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Dinoflagellate infections of *Favella panamensis* from two North American estuaries

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Abstract *Favella panamensis* Kofoid and Campbell, 1929 is seasonally abundant in meso- to polyhaline waters of Chesapeake Bay and Indian River, Florida, USA, where it reaches densities of 10^3 cells l^{-1}. During the summers of 1986–1992, *F. panamensis* populations of the two estuaries were commonly infected by the parasitic dinoflagellate *Duboscquella aspida* Cachon, 1964. The intracellular phase of the parasite reached maturity in ~21 h (30 °C) and consumed ~35% of the host's biomass. Infections were not typically lethal to *F. panamensis*, but sometimes forced the host from its lorica. Several *D. aspida* were found in the cytoplasm of many hosts, and the number of parasites infection^{-1} was directly related to infection level. Parasite prevalence averaged 24.0 and 11.5% with mean number of parasites infection^{-1} being 1.5 and 1.3 for Chesapeake Bay and Indian River samples, respectively. *D. aspida* was estimated to remove up to 68% of host standing stock d^{-1} with a mean of ~10% for all samples. The average impact of parasitism on *F. panamensis* populations was somewhat less than would be expected from copepod grazing.

Introduction

Ciliated protozoa are widely recognized as playing a critical role in the trophodynamics of planktonic food webs (Stoecker and Capuzzo 1990; Gifford 1991; Reid et al. 1991; Pierce and Turner 1992). These small heterotrophs mediate the transfer of energy and matter from primary producers to higher trophic levels and act as a major source of nutrient regeneration. In contemporary models of the marine planktonic food web, ciliates represent a two-way link of primary production to the larger zooplankton (Sherr et al. 1988). They form a direct connection by repackaging the nanophytoplankton into a size fraction that is more readily grazed by the macrozooplankton and represent an indirect connection by forming the terminal link in a multiple step pathway between secondary bacterial production and zooplankton, the "microbial loop" (Azam et al. 1983).

Metazoan consumers are not, however, the only predators of planktonic ciliates. A variety of heterotrophic protists including fungi (Ball 1969), sarcodines (Caron and Swanberg 1990; Swanberg and Caron 1991), dinoflagellates (Hansen 1991; Schnepf and Elbrächter 1992), and other ciliates (Dolan and Coats 1991a, b) utilize ciliated protozoa as a food source. Even some typical phytoplankton species that were conventionally viewed as strict phototrophs are now known to feed on small choreotrich ciliates (Bockstahler and Coats 1993 a, b). Grazing pressure exerted on ciliates by protozoan predators may at times rival that generated by larger zooplankton. For example, epidemic infections of *Eutintinnus pectinis* by the parasitic dinoflagellate *Duboscquella cachoni* may remove in excess of 50% of the host standing stock d^{-1} in Chesapeake Bay (Coats and Heisler 1989). The annual impact of this parasite on host populations was estimated to equal that of the dominant metazoan grazer, *Acartia tonsa*.

Several other heterotrophic dinoflagellates act as intracellular parasites of loricate and aloricate ciliates (Cachon and Cachon 1987). Among these, *Duboscquella aspida* has been most thoroughly studied with infections reported for a number of tintinnids including *Coxliella lacinosa*, *Codonella campanula*, *Favella ehrenbergii*, and *Eutintinnus fraknóii* (Cachon 1964). The life cycle of *D. aspida*, as described for infections in *F. ehrenbergii* (Cachon 1964), consists of an intracellular vegetative phase followed by an extracellular reproductive/dispersal phase. During the trophont (i.e., vegetative) stage, the parasite increases in size and forms a large yellowish mass within the cytoplasm of the host. This intracellular phase of the parasite's life cy-

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cle is terminated when the trophont ruptures through the cortex of the host, and in the process phagocytizes a large portion of the ciliate's cytoplasm. Cachon noted that emergence of the trophont through the host's cortex was often, but not always, fatal to *F. ehrenbergii*. Once outside the host, the parasite undergoes a rapid series of divisions to produce numerous biflagellate dinospores that initiate new infections if ingested by another susceptible host.

While *Duboscquella aspida* is not host specific, it appears to parasitize *Favella* spp. more frequently than other tintinnids, with infection levels in *F. ehrenbergii* sometimes approaching 100% (Cachon 1964). Cachon also suggested that epidemic outbreaks of *D. aspida* in the plankton of Algiers harbor caused abrupt decreases in the abundance of *F. ehrenbergii*, even though some host organisms were capable of surviving infection.

Here we document the occurrence of *Duboscquella aspida* in populations of *Favella panamensis* from two North American estuaries, Chesapeake Bay and Indian River, Florida. Data on parasite and host morphology, parasite generation time, and in situ infection levels are used to estimate the potential impact of this parasite on host populations.

Materials and methods

Parasitism of *Favella panamensis* Kofoid and Campbell, 1929 by the dinoflagellate *Duboscquella aspida* Cachon, 1964 was studied by examining specimens collected from Chesapeake Bay and Indian River, Florida, USA. Samples from Chesapeake Bay were taken at routine stations located along the major axis of the Bay during cruises in 1986 to 1991. Station protocol followed established procedures (Coats and Heisler 1989) and included conductivity-temperature-depth profiles, Niskin bottle samples at eight to ten depths, and vertical net tows (30-μm mesh). Plankton collections containing sufficient numbers of *F. panamensis* for the present study were only obtained from the meso- to polyhaline portion of the Bay south of the Choptank River (i.e., between 38°34′ and 37°07′ N latitudes).

Samples from the Indian River, a shallow polyhaline estuary on the southeastern coast of Florida, were obtained in June and September 1991, and in August 1992. On each occasion, 18 stations evenly spaced along a 40-mile transect from Sebastian Inlet (27°52′ N lat.) to St. Lucie Inlet (27°10′ N lat.) were sampled over a 1- to 2-wk period. Whole-water and net tow (35-μm mesh) samples were collected near the surface of each station, and measurements were made for temperature and salinity.

Host abundance was determined by inverted microscopy (×200) using replicate 50-ml aliquots of whole-water samples preserved with a modified Bouin's solution (Coats and Heinbokel 1982). For assessment of parasite prevalence, host cells collected in net tows or by screening 2 to 4 liters of sample onto 20-μm Nitex were fixed in modified Bouin's and stained with acidulated alum hematoxylin (Galigher and Kozloff 1971) or by Protargol silver impregnation (Montagnes and Lynn 1987). Parasite prevalence was obtained by scoring the number of *Duboscquella aspida* present in each of 100 *Favella panamensis* sample^{-1}.

For observations of living parasites, infected ciliates were washed by micropipetting cells through several changes of 0.45-μm filtered water and individually transferred to wet-mount slide preparations. Wet mounts consisted of ~ 0.1 ml of filtered estuarine water sealed beneath a coverslip and enclosed within a vaspar ring (50% petroleum jelly; 50% paraffin) of sufficient thickness to provide an ample air space. Isolated specimes were placed in humidity chambers, held in a Percival incubator at 25 °C and periodically examined and photographed. These direct observations provided data on developmental events and the duration of sporogenesis.

Information on the population dynamics of *Duboscquella aspida* was obtained through the study of a natural host-parasite assemblage. A 120-liter cylindrical plastic tub was filled with Indian River water containing parasitized *Favella panamensis* and maintained at ambient temperature (~30 °C) in a flowing estuarine water bath. The isolated population was exposed to the natural light regime and sampled at 2- to 4-h intervals over 34 h. For each sample, 4 liters of water were concentrated on 20-μm mesh Nitex, back-flushed to 25 ml using multiple rinses, and preserved in Bouin's fluid. *F. panamensis* abundance, number of host loricae without ciliates, and number of loricae containing parasite sporogenic stages were determined by examining 2-ml aliquots of preserved sample (=320 ml whole-water) using inverted microscopy (×200). Parasite prevalence, number of parasites infection^{-1}, and the relative occurrence of four sequential stages (I–IV, respectively) in the intracellular, trophic phase of *D. aspida* were determined from hematoxylin preparations with 100 hosts examined sample^{-1}. The abundance of parasite trophonts at each sampling time was calculated by multiplying mean number of intracellular parasites host^{-1} by host density.

An estimate for the duration of the parasite's trophic phase was derived from data on the temporal occurrence of different intracellular stages of *Duboscquella aspida* during this experiment. Data analysis was similar to that used by Heinbokel (1988) for determining duration of ciliate reproductive stages. The percentages of parasites represented by stage I–II and III–IV infections were plotted separately, and the time interval during which those percentages exceeded their respective mean values was determined by linear extrapolation. The mid-point of each interval provides an objective estimate for the time of maximum occurrence of each stage, and the difference between the two mid-points equals half the intracellular development time.

The impact of parasitism on *Favella panamensis* populations, expressed as the percent of host standing stock removed d^{-1}, was calculated as:

$$V_{P/T} \times N \times D^{-1},$$

where $V_{P/T}$ is the mean value for the volume of the mature parasite divided by total parasite-host volume (=0.35); N is the number of parasites (=infection level multiplied by parasites infection^{-1}); and D is the development time for the intracellular phase of the parasite in days adjusted for sample temperature using a Q_{10} of 2.

Living and stained specimens were viewed and photographed using Zeiss microscopes equipped with brightfield, phase contrast, and differential interference contrast optics. Intracellular parasite stages were measured on hematoxylin stained cells using a filar micrometer; means are reported with standard errors (± SE) and sample sizes (n). Cell volumes for hosts and the post-phagotrophic stage of *Duboscquella aspida* were calculated using appropriate geometric formulae and cell dimensions obtained from photographic images of living specimens.

Results

Parasite life history

Four morphologically distinct stages in the trophic phase of *Duboscquella aspida* were identified in hematoxylin stained specimens. Very early infections, stage I organisms, were spherical to ovoid cells (9.6±0.5 by 8.3±0.4 μm; n=28) that had a prominent cytoplasmic vacuole and a large nucleus (6.4 ±0.4 μm diameter; n=28) containing a single nucleolus (Fig. 1). Stage II parasites (Fig. 2) were noticeably larger (17.5±0.7 by 13.2±0.4 μm; n=32), had two or more cytoplasmic vacuoles and multiple nucleoli (nuclear diameter=10.1±0.3 μm; n=32), but were otherwise simi-

lar to stage I cells. Mid- to late infections (stage III) were characterized as individuals that had highly vacuolated cytoplasm and numerous pseudopod-like protrusions of the cell surface (Fig. 3). These irregularly shaped cells measured 30.0±0.9 by 25.7±1.0 μm and had a nuclear diameter of 16.9±0.6 μm (n=30). As the trophont approached maturity (stage IV), all pseudopod-like structures were resorbed and the cytoplasm took on an homogeneous smooth to finely granular appearance (Fig. 4). Stage IV trophonts were roughly hemispherical in profile (Fig. 5) with a maximum dimension of 46.4±1.5 μm and a nuclear diameter of 23.0±0.6 μm (n=40).

The morphogenetic process resulting in the release of *Duboscquella aspida* from the host only required 3 to 4 min and usually ruptured the pedicel that anchored the tintinnid to its lorica (Fig. 6). In some instances, the host remained within the lorica as the parasite continued to develop, but often the ciliate swam out of the lorica after its pedicel was broken. The post-phagotrophic stage of the parasite averaged 66.1±2.7 μm in diameter (n=21) and contained a conspicuous food vacuole (Fig. 6). This stage of the parasite had a volume of 1.67×10^5 μm^3 (±0.2; n=21) and represented 35%±2.1 (n=21) of total host-parasite biomass; host biovolume after emergence of the parasite was 3.19×10^5 μm^3 (±0.4; n=21). Sporogenesis (Figs. 7 to 9) lasted 4.9±0.7 h (n=7) at 25 °C and gave rise to 2000–3000 dinospores (Fig. 10) that averaged 7.0±0.1 by 4.2±0.1 μm (n=20). We could not count dinospores accurately in wet-mount preparations, but the volume ratio for post-phagotrophic parasites and dinospores indicated that ca. 2600 dispersal cells were formed infection^{-1}.

When followed in the laboratory, the transition of the parasite from intracellular to extracellular phase did not kill the host. The ability of *Favella panamensis* to survive infections was also evident in plankton samples, where sporogenic stages were frequently observed in the loricae of actively swimming hosts that, aside from being small, had a normal appearance. Sporogenic stages of *Duboscquella aspida* were sometimes observed in loricae that were not inhabited by a host. In such cases, the ciliate may have been killed by the infection, or may have swum out of the lorica after the pedicel was severed by the parasite.

Parasitism did not appear to prevent reproduction of the host, as cytological preparations revealed that all stages of the ciliate's cell cycle including mitosis could harbor well developed infections. Interestingly, food vacuoles of post-phagotrophic parasites sometimes contained one of the host's macronuclei. The reproductive competency of individual *Favella panamensis* that have lost one or more macronuclei is unknown.

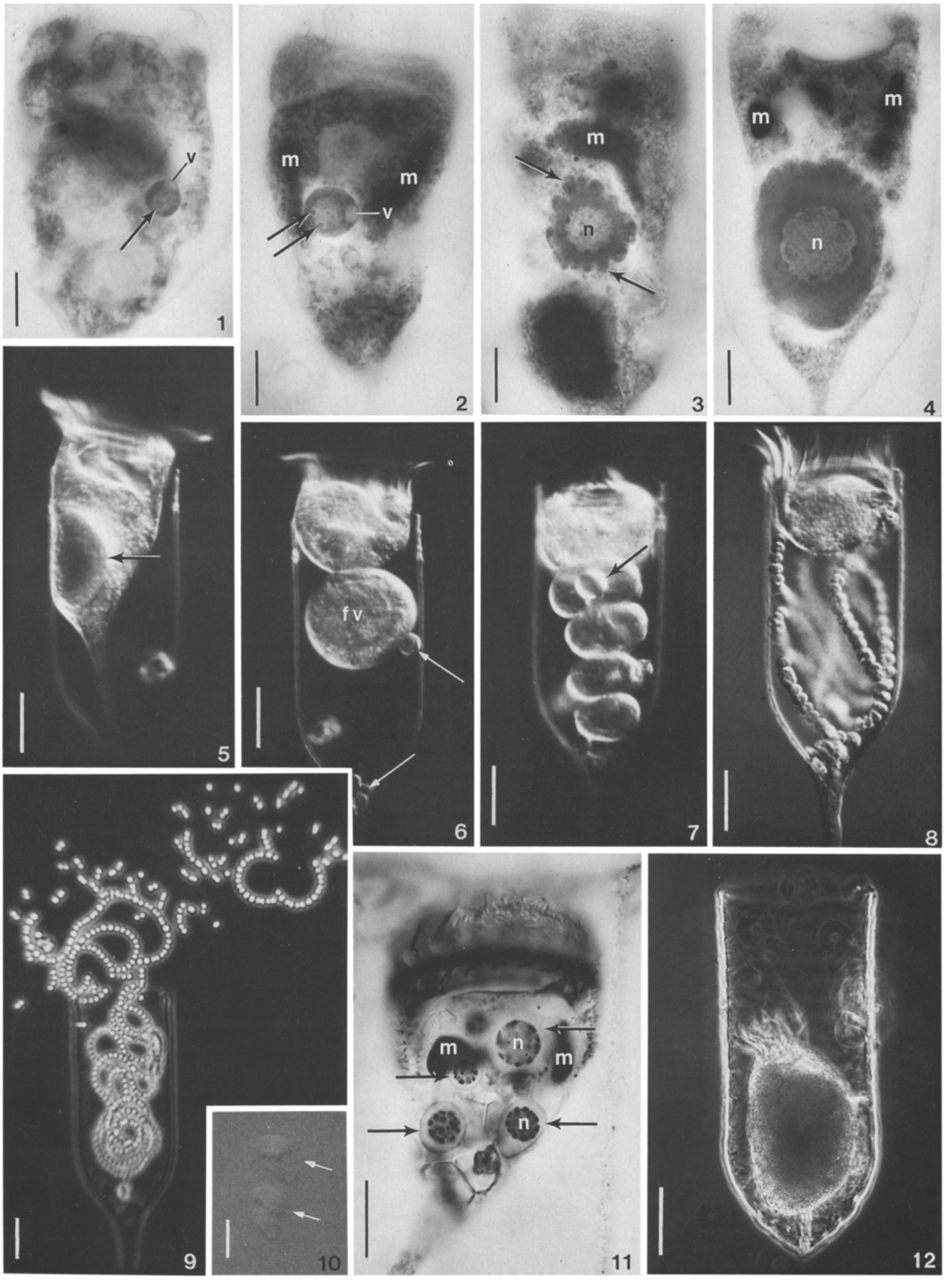
Favella panamensis was often infected by multiple parasites (Fig. 11), with up to 13 *Duboscquella aspida* observed in a single host. Multiple infections were manifested as either several parasites of the same developmental stage or parasites in two or more stages. In some instances, the cytoplasm of host organisms contained trophonts of *D. aspida* in all four stages of development, while outside the ciliate, but still within the lorica, another parasite was undergoing sporogenesis.

Host-parasite population dynamics

Information on the population dynamics of *Favella panamensis* and *Duboscquella aspida* was obtained by monitoring a natural host-parasite assemblage during a 34-h incubation study (ambient temperature ~30 °C). The study was initiated in late afternoon (17:00 hrs, ~3 h before sunset) using Indian River plankton that contained 240 hosts l^{-1} with an infection level of 31%. Parasite prevalence decreased to about a third of the initial level over the 7.5 h of the incubation (T_0 to $T_{7.5}$), then showed a step increase to ~40% near the end of the first dark period, followed by a second jump to ~70% at the beginning of the second dark period (Fig. 13A). By comparison, *F. panamensis* steadily increased in number early in the incubation and reached a maximum density of ~1200 cells l^{-1} at T_{16} (Fig. 13A); doubling time =6.7 h. No growth of the host population was evident following the first increase in parasite prevalence, and an abrupt decline in *F. panamensis* abundance to ~400 cells l^{-1} coincided with the second rise in infection level. While the percent of hosts infected by *D. aspida* decreased between T_0 to $T_{7.5}$, the absolute number of intracellular and extracellular stages of the parasite present during this period remained relatively constant (Fig. 13B). Thus, the decline in parasite prevalence probably resulted from growth of the host population without the spread of infections, rather than from the loss of parasites.

Duboscquella aspida exhibited two distinct peaks in sporogenesis, with each peak followed by a sharp rise in the abundance of intracellular parasite stages (Fig. 13B). The short lag between sporogenesis and the subsequent occurrence of new infections (3 and 6 h for the first and second peak, respectively), along with the stepwise increase in infections, suggests that dinospores of *D. aspida* disperse rapidly, but have a relatively narrow interval during which they are competent to establish infections (cf. Fig. 13A, B). Were each of the parasites undergoing sporogenesis during the first peak (~30 l^{-1} at $T_{12.5}$) to release 2600 dinospores as estimated above, then successful infection of hosts by 8 to 10% of the dinospores would account for the number of intracellular parasites observed in subsequent samples (630 to 830 l^{-1} between T_{16} and $T_{21,5}$; Fig. 13B).

The presence of well defined peaks in sporogenesis of *Duboscquella aspida* indicates that at least a portion of the parasite population was developing in synchrony. Additionally, a pronounced oscillation in the relative occurrence of early (stage I & II) and late (stage III & IV) trophonts of *D. aspida* was evident from the onset of the incubation and provides further evidence for phased development of the parasites (Fig. 13C). Mean values for percent early and late trophonts during the incubation were 49.0±6.4 and 43.7±5.5 (n=13), respectively (mean parasites in sporogenesis =7.3±2.1%). These values are indicated by the horizontal lines that intersect the two plots of Fig. 13C. The intervals indicated by these lines represent the period when successive data for percent early and late infections exceeded their mean values. The mid-point of each interval is marked by a short vertical line, and the time increment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between mid-points (10.3 h) is an objective estimate for half the duration of the parasite's intracellular phase. Thus, the time from initial infection until *D. aspida* erupted through the cortex of *Favella panamensis*, was ~21 h at 30 °C.

At the beginning of the incubation, 23% of infected hosts (=7% of all hosts) were parasitized by more than one *Duboscquella aspida* with an average of 1.3±0.09 (n=31) parasites infection^{-1}. Multiple infections became progressively less common prior to the first peak in sporogenesis but were frequently encountered thereafter with a maximum of 59% (at T_{34}) of host cells containing multiple parasites. Values for number of parasites per infected *Favella panamensis* ranged from 1.0 at T_{11} to 2.2 at T_{34} and showed a strong correlation with infection level (Fig. 14).

The loricae of all *Favella panamensis* examined prior to the first peak in sporogenesis had a structure that was typical for the species (i.e., goblet-shaped with uniform wall thickness and a distinctive aboral horn; see Fig. 8); however, at $T_{12.5}$, a number of *F. panamensis* had spiral-walled loricae that lacked an aboral horn (Fig. 12). The occurrence of spiralled loricae was positively correlated with parasite prevalence (r=0.85; p<0.01; n=13) and reached a frequency of 9.8% at T_{34}.

Parasite prevalence in situ and impact on host populations

The occurrence of *Duboscquella aspida* in Chesapeake Bay and Indian River populations of *Favella panamensis* was determined for samples where host abundance was ≥10 cells l^{-1}. Concentrations of *F. panamensis* above 10 cells l^{-1} were only encountered in the mid- to lower part of Chesapeake Bay at salinity of 14 to 27‰ and temperature of 18 to 29 °C. By contrast, dense patches of hosts were observed throughout the Indian River study area where salinity ranged from 21 to 33‰ and temperature was 28 to 32 °C.

Data on parasite prevalence were obtained for 36 Indian River samples of which 13 were collected in June and September of 1991 and 23 in September 1992. Infection levels were typically between 1 and 40%; however, parasites were below detectable levels (<1%) on seven occasions and infected 69% of the hosts in one sample; mean parasite prevalence was 14.4% (Table 1). Infection levels for 1991 and 1992 samples were not significantly different, averaging 12.5±5.3 and 15.5±2.3, respectively.

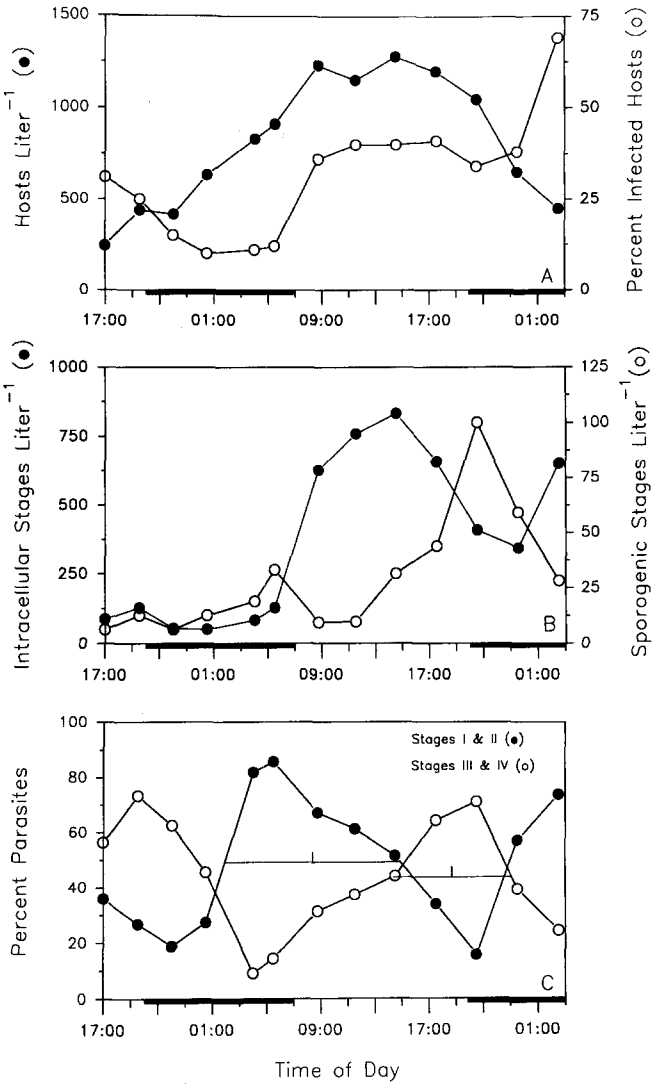

Fig. 13 *Favella panamensis* parasitized by *Duboscquella aspida*. Incubation study of Indian River plankton; interval between sunset and sunrise indicated by dark area on abscissa. (A) *F. panamensis* density and parasite prevalence. (B) Abundance *D. aspida* present as intracellular (trophonts) and extracellular (sporogenic) life history stages. (C) Relative occurrence of early (stage I and II) and late (stage III and IV) trophonts of *D. aspida*. Horizontal lines represent interval when successive data for percent early and late trophonts exceeded their mean values (49 and 44%, respectively); short vertical line indicates mid-point of each interval. Time between mid-points (10.3 h) equals half the duration of the parasite's intracellular phase

Figs. 1–12 *Favella panamensis* infected by *Duboscquella aspida*. **Fig. 1** Stage I of trophont development showing a conspicuous nucleolus (arrow) and vacuole (v); hematoxylin stain; scale =20 µm. **Fig. 2** Stage II parasite with multiple nuclei (arrows) and large vacuole (v); macronuclei of host (m); hematoxylin stain; scale =20 µm. **Fig. 3** Stage III trophont with numerous pseudopod like extensions (arrows); parasite nucleus (n); host macronucleus (m); hematoxylin stain; scale =20 µm. **Fig. 4** Stage IV in the intracellular phase of *D. aspida*; parasite nucleus (n); host macronuclei (m); hematoxylin stain; scale =20 µm. **Fig. 5** Stage IV trophont (arrow) just before emergence from the host; differential interference contrast (DIC) microscopy of a living specimen; scale =40 µm. **Fig. 6** Same specimen as Fig. 5 immediately after emerging from the host; parasite food vacuole (fv); pieces of the ruptured pedicel (arrows); DIC live specimen; scale =40 µm. **Fig. 7** Four-cell stage of sporogenesis; parasite food vacuole (arrow); DIC live specimen; scale =40 µm. **Fig. 8** Late sporogenesis of *D. aspida* with host still occupying lorica; DIC live specimen; scale = 40 µm. **Fig. 9** Release of sporocytes from host lorica; phase contract microscopy of live specimen; scale =40 µm. **Fig. 10** Mature macrospores showing bipartite structure and transverse flagella (arrows); DIC live specimens; scale =10 µm. **Fig. 11** *F. panamensis* infected by four trophonts (arrows) of *D. aspida;* parasite nucleus (n); host macronuclei (m); Protargol silver-impregnated specimen; scale =20 µm. **Fig. 12** *F. panamensis* with spiralled lorica; Bouin's fixed specimen; phase contrast microscopy; scale =40 µm.

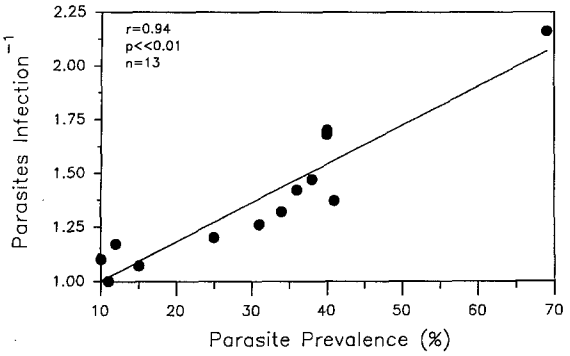

Fig. 14 *Duboscquella aspida* infection of *Favella panamensis*. Correlation between the number of parasites per infected host and parasite prevalence during incubation of Indian River plankton

Table 1 *Favella panamensis* infected by *Duboscquella aspida*. Infections in *F. panamensis* collected from the Indian River and Chesapeake Bay

	Host abundance	Parasite prevalence	Parasites infection^{-1}	% standing stock removed d^{-1}
Indian River, Florida (n=36)				
Median	175	11.5	1.3	6.9
Mean	230	14.4	1.3	8.5
SE	36	2.4	0.06	1.6
Range	10–1070	0–69	1.0–2.0	0–50
Chesapeake Bay (n=8)				
Median	115	24.0	1.7	7.3
Mean	120	32.1	1.5	18.3
SE	29	11.1	0.16	8.0
Range	10–220	0–86	1.1–2.1	0–68

Only eight Chesapeake Bay samples had sufficient numbers of *Favella panamensis* to determine parasite prevalence. *Duboscquella aspida* was detected in five of these collections with infection level exceeding 50% on three occasions; mean parasite prevalence was 32.1% (Table 1). Average infection level in *F. panamensis* from Chesapeake Bay was about twice that of Indian River populations; however, mean values for the two estuaries were not significantly different and the average for all samples was 17.6±2.7; n=44. Also, parasite prevalence was not correlated with host density (p>0.05) in either system.

Multiple infections were common in *Favella panamensis* collected from both estuaries with as many as 53% of infected hosts containing more than one parasite (mean =20±2.9%, n=34). As in the preceding incubation study, the number of *Duboscquella aspida* per infected host was closely correlated with parasite prevalence (r=0.65; p<0.01; n=34). Mean number of parasites infection^{-1} ranged from 1.0 to 2.1 with Indian River and Chesapeake Bay populations averaging 1.3 and 1.5, respectively (Table 1).

Estimates for the impact of parasitism on *Favella panamensis* populations, expressed as percent of host standing stock removed d^{-1}, are presented in Table 1. Mean values for Chesapeake Bay and Indian River were not significantly different (18.3±8.0 and 8.5±1.6, respectively), and the average for all samples was 10.3±2.1; n=44. On the four occasions on which parasite prevalence exceeded 50%, *Duboscquella aspida* was estimated to remove 28 to 68% of the host's standing stock d^{-1}.

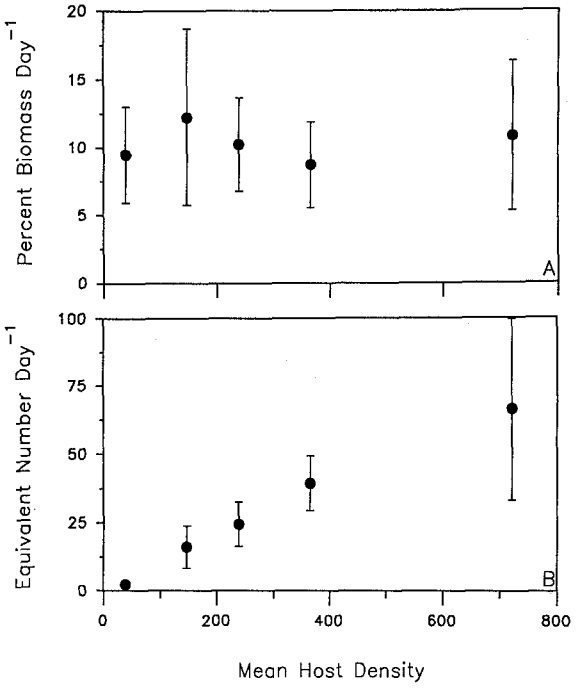

Fig. 15 *Duboscquella aspida* impact on *Favella panamensis*. (A) Proportion of host standing stock removed d^{-1} as a function of mean host density. (B) Biomass consumed by the parasite expressed as an equivalent number of host cells and plotted against mean host density. Mean host densities are for Indian River and Chesapeake Bay data grouped by host abundance into five categories: ≤100 l^{-1} (n=15); >100 to 200 l^{-1} (n=10); >200 to 300 l^{-1} (n=10); >300 to 400 l^{-1} (n=5); >400 l^{-1} (n=4). Error bars represent standard error of the mean

When grouped according to host abundance, estimates for the proportion of *Favella panamensis* standing stock removed by *Duboscquella aspida* had comparable averages at mean host densities of 40 to 725 cells l^{-1} (Fig. 15A). The total amount of host biomass utilized by the parasite increased with mean host density and was equivalent to removing 2 to 70 *F. panamensis* d^{-1} (Fig. 15B).

Discussion

Loricate choreotrichs of the genus *Favella* are predominantly neritic ciliates that are most common in temperate waters (Campbell 1942; Pierce and Turner 1993) and often associated with dinoflagellate blooms (Stoecker et al. 1981, 1984; Sellner and Brownlee 1990). Most *Favella* spp. are large and densities rarely exceed 10^3 cells l^{-1}, yet they are a consistent and potentially important component of the microzooplankton in many areas. *Favella* spp. are

known to feed on dinoflagellates in preference to other phytoplankton taxa and are, in turn, a choice prey of copepods (Robertson 1983; Stoecker and Sanders 1985; Ayukai 1987; Stoecker and Egloff 1987). Ciliates of this genus are also consumed by gelatinous zooplankton, decapod zoea, and fish (Robertson 1983; Stoecker and Govoni 1984; Stoecker et al. 1987 a, b).

Favella spp. have long been known to harbor parasitic dinoflagellates including *Duboscquella aspida* (Duboscq and Collin 1910; Chatton 1920; Cachon 1964), but the effect of parasitism on host populations has been largely overlooked. Cachon (1964) was the first to point out potential ecological consequences of parasitic dinoflagellates when he suggested that epidemic outbreaks of *D. aspida* might regulate the abundance of *Favella ehrenbergii*. More recently, dinoflagellate infections have been linked to low in situ growth rates of an unidentified species of *Favella* (Stoecker et al. 1983). Coats and Heisler (1989) have shown parasitic dinoflagellates to be widespread in Chesapeake Bay, where parasite-induced mortality of another loricate ciliate, *Eutintinnus pectinis*, averaged ~10% of the host standing stock d^{-1}.

The dinoflagellates infecting *Favella panamensis* in the current study had morphological features that corresponded closely to the description of *Duboscquella aspida* (Cachon 1964) for infection in *F. ehrenbergii*; however, various aspects of parasite development differed between the two host species. Some of these inconsistencies probably reflect the different temperatures at which observations were made (25 to 30 °C in our study; ~20 °C for Cachon's work). For example, the intracellular phase of the parasite had a duration of ~21 h in *F. panamensis* with sporogenesis requiring ~5 h, whereas trophont development and sporogenesis were reported to take 3 to 4 d and 2 to 3 d, respectively, in *F. ehrenbergii* (Cachon 1964). Other discrepancies may reflect strain variations in the parasites or differences in host–parasite interactions. For example, Cachon (1964) reported two size classes of dinospores for infections of *F. ehrenbergii* with each parasite producing either ~1000 macrospores (6 to 7 μm long) or >50000 microspores (2 to 3 μm). Only macrospores (7 × 4 μm) were formed by parasites of *F. panamensis* with each infection liberating 2000 to 3000 daughter cells. Also, infections usually killed *F. ehrenbergii* (Cachon 1964), but were often not lethal to *F. panamensis*.

Progression of *Duboscquella aspida* from an intracellular to the extracellular phase of the life cycle was not typically lethal to *Favella panamensis* under laboratory conditions, although emergence of the parasite usually severed the pedicel that anchored the host to its lorica. That *F. panamensis* also survives infection in the field was evident from the occurrence of loricae containing host cells and sporogenic stages of the parasite. *F. panamensis* was sometimes dislodged from its lorica as the parasite underwent sporogenesis, and loricae containing only parasite developmental stages were not uncommon in field samples. Cachon (1964) suggested that absence of a host cell and presence of parasites indicated that *D. aspida* was lethal to *F. ehrenbergii*. Alternatively, this situation may simply result from abandonment of the lorica once the pedicel of the ciliate is severed. Laval-Peuto (1981) has shown that *F. ehrenbergii*, when removed from its lorica, forms a new lorica that has a distinctive spiralled appearance. The presence of spiralled loricae in *F. panamensis* was positively correlated with infection level and suggests that hosts forced from their loricae by developing parasites survive to form new loricae.

Multiple infections with as many as 13 parasites in an individual host were frequently encountered in *Favella panamensis*. The number of parasites $infection^{-1}$ was positively correlated with infection level, and at maximum infection level, each infected host had an average of two parasites. In some instances, all the infections were of equivalent age (i.e., same stage of trophont development) and were probably established within a few hours of each other. Hosts that survive infection and remain with their loricae would be exposed to a large number of infective stages over a very short period and could develop multiple infections of the same age. Alternatively, the infective dinospores of the parasite might remain clustered after leaving the lorica and be encountered in patches. Other multiple infections contained parasites of differing age. These probably resulted from sequential acquisition of infections; however, interaction among trophonts might retard the growth of some *Duboscquella aspida* and shift the apparent age distribution of the parasites.

Favella panamensis populations of Chesapeake Bay and Indian River, Florida were often heavily infected by *Duboscquella aspida*, with maximum parasite prevalence of 69 and 86% for the two estuaries, respectively. These values are substantially higher than peak parasite prevalence reported for *D. cachoni* infections in *Eutintinnus pectinis* populations of Chesapeake Bay (Coats and Heisler 1989). Infection levels above 50% were never observed in *E. pectinis*, but were encountered in ~10% of *F. panamensis* samples. That *F. panamensis* commonly survives infections and is subject to reinfection may explain the frequent occurrence of very high parasite prevalence. *E. pectinis* on the other hand is always killed by its parasite (Coats and Heisler 1989). Thus, very high infection levels in that host species could persist for only a short time and would be infrequently encountered.

The impact of *Duboscquella aspida* on *Favella panamensis* populations, expressed as percent host standing stock removed d^{-1}, averaged 18.3 and 8.5 for Chesapeake Bay and Indian River samples, respectively, with epidemic infections (>50%) cropping 28 to 68% of host standing stock d^{-1}. By comparison, Coats and Heisler (1989) estimated 7 to 24% of the *Eutintinnus pectinis* population of Chesapeake Bay was removed by *D. cachoni*. While *F. panamensis* supports higher infection levels and has more parasites $infection^{-1}$ than *E. pectinis*, trophonts of *D. aspida* only utilize ~35% of the host cell, whereas those of *D. cachoni* consume the entire host. As a consequence, the effect of parasitism on standing stocks of the two host species is roughly comparable.

Estimates for impact of parasitism on *Favella panamensis* populations assumed that all parasites, whether present

as single or multiple infections, matured to the post-phagotrophic stage, had comparable development times, and utilized the same proportion of host biomass. These would be over-estimated should older trophonts of multiple infections decrease the success or increase development time of younger parasites. A more conservative approach would be to assume that only one parasite of each multiple infection reaches maturity, which would reduce the impact of *D. aspida* by an average of 34%. Our estimates also assume that parasite prevalence was independent of sampling time, but the incubation study of a natural plankton assemblage showed significant temporal fluctuation in infection levels associated with phased development and spread of infections. Decreasing infection levels in the evening were followed by abrupt increases in parasite prevalence at night and relatively steady levels of parasitism through the day. If this pattern is characteristic of field populations, our estimates of parasite impact should not be biased as all samples were collected during the day.

Ingestion rates for copepods feeding on *Favella* spp. range from 10 to ~264 prey copepod^{-1} d^{-1} at prey densities of 250 to 3400 l^{-1} (Stoecker and Sanders 1985; Ayukai 1987; Stoecker and Egloff 1987). Robertson (1983) reported values of 25 and 90 prey copepod^{-1} d^{-1} for *Acartia tonsa* feeding on *F. panamensis* at 250 and 1000 cells l^{-1}, respectively, and 160 *F. panamensis* copepod^{-1} d^{-1} for *Tortanus setacaudatus* exposed to 1000 prey l^{-1}. Utilization of host biomass by *Duboscquella aspida* was equivalent to removing an average of 2 to 70 *F. panamensis* d^{-1} at mean host densities of 40 to 720 cells l^{-1}. Thus, the average effect of parasitism of *F. panamensis* standing stock was comparable to the grazing pressure exerted by a copepod density of one individual l^{-1}. Since copepod densities often exceed one individual l^{-1} in enriched coastal systems (e.g. *A. tonsa* abundance in Chesapeake Bay during summer ranges from 4 to 20 copepods l^{-1}; Brownlee and Jacobs 1989), *D. aspida* would have a lower impact on *F. panamensis* populations than would metazoan grazers, except during epidemic outbreaks when the parasite is capable of removing 68% of *F. panamensis* standing stock d^{-1}.

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