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Short Communication

Prymnesium parvum, an Ichthyotoxic Alga in an Ornamental Fish Farm in Southern Israel

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Abstract

The haptophyte alga *Prymnesium parvum* (Prymnesiophyceae) caused “creeping” mortality in ornamental fish, molly (*Poecilia* sp.) and koi (Japanese carp, *Cyprinus carpio*), farmed in the Arava Valley in southern Israel. The toxicosis occurred when the water system was changed from flow-through to closed circulation. A moderately high temperature, three-fold increase of salinity, and probable rise in eutrophication created conditions suitable to *P. parvum* blooms. The system was treated with 10 ppm ammonium sulfate and fish mortality ceased.

Prymnesium parvum is a relatively small haptophyte phytoflagellate that produces toxins of lethal effect on gill-breathing animals and extensive fish mortality in brackishwater fishponds. It was first associated with ichthyotoxicity in the Netherlands (Liebert and Deerns, 1924) and some years later in Denmark (Otterstrom and Steemann-Nielsen, 1939). In Israel, the flagellate was first described in carp ponds in the Beit She’an (Beisan) Valley in the northern part of the country in the fall of 1945 (Reich and Aschner, 1947). Fish mortality was always preceded by a typical change of water color from transparent light green in healthy ponds to a dirty yellow-brown. The condition gradually spread south and in the following winter reached the ponds of Beit Ha’arava, a settlement north of the Dead Sea. Although situated some 100 km apart, the two locations shared the same water supply. Soon after this first case report, the toxicosis became enzootic, affecting an estimated 3,000 acres, 60% of the total Israeli pond area (Sarig, 1971).

The toxicity of *P. parvum* was extensively studied in Israel (Yariv and Hestrin, 1961; Bergmann et al., 1963; Parnas, 1963; Reich et al., 1965; Dafni and Shilo, 1966; Shilo, 1967). The alga produces prymnesin, a com-

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plex of highly toxic compounds whose synthesis is greatest during the late stages of logarithmic growth and stationary phases (Shilo, 1967). The primary site of action of prymnesin appears to be the gill membrane, whose permeability becomes severely compromised (Yariv and Hestrin, 1961; Ulitzur and Shilo, 1966). Fish may die within hours without presenting any particular anatomo-pathological lesions. *Prymnesium parvum* toxins can kill other aquatic organisms as well, including protozoans, cnidarians, turbellarians, rotifers, oligochaetes, polychaetes, irudineans, cladocerans, amphipods, isopods, gastropods, and amphibians in the gill-breathing stage (Valkanov, 1964; Paster, 1973).

Although euryhaline and able to survive in fresh water (250 mg chloride per l water; Kimor, 1948), *P. parvum* requires a salinity of 0.1-3% to grow (Shilo and Shilo, 1962; Larsen and Bryant, 1998). The optimum salinity for growth as well as the environmental factors that influence toxin production are uncertain (Larsen and Bryant, 1998), however, stagnating brackish water, raised nutrient concentrations, and a mild climate provide an ideal ecological niche for *P. parvum* and favor blooms (Collins, 1978; Holdway et al., 1978; Baker et al., 2007).

In the past, confirmation of *P. parvum* was made by challenging small fish that are particularly sensitive to the toxin (such as *Gambusia affinis*) with an activator (3,3 diaminodipropylamine/DADPA) that lowers the minimal toxic concentration. Presence of at least 1 ITU (ichthyo-toxic unit) was confirmed by the death of all test fish within two hours (Ulitzur and Shilo, 1966). *Prymnesium parvum* blooms were controlled mainly by using 10 ppm ammonium sulfate (Reich and Aschner, 1947), 10-15 ppm liquid ammonia (Sarig et al., 1960), or 2-3 ppm copper sulfate (Sarig, 1971).

The present report describes a case brought to our attention in January 2007. Continuous, “creeping” mortality in an ornamental fish farm in the desert area of the Arava Valley started in 2003 when the water system, fed by local aquifers, was changed from flow-through to closed circulation. The fish were raised in a variety of ponds (concrete, plastic-lined, and earthen). The farm water temperature was maintained at 25±1.5°C and salinity at the water inlet fluctuated within a range of 300-500 mg chloride per l water. After the change, effluent water flowed through a sedimentation pond and recirculated back to the fishponds. Fresh water was added to compensate for evaporation. Chloride in the ponds rose to 700-1100 mg/l (1.3-2‰) during the algal toxication.

Neither gross pathology nor parasitological or histopathological analysis of a sample of about 20 molly and koi revealed the presence of pathogens or any particular abnormalities, as in similar cases (Reich and Aschner, 1947; Sarig, 1971). Microscopic examination of the water, however, revealed a relative abundance (over 1 x 10^5 cells/ml) of *P. parvum* (length 13.5±1.5 µm, width 9±1 µm; Fig. 1). A bloom of the green microalga *Kirchneriella lunaris* was also observed, as was the presence of the diatoms *Chaetoceros* and *Skeletonema* and the microflagellates *Cryptomonas* and *Chlamydomonas*.

No mortality occurred in ten fish that were transferred to an aquarium containing fresh uncontaminated water and kept under observation for over a month, indicating that the fish were in an initial and still reversible stage of intoxication, a feature already noted by other authors (Ulitzur and Shilo, 1966). The system was treated with 10 ppm ammonium sulfate according to the recommendations of Reich and Aschner (1947) and the fish mortality stopped.

The propagation of the *P. parvum* was quite clearly triggered by the up to three-fold increase in salinity and favored by the moderately warm water of the fishpond, probably together with the rise in eutrophication caused by recirculating the water. The renewed occurrence of *P. parvum* in this region is worrisome and may require new management strategies to control its spread.

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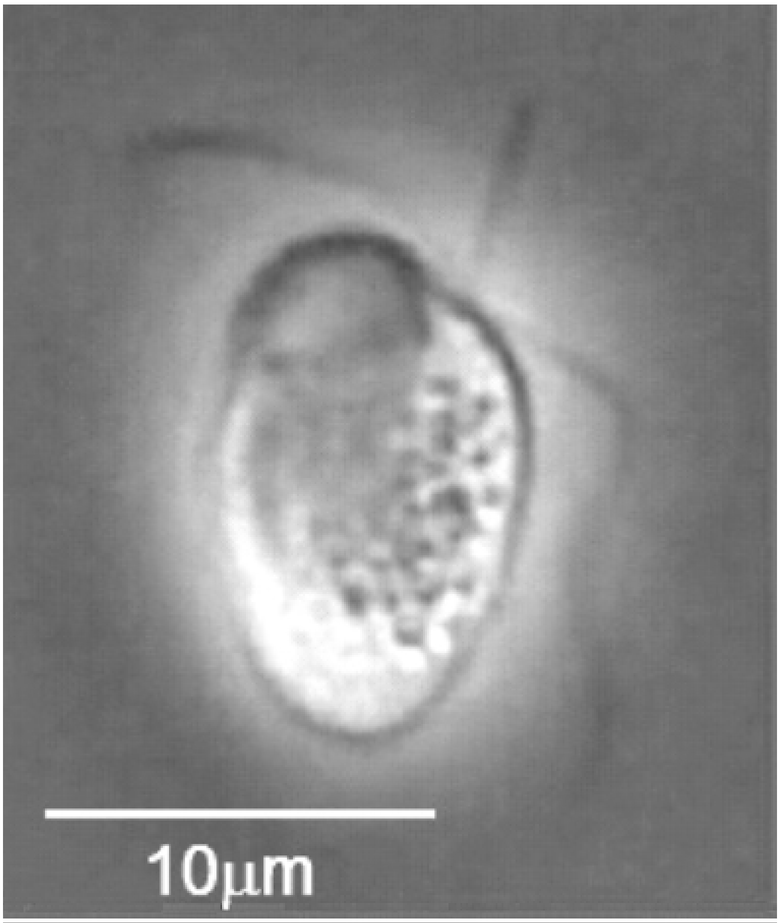

Fig. 1. *Prymnesium parvum* Carter from the Arava Valley in southern Israel.

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