

# Effects of parasitism on diel vertical migration, phototaxis/geotaxis, and swimming speed of the bloom-forming dinoflagellate *Akashiwo sanguinea*

Myung Gil Park<sup>1,\*</sup>, Sean K. Cooney<sup>1</sup>, Jae Seong Kim<sup>2</sup>, D. Wayne Coats<sup>1</sup>

<sup>1</sup>Smithsonian Environmental Research Center, PO Box 28, Edgewater, Maryland 21037, USA

<sup>2</sup>Department of Oceanography, College of Ocean Science and Technology, Kunsan National University, Kunsan 573-701, Republic of Korea

**ABSTRACT:** Eukaryotic parasites are believed to play important roles in bloom dynamics of red-tide dinoflagellates; however, little is known about their impact on host behavior. To address this issue, we examined the influence of the parasitic dinoflagellate *Amoebophrya* sp. on diel vertical migration (DVM), phototaxis/geotaxis, and swimming speed of the bloom-forming dinoflagellate *Akashiwo sanguinea*. Uninfected *A. sanguinea* exhibited strong DVM, yielding subsurface aggregations during the day and evenly dispersed populations at night. DVM of parasitized *A. sanguinea* was comparable to that of uninfected host for much of the infection cycle; however, subsurface accumulations of host cells were dramatically reduced or eliminated as parasites matured. The absence of DVM in the latter part of the infection cycle appeared to be associated with changes in phototactic response and swimming speed of parasitized cells. While uninfected *A. sanguinea* exhibited strong positive phototaxis, hosts with late-stage infections exhibited strong negative phototaxis. By contrast, infected and uninfected *A. sanguinea* did not differ in geotaxis or sinking. Swimming speed of infected *A. sanguinea* decreased with parasite age, differing significantly from that of uninfected hosts 24 h after initiation of infections. *A. sanguinea* with late-stage infections exhibited a 37.3% lower swimming speed than uninfected hosts. Results indicate that parasitism by *Amoebophrya* sp. disrupts 'internal' factors that control DVM in dinoflagellates. Data also suggest that modification of host behavior by parasitism may contribute to vertical variations in phytoplankton species-composition and -abundance, particularly during epidemics when late-stage infections are prevalent.

**KEY WORDS:** Parasitism · Diel vertical migration · Taxis · Swimming speed · Behavior · Red tide · *Akashiwo sanguinea*

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## INTRODUCTION

Parasitic dinoflagellates have long been thought to play important roles in bloom dynamics of red-tide dinoflagellates (for recent review, see Elbrächter & Schnepf 1998, Coats 1999). The genus *Amoebophrya* has received particular attention, as it is widely distrib-

uted in coastal environments and has been reported from numerous host species (Cachon 1964, Taylor 1968, Elbrächter 1973, Nishitani et al. 1985, Cachon & Cachon 1987, Fritz & Nass 1992, Coats & Bockstahler 1994, Coats et al. 1996, Maranda 2001). Prior studies have emphasized parasite morphology and life history (Coats & Bockstahler 1994), prevalence and distribution of infections (Taylor 1968, Elbrächter 1973, Nishitani & Chew 1984, Nishitani et al. 1984, 1985, Fritz & Nass 1992, Coats & Bockstahler 1994, Coats et al. 1996), and impact of parasitism on host populations (Coats & Bockstahler 1994, Coats et al. 1996). Recently,

\*Present address: Red Tide Research Center, Kunsan National University, Kunsan 573-701, Republic of Korea. E-mail: myungpark@kunsan.ac.kr

Yih & Coats (2000) examined the influence of host quality on the success of *Amoebophrya* sp. ex *Akashiwo sanguinea* and suggested that the parasite was well adapted to exploit dinoflagellate populations in nutrient enriched environments. Further, Park et al. (2002) found that photosynthetic performance and photophysiological properties of the bloom-forming dinoflagellates *A. sanguinea* and *Gymnodinium instriatum* were significantly altered following infection by parasites of the genus *Amoebophrya*. Despite growing interest in the ecological importance of parasites in red-tide dinoflagellates (Anderson 1997, Coats 1999, Norén et al. 1999, Erard-Le Denn et al. 2000), little is known about the influence of parasitism on host behavior.

This study was undertaken to test the hypothesis that parasitism by *Amoebophrya* sp. alters host behavior. To test the hypothesis, we compared diel vertical migration (DVM), phototaxis/geotaxis, and swimming speed of infected and uninfected cultures of *Akashiwo sanguinea*. The host-parasite system was isolated from Chesapeake Bay and is the same as that previously referred to as *Amoebophrya ceratii* ex *Gymnodinium sanguineum* (Coats et al. 1996), *Amoebophrya* sp. ex *Gymnodinium sanguineum* (Gunderson et al. 1999, Yih & Coats 2000), and *Amoebophrya* sp. ex *Akashiwo sanguinea* (Park et al. 2002).

## MATERIALS AND METHODS

**Laboratory cultures.** Chesapeake Bay isolates of *Akashiwo sanguinea* (formerly *Gymnodinium sanguineum*) and the parasitic dinoflagellate *Amoebophrya* sp. were maintained as stock cultures in  $\frac{1}{2}$ -Si medium (Guillard & Ryther 1962) formulated using 15‰ bay water plus soil extract (5% v/v). Parasite cultures were propagated by sequentially transferring aliquots of infected *A. sanguinea* into uninfected host culture at 2 to 3 d intervals. Stock and experimental cultures were maintained at 20°C under a 14 h light:10 h dark cycle of cool-white fluorescent light at an irradiance of 175  $\mu\text{mol photons m}^{-2} \text{s}^{-1}$ .

**Diel vertical migration.** Vertical migration of infected and uninfected *Akashiwo sanguinea* was examined using populations maintained in 2 l plastic DVM columns measuring 46 cm in height and 8 cm in diameter. Each DVM column was wrapped with black tape and had sampling ports spaced at 4.5 cm intervals from 1 cm below the top (Port 1), to the bottom (Port 11) of the column. Ports were equipped with short tubes to permit collection of samples from the center of the column. A 12th refill port for addition of culture medium was positioned at the bottom of the column opposite sampling Port 11. Columns were maintained in a walk-

in incubator at 20°C and illuminated from above using cool-white fluorescent light bulbs on a 14 h light:10 h dark cycle. Light intensity decreased exponentially from 80  $\mu\text{mol photons m}^{-2} \text{s}^{-1}$  at the uppermost sampling port to near zero at the bottom of the column.

Prior to initiation of the experiment, 800 ml of *Akashiwo sanguinea* culture in exponential growth was brought to 8000 ml by the addition of 15‰ filtered (Whatman GF/F) and sterilized Chesapeake Bay water. The 10-fold dilution of stock culture resulted in a host density of approximately 250 cells  $\text{ml}^{-1}$  and reduced growth medium to  $\frac{1}{20}$  nutrient concentrations. The diluted culture was gently mixed, equally distributed to 4 DVM columns, and allowed to acclimate for ca. 5 d. At circadian time (CT) 19 on the fifth day (CT is the time corresponding to the hour after the onset of the light period; during this study, the lights came on at CT 0 and went off at CT 14), 10 ml aliquots were taken from sampling Ports 1, 3, 5, 7, 9, and 11 of each column, and 60 ml of 15‰ sterile, filtered bay water was then added through the refill port. Samples were preserved with acid Lugol's fixative and used to determine *A. sanguinea* abundance following the methods of Park et al. (2002). At CT 20, 2 columns were inoculated with recently formed ( $\leq 6$  h old) dinospores of *Amoebophrya* sp., while the other 2 columns served as uninfected controls. Recently formed dinospores were harvested and their abundance determined following established procedures (Park et al. 2002). Dinospores were injected uniformly through all 11 sampling ports of the 2 treatment columns to yield a dinospore:host ratio of 20:1, while equal volumes of dinospore filtrate (Whatman GF/F) were added through each port of the 2 control columns.

The vertical distribution of *Akashiwo sanguinea* in the 4 columns was determined 23 h (CT 19) after columns were inoculated and at approximately 5 h intervals thereafter (sampling times were CT 0, 4.5, 9, 14, and 19). At each sampling period, 10 ml aliquots were taken from Ports 1, 3, 5, 7, 9, and 11 (depths of 1, 10, 19, 28, 37, and 46 cm) of each column, and an equivalent volume (60 ml) of 15‰ sterile, filtered bay water was replaced through the refilled port. Samples were preserved with modified Bouin's fixative (Coats & Heinbokel 1982) and used to obtain host abundance and parasite prevalence following the methods of Park et al. (2002). Samples were stained with quantitative protargol staining (Montagnes & Lynn 1993) and infections were classified as early and late following the criteria of Coats & Bockstahler (1994).

**Phototaxis, geotaxis, and sinking.** To examine phototaxis, geotaxis, and sinking of infected and uninfected *Akashiwo sanguinea*, 4 DVM columns (2 treatments and 2 controls) were set up following the protocol described above. Sixty-two hours after the columns

were inoculated (CT 9), a 400 ml sample was taken through Port 3 of each column. Each sample was gently mixed and used to fill six 21 ml glass test tubes that had been wrapped with black tape. Three tubes per column were sealed with clear plastic film for the phototaxis study, while the other 3 were covered with metal caps for the geotaxis experiment. The remaining sample from each column was preserved with CaCO<sub>3</sub> buffered formalin (1% final concentration) and distributed to triplicate metal-capped tubes to determine sinking.

The 12 plastic-sealed 'phototaxis' tubes were placed horizontally on a shelf in a 20°C incubator, with the transparent end of the tubes facing cool-white fluorescent light bulbs. Irradiance at the transparent end of the tubes was equivalent to that available 10 cm below the surface (Port 3) of the DVM columns (i.e. 25 μmol photons m<sup>-2</sup> s<sup>-1</sup>). The 12 metal-capped 'geotaxis' tubes were placed vertically on an adjacent shelf, while the 12 metal-capped 'sinking' tubes were held in the laboratory at room temperature (ca. 20°C). After a 2 h incubation, the phototaxis tubes were turned upright, and the fluid from the upper, middle, and lower thirds of each tube was transferred via syringes to 3 separate 20 ml glass scintillation vials. The geotaxis tubes were incubated for only 30 min to minimize the effects of sinking but were sampled following the same protocol. Sinking tubes were likewise sampled after 30 min. Samples for phototaxis and geotaxis experiments were preserved with Bouin's fixative for determination of host abundance, parasite prevalence, and parasite developmental stage as above. *Akashiwo sanguinea* densities were similarly obtained for formalin-fixed samples from the sinking experiment.

**Swimming speed.** Two 500 ml flasks containing 250 ml of *Akashiwo sanguinea* culture at ca. 1000 cells ml<sup>-1</sup> were used to assess swimming speed of infected and uninfected host cells. One flask was inoculated at a dinospore:host ratio of 10:1, while the other served as an uninfected control. Flasks were maintained under growth conditions used for stock cultures and sampled at 8 h intervals after inoculation. At each sampling period, 10 ml aliquots were taken from each flask and preserved with Bouin's fixative for determination of parasite prevalence and stage of infection. In addition, 10 subsamples (1 ml each) were transferred from each flask to separate multi-well plates. Multi-well plates were examined using a Lica/Wild stereoscope equipped with darkfield illumination and focused near the vertical center of the sample. Specimens swimming in the plane of focus of each 1 ml subsample were videotaped over a 2 min period using a Sony DXC-760MD camera and JVC VHS Model HR-S4500U recorder capturing 30 frames s<sup>-1</sup>. Tapes were viewed on a Sony Trinitron PVM

1344Q monitor (140× total magnification at viewing screen) using frame-by-frame playback. Specimens that maintained a linear path for ≥1 s were traced onto acetate overlays. Swimming speed was determined from the distance traversed in 1 s, with care taken to analyze each specimen only once. This approach provided estimates of swimming speed for 10 specimens for each of 10 wells, with data obtained for 100 specimens for each flask at each sampling time.

**Statistical analysis.** For comparison of cell densities in replicate tubes of the taxis and sinking studies, data were log<sub>10</sub> transformed to meet assumptions of normality and homogeneity of variance and then compared by 2-way ANOVA, with DVM column and replicate number being independent variables and cell density being the dependent variable. Percentage data for infected treatments and uninfected controls of the phototaxis, geotaxis, and sinking experiments were arcsine square-root transformed and compared by 2-way ANOVA, where tube position and presence or absence of parasites were independent variables and percentage of cells was the dependent variable. Data for swimming speeds of cells from infected treatments and uninfected controls were also compared by 2-way ANOVA, where elapsed time and presence or absence of parasites were independent variables and swimming speed was the dependent variable. Statistical comparisons were made using SigmaStat 3.0 (SPSS). Unless otherwise stated, data are reported as mean ± standard error of the mean (SEM) for untransformed data from duplicate DVM columns.

## RESULTS

### Diel vertical migration

Mean abundance of *Akashiwo sanguinea* in treatment (infected) and control (uninfected) DVM columns was 170 ± 10.9 and 94 ± 23.9 cells ml<sup>-1</sup>, respectively, at T<sub>23</sub> (where T<sub>n</sub> is the time in hours after the start of the experiment). By the end of the experiment (T<sub>71</sub>), *A. sanguinea* abundance had declined to 73 ± 14.6 and 49 ± 7.4 cells ml<sup>-1</sup> in the treatment and control groups, respectively, due to removal of cells during sampling and parasite induced mortality of hosts. Parasite prevalence increased quickly in infected treatments, with all samples having 100% infection levels from T<sub>18</sub> to the end of the experiment. The proportion of parasitized hosts exhibiting late-stage infections increased dramatically between the first and second light cycles (from 8 ± 2.5% at T<sub>42</sub> to 69 ± 2.5% at T<sub>56.5</sub>) and reached 83 ± 2.0% by the end of the second light period (T<sub>66</sub>).

Uninfected *Akashiwo sanguinea* had strong DVM over the 2 light:dark cycles, with subsurface (10 cm

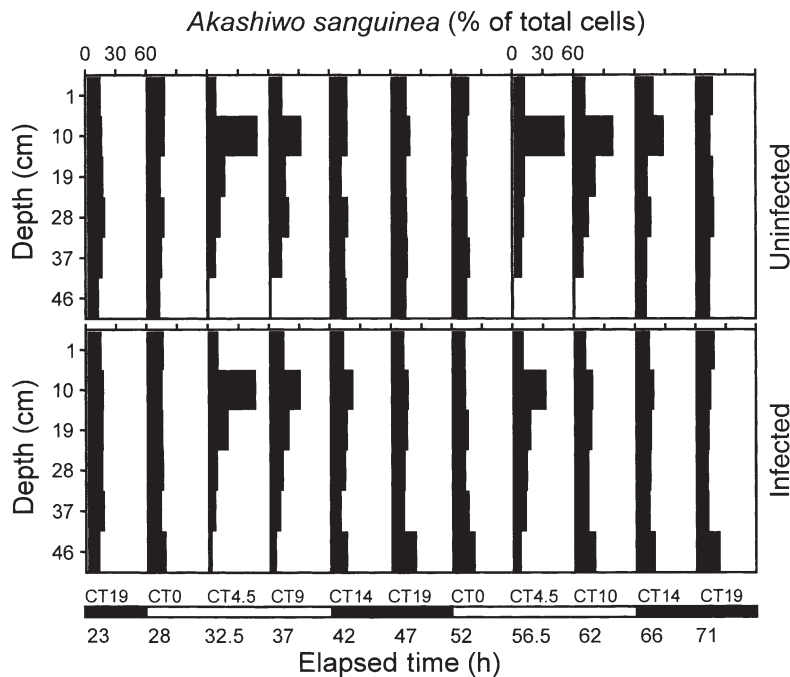


Fig. 1. *Akashiwo sanguinea*. Diel vertical migration of infected and uninfected *A. sanguinea*. Data are expressed as a percentage of total column cells using mean cell abundance of replicate columns. CT: circadian time, the time corresponding to the hour after the onset of the light period. During this study, light came on at CT 0 and off at CT 14. White and black areas of the bar under the x-axis represent the light and dark periods, respectively

depth) aggregations occurring during the day and populations being evenly dispersed at night (Fig. 1, upper panels). Peak subsurface accumulations occurred at CT 4.5 of both light periods and accounted for  $49 \pm 5.0$  and  $52 \pm 23.2\%$  of the *A. sanguinea* population at  $T_{32.5}$  and  $T_{56.5}$ , respectively. Infected *A. sanguinea* also exhibited strong DVM over the first light:dark cycle (Fig. 1, lower panels), with peak subsurface accumulations at CT 4.5 accounting for  $47 \pm 4.9\%$  of total cells present at  $T_{32.5}$ . Later in the infection cycle, however, parasitized cells appeared to lose their ability to migrate, as only a limited subsurface aggregation of cells ( $33 \pm 9.3\%$  of the population) was present at  $T_{56.5}$  (CT 4.5), and no accumulations were evident thereafter.

#### Taxis and sinking

A separate set of treatment and control DVM columns was used to examine *Akashiwo sanguinea* phototaxis, geotaxis, and sinking. Infected and uninfected populations of *A. sanguinea* collected from the upper 10 cm of the DVM columns at  $T_{62}$  averaged  $130 \pm 19.8$  and  $763 \pm 330.5$  cells  $\text{ml}^{-1}$ , respectively. Not

surprisingly, therefore, cell densities of *A. sanguinea* in test tubes used for phototaxis, geotaxis, and sinking studies differed by treatment and in some cases within replicates. Two-way ANOVA conducted separately for the 3 experiments consistently showed significant differences in *A. sanguinea* density between uninfected and infected cultures ( $p < 0.001$ ). *A. sanguinea* density did not differ ( $p > 0.05$ ) between replicates for the phototaxis and sinking studies, but did differ significantly ( $p < 0.001$ ) between replicates for the geotaxis experiment. Thus, *A. sanguinea* abundance at different positions in the phototaxis, geotaxis, and sinking tubes at the end of incubations could not be compared directly. Instead, cells present in each region were normalized to total number of cells to give the percentage of the *A. sanguinea* population at different levels in the tubes and then compared. The parasite prevalence in infected populations was  $100 \pm 0.5\%$ , with most of the infections ( $71 \pm 2.3\%$ ) being in late-stage development.

*Akashiwo sanguinea* from treatment and control columns showed opposite responses to light, as indicated by cell distributions in the phototaxis experiment (Fig. 2). Infected *A. sanguinea* exhibited negative phototaxis, with cell densities 2.8 times higher in the darkest third of the tube than in the third nearest the light. In contrast, uninfected *A. san-*

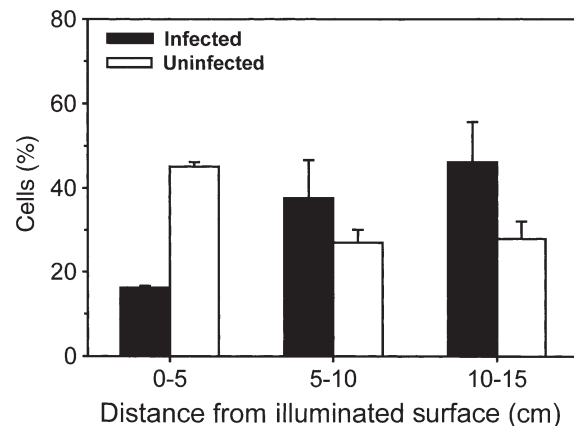


Fig. 2. *Akashiwo sanguinea*. Plot of phototaxis-precision strength in infected and uninfected *A. sanguinea* at 62 h after the parasite infection. Data are expressed as % of total tube cells. Error bars represent SEM of duplicate tubes

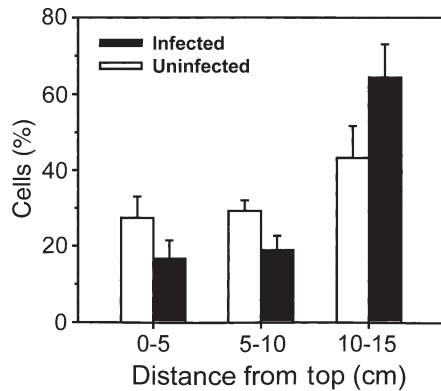


Fig. 3. *Akashiwo sanguinea*. Plot of geotaxis precision strength in infected and uninfected *A. sanguinea* at 62 h after the parasite infection. Data are expressed as % of total tube cells. Error bars represent SEM of duplicate tubes

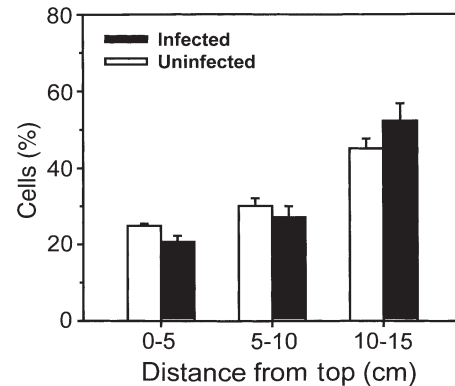


Fig. 4. *Akashiwo sanguinea*. Plot of sinking precision strength in infected and uninfected *A. sanguinea* at 62 h after the parasite infection. Data are expressed as % of total tube cells. Error bars represent SEM of duplicate tubes

*guinea* showed positive phototaxis, with 1.6 times as many cells at the illuminated end of the tube as at the dark end. Two-way ANOVA showed no significant difference ( $p > 0.05$ ) in mean percentage of cells by host types (infected versus uninfected) or by position in the phototaxis tube (illuminated third, middle third, darkest third) but did show a significant interaction between host type and position ( $p = 0.013$ ), indicating that the distribution of cells within the tubes depended on whether the hosts were infected. *A posteriori* pairwise comparison (Tukey's test) revealed a significant difference ( $p = 0.009$ ) in the percentage of infected versus uninfected *A. sanguinea* at the illuminated end of the phototaxis tubes. There was also a significant difference ( $p = 0.018$ ) in the percentage of infected cells in the illuminated third versus darkest third of the tubes.

Both infected and uninfected *Akashiwo sanguinea* showed positive geotaxis (Fig. 3), with substantially more cells at the bottom of the tube ( $64 \pm 9$  and  $43 \pm 8\%$ , respectively) than at the top ( $17 \pm 5$  and  $27 \pm 6\%$ , respectively). Two-way ANOVA indicated no significant difference ( $p > 0.05$ ) in mean percentage of cells by host type, but did reveal a significant difference ( $p = 0.003$ ) between regions of the geotaxis tube. There was no significant interaction between host type and position ( $p > 0.05$ ). *A posteriori* pairwise comparison indicated that the proportion of cells in the bottom third of the geotaxis tubes differed significantly from that in the top ( $p = 0.005$ ) and middle ( $p = 0.008$ ) thirds. Infected and uninfected *A. sanguinea* also exhibited similar sinking patterns (Fig. 4), with 2-way ANOVA showing a significant difference ( $p < 0.001$ ) in mean percentage of cells by position in the sinking tube, but not by host type ( $p > 0.05$ ), and there was no significant interaction between position and host type ( $p > 0.05$ ). *A*

*posteriori* pairwise comparison indicated that there were significantly more cells in the bottom third of the sinking tubes than in the top or middle third ( $p < 0.001$ ). Accumulation of infected and uninfected host cells in the bottom third of sinking tubes was 81 and 104%, respectively, of that in geotaxis tube.

### Swimming speed

The swimming speed of uninfected *Akashiwo sanguinea* remained stable during the experiment (Fig. 5), with values averaging  $298 \pm 3.8 \mu\text{m s}^{-1}$  ( $n = 8$ ) over the course of the experiment. In contrast, the swimming speed of parasitized *A. sanguinea* decreased during the experiment. Two-way ANOVA revealed signifi-

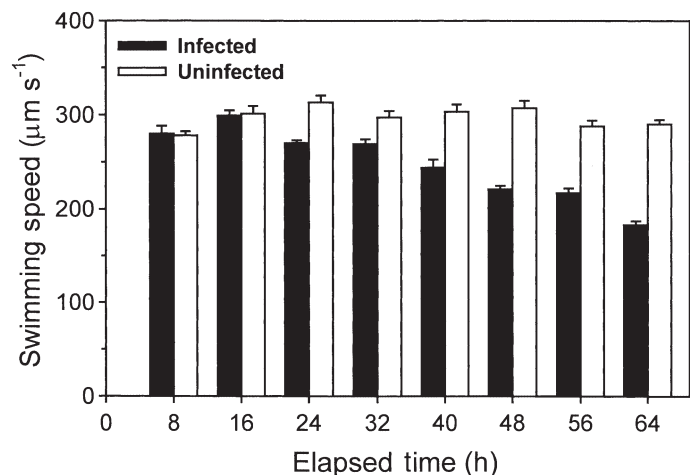


Fig. 5. *Akashiwo sanguinea*. Swimming speed of infected and uninfected *A. sanguinea* over the infection cycle of the parasite. Error bars represent SEM of duplicate tubes



cant differences ( $p < 0.001$ ) in swimming speed by host type (infected versus uninfected) and elapsed time after infection, with a significant ( $p < 0.001$ ) interaction effect between the two. *A posteriori* pairwise comparison indicated no significant difference ( $p > 0.05$ ) in swimming speed between infected and uninfected cells at  $T_8$  and  $T_{16}$ , but showed significant differences ( $p < 0.05$ ) at all subsequent sampling times. Parasite prevalence at  $T_8$  and  $T_{16}$  was only 12 and 85%, respectively. One day after inoculation ( $T_{24}$ ), parasite prevalence had increased to 97%, with all parasites being in the early part of the infection cycle. Swimming speeds of infected *A. sanguinea* decreased steadily at subsequent sample times ( $T_{32}$  to  $T_{64}$ ), with speeds 37% lower than those of uninfected cells at the end of the experiment. Parasite prevalence over that interval averaged  $99.6 \pm 0.24\%$  and most parasites (ca. 80%) had reached the late phase of the infection cycle before sampling was terminated.

## DISCUSSION

Uninfected cultures of *Akashiwo sanguinea* exhibited strong DVM similar to that reported for many dinoflagellates in previous field and laboratory studies (Cullen & Horrihan 1981, Heaney & Eppley 1981, Dortch & Maske 1982, Kamykowski et al. 1998b). Parasitized *A. sanguinea* exhibited comparable DVM patterns throughout most of the infection cycle, but failed to form subsurface accumulations during late stages of parasite development. The loss of DVM during late infection appeared to reflect parasite-induced changes in host behavior, as uninfected hosts exhibited positive phototaxis during the light cycle, while hosts in late-stage infection showed negative phototaxis. Infected and uninfected populations of *A. sanguinea* appeared to move downward in the absence of light, supporting earlier reports that DVM encompasses a combination of phototaxis and geotaxis (Kamykowski et al. 1998b). Thus, negative phototaxis and positive geotaxis may work synergistically to promote downward migration of infected *A. sanguinea*. Sinking was also pronounced during the time frame of our geotaxis experiments (30 min), raising the possibility that passive forces contributed to downward movement of cells. Sinking, however, probably had little influence on results of geotaxis experiments for uninfected *A. sanguinea*, as cells swam at relatively constant rates throughout the light:dark cycle ( $298 \pm 3.8 \mu\text{m s}^{-1}$ ). In contrast, swimming speeds of infected hosts decreased markedly (by as much as 37% relative to uninfected *A. sanguinea*), which may have inhibited the ability of parasitized cells to migrate toward the surface.

Coats & Bockstahler (1994) reported sharp vertical separation of infected and uninfected *Akashiwo sanguinea* in Chesapeake Bay, and noted a high prevalence of late-stage infections near the pycnocline, several meters beneath dense, but lightly infected, surface populations. They argued that vertical uncoupling of host and parasite populations reflected either rapid sinking of infected *A. sanguinea* or failure of parasitized hosts to migrate upward during the day (i.e. loss of phototaxis). While our results do not exclude sinking as having an influence on distributions of parasitized hosts in the field, they strongly favor the interpretation that the concentration of *Amoebophrya* sp. at depth reflects a loss of phototaxis and DVM. Our data also indicate that active downward swimming of infected cells (geotaxis) contributes to the vertical distribution of parasite populations in the field. Nonetheless, other factors may also contribute to vertical heterogeneity of infected dinoflagellate hosts. For example, Johansson & Coats (2002) recently showed that grazing by ciliates can limit the success of dinospores from *Amoebophrya* sp., with differential grazing pressure potentially influencing parasite prevalence.

The loss of phototaxis during late infection suggests that *Amoebophrya* sp. blinds the photoreceptor and/or eyespot of *Akashiwo sanguinea*, thereby preventing infected cells from sensing light and orienting themselves toward the stimulus. Interestingly, *Amoebophrya* sp. ex *A. sanguinea* always develops inside the nucleus of its host (Coats & Bockstahler 1994). While migration of dinospores through the host cytoplasm is required prior to invading the nucleus, parasites may not come into direct contact with host cytoplasmic structures once infections are established. Thus, blinding of the host's photoreceptor and/or eyespot may not involve direct interactions like ingestion or digestion of structural elements. Rather, indirect interference seems more likely and may involve disruption of physiological or biochemical states in the signal transduction chain from the photoreceptor to flagella (Kreimer 1994). Alternatively, growth of the parasite may alter phototaxis by shading the host's photoreceptor. This seems plausible, as growth of *Amoebophrya* sp. ex *A. sanguinea* causes enlargement of the host's episome and may partially block light from reaching the photoreceptor, which in many dinoflagellates is located on the ventral surface, just under the sulcus or beneath the base of the longitudinal flagellum near the junction of the sulcus and cingulum (Levandowsky & Kaneta 1987, Kreimer 1994).

While the swimming speed of uninfected *Akashiwo sanguinea* was more or less stable during the experiment, the swimming speed of parasitized *A. sanguinea* decreased steadily after the early stage of the infection cycle. Forward et al. (1986) reported that the swim-

ming speed of *A. sanguinea* (*Gymnodinium splendens*) varied up to 2-fold in response to temperature changes between 13 and 25°C and showed that the effect of temperature on swimming speed was especially dramatic between 19 and 21°C. Since our cultures were grown at 20°C, subtle changes in temperature resulting during microscopic examination or video recording could have influenced our estimates of swimming speed. Since infected and uninfected treatments were handled in the same way, it seems unlikely that any change in swimming speed due to change in temperature would have biased experimental outcomes; however, differential effects of temperature on the swimming speed of uninfected and infected *A. sanguinea* cannot be ruled out and need to be addressed in future studies. There are at least 3 other possible explanations for the reduced speed in infected cells: (1) energy diverted to growth of the parasite; (2) mechanical effects caused by the parasite inside the host cell; or (3) changes in host cell size, drag, or mass. Resolution of the possibilities will also require additional study.

In field populations of dinoflagellates, DVM patterns can be influenced by a variety of external environmental and internal cellular factors, including light intensity (Eppley et al. 1968, Blasco 1978, Heaney & Eppley 1981, Passow 1991), temperature (Kamykowski 1981), salinity (Kamykowski 1981, Tyler & Seliger 1981), oxygen (Harris et al. 1979), nutrient concentrations (Cullen & Horrigan 1981, Heaney & Eppley 1981, MacIntyre et al. 1997), and cellular biochemical pools (Cullen 1985, Cullen et al. 1985, Kamykowski et al. 1998a). Our result suggests that parasitism is yet another internal factor that can alter DVM of dinoflagellates. Furthermore, results from this study suggest that modification of host behavior by parasitism may contribute to vertical variations in phytoplankton species composition and abundance, particularly during epidemics when late-stage infections are prevalent. The energy budgets of the host cell may also be expected to vary over the infection cycle. For example, infected host cells might invest more energy than uninfected cells in maintaining an appropriate vertical position within the water column. In addition, parasitism can have significant impacts on host physiology (Park et al. 2002) and may alter the photosynthetic potential of different cell types in the water column.

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*Editorial responsibility: William Li,  
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