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Occurrence of the Parasitic Dinoflagellate *Amoebophrya ceratii* in Chesapeake Bay Populations of *Gymnodinium sanguineum*

D. WAYNE COATS[1] and KATRIN R. BOCKSTAHLER

Smithsonian Environmental Research Center, Box 28, Edgewater, Maryland 21037

ABSTRACT. Chesapeake Bay populations of the red-tide dinoflagellate *Gymnodinium sanguineum* were regularly infected by the parasitic dinoflagellate *Amoebophrya ceratii* during the summers of 1988–1991. Infections developed inside the nucleus of *G. sanguineum* and were always lethal to the host. Parasite generation time was ~ 40 h at 23° C, with the intracellular, trophont phase lasting 39.5 ± 0.3 h, and the extracellular, vermiform stage persisting for ~ 20 min. Near surface accumulations of *G. sanguineum* sometimes exceeded 1,000 cells/ml; however, host abundance was relatively low when integrated over the surface mixed layer of each station (mean = 12.2 cells/ml ± 2.96 SE; n = 60). Parasitized hosts were encountered in 75% of the samples where host abundance was ≥ 1 per ml, and epidemic outbreaks (20–40% hosts infected) were observed on several occasions. Epidemic infections were generally located several meters below surface accumulations of *G. sanguineum* and were always restricted to a narrow region near the pycnocline. Consequently, integrated station values for parasite prevalence were low, with an average 2.7% (± 0.31 SE; n = 60). Parasite induced mortality removed up to 8% of *G. sanguineum* populations per day, but averaged < 2% of host biomass throughout the Bay. Thus, parasitism by *A. ceratii* does not appear to be a major factor regulating *G. sanguineum* bloom in the main stem of Chesapeake Bay.

Supplementary key words. Infection level, parasitism, population dynamics, red-tide.

THE heterotrophic dinoflagellate *Amoebophrya ceratii* is an obligate, intracellular parasite of other dinoflagellates that is known to occur in coastal waters of the North Atlantic [1, 8, 9, 12], the North Pacific [14, 15], and the Mediterranean Sea [2]. *A. ceratii* is not a host-specific parasite, as infections have been reported in species of more than 15 dinoflagellate genera including a few toxic forms [2, 4, 11, 12, 14]. Dinoflagellates infected by *A. ceratii* become reproductively incompetent [8, 14] and are eventually killed by the parasite. The lethal nature of *A. ceratii* and its high infection levels in some host species have led to the suggestion that this parasite might serve as an agent for the biological control for toxic dinoflagellate blooms [15, 16].

Amoebophrya ceratii infections are initiated when dinospores, the biflagellated dispersal stage of the parasite, attach to the outer membrane and penetrate into the cytoplasm of the host. As many as 12 dinospores can attack a single host, but rarely does more than one reach maturity [2]. Once inside the host, the parasite differentiates into the trophont (vegetative) stage that may remain in the cytoplasm or may invade the host's nucleus. In either case, the parasite soon increases in size and begins a series of nuclear divisions that are accompanied by flagellar replication, but proceed without cytoplasmic fission. With continued growth, the anterior portion of the parasite (the episome) is partially enclosed by the posterior hyposome to form a flagellar cavity, the mastigocoel [3]. Thus, trophonts of late infections are large, polynucleate and multiflagellate organisms that occupy most of the host cell and have a characteristic "beehive" [9] appearance. At maturity, the trophont ruptures through the host's pellicle and transforms into a strongly motile vermiform stage that divides to produce numerous infective dinospores.

Cachon [2] reported that *Amoebophrya ceratii* occurred sporadically in a number of Mediterranean host species, but highest infection levels usually occurred near the end of dinoflagellate blooms. Heavy infections (30–40% in *Gonyaulax cantenella*; 80% in *Ceratium fusus*) were also observed along the western coast of North America, where parasitism by *A. ceratii* was linked to rapid declines in host populations and implicated as

[1] To whom correspondence should be addressed.

an important factor in preventing bloom formation [14, 15]. By contrast, Fritz and Nass [9] found that ≤ 2% of *Dinophysis norvegica* collected from coastal waters of Nova Scotia were infected by *A. ceratii* and argued that parasitism had little effect on host populations. The same study reported infection levels of 50% in *Scrippsiella trochoidea.*

To reliably assess the effect of *Amoebophrya ceratii* on host populations requires recognition of all parasite life history stages and knowledge of parasite development time. Heavy infections would be of little consequence if the duration of infection greatly exceeded host generation time. Were the reverse true, then even low infection levels could have a significant influence on host populations. Thus far, only Nishitani et al. [14] have estimated parasite prevalence using techniques that reveal all intracellular stages of the parasite, and data on development time of *A. ceratii* have been unavailable.

Here we present data on the occurrence of *Amoebophrya ceratii* in Chesapeake Bay populations of *Gymnodinium sanguineum* over a four-year period. We also provide information on parasite morphology and development time, and estimate the impact of *A. ceratii* on this host species.

MATERIALS AND METHODS

Field samples for documentation of temporal and spatial occurrence of *Amoebophrya ceratii* in Chesapeake Bay populations of *Gymnodinium sanguineum* were collected during cruises conducted at approximately monthly intervals between June and October of 1988–1991. On each of 15 cruises, vertical CTD (conductivity-temperature-depth) -Niskin bottle casts were taken at 10 stations located along the longitudinal axis of the Bay (Fig. 1). CTD profiles provided data on conductivity and temperature, which defined the depth of the pycnocline and facilitated sampling within the surface mixed layer. The number and vertical position of Niskin samples depended on the depth and stratification of the water column, but six to eight were taken at most stations with the upper four to five positioned at 2 to 3-meter intervals between the surface and the pycnocline. A 500-ml subsample was taken from each Niskin bottle and preserved in modified Bouin's solution [15].

Estimates of *Gymnodinium sanguineum* abundance were obtained by enumerating cells in 1-ml aliquots of Bouin's preserved whole-water sample using Sedgwick-Rafter chambers and a Zeiss microscope (×125). Samples that contained ≥ 1 dinoflagellate/ml were processed by the quantitative protargol staining (QPS) technique [13] and examined at 500× using Zeiss oil-immersion optics. Parasite prevalence (i.e. percent *G. sanguineum* infected by *Amoebophrya ceratii*) was determined by examining ≥ 50 (usually 100) protargol-stained hosts per sample, and the number of infected hosts per ml was calculated as host abundance multiplied by parasite prevalence.

Integrated values for the number of *Gymnodinium sanguineum* per ml and number infected hosts/ml were generated for the surface mixed layer of each station as depth-weighted averages of data for samples taken between the surface and the greater of two depths, represented by either the pycnocline or the deepest sample containing ≥ 1 *G. sanguineum* per ml. Integrated data were used to calculate station values for parasite prevalence, and parasite induced mortality of *G. sanguineum* was estimated as:

$$\text{Proportion of host's population killed per day} = \frac{\text{integrated parasite prevalence}}{\text{infection time}}$$

where infection time = duration of the parasites' intracellular phase (1.65 days at 23° C) corrected for ambient temperature using a Q_{10} of 2. Station values for hosts killed per day were divided by integrated host abundance to give the proportion of *G. sanguineum* removed per day. Cruise averages were calculated as the mean of integrated station data.

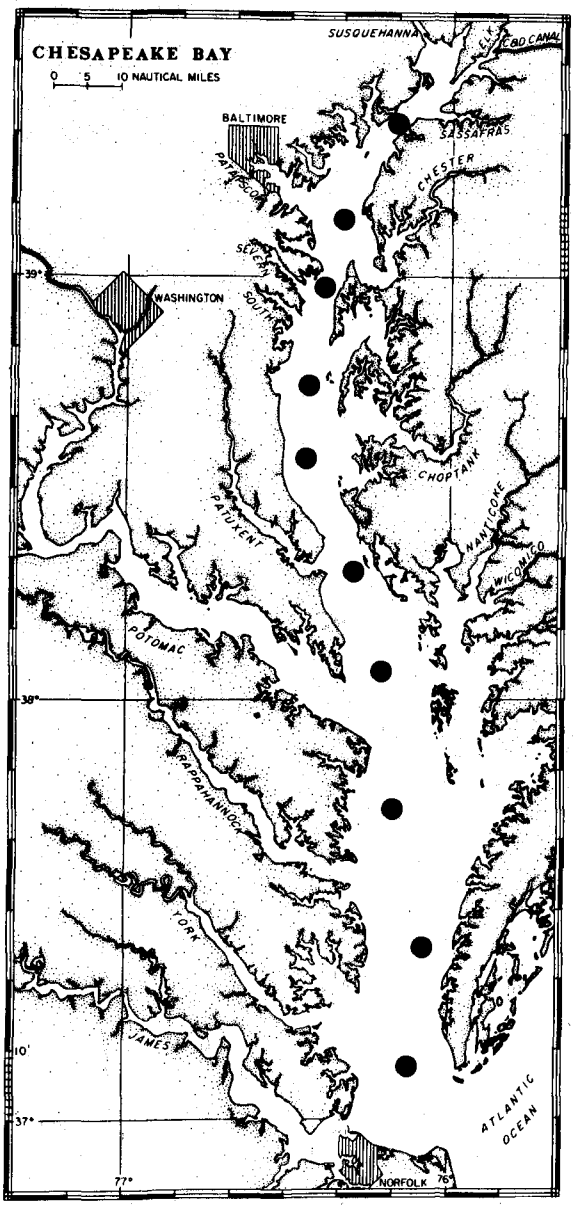
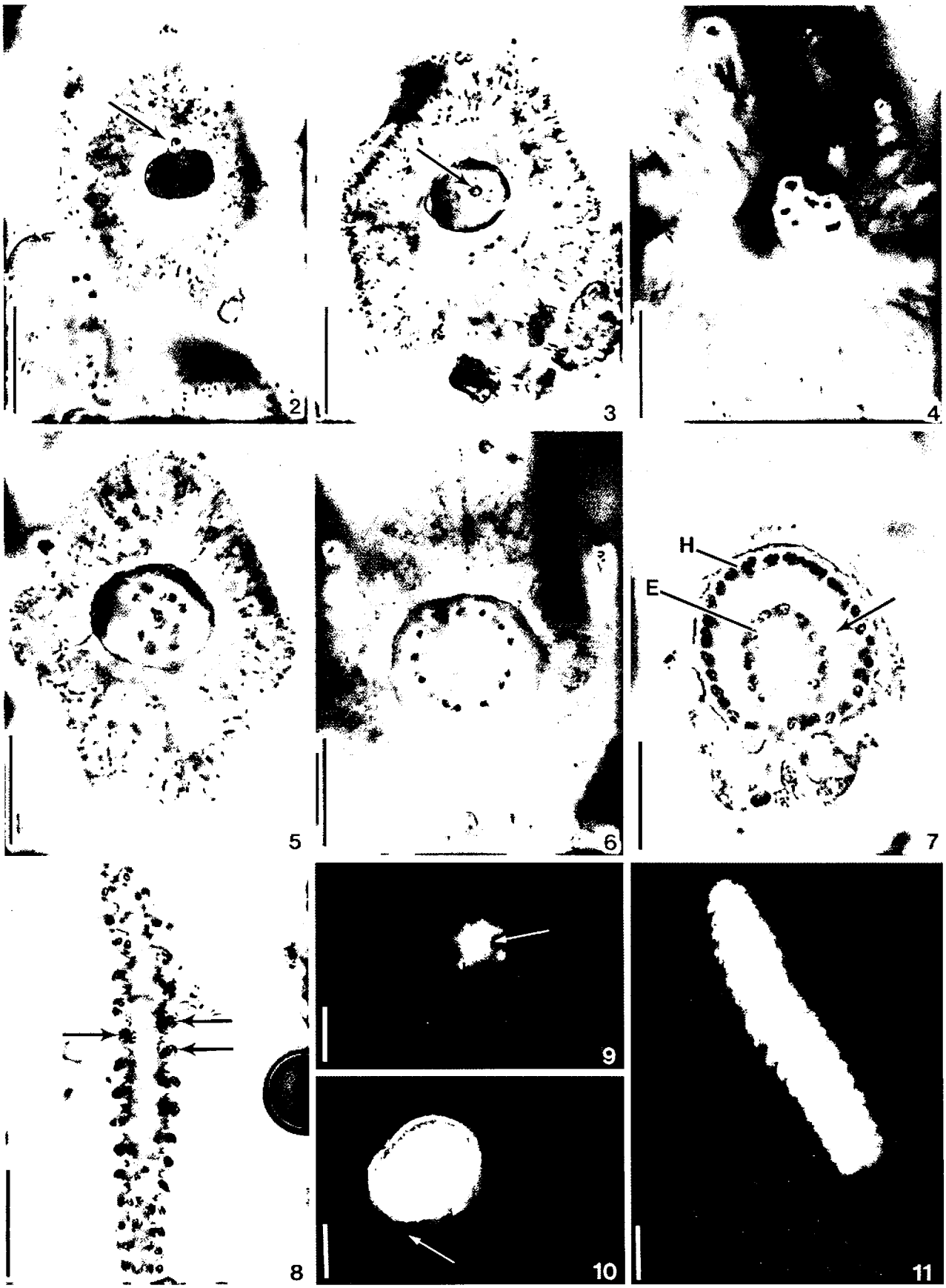


Fig. 1. Map of Chesapeake Bay showing routine stations (●) for research cruises in 1988–1991. Stations are designated from north to south as 921 (39° 21′ N. Lat.; 76° 20′ W. Long.), 908 (39° 08′ N. Lat.; 76° 20′ W. Long.), 858 (38° 58′ N. Lat.; 76° 23′W. Long.), 845 (38° 45′ N. Lat.; 76° 26′ W. Long.), 834 (38° 34′ N. Lat.; 76° 26′ W. Long.), 818 (38° 18′ N. Lat.; 76° 17′ W. Long.), 804 (38° 04′ N. Lat.; 76° 13′ W. Long.), 744 (37° 44′ N. Lat.; 76° 11′ W. Long.), 724 (37° 24′ N. Lat.; 76° 05′ W. Long.), and 707 (37° 07′ N. Lat.; 76° 07′ W. Long.).

For determination of parasite development time, two 1-liter flasks containing 500 ml of cultured *Gymnodinium sanguineum* (1,250 cells/ml) were inoculated with ~ 30 infected hosts that were individually isolated from field samples using a micropipette and inverted epifluorescence microscopy (see below). Flasks were incubated at near ambient temperature (23° C) on a 14:10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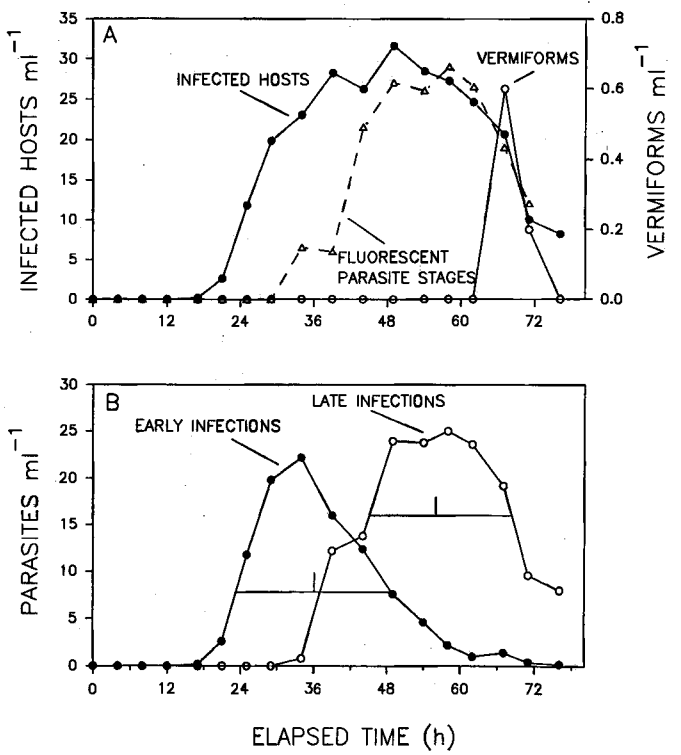

Fig. 12. Parasitism of *Gymnodinium sanguineum* by *Amoebophrya ceratii* during a three-day culture experiment. **A.** Total number of infected hosts, number of hosts containing green fluorescing parasite stages, and number of *A. ceratii* vermiforms. **B.** Number of *A. ceratii* trophonts sorted as early and late infections. Horizontal lines indicates interval when successive data for number early and late infections exceeded their mean values (7.9 and 16.0/ml, respectively); short vertical line marks the mid-point of each interval. Time between mid-points (19.9 h) equals half the duration of the parasites intracellular phase.

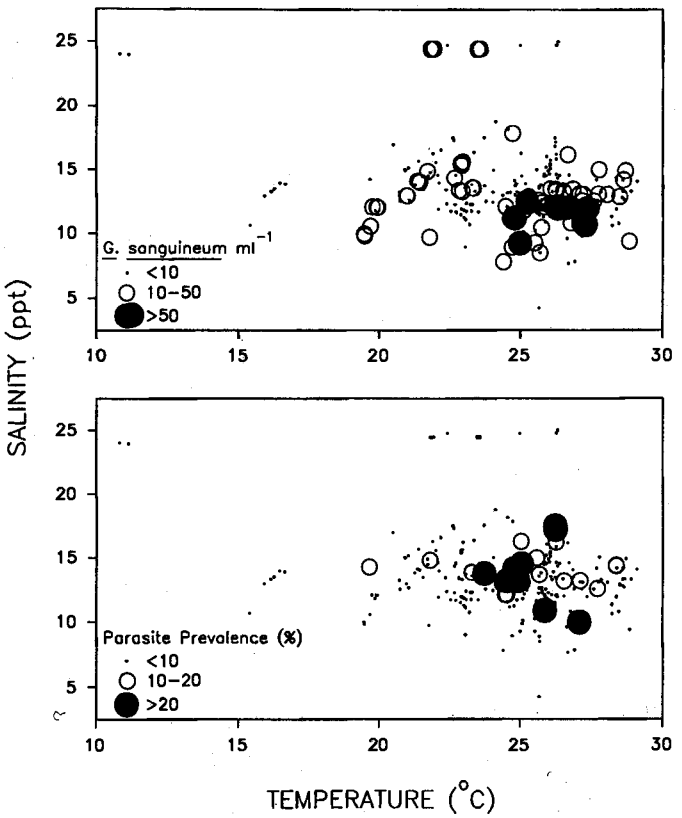

Fig. 13. Host abundance and parasite prevalence for Chesapeake Bay samples plotted on a temperature-salinity parameter space; data grouped into low (•), intermediate (○), and high (●) categories. **A.** *Gymnodinium sanguineum* per ml. **B.** Percent hosts infected by *Amoebophrya ceratii.*

light-dark cycle and sampled at 4 to 6-h intervals over three days. At each interval, cultures were gently mixed, and two sets of samples were taken from both flasks. One set of samples was fixed with Bouin's fluid and processed as 5-ml QPS preparations. The number of early infections (prior to circular configuration of parasite nuclei), late infections (all other stages leading to death of the host), and vermiforms present in QPS preparations were determined by scanning the entire filter at 500× as above. The second set of samples was preserved with 5% $CaCO_3$-buffered formalin and settled in 2-ml Ütermohl chambers. Hosts that had green fluorescent nuclei were enumerated by scanning settled samples using a Leitz Diavert inverted microscope (×100) equipped with a 100 W mercury lamp (exciting wavelength of 455–490 nm). Formalin fixed samples were processed within 30 min, as autofluorescence of the parasite decreased significantly when samples were stored at room temperature for a few hours; fluorescence also faded when samples were stored in the dark at 4° C.

RESULTS

Parasite morphology and development. *Gymnodinium sanguineum* rarely contained more than one trophont of *Amoebophrya ceratii,* and all parasites matured inside the nucleus of the host. Recently established infections appeared in protargol preparations as small (8–10 μm) ovoid inclusions within the cytoplasm of *G. sanguineum* and were often located near or attached to the nuclear envelope of the host (Fig. 2). This early stage of the parasite had a spherical nucleus that contained a prominent nucleolus and exhibited little or no growth prior to entering the host's nucleus. Once in the nucleoplasm of *G. sanguineum,* young trophonts were distinguished by the presence of a single densely staining nucleus surrounded by a translucent halo of parasite cytoplasm (Fig. 3). Growth of the parasite began

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Fig. 2–11. Life history stages of *Amoebophrya ceratii* in *Gymnodinium sanguineum*; protargol impregnations of field specimens and epifluorescence of cells from infected cultures; scales = 25 μm. **2.** Recently established infection showing very early trophont of *A. ceratii* (arrow) attached to the nuclear envelope of *G. sanguineum.* **3.** Young trophont visible as a lightly staining circle inside the host's nucleoplasm; arrow indicates parasite nucleus. **4.** Early infection showing irregularly arranged parasite nuclei. **5.** Mid- to late stage of parasitism after parasite nuclei have started to form a spiral. **6.** Late infection where parasite nuclei exhibit a distinct circular arrangement. **7.** Nearly mature trophont that occupies most of the host cell and has a well developed mastigocoel (arrow) positioned between the hyposome (H) and episome (E). **8.** Vermiform stage of *A. ceratii* with parasite nuclei (arrows) distributed in a tight spiral around the longitudinal axis of the cell. **9.** Early to mid-infection where the green autofluorescing parasite is surrounded by red fluorescing chloroplasts of *G. sanguineum.* **10.** Epifluorescence of a mature trophont; arrow indicates remnants of the host's chloroplasts. **11.** Vermiform of *A. ceratii* viewed with epifluorescence microscopy.

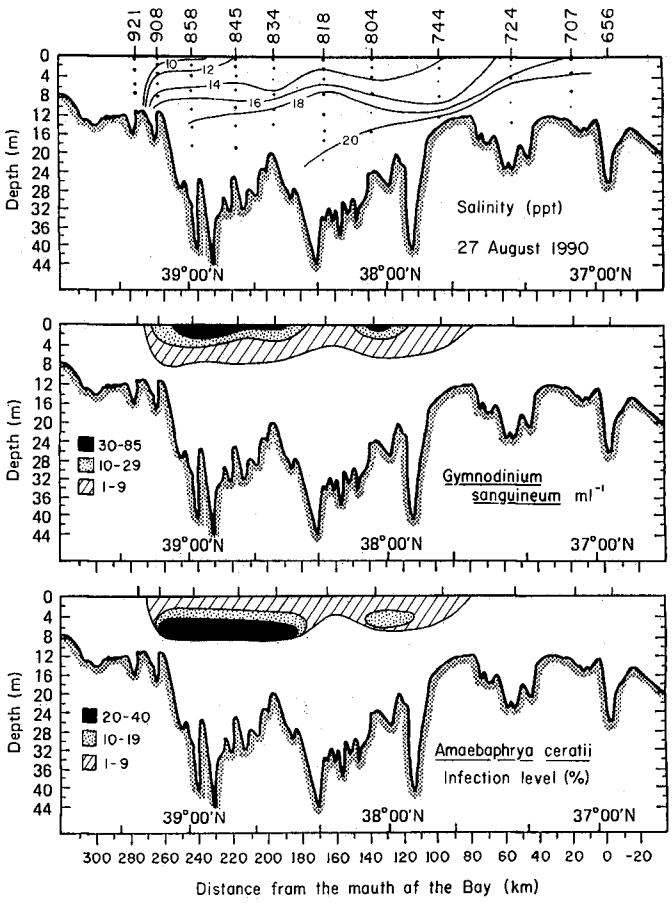

Fig. 14. Distributional maps for a representative cruise in 1990 showing **A**. salinity isopleths. **B**. Host abundance. **C**. Parasite prevalence.

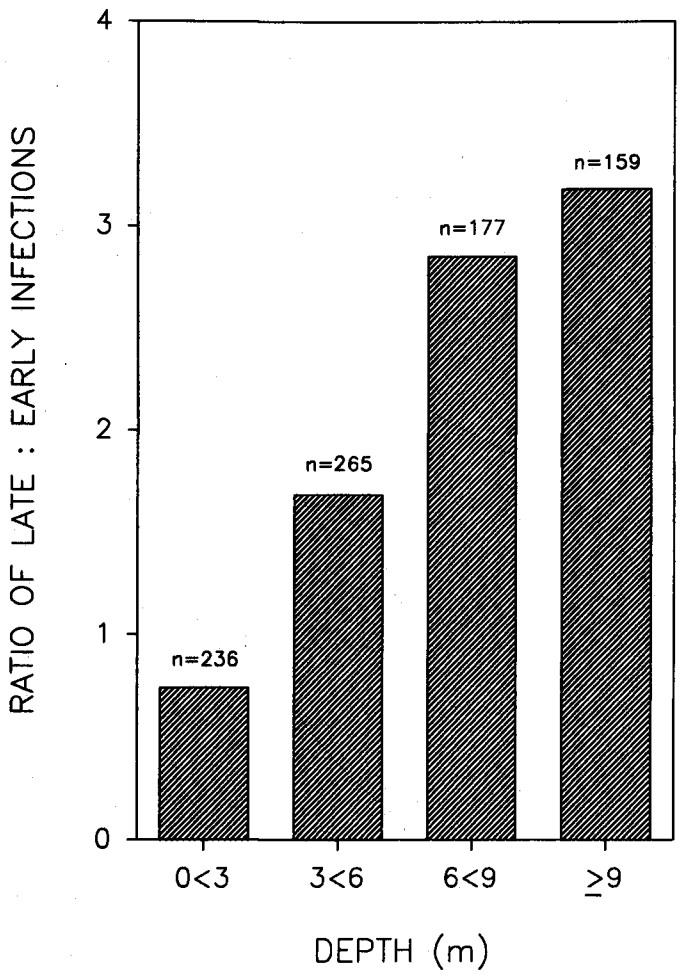

Fig. 15. Vertical distribution of *Amoebophrya ceratii* life history stages. Data expressed as the proportion of late to early infections; sample sizes (n) give number of infected hosts examined for each depth interval.

inside the host's nucleus and was accompanied by a series of nuclear divisions that started when the trophont reached a diameter of ~ 20 μm. Nuclei resulting from the first few divisions were irregularly distributed within the parasite's cytoplasm (Fig. 4), but continued karyokinesis resulted in a circular to spiraled arrangement of nuclei (Fig. 5, 6). Additional growth of the parasite produced a large mass that filled much of the host cell and had the characteristic "beehive" appearance of late infections (Fig. 7). Vermiforms were easily identified in protargol preparations by their elongated form and distinctive nuclear arrangement (Fig. 8), but were rarely encountered in field samples. Those that were observed in natural populations may have originated from infected *G. sanguineum* or some other host species and thus were not included in estimates of parasite prevalence.

Parasitized *Gymnodinium sanguineum* continued to swim throughout the infection cycle and were not easily distinguished from uninfected hosts when living specimens were viewed with brightfield microscopy. Very advanced infections (comparable to that shown in Fig. 7) could be recognized at low magnification (100×) by the presence of a swollen, dome-shaped episome; however, most field samples contained few of these stages. When living specimens were viewed by epifluorescence microscopy, uninfected hosts had a uniform red color due to presence of Chl *a*. By contrast, autofluorescence of the parasite made the nuclei of infected hosts appear as bright green spheres (Fig. 9, 10). Vermiform (Fig. 11) and dinospore stages of *Amoebophrya ceratii* were also strongly autofluorescent.

Amoebophrya ceratii only required 1–2 min to rupture through the host cell and emerge as a fully developed and motile vermiform. *Gymnodinium sanguineum* was always killed during this process and host material that was not utilized by the trophont was discarded as the vermiform began to swim. The vermiform stage persisted for about 20 min, during which time it slowly extended into a 200–300 μm long string of loosely connected cells that eventually separated to form dinospores.

Generation time of *Amoebophrya ceratii*. *Amoebophrya ceratii* that were isolated from field samples and introduced into uninfected cultures of *Gymnodinium sanguineum* required about 18 h to mature before establishing new infections (Fig. 12A). Thereafter, the number of infected hosts, as determined from protargol stains, increased quickly to ~ 30 cells/ml, remained near that level for 24–30 h, and then declined abruptly. Vermiforms never exceeded 1/ml and were only observed after the number of infected hosts began to decline. Hosts containing fluorescent nuclei were encountered ~ 12 h after infections were evident in protargol stains, suggesting that some development of the parasite was required before infections could be detected by epifluorescence microscopy. Early trophonts may either lack autofluorescence, or their fluorescence may have been masked by that of the host's chloroplasts.

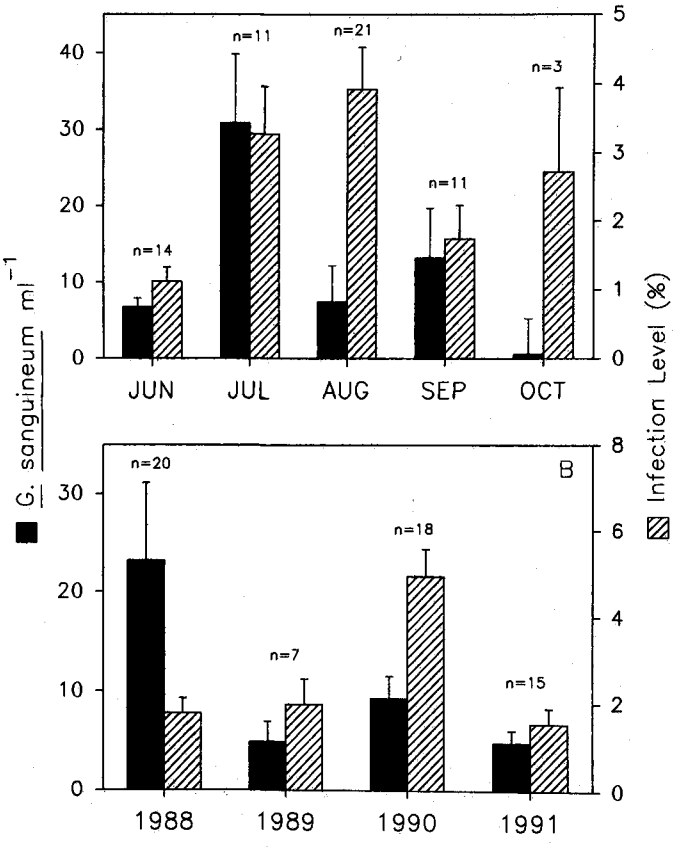

Fig. 16. A. Seasonal and B. annual fluctuations in Chesapeake Bay populations of *Gymnodinium sanguineum* and *Amoebophrya ceratii*. Data represent means and standard errors for integrated station values; n = number of stations for each category.

The time between the onset of new infections and subsequent appearance of vermiforms suggests that intracellular development of *Amoebophrya ceratii* required ~ 45 h (Fig. 12A). To obtain better resolution of parasite development time, data for abundance of early (i.e. prior to circular configuration of parasite nuclei) and late (i.e. all other stages leading to death of the host) infections were plotted separately (Fig. 12B). The intervals during which early and late infections exceeded their respective mean values (horizontal lines crossing peaks in Fig. 12B) were of comparable length, 25.7 and 21.9 h, respectively, and indicate that the two categories were of similar duration. The mid-point of each interval (short vertical lines) provided an objective estimate for the time of maximum occurrence of each stage, and the difference between the two mid-points was taken as half the intracellular development time. Estimates for duration of the parasites's trophont stage from replicate experiments were in close agreement and averaged 39.5 ± 0.3 h at 23° C. Since the vermiform stage lasted ~ 20 min before production of dinospores, total parasite generation time equaled ~ 40 h.

Spatial and temporal patterns in parasitism. *Gymnodinium sanguineum* and *Amoebophrya ceratii* were broadly distributed in the mesohaline portion of Chesapeake Bay during the summers of 1988–1991. Host densities ≥ 1/ml were typically encountered between the Bay Bridge (station 858) and the Potomac River (station 804), and *A. ceratii* infections were present in 75% of those samples (n = 238). Parasite prevalence was generally low (< 10%), but epidemic infection levels (20–40%) were encountered on several occasions; mean parasite prevalence for discrete samples was 4.0 ± 0.4% (n = 238).

Table 1. *Amoebophrya ceratii* infections in *Gymnodinium sanguineum* during 1988–1991.

	Parasite prevalence (%)	Host abundance (#/ml)	Percent host population removed per day
Integrated station data (n = 60)			
Mean	2.7	12.2	1.9
SE	0.31	2.96	0.23
Mode	1.8	6.2	1.3
Range	0–11	0.1–138	0–8
Cruise averages (n = 15)			
Mean	2.6	10.2	1.8
SE	0.44	3.14	0.34
Mode	2.0	7.1	1.3
Range	0.8–6.3	0.6–50.5	0.4–4.9

Bloom concentrations of *Gymnodinium sanguineum* (> 50/ml) occurred at salinities of 9–12‰ when water temperature was above 25° C (Fig. 13A), whereas epidemic outbreaks of *Amoebophrya ceratii* were present at somewhat higher salinity (10–17‰) and at slightly lower temperature (23–28° C; Fig. 13B). The subtle difference in salinity-temperature conditions associated with maximum occurrence of host and parasite populations resulted from the vertical distribution of the two species. Peak abundances of *Gymnodinium sanguineum* were generally restricted to the upper 3–4 m of the surface mixed layer, while highest infection levels typically occurred in samples taken at depths of 8–12 m (Fig. 14). *A. ceratii* epidemics always occurred several meters below dense surface accumulations of *G. sanguineum* and were generally located near the pycnocline. As a result, estimates of host abundance and parasite prevalence for discrete samples were negatively correlated ($P < 0.01$, n = 238), while integrated station values showed no significant correlation.

Life history stages of *Amoebophrya ceratii* also displayed a striking vertical zonation. Early stages of parasitism were more common than late infections in the upper three meters of the water column, but became proportionally less frequent with depth (Fig. 15). Late infections were about three times more numerous than early infections at depths ≥ 6 meters, and vermiform stages were most often observed in samples taken near the pycnocline.

Considerable seasonal and interannual variation in host density and parasite prevalence was evident during the four year study period. Monthly averages for integrated station data showed a sharp peak in *Gymnodinium sanguineum* abundance during July, with relatively low numbers in other months (Fig. 16A). Corresponding data for parasite prevalence revealed a steady increase in *Amoebophrya ceratii* infections through late summer, with moderately lower infection levels in fall. Yearly averages showed more than a two-fold difference in host abundance and parasite prevalence (Fig. 16B).

Gymnodinium sanguineum occasionally formed dense surface accumulations of > 1,000 cells/ml, yet abundances were relatively low over most of the surface mixed layer as indicated by integrated station and cruise averages (Table 1). Station and cruise averages for parasite prevalence were similarly low with mean infection levels just over 2.5% and upper limits of 11% and 6%, respectively (Table 1). Estimates of parasite induced mortality indicate that *Amoebophrya ceratii* was capable of removing up to 8% of hosts during localized outbreaks and 5% of the *G. sanguineum* standing stock throughout the Bay (Table 1). On average, *A. ceratii* killed < 2% of the population per day.

DISCUSSION

Three species of *Amoebophrya* have been described as parasites of other dinoflagellates. Of these, *A. leptodisci* and *A. grassi* are more or less host-specific, as each infects dinoflagellates of a single genus. *A. leptodisci* parasitizes the heterotrophic dinoflagellate *Pratjetella medusoides,* while *A. grassi* infects parasitic dinoflagellates of the genus *Oodinium* [3]. The third species, *A. ceratii,* is not host-specific and infections have been reported in more than 20 species of free-living dinoflagellates [2, 4, 8, 9, 11, 12, 14].

Amoebophrya ceratii was originally described as a suctorian parasite of *Ceratium* spp. [11], and its dinoflagellate affinity was not recognized until the work of Chatton and Biecheler [4]. Some years later, Cachon [2] studied infections in a number of host genera and provided the first detailed analysis of the morphology and life cycle of *A. ceratii.* He reported intracytoplasmic infections in athecate dinoflagellates and intranuclear infections in thecate species (except for *Prorocentrum micans*), and noted conspicuous differences in parasite development depending on the site of infection. Specifically, chromosome replication was immediately followed by karyokinesis in parasites that developed in the host's cytoplasm, whereas division was delayed in intranuclear infections and led to the formation of a polyploid stage that had a distinctive tubular nucleus. Cachon also found considerable variation in trophont and dinospore morphology and acknowledged the possibility that infections might have been caused by more than one parasite species.

We observed very early trophonts in the cytoplasm of *Gymnodinium sanguineum,* but growth and nuclear division of the parasite started only after it had invaded the host's nucleus. Karyokinesis was not delayed in these intranuclear trophonts, and we saw no indication of the polyploid, tubular nucleus described by Cachon [2]. Thus, intranuclear infections can occur in both thecate and athecate dinoflagellates, and nuclear development of the parasite is not strictly regulated by location within the host cell. These different types of infections (viz., cytoplasmic vs. intranuclear; normal vs. delayed karyokinesis) may all be produced by *Amoebophrya ceratii* as suggested by Cachon [2], or may represent multiple parasite species. Until this issue is resolved, we believe it prudent to attribute infections of *G. sanguineum* in Chesapeake Bay to *A. ceratii.*

Chesapeake Bay populations of *Gymnodinium sanguineum* were regularly infected by *Amoebophrya ceratii* during the summers of 1988–1991; however, infection levels were generally low, with cruise averages for the surface mixed layer ranging from 0.8–6.3%. Vertically integrated estimates of host abundance were also low, with cruise averages ranging from 0.6 to 50.5 cell/ml; however, surface accumulations of $\geq$ 1,000/ml were observed at some stations. Epidemic outbreaks with 20–40% of the hosts infected by *A. ceratii* were encountered on several occasions, but were always restricted to a narrow zone near the pycnocline and several meters below peak concentrations of *G. sanguineum.* Consequently, estimates of host abundance and parasite prevalence for discrete samples were negatively correlated, while integrated station values showed no significant relationship.

The vertical separation of dense host populations and high levels of parasitism suggests that infected hosts quickly sink from surface waters; yet, in laboratory studies, hosts continued to swim throughout the infection cycle. Since many dinoflagellates including *Gymnodinium sanguineum* undergo diel vertical migrations [6, 7, 10], observed distributional patterns might reflect a loss of phototaxis during the later stages of infection. Following accumulation near the pycnocline at night, non-parasitized and recently infected hosts could return to the surface, while the lack of phototaxis in mid- to late infection would interfere with their upward migration. Alternatively, environmental conditions at the pycnocline may simply favor the growth and spread of *Amoebophrya ceratii.* In that case, parasites that we recognized as early and late infections should be equally represented near the pycnocline as these stages were of similar duration. That late trophonts of *A. ceratii* were three times more numerous than early infections at depths below 6 m indicates that parasitized hosts were either sinking out of surface waters or failing to migrate upward during the day.

The accumulation of late trophonts near the pycnocline may influence the rate at which infective dinospores encounter susceptible hosts and thereby affect host-parasite population dynamics. *Gymnodinium sanguineum* would presumably be at greatest risk to infection at night when cells migrate to the pycnocline, while near surface waters should provide a temporary refuge from parasitism during the day. The extent to which this refuge benefits host populations would depend on the longevity of dinospores and possible temporal patterns in parasite development. Nonetheless, the vertical segregation of host and parasites during the day may have contributed to the relatively low infection levels observed in Chesapeake Bay.

Our estimates of parasite-induced mortality indicate that *Amoebophrya ceratii* had minimal impact on *Gymnodinium sanguineum* populations in Chesapeake Bay. Parasite prevalence in near surface water remained low even when infection levels approached 40% near the pycnocline, and *A. ceratii* never removed more than 8% of host standing stock per day at any station. Bay-wide, *A. ceratii* utilized $\leq$ 5% of *G. sanguineum* biomass per day. This parasite may, however, have a significant impact in systems that promote high infection levels throughout the water column. For example, Nishitani et al. [14] reported an infection level of 32% in vertically integrated samples of *Gonyaulax catenella* taken from Puget Sound during the decline of a bloom. Assuming a parasite development time equivalent to that in *G. sanguineum, A. ceratii* would have removed 19% of the *G. catenella* per day. Given the relatively slow growth rate of *G. catenella* during that period (15% uninfected cells dividing per day [14], equivalent to 10% total cells dividing per day), *A. ceratii* would have removed 190% of host production and caused a 9% reduction in host abundance per day.

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