Some Preliminary Observations on the Incidence of Infection and Pathological Effect of the Parasitic Copepod, Mytilicola orientalis Mori, in the Pacific Oyster (Crassostrea gigas (Thunberg)) on the West Coast of the United States.

by

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Introduction:

Mytilicola orientalis Mori was described from the gut of the oyster Crassostrea gigas (Thunberg) from the Inland Sea of Japan (Mori, 1935). Mori stated that it also occurred in Mytilus crassitesta Tischke in the same area. Wilson (1938) overlooked Mori's description of M. orientalis and described it as M. ostrea from C. gigas in Puget Sound, Washington. He believed that the copepod was not a true parasite because, in his opinion, it did not harm the host and its mouth parts were not suited for sucking blood or biting body tissues. He did point out, however, that Mytilicola maintained its position in the oyster by attaching to the gut wall with the distal segments of the second antennae which are provided with two spine-like setae and terminate in a stout curved claw. Kincaid (personal communication, 1961) found M. orientalis in C. gigas at Samish Bay about thirty years ago and identified it as the Japanese species. Odlaug (1946) studied the effect on condition of Ostrea lurida Carpenter by M. orientalis and found that a significantly lower index of condition occurred with infection by the copepod, but he did not study the effect of the copepod on the tissues of the host. Odlaug found M. orientalis to be more numerous in Mytilus edulis than in O. lurida and concluded that the mussel was the normal host in Washington waters. Rankin, in an unpublished manuscript (1943), reported that heavy infections, more than five copepods per oyster, resulted in weak, watery oysters and mortalities occurred with infections of twelve or more parasites. He observed an erosion of the gut wall, in one instance, associated with the presence of a Mytilicola and surmised that it resulted from ingestion of host tissue by the parasite.

Sparks (1962) described certain metaplastic changes in the gut of C. gigas associated with the presence of M. orientalis in which the normal tall columnar epithelium with a heavy brush border was reduced to a low cuboidal or squamous epithelum and the cilia were depressed or lost in areas of apposition of the copepod. The mucosa was occasionally observed to be completely destroyed and appendages of the parasite to penetrate into the underlying connective tissue. Additionally, an apparent tendency for development of a fibrosis of the underlying connective tissue was noted.

The mortality effect of the related species, Mytilicola intestinalis Steur, on Mytilus edulis has been well documented by numerous European investigators, but only infrequently infects oysters and apparently causes no mortalities (Korringa, personal communication 1949).

While the paper by Sparks was in press, a laboratory technician in the laboratories of the State of California Department of Public Health, while opening oysters for routine bacteriological examination, discovered "red worms" in oysters which, on subsequent examination, proved to be M. orientalis. The State Health Department of California, which has the dual responsibility of human disease prevention and prevention of adulteration of pure food, considered banning oysters infected with Mytilicola from sale if not controlled by the industry.

A conference of Mytilicola in oysters was held in Berkeley, California on September 20, 1961 which was composed of interested parties representing the United States Public Health Service, United States Food and Drug Administration, The Oyster Institute of North America, The Pacific Coast Oystergrowers Association, and Public Health and Fisheries departments of the States involved in the problem. It was agreed at this conference that no human disease risk was involved with oysters infected with Mytilicola, but that the esthetic value of such infected oysters of the consuming public might be impaired and that the effect on survival, fatness and growth of infected oysters should be studied. The author was requested to solicit support from federal funds to study the seasonal incidence of infection and effect on the well-being of oysters. This

was done through a proposal to the U. S. Public Health Service, which was kindly reviewed by Dr. Korringa, and funds were granted for a two-year study to begin September 1, 1962. This investigation has been initiated.

Materials and Methods:

In a growth and mortality study of the Pacific oyster, C. gigas in Washington (Sparks and Chew, 1961), 1,000 oysters were placed in woven-wire baskets (250/basket) in floats in three oyster growing areas of Washington, Oyster Bay in southern Puget Sound, Point Whitney in Hood Canal, and at Nahcotta in Willapa Bay. All gapers (dead and dying oysters) recovered and three normal oysters at the twice monthly station checks were fixed in Zenker's Fluid, embedded in paraffin, sectioned through the palp region and stained in Harris Hematoxylin and Eosin. The incidence of infection by Mytilicola was ascertained by reading a 7 μ section, thus infections in which no copepods were present in those portions of the gut in the palp region were registered as negative. A total of 654 oysters were processed between March 1959 and September 1961. Studies of the pathological effect of Mytilicola on the gut of the oyster were also conducted from these preparations.

Some data is available on the incidence of infection and the number of Mytilicola present in California oysters during the 1961-62 season. These data were obtained by the State of California Department of Public Health and represent the result of gross examination of whole oysters rather than microscopic examination of sectioned material.

Results:

Incidence of Infection -

The percentage of the sample infected from each experimental station for each month in the Washington studies are shown in Figure 1. These figures are based on very limited sampling, generally of a minimum of six oysters from each area each month, therefore the percentages of infection are of questionable validity as representing the entire population. Also, some infected oysters, particularly with light infections,

would undoubtedly not have Mytilicola in that portion of the gut sectioned for microscopic examination. Despite the unreliability of the data, it is believed that some general idea of the seasonal incidence of infection can be obtained from examination of these data. It appears that peaks of infection occurred in the oysters during each year in each area followed by a decline. Examination of the data revealed that the trends of infection in all areas were similar though there were variations in both the levels and the periodicity. Because of the similar trends, the data for all three stations were combined (Table 1) which provided a much larger and, hopefully, more valid sample which still represented the seasonal incidence of infection. When these combined data are plotted (Fig. 2), we see more clearly that two peaks of infection occur each year: one, in the spring is followed by a sharp decline in midsummer, with very low levels of infection in July of each year. Infection rates climbed sharply each year reaching a high peak in August or September and then tapering off during the fall and winter, but building up again in the early spring.

The Sanitation Laboratory of the California Department of Public Health has conducted, since October 1961, a study of the infestation of shellstock of California from Drakes Bay and Humbolt Bay (Miller, 1962) and have also found a marked variation in the percentage of infected oysters (Fig. 3), but with the seasonal trend not so marked as in Washington oysters. It is interesting that the low percent of infection noted in Washington in July is also reflected in the California data.

These data seem to indicate that reproduction of the copepod takes place in two peaks, with the infective stages present in the water during the early spring and late summer. It further indicates that the parasite is not capable of maintaining itself in the gut of the oyster or that the normal life span of the copepod is quite short. These phenomena, along with a study of other possible hosts, will be studied in the investigation just begun.

Table 1. Combined Percentage of Infection of Oysters With Mytilicola From Three Areas In Washington.

	1959		
	Pos.	Neg.	% Inf.
March	5	6	45.5
April	12	11	52.1
May	8	8	50.0
June	3	8	27.3
July	3	14	17.6
August	5	12	29.4
September	4	5	44.4
October	15	21	41.7
November	5	11	31.3
December	2	10	16.8

	1960		
	Pos.	Neg.	% Inf.
January	5	19	20.8
February	2	13	13.3
March	5	15	25.0
April	8	31	20.5
May	4	13	23.5
June	5	13	27.8
July	5	25	16.8
August	6	12	33.3
September	1	12	7.7
October	3	27	10.0
November	-	-	-
December	-	-	-

	1961		
	Pos.	Neg.	% Inf.
January	-	-	-
February	2	7	22.2
March	2	5	28.6
April	1	31	3.2
May	3	25	10.7
June	7	24	22.2
July	1	32	3.0
August	4	24	14.3
September	10	17	37.3

Effect of Infection on the Oyster -

As noted previously, Mytilicola orientalis has been demonstrated to cause a lowering of Condition Index in Ostrea lurida (Odlaug, 1946) and mortalities in the same host with heavy infections (Rankin, 1943). Sparks reported pathological effects of M. orientalis in C. gigas, but no data has been published demonstrating a lowering of condition or increase in mortalities in C. gigas resulting from infection with M. orientalis. In the growth mortality study from which the present data were obtained, the growth rates, Condition Index and mortality rates were studied during the same period in which the oysters were prepared for this study (Sparks and Chew, 1961 and Chew, 1962). There is no apparent correlation between the pattern of infection and the Condition Index at each station, but the highest overall percentage of infection was found at Willapa Bay (32.95%) which was correlated with the consistently lowest Condition Index of the three areas. The relative Condition Indices at the Hood Canal and Oyster Bay stations varied with higher levels in the oysters from Hood Canal from October, 1959 to July, 1960 and in Oyster Bay oysters during the rest of the period, but there was, again, no apparent relationship between the Condition Index and the level of infection at any particular time.

During this period, growth was best at Oyster Bay, next best at Hood Canal, and poorest at Willapa Bay, which was correlated with the level of average infection with Mytilicola, these being 10.68 percent, 27.45 percent and 32.95 percent, respectively. Whether the presence of Mytilicola was related to the growth rate or condition cannot be answered at this time.

In an effort to determine if the presence of Mytilicola in the Pacific oyster caused significant mortalities, comparisons by chi-square of the presence of Mytilicola in sections from the normal oysters and gapers from each station were calculated. In no instance was there a significant difference between the incidence of infection in gapers and normal oysters which seems to indicate that Mytilicola is not a mortality factor in Pacific oysters.

Summary:

The preliminary data reported here demonstrate that high levels of infection of Mytilicola orientalis in the gut of Crassostrea gigas occur during most of the year in Washington and California, but that sharp peaks of infection occur which are followed by rapid loss of infection which indicates that reproduction of the copepod takes place during the fall and spring and that Mytilicola is incapable of maintaining itself in the gut of C. gigas for prolonged periods. There is circumstantial evidence which implies that heavy infections may cause a lowering of condition and, perhaps, a lessening of the growth rate. Despite the demonstration of pathological conditions in the gut of infected oysters, statistical comparison of the level of infection of gapers and live oysters shows that no significant mortality due to Mytilicola occurred in the 654 oysters examined in this study. Further work is underway to ascertain the geographic distribution and seasonal incidence of infection in both C. gigas and O. lurida in Washington, Oregon, and California, the effect of infections on fatness and on survival, and the pattern of incidence of pathological effect in Mytilus edulis and other host reservoirs.

Acknowledgments

Most of the oysters used in this study were collected and fixed by Dr. Kenneth K. Chew and Mr. Stanley C. Katkansky and were sectioned and stained by Mr. Katkansky and Mrs. Daphne Pereyra. The author was aided in reading the slides by Mrs. Pereyra, Mr. Katkansky, and Mr. Gilbert Pauley, all of the College of Fisheries, University of Washington. The data from California is used through the courtesy of the California Department of Public Health.

Literature Cited

Mori, T. 1935. Mytilicola orientalis, a new species of parasitic copepoda. Dobyts. Zasshi., 47, 687-689.

Wilson, C. B. 1939. A new copepod from Japanese oysters transplanted to the Pacific Coast of the United States. J. Wash. Acad. Sci. 28, 284-288.

Kincaid, Trevor 1962. Personal communication.

Odlaug, T. O. 1946. The effect of the copepod, Mytilicola orientalis upon the Olympia oyster, Ostrea lurida. Trans. Am. Microscop. Soc. 65, 311-317.

Rankin, J. S. 1943. A biological report of the conditions in the waters of Oyster Bay, Little Skookum, and Oakland Bay. July-December, 1942. Unpublished manuscript, 19 pp.

Sparks, Albert K. 1962. Metaplasia of the gut of the oyster Crassostrea gigas (Thunberg) caused by infection with the copepod Mytilicola orientalis Mori. Journal of Insect. Pathology, Vol. 4, No. 1, March 1962.

Korringa, P. 1949. Letter to Dr. Trevor Kincaid

Sparks, A. K. and Chew, K. K. 1961. Preliminary report on growth and survival of the Pacific oyster in Washington waters. Proc. Natl. Shellfish Assoc., 50, 125-132.

Chew, K. K. 1962. The growth of a population of Pacific oysters (Crassostrea gigas) when transplanted to three different areas in the State of Washington. PhD Thesis, College of Fisheries, University of Washington.

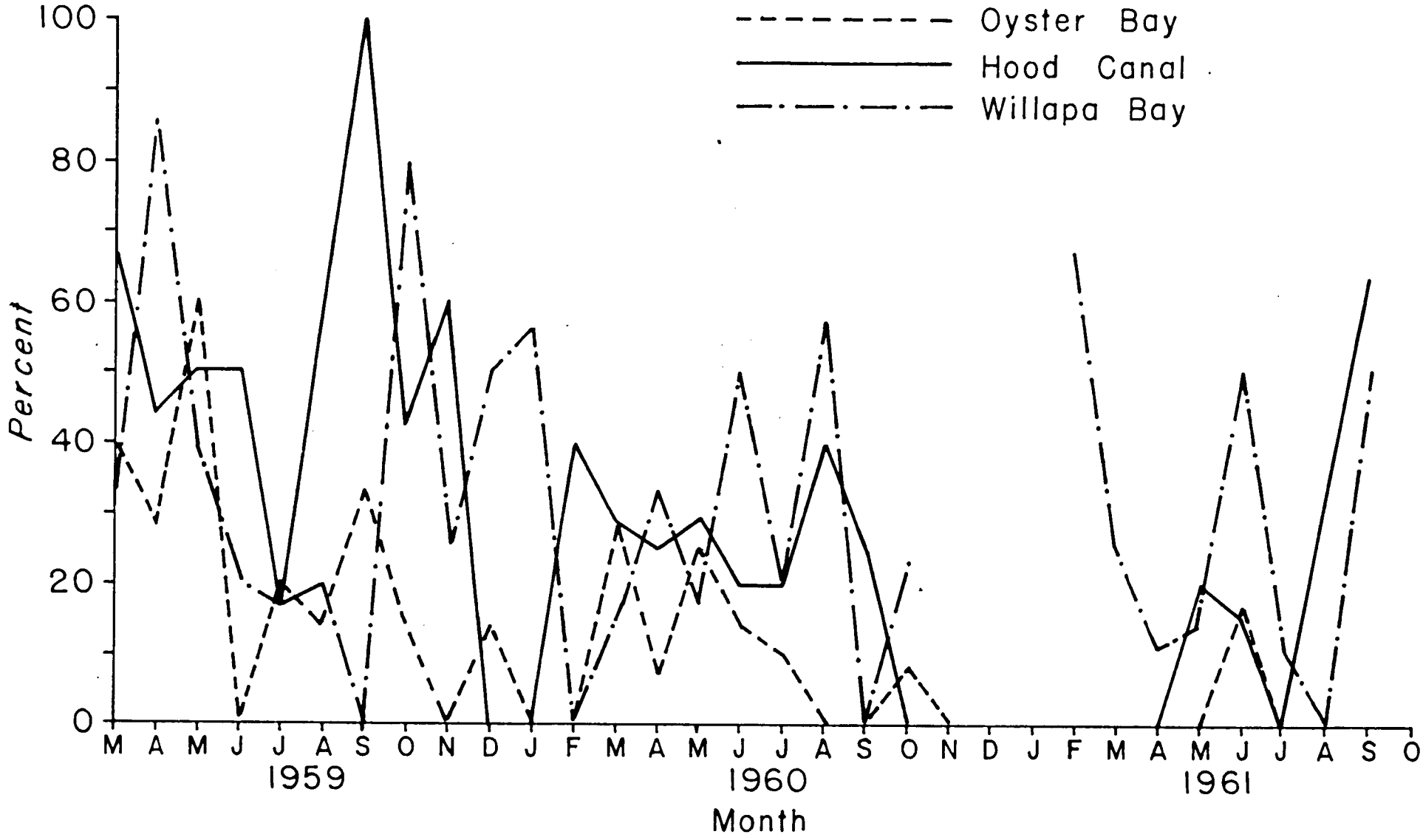

Fig. 1 Incidence of Mytilicola in C. gigas — Oyster Bay, Hood Canal, Willapa Bay.

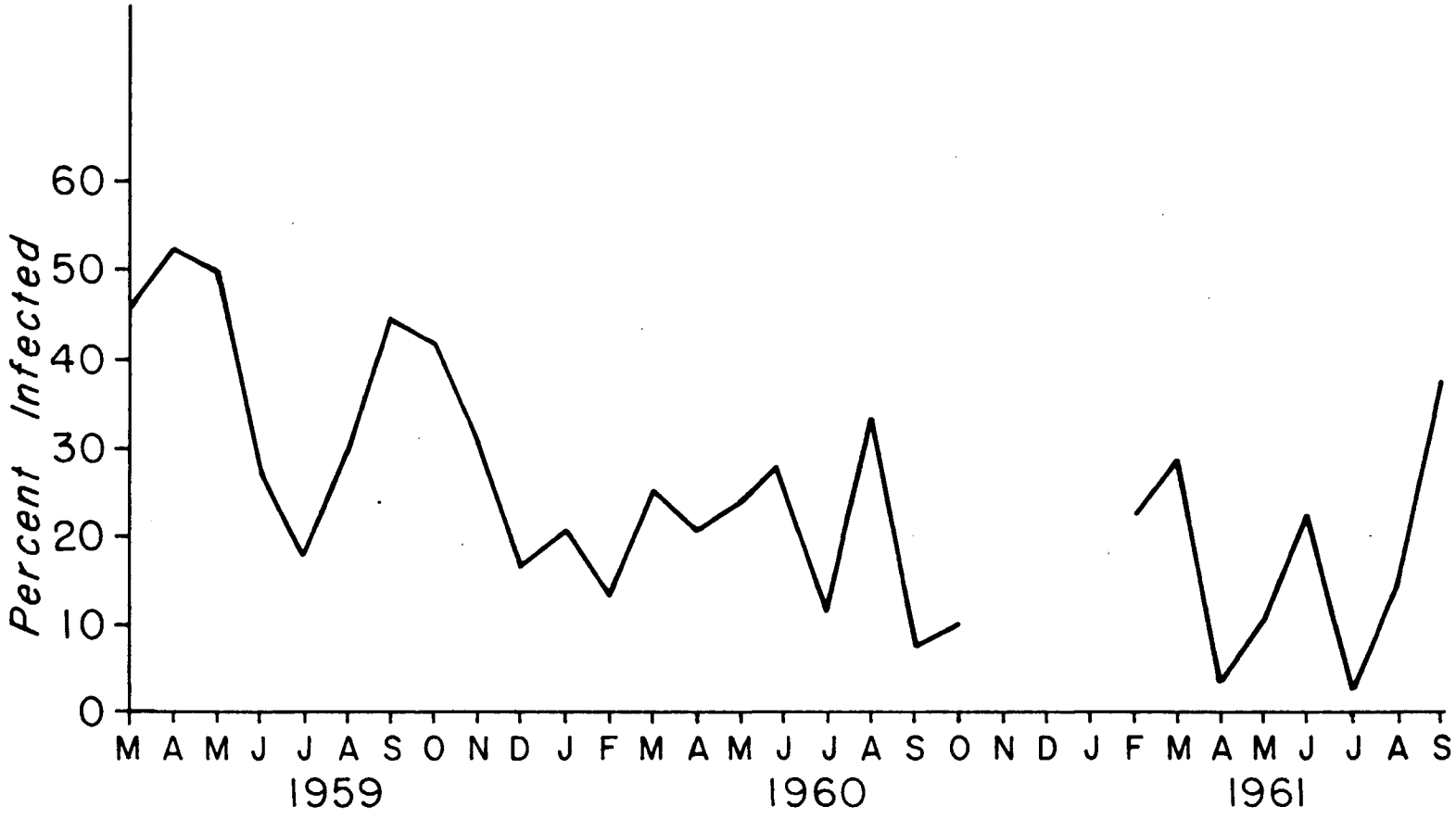

Fig. 2 Seasonal incidence of infection of Mytilicola in Washington (3 areas combined).

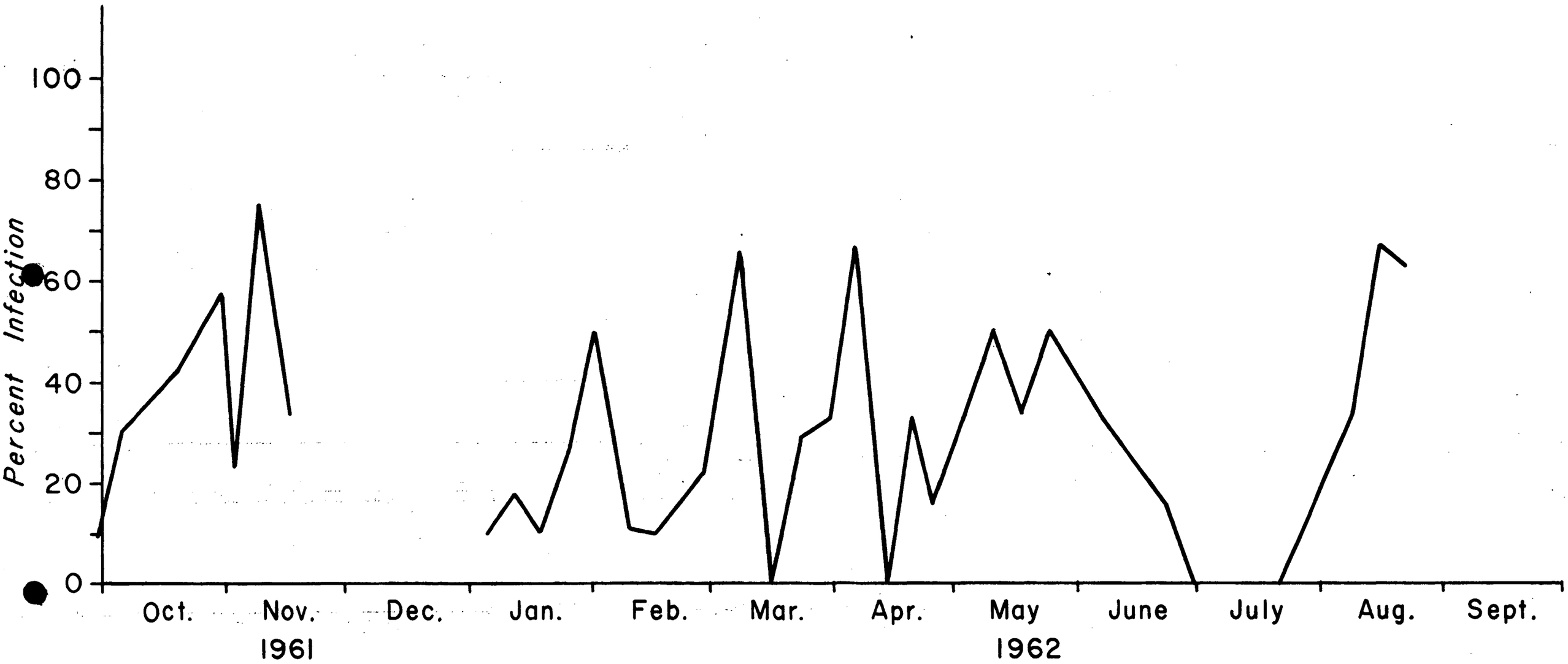

Fig. 3 Incidence of infection of C. gigas with Mytilicola in California