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Distribution of bitter crab dinoflagellate syndrome in southeast Alaskan Tanner crabs *Chionoecetes bairdi*

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ABSTRACT: The distribution of bitter crab syndrome in southeast Alaskan Tanner crabs *Chionoecetes bairdi* was determined from population surveys conducted by the Alaska Department of Fish and Game and commercial catch census data provided by seafood processors and fishermen. Both sources of data indicate the disease syndrome is widespread, affecting 1/3 of the total subdistricts fished during the 1988/1989 season, with the upper Lynn Canal areas having the highest prevalences (up to 95 %). A conservative estimate of the total economic loss to fishermen due to rejected diseased crabs was about $220 500, representing 5 % of the total catch for the season. Disease prevalence in male and female crabs was nearly equal, with a significantly higher prevalence found in crabs of both sexes that have molted within the year (newshell). The latter finding was new information suggesting further considerations regarding the seasonality and transmission of the parasite. Increasing prevalences and spread of the disease syndrome to new areas is suggested by the commercial data but needs further verification from additional population surveys. Diseased crabs are known to have been introduced into some areas through improper disposal by processors and fishermen. Management of bitter crab syndrome may be possible by harvesting crabs earlier in the year when fewer crabs are severely parasitized and meats are more marketable. Promotion of increased public awareness of the disease syndrome and proper disposal of infected crabs are also necessary components in controlling dissemination of this parasite.

INTRODUCTION

Bitter crab syndrome in Tanner crabs *Chionoecetes bairdi* and *C. opilio* is caused by a *Hematodinium*-like dinoflagellate organism that infects the hemolymph and subsequently invades all host tissues (Meyers et al. 1987, Meyers 1990). The parasite has a vegetative stage, occurring within the host, that sporulates into one of 2 types of motile dinospores which escape into the environment during morbidity and after crab death.

External gross signs of the disease syndrome include abnormally bright pink coloration of the carapace, abdomen and intersegmental membranes of the walking legs with light-colored streaks along the mid-ventral aspect of the merus leg sections. Internal signs consist of opaque hemolymph and milky emaciated musculature in uncooked crabs. Cooked meats from infected crabs characteristically elicit a chalky texture with an astringent after-taste. Southeast Alaskan Tanner crab fishermen noticed many of these signs as early as 1974, but attributed them to such other causes as a premolt condition. The dinoflagellate was not identified as the causative agent of these gross signs until 1986 (Meyers et al. 1987). Subsequently, the parasite has been discovered in *Chionoecetes opilio* from the Bering Sea (Meyers 1990).

Bitter crab syndrome is economically and biologically significant. Tanner crabs in the advanced stages of infection are harmless to humans but unsuitable for human consumption because of poor meat texture and the aforementioned bitter after-taste, hence the name 'bitter crab'. Unmarketability of infected crabs causes direct economic losses to both fishermen and processors. All sizes and both sexes of crabs are susceptible to

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the disease which is ultimately fatal. Consequently, the resulting mortality from this disease syndrome threatens the future recruitment and viability of susceptible Tanner crab populations.

Since 1986 the Commercial Fisheries Division of the Alaska Department of Fish and Game (ADF&G) has collected hemolymph samples from *Chionoecetes bairdi* caught during red king crab (*Paralithodes camtschatica*) stock assessment surveys. Other hemolymph samples were taken during test fishing surveys designed specifically to collect Tanner crabs and from crabs caught during regular commercial seasons. Additional distribution data have originated from catch census information provided by fishermen and processors. Diagnostic evaluation of all stained hemolymph smears has been conducted by the ADF&G Fish Pathology Section within the Fisheries Rehabilitation Enhancement and Development (FRED) Division. These hemolymph samples, in conjunction with the interview information provided by fishermen and processors, define the known distribution and prevalence of bitter crab syndrome in southeast Alaska. The purpose of obtaining these data is to provide the basis for development of proactive resource management strategies intended to minimize economic losses due to infected crabs and discourage the dissemination of the disease through unwise fishing and processing practices.

MATERIALS AND METHODS

Systematic population surveys in 0.5 km grid patterns were conducted within various harvest areas using 20 commercial king-crab pots as described by Meyers et al. (1987). Pots baited with whole herring were set at individual depths ranging from 22 to 192 m with fishing times of 15 to 22 h. Average depth per sample site ranged from 26 to 123 m. Capture of Tanner crabs in this manner allowed various size classes and both sexes to be represented and exact determination of parasite prevalence per area sampled. The 18 March 1987 sample at Site 5 (Sullivan Island) was collected by a 12-m-wide beam trawl used for fishing shrimp. Five separate tows were made with bottom times of 20 to 60 min at a speed of 1.5 knots. Infected crabs from all sites were identified by stained hemolymph smears prepared and examined for the bitter crab dinoflagellate according to the methods described by Meyers et al. (1987). An alternate method of smear preparation consisted of expressing a drop of hemolymph onto a slide from a disjointed crab leg rather than using a needle and syringe.

The total numbers of crabs examined and those parasitized from all positive sites were added separately for statistical comparisons of parasite prevalences in male and female crabs as well as in new and oldshell individuals. Significance was determined using the arcsine transformation of proportions and test of equality of percentages for determining adequate sample sizes (Sokal & Rohlf 1969). Newshell crabs were those judged to have molted within a year as determined by examination of the carapace showing lighter color, more iridescence, minimum hardness and lack of epibiont organisms giving a clean appearance to the shell surface. Oldshell crabs had not molted within the year having much darker colored, harder shells often with attached fouling organisms.

Catch census data provided by commercial fishermen and information supplied by the processors for the 1988/1989 fishing season provided overall estimates of the economic losses to the industry from infected crabs that were rejected. In addition, these data provided a second source of bitter crab distribution information. The raw data consisted of kg of crab delivered and kg rejected (deadloss) by processors, the latter consisting of dead crabs and those with other gross signs of bitter crab syndrome. Because the disease syndrome severely debilitates the crab hosts, the primary gross sign observed by processors is often excessive mortality within the live wells of crab vessels. All rejected crabs were presented as percent deadloss per specific fishing subdistrict. Only those subdistricts producing bitter crabs detected by gross examination and/or having dead-losses of ≥ 454 kg were included as positive sites. This selected limit for deadloss was considered excessive to normal handling mortality, yet conservative regarding distribution of parasitized crabs because a low prevalence of the disease within a single harvest of crabs or previous culling of infected crabs by fishermen may not result in high mortality at delivery. Catch census data relate only to male legal-sized crabs ≥ 140 mm in carapace width. Collectively these data are also conservative regarding occurrence of bitter crabs, again because of the culling procedure above and not all deadlosses are fully reported by fishermen. The greater awareness of the disease problem by the industry in 1988/1989 initiated more rigorous sorting of infected crabs by fishermen and processors than in past seasons.

RESULTS

Disease prevalence data for locations examined in the ADF&G population surveys are presented in Table 1 according to total number of crabs sampled, sex and shell condition. Fig. 1 illustrates the location of each site examined. In several samples the prevalence data are based upon a smaller subset of those crabs examined for carapace-width statistics, as noted in the table footnotes. Previous data have shown no significant differences in dinoflagellate prevalence with depth of pot set,

Table 1. *Chionoecetes bairdi*. Prevalences of bitter crab syndrome in populations from various locations in commercial fishing subdistricts of southeast Alaska as determined by hemolymph smears collected by the Alaska Department of Fish and Game from 1986 through 1989. Locations (and area by statistical code) examined include 1: Icy Bay (186); 2: Glacier Bay (114–75); 3: Lutak Inlet (115–33); 4: Chilkoot Inlet (115–34); 5: Sullivan Island (115–31); 6: St. James Bay/Lynn Sisters (115–10); 7: Eagle River (111–50); 8: Excursion Inlet (114–80); 9: Barlow Cove (111–50); 10: Point Hilda (111–40); 11: Port Frederick (114–31, 33, 34); 12: Deadman Reach (113–55); 13: Seymour Canal (111–11, 14); 14: Gambier Bay (110–23); 15: Pybus Bay (110–22); 16: Port Camden (109–43); 17: Duncan Canal (106–43). (–) Data not examined

Date	Site	Total[a]	(%)	Male[b]	Female	Newshell	Oldshell
11 Mar 1987	1	0/50	(0)	0/50 (144 ± 4.0)[b]	–	0/50	–
31 Jan 1987	2	0/41	(0)	0/41 (149 ± 5.1)[c]	–	0/33	0/8
31 Jan 1987	3	37/46	(80)	37/46 (143 ± 16.7)	–	27/29	10/17
31 Jan 1987	4	11/45	(24)	10/29 (151 + 16.8)	1/16 (105 ± 9.4)	6/10	5/35
01 Jul 1986	5	141/149	(95)	84/90 (134 + 20.3)[d]	57/59 (93 ± 8.3)[e]	138/145	3/4
18 Mar 1987	5	17/46	(37)	5/19 (101 + 32.3)	12/27 (74 + 23.6)	14/41	3/5
15 Oct 1987	5	41/69	(59)	39/62 (127 + 14.2)[f]	2/7 (95 + 12.7)	39/53	2/16
08 Jun 1988	5	280/365	(77)	238/314 (138 + 17.4)[g]	42/51 (95 + 8.4)[h]	–	–
05 Oct 1988	6	13/100	(13)	12/81 (142 + 21.4)	1/19 (94 + 6.9)	12/80	1/20
19 Jan 1987	7	0/50	(0)	0/50 (156 + 9.0)[i]	–	0/45	0/5
24 Jul 1987	7	11/88	(13)	11/82 (145 + 13.2)[j]	0/6 (100 + 6.5)	11/55	0/33
30 Sep 1988	7	18/100	(18)	15/90 (145 + 15.6)	3/10 (88 + 13.4)	18/83	0/17
22 Jul 1987	8	0/91	(0)	0/65 (136 + 18.6)[k]	0/26 (97 + 7.2)	0/38	0/53
07 Oct 1988	8	5/90	(6)	2/67 (152 + 19.2)[l]	3/23 (97 + 9.1)	5/61	0/29
23 Jul 1987	9	9/100	(9)	8/84 (132 + 16.5)	1/16 (93 + 9.0)	8/79	1/21
29 Sep 1988	9	3/100	(3)	1/81 (136 + 14.1)	2/19 (98 + 7.4)	3/65	0/35
19 Jan 1987	10	5/50	(10)	5/50 (155 + 10.6)[m]	–	5/46	0/4
09 Oct 1988	11	5/100	(5)	4/87 (131 + 17.8)	1/13 (96 + 9.3)	5/84	0/16
18 Jul 1987	12	11/93	(12)	6/65 (128 + 19.1)[n]	5/28 (92 + 10.4)[o]	9/36	2/57
13 Oct 1988	12	2/100	(2)	1/56 (128 + 13.8)	1/44 (94 + 6.8)	2/20	0/80
26 Sep 1988	13	0/100	(0)	0/77 (144 ± 21.7)	0/23 (104 ± 8.6)	0/68	0/32
23 Sep 1988	14	12/100	(12)	12/71 (144 ± 17.8)	0/29 (96 ± 8.2)	9/60	3/40

Table 1 (Continued)

Date	Site	Total[a]	(%)	Male[b]	Female	Newshell	Oldshell
04 Feb 1987	15	0/49	(0)	0/49 (154 ± 9.1)	–	0/40	0/9
14 Jul 1987	15	15/80	(19)	14/75 (128 ± 20.0)[p]	1/5 (103 ± 9.0)[q]	15/60	0/20
21 Sep 1988	15	0/100	(0)	0/82 (138 ± 16.5)	0/18 (98 ± 10.7)	0/78	0/22
09 Feb 1987	16	0/50	(0)	0/50 (148 ± 6.0)[r]	–	0/40	0/10
06 Jun 1989	17	0/98	(0)	0/92 (125 ± 11.9)[s]	0/6 (105 ± 7.5)	0/78	0/20

[a] No. infected/No. examined

[b] Average carapace width in mm and standard deviation based upon a total of 120 male crabs measured; [c] 123 males; [d] 97 males; [e] 64 females; [f] 66 males; [g] 312 males; [h] 59 females; [i] 124 males; [j] 94 males; [k] 74 males; [l] 77 males; [m] 149 males; [n] 69 males; [o] 31 females; [p] 84 males; [q] 7 females; [r] 101 males; [s] 94 males. Unless otherwise noted, all SD derived from total crabs examined for prevalence

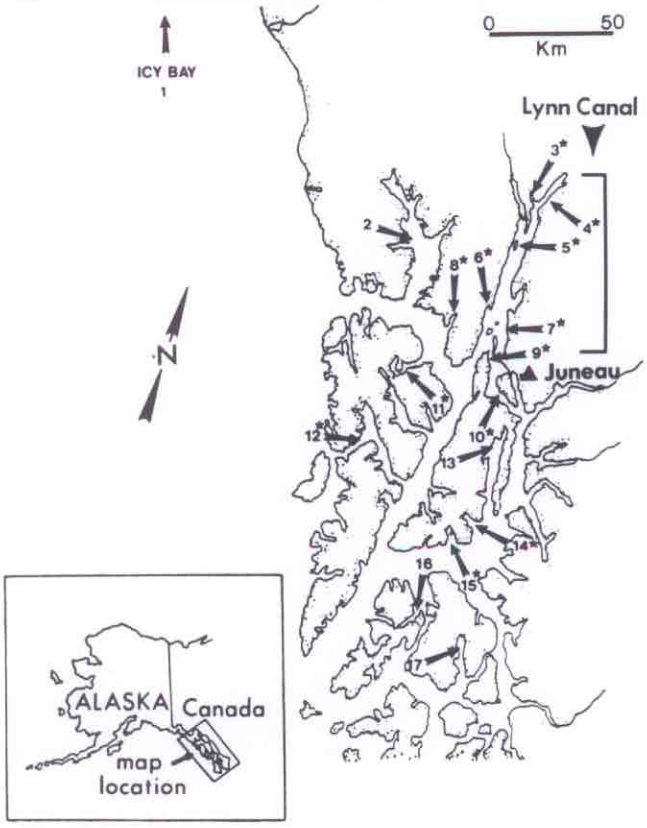

Fig. 1. Site locations in southeast Alaska, as numbered in Table 1, where the causative organism of bitter crab syndrome has been found in hemolymph smears from *Chionoecetes bairdi* collected by the Alaska Department of Fish and Game during 1986 through 1989. Numbered areas with crabs positive for the disease are accompanied by an asterisk

between sexes and among size classes of *Chionoecetes bairdi* crabs greater than 70 mm carapace width (Meyers et al. 1987). Again in this study, the parasite prevalences in male (34.4 %) and female (35.5 %) crabs from positive sites were not significantly different. However, differences in parasite prevalence in new (32.4 %) vs old (6.7 %) shell crabs from infected areas were significant at the 99 percentile with a probability of $p = 0.001$. Sample sizes were statistically valid.

Table 2 presents the various commercial subdistricts found to be positive for bitter crabs based upon the commercial catch census data. Fig. 2 illustrates the locations of only the positive areas. In the 1988/1989 season 6 districts from a total of 9 had subdistricts with harvested bitter crab. More specifically, 20 of the 60 subdistricts fished were positive for bitter crab syndrome. In most cases grossly diseased crabs were observed to be associated with the excessive deadlosses. Four sites yielded harvests with no dead crabs or considerably less deadloss than the limit considered excessive, but were still found to have some infected crabs after examination. These harvests of crab may reflext a low prevalence of the disease or the efficiency of some fishermen in culling diseased crabs. The total Tanner crab harvest during the 1988/1989 season in southeast Alaska was 737 006 kg of which a conservative estimate of 36 372 kg (4.9 %) were rejected mostly as bitter crabs. This total weight of rejected crabs is slightly more than the sum of rejected weight in Table 2 because deadlosses less than 454 kg were also included. The wholesale value of the crabs rejected was \$220 514 (at \$6.06 kg^{-1}).

DISCUSSION

The distribution of bitter crab syndrome appears similar in both data sets and the numerous positive sites indicate that parasite occurrence is widespread. In

Table 2. *Chionoecetes bairdi*. Commercial fishing subdistricts (and area codes) in southeast Alaska where bitter crab syndrome was detected in legal-sized male crabs during the 1988/1989 season. Positive sites were determined primarily by observation of grossly diseased crabs, but in some cases by excessive deadlosses ≥ 454 kg in delivered harvests of crabs. Occurrence of bitter crab by taste testing of cooked crabs purchased by one processor in 1986 is also noted by subdistrict

Location (Area code)	Deadloss[a] (%)	1986 Bitter flavor
Burro Ck (115–35)	48.0 (564/1174)	NA[b]
Chilkoot Inlet (115–34)	33.7 (2310/6861)	Yes[c]
Chilkat Inlet (115–32)	44.0 (1664/3779)	Yes[c]
Sullivan Island (115–31)	36.3 (6312/17 412)	Yes
Berners Bay (115–20)	2.5 (119/4844)	NA
St. James Bay (115–10)	3.2 (453/13 993)	Yes
Little and Shelter Islands, Auke Bay, Fritz Cove (111–50)	3.2 (1479/46 324)	NA
North Stephens Passage Greens Cove to Pt. Hilda (111–40)	6.7 (5675/84 889)	NA
Taku Inlet (111–32)	10.2 (2385/23 476)	NA
Middle Stephens Passage (111–31)	7.6 (841/11 034)	NA
Gilbert Bay (111–35)	2.8 (46/1652)	NA
South Stephens Passage (111–20)	NA	Yes
Tracy and Endicott Arms (111–21)	8.1 (2701/33 284)	NA
Gambier Bay (110–23)	1.8 (507/28 663)	NA
Pybus Bay (110–22)	1.9 (919/47 319)	NA
Farragut River (110–14)	6.4 (669/10 500)	NA
Thomas Bay (110–12)	8.6 (2467/28 548)	NA
Wrangell Narrows (106–44)	10.7 (685/6424)	NA
Wrangell (108–40)	19.7 (3480/17 634)	NA
Pt. Sophia/Icy Strait (114–27)	0.0 (0/12 300)	Yes
Port Frederick (114–31, 33, 34)	NA	Yes
Cross Sound (114–21)	1.6 (55/3514)	Yes

[a] kg deadloss per kg delivered crab
[b] NA: data not available
[c] Bitter crab also detected in 1985 from these areas including Lutak Inlet (115–33)

1988/1989, 1/3 of the commercially fished areas were found to be positive for bitter crabs. Examinations by the ADF&G in 1987 and 1988 extended the range of parasitism to 2 additional subdistricts (Excursion Inlet and Deadman Reach) that were fished in 1988/1989 with no bitter crabs evident in the commercial catch census data. Clearly, both the ADF&G and commercial data showed that the highest prevalences of the disease occur in upper Lynn Canal beginning at Sullivan Island and extending northward to the canal terminus at Skagway.

This high prevalence may be due to increased exposure to the parasite when crabs inhabit confined areas such as embayments or sites where less tidal or current flushing may occur. Another contributing practice is the harvest of only healthy crabs with rejection of bitter crabs in the more intensely fished upper Lynn Canal areas which may have concentrated the numbers of diseased crabs within certain populations. Finally, annual fishing effort of such high intensity may have stressed these Tanner crab stocks overall, further exacerbating the disease problem.

In those sites in Table 1 where multiple monthly sampling has been done during the same and separate years, (i.e. Sullivan Island) the prevalence data continued to suggest a yearly cycle for the disease syndrome as discussed in previous studies (Meyers et al. 1987). However, the true seasonality of the bitter crab agent needs to be confirmed by rigorous monthly hemolymph sampling of crabs for study of prevalence and parasite life stages at a single positive site through the course of a year or more. As observed before, the prevalences and intensities of the parasite in this study tended to be highest during July through October, represented by prespores and spores as well as vegetative stages. Sporulation is the end stage of infection that kills the crab hosts releasing the putatively infectious spore stages to infect new crab hosts (Meyers et al. 1987). As this occurs, the prevalence may drop off or be eclipsed within an area until the new infections can build up to the detectable levels seen in the late winter and early spring (Meyers et al. 1987).

However, new data in this study justify reconsideration of the parasite's seasonality and mode of transmission as previously proposed. The significantly higher disease prevalence in newshell crabs suggests that the molting process may be a predisposing factor for the infection of crab hosts by the parasite. The loss of the

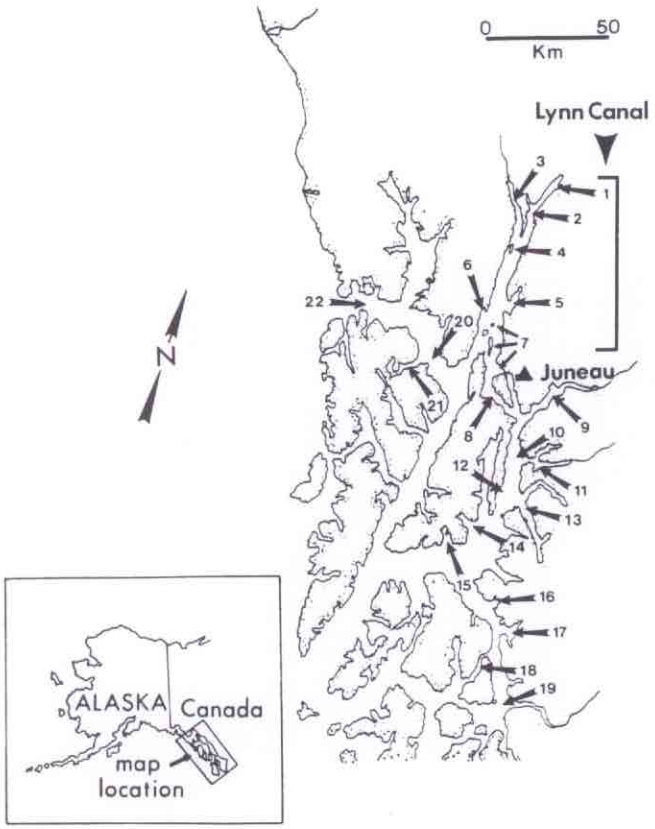

Fig. 2. Site locations in southeast Alaska, as numbered in Table 2, where bitter crab syndrome occurs according to commercial catch census data. Only positive areas appear on the map

hard shell barrier both externally and internally for a short period after molting could allow infectious stages of the parasite easier access to the internal tissues of the crab host through minor breaks in the cuticle. Another possible route of transmission is by cannibalism. Tanner crabs would also be especially vulnerable to cannibalism during the molting cycle. Both modes of transmission have been postulated for the amoebic pathogen *Paramoeba perniciosa* causing gray crab disease in the blue crab *Callinectes sapidus* from the Atlantic and Gulf coasts of the United States (Couch & Martin 1982, Couch 1983).

Should the parasite spores be considered the infectious stage for the bitter crab syndrome then there is a major conflict within the theory of transmission during host ecdysis. Spore formation and the molting cycle of the host would have to be synchronous for this to be true. This is not the case since the molting of Tanner crabs in southeast Alaska begins in mid-March with most occurring in April. This is at least 3 mo earlier than when the supposedly infectious spore stages are known to develop. Laboratory studies further suggest that at least one of the spore types is not infectious. Vegetative stages that sporulated into the small spore type 63 d post-injection in 2 Tanner crabs failed to produce a detectable infection after 214 d (Meyers et al. 1987). A more plausible argument is that the vegetative stages may be responsible for parasite transmission primarily during molting of the host crab. Although the non-motile vegetative stages are only passively infectious, it may be possible for such stages to be released in large numbers from an infected crab as well as gain access to a new crab host during or immediately after molting. Vegetative stages remain viable in seawater for at least 5 d and probably longer (Meyers et al. 1987). If transmission of vegetative stages were a major occurrence in the spring then the disease would not likely have time to progress to sporulation by late summer or early fall. This premise is supported by the fact that when massive numbers of vegetative stages are injected into crabs, it takes up to 3 mo for the infection to become detectable (Meyers et al. 1987). Infective doses in nature are not likely to approach the 5 to 6 $\log_{10}$ concentrations of the organism used experimentally. Thus, replication of the parasite to detectable levels in naturally infected crabs may take many months more than seen in the laboratory. Consequently, those crabs sustaining parasite sporulation in the fall may have been infected during the spring of the previous year with the pathogenesis of the disease taking approximately 15 to 18 mo rather than 12. This would also explain some of the overlap of moderately infected crabs seen in October which are too far along in disease progression to have been infected in August but do not have infection intensities high enough to sporulate (Meyers unpubl.).

Should this speculation be considered further, the function of the parasite spores would need to be re-evaluated. Perhaps the motile spores are not infectious but rather serve a resting or disseminatory function for the parasite. The question of why 2 spore types occur is still an enigma regardless of their purpose. The 2 spore types are greatly different regarding size, internal structure and behavior (Meyers et al. 1987) which might be adaptive mechanisms for parasite survival when certain environmental conditions favor one spore type over the other. There is still a possibility that the different spores represent 2 different parasite species but this would be unlike the parasitic dinoflagellates reported to cause pandalid embryo peridinienosis (Stickney 1978, Holmes et al. 1980, J. Hibbits & D. Porter unpubl.) and syndiniasis of radiolarians (Hollande 1974). There is also some evidence to suggest that the different spores may not represent separate sexes (W. Eaton unpubl.). Again, additional work on parasite transmission must be done to fully unravel the true nature of this dinoflagellate life cycle.

Bitter crab syndrome is reported by fishermen and processors to be either spreading to uninfected crabs in

other areas or increasing in prevalence in certain areas. Although evidence of increasing disease dissemination cannot be determined from the data in this report, catch statistics suggest this to be occurring. The percent of unmarketable *Chionoecetes bairdi* Tanner crabs within the yearly total catches in southeast Alaska has steadily increased from 0.05 to 5.2% from 1985/1986 to 1988/1989, respectively (ADF&G unpubl. data). Whether this increase in rejected crabs is due to better recognition and reporting or spread of the disease remains to be determined. Continued yearly sampling from certain areas with and without diseased crabs would establish if the parasite is increasing in prevalence or extending its range to other crab populations. It is known that in the past 2 yr since crabs have been actively sorted for this disease, infected specimens have been thrown back into seawater by fishermen and processors in areas geographically far removed from where the crabs were harvested. In these areas increased exposure of resident Tanner crabs to the parasite is a certainty. Auke Bay in Area 111-50 (Table 1) is an example of where prevalence of the disease has apparently increased from this practice according to local sport and commercial fishermen. Dissemination of bitter crab syndrome may be reduced by increased awareness of both fishermen and processors through educational programs and materials explaining the nature of the parasite and proper disposal of infected crabs. Proper disposal would include burial or incineration. Cooking of infected crabs to industry sanitation standards would also be an alternative method of disposal if such wastes are to be discharged into seawater.

Management of the disease may be possible despite uncertainty as to whether the parasite life cycle requires 12 or 16 mo for completion. Harvesting crabs during early winter rather than in the early spring regardless of whether they become infected in April or August/October would allow for interception of crabs earlier in the disease process when meats may yet be marketable with less need for culling.

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