in similar habitats. Yet, both species vomit when they ingest PGA_2. Prostaglandins also induce vomiting in several distantly related species of mammals (Smith and Mason, 1974; Robert and Yankee, 1975). Thus, prostaglandins have emetic properties in a very wide range of organisms. The 15(S) and 15(R)-PGA_2 found in *P. homomalla*, therefore, can probably produce vomiting not only in killifish and yellowhead wrasses, but in fish in general.

Table 6. Data from a typical run of the *in situ* experiment with yellowhead wrasses

Pellet #	Pellet type	Results
1	15 (S)–PGA_2	Eaten by a yellowhead wrasse, which vomited 4 times
2	Control	Eaten by a yellowhead wrasse. Fish showed no unusual behavior over the next 10 min
3	15 (S)–PGA_2	Eaten by a yellowhead wrasse, which vomited twice
4	Control	Eaten by a yellowhead wrasse. No unusual behavior
5	15 (S)–PGA_2	Eaten by a yellowhead wrasse, which vomited 3 times
6	Control	Eaten by a yellowhead wrasse. No unusual behavior
7	15 (S)–PGA_2	Mouthed and rejected a total of 3 times by various yellowhead wrasses
8	15 (S)–PGA_2	Mouthed and rejected by 1 yellowhead wrasse. Examined but not touched 6 times by various yellowheads
9	Control	Eaten by a yellowhead wrasse. No unusual behavior
10	15 (S)–PGA_2	Ignored by several yellowheads
11	15 (S)–PGA_2	Ignored
12	15 (S)–PGA_2	Ignored
13	Control	Eaten by a yellowhead wrasse. No unusual behavior

Other 15(R) prostaglandins are inactive when given intravenously, but active when given orally (Karim, 1973). This is due to acid-catalyzed conversion of the inactive (R) form to the active (S) form in low-pH gastric environments (Robert and Yankee, 1975). Killifish, however, possess basic intestinal tracts with a pH of about 8, and lack a stomach (Babkin and Bowie, 1928). Thus, since 15(R)-PGA_2 induces vomiting in killifish, acid-catalyzed conversion does not seem to be needed for this compound to be an effective emetic when given orally to fish.

Vomiting was produced in the fish by ingested PGA_2, and not by external PGA_2 in the surrounding water. This is demonstrated by the control experiments in the killifish vomiting experiment. In the *in situ*

experiment with yellowhead wrasses, the test fish was surrounded by an essentially infinite volume of water. Any PGA_2 leached from the pellet was diluted tremendously, making it highly unlikely that traces of external PGA_2 were responsible for the vomiting.

Both killifish and yellowhead wrasses began to reject food pellets which contained PGA_2. This rejection behavior was not due to satiation of the fish, because the fish continued to accept control pellets (see Fig. 2 and Table 6). Nor is this behavior due to unpleasant taste of the prostaglandins, for the first prostaglandin pellets offered to fish were nearly always accepted and eaten. Rather, this phenomenon seemed to be a learned aversion which developed because the fish were made ill by the PGA_2-containing food pellets. Yellowhead wrasses started to reject PGA_2-containing fish pellets after the third pellet offered in the trial. There were more wrasses in the study area than the 3 which ate the pellets. When a fish vomited up a pellet, however, other fish often came in and ate the vomited fragments. I did not observe vomiting in any of these fish, but I did not closely follow their behavior. Many of the fish in the area were thus exposed to PGA_2-containing food items after the first 3 pellets were eaten and vomited. The wrasses may have distinguished the PGA_2 pellets from the control pellets on the basis of color (PGA_2 pellets had a more yellowish tint), smell/taste, or a combination of these 2 characters.

Labrid fishes occasionally nip at gorgonians, including *Plexaura homomalla* (Randall, 1967; Kinzie, 1974; Gerhart, own obs.). This behavior may represent trial feedings on the gorgonian, or mucous feeding, or feeding on the epibenthic organisms occupying the colony. The stomachs of labrids, however, rarely contain gorgonian tissue. Labrids are omnivorous fish with dentition which allows them to feed on prey with hard skeletal elements (Randall, 1967). Thus, the spicules of gorgonians would probably not provide an effective defense against predation by these fish. Therefore, the protection provided by PGA_2 against predation by labrids may have ecological significance. Furthermore, a wide range of organisms learn to avoid prey which produce nausea and vomiting (Nicolaus et al., 1983 and references therein). Since prostaglandins probably can induce vomiting in many different species of fish, the PGA_2 of *P. homomalla* may provide this gorgonian with an effective chemical defense against a wide range of carnivorous reef fish.

Prior to this study, the observation that *Plexaura homomalla* contained large amounts of an apparently inactive prostaglandin derivative was perplexing. The results of these experiments, however, show that oral doses of either 15(R)-PGA_2 or 15(S)-PGA_2 can effectively discourage fish predation. This result supports the hypothesis that the PGA_2 derivatives of *P. homomalla* function as a chemical defense against predatory fish.

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LITERATURE CITED

Ayer, S. W., Anderson, R. J. (1982). Steroidal antifeedants from the dorid nudibranch *Aldisa sanguinea cooperi*. Tetrahedron Lett. 23: 1039–1042

Babkin, B. P., Bowie, D. J. (1928). The digestive system and its function in *Fundulus heteroclitus*. Biol. Bull. mar. biol. Lab., Woods Hole 54: 254–277

Bakus, G. J. (1974). Toxicity in holothurians: a geographical pattern. Biotropica 6: 229–236

Bakus, G. J. (1981). Chemical defense mechanisms on the Great Barrier Reef, Australia. Science, N.Y. 211: 497–499

Christ, E. J., van Dorp, D. A. (1972). Comparative aspects of prostaglandin biosynthesis in animal tissues. Biochem. biophys. Acta 270: 537–545

Ciereszko, L., Karns, T. K. B. (1973). Comparative biochemistry of coral reef coelenterates. In: Jones, O. A., Endean, R. (ed.) The biology and geology of coral reefs, Vol. II, Biology I. Academic Press, New York, p. 183–203

Coll, J. C., LaBarre, S., Sammarco, P. W., Williams, W. T., Bakus, G. J. (1982). Chemical defences in soft corals (Coelenterata: Octocorallia) of the Great Barrier Reef: a study of comparative toxicities. Mar. Ecol. Prog. Ser. 8: 271–278

Dominguez, J. N., Adams, D. R., Flamerich, J. (1980). Isolation of prostaglandins from the gorgonian *Plexaura homomalla* collected off the Venezuelan coast. Rev. Latinoam. Quim. 11: 56–58

Fenical, W. (1982). Natural products chemistry in the marine environment. Science, N.Y. 215: 923–928

Green, G. (1977). Ecology of toxicity in marine sponges. Mar Biol. 40: 207–215

Hamberg, M., Samuelsson, B. (1966). Prostaglandins in human seminal plasma. J. biol. Chem. 241: 257–263

Karim, S. M. M., Carter, D. C., Bhana, D., Ganesan, P. A. (1973). Effects of orally and intravenously administered prostaglandin 15(R)-15-methyl E_2 on gastric secretion in man. Adv. Biosci. 9: 255–264

Kinzie, R. A. (1974). *Plexaura homomalla:* the biology of a harvestable marine resource. In: Bayer, F. M., Weinheimer, A. J. (ed.) Prostaglandins from *Plexaura homomalla:* ecology, utilization and ecology of a major medical marine resource. University of Miami Press, Miami, p. 22–38

Lee, J. B., Cheng, W. D., Bianchi, R. G., Mollison, K., Hansen, J. (1973). Effects of oral administration of PGE_2 on gastric

secretion and experimental peptic ulceration. Prostaglandins 3: 29–45

Light, R. J., Samuelsson, B. (1972). Identification of prostaglandins in the gorgonian *Plexaura homomalla*. Eur. J. Biochem. 28: 232–240

Lucas, J. S., Hart, R. J., Howden, M. E., Salathe, R. (1979). Saponins in eggs and larvae of *Acanthaster planci* (L.) (Asteroidea) as chemical defences against planktivorous fish. J. exp. mar. Biol. Ecol. 40: 155–165

Mackie, A. M., Singh, H. T., Fletcher, T. C. (1975). Studies on the cytolytic effects of seastar *(Marthasterias glacialis)* saponins and synthetic surfactants in the plaice *Pleuronectes platessa*. Mar. Biol. 29: 307–314

Nakano, J. (1969). Cardiovascular effect of a prostaglandin isolated from a gorgonian *Plexaura homomalla*. J. Pharmacol. 21: 782–783

Nakano, J., Kessinger, J. M. (1970). Effect of 15(R)-PGA_2 on the cardiovascular responses to PGA_2 in dogs. Clin. Res. 18: 117

Nicolaus, L. K., Cassel, J. F., Carlson, R. B., Gustavson, C. R. (1983). Taste-aversion conditioning of crows to control predation on eggs. Science, N.Y. 220: 212–214

Nomura, T., Ogata, H. (1976). Distribution of prostaglandins in the animal kingdom. Biochem. Biophys. Acta 431: 127–131

Paul, V. J., McConnell, O. J., Fenical, W. (1980). Cyclic monoterpenoid feeding deterrents from the red marine alga *Ochtodes crockeri*. J. org. Chem. 45: 3401–3407

Randall, J. E. (1967). Food habits of reef fishes of the West Indies. Stud. Trop. Oceanogr. 5: 665–847

Robert, A., Yankee, E. W. (1975). Gastric antisecretory effect of 15(R)-15-methyl PGE_2, methyl ester (38707). Proc. Soc. exp. Biol. Med. 148: 1155–1158

Schneider, W. P., Bundy, G. L., Lincoln, F. H., Daniels, E. G., Pike, J. E. (1977). Isolation and chemical conversions of prostaglandins from *Plexaura homomalla:* preparation of prostaglandin E_2, prostaglandin $F_{2\alpha}$, and their 5,6-trans isomers. J. Am. chem. Soc. 99: 1222–1232

Smith, E. R., Mason, M. M. (1974). Toxicology of the prostaglandins. Prostaglandins 7: 247–268

Spraggins, R. L. (1972). PGA_2 and isomers from coral prostaglandins. Tetrahedron Lett. 42: 4343–4346

Still, W. C., Kahn, M., Mitra, A. (1978). Rapid chromatographic technique for preparative separations with moderate resolution. J. org. Chem. 43: 2923–2925

Tursch, B., Braekman, J. C., Daloze, D., Kaisin, M. (1978). Terpenoids from coelenterates. In: Scheuer, P. J. (ed.) Marine natural products, Vol. II. Academic Press, New York, p. 247–296

Vermeij, G. J. (1978). Biogeography and adaptation: patterns of marine life. Harvard University Press, Cambridge, Mass., p. 90

Weinheimer, A. J., Spraggins, R. L. (1969). The occurrence of two new prostaglandin derivatives (15-epi-PGA_2 and its acetate, methyl ester) in the gorgonian *Plexaura homomalla*. Chemistry of coelenterates XV. Tetrahedron Lett. 59: 5185–5188

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