

The Effects of Phenylmercuric Acetate on the Growth of *Chlamydomonas variabilis* Dang

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In recent years, and for evident reasons of pollution prevention, several studies (KAMP NIELSEN, 1971, BEN BASSAT et al. 1972, VAIDEHI et al. 1974) have been made regarding the toxicity of mercurial compounds to phytoplanktonic organisms of continental waters. This paper aims to complete these data.

MATERIALS AND METHODS

The axenic strain of *Chlamydomonas variabilis* Dang N^o AV 511-5 was obtained from the "algotheque" of the French C.N.R.S. It was maintained aseptically on the L-C Lefevre medium. Before each experiment the algae were cultured for eight days on a synthetic medium whose composition has been previously reported (DELCOURT et al. 1974, VAIDEHI et al. 1974).

During experiments, flasks containing 50 ml of algal suspension were placed in a phytotronic basin at 23°C, and shaken at 80 oscillations per minute. The cultures were continuously illuminated at 5200 lux by fluorescent light.

Cell growth was determined daily by cell counts using a Cytograf 6301 (Biophysics). Triplicate experiments were performed at each concentration of phenylmercuric acetate with control flasks included each time and under the same conditions.

RESULTS

Regardless of the initial cell concentration in the control flasks (2000, 4000, 10,000, 20,000, 50,000 or 100,000 cells per ml) growth curves were linear with no evident lag phase. (Figure 1.)

Growth curves were also produced for these initial concentrations of cells in the presence of various phenylmercuric acetate concentrations of 10^{-9} , 2.5×10^{-9} , 5×10^{-9} , 7.5×10^{-9} , 10^{-8} , 2.5×10^{-8} , 7.5×10^{-8} M.

The growth of a culture of *C. variabilis* with an initial concentration of 20,000 cells/ml. as affected by various concentrations of phenylmercuric acetate is illustrated in figure 1. Growth in the presence of concentrations below 5×10^{-9} M is the same as that of the control. The only apparent effect is the occurrence of a lag phase before the exponential growth phase. The length of the lag phase

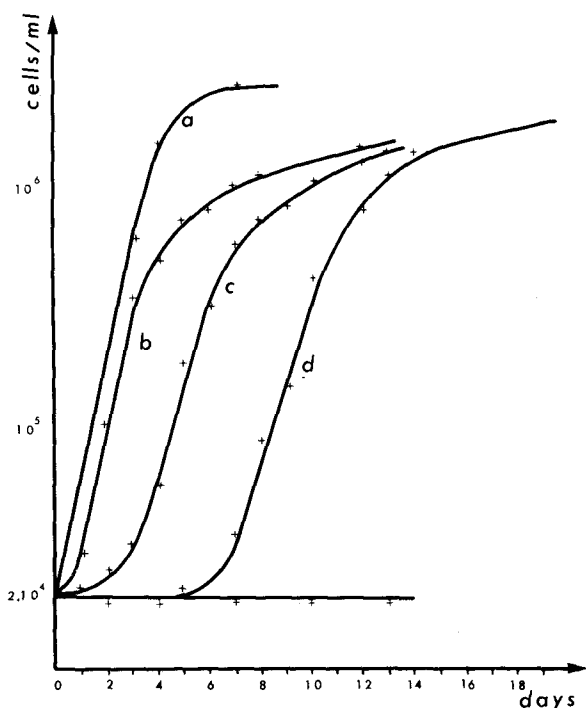


Figure 1. Growth of *Chlamydomonas variabilis* in media containing various concentrations of phenylmercuric acetate; a, control, b, 5×10^{-8} M, c, 10^{-8} M, d 2.5×10^{-8} M.

is proportional to the phenylmercuric acetate concentration. (Fig. 1)

With 5×10^{-8} M phenylmercuric acetate and above, the cells (Fig 1) did not continue to increase after 13 days, but it is not proven that they will not resume again later. They have, indeed, the same microscopic appearance as those in the lag phase (loss of motility, absence of coloration, and for some, an increased volume).

The toxic action of phenylmercuric acetate is shown by another modification of algal growth. Although the slope of curves are identical with the control, the duration of exponential phase decreases as soon as the threshold concentration, 5×10^{-8} M is exceeded, i.e. at the same concentration where the lag phase appears (Fig.1). Nevertheless the decrease in length of the exponential phase does not modify the final cell concentration. Only the time to this final cell concentration is increased. The results are similar for all initial cell concentrations we have tested, and the only difference is that the threshold concentration of phenylmercuric acetate increases with the increase in

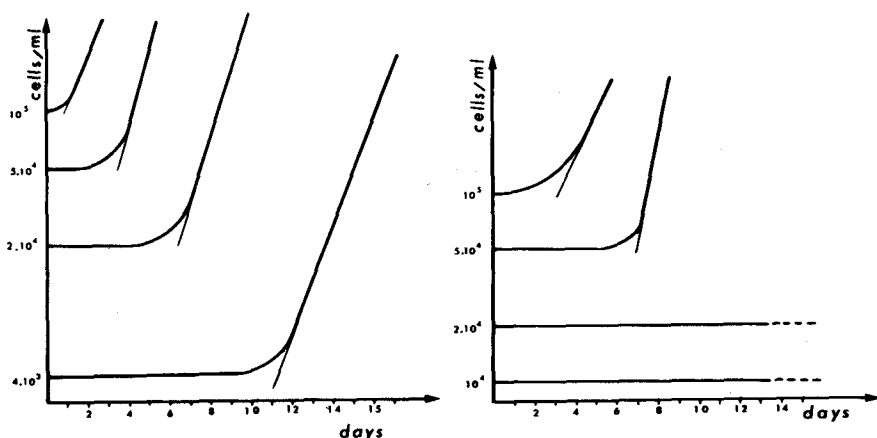


Figure 2 & 3. Variations of the length of the lag phase in populations of Chlamydomonas varabilis in relation to the initial cell concentration.

Fig.2: Left: Medium containing 2.5×10^{-8} M phenylmercuric acetate. Fig.3: Right: Medium containing 5×10^{-8} M phenylmercuric acetate

initial cell concentration. Thus the toxicity is higher at low algal cell concentrations. Figures 2&3 show this increase in toxicity of the same concentration of phenylmercuric acetate.

Thus, two characteristics of the toxic action of phenylmercuric acetate on the growth of C.variabilis have been determined:

- The existence of a threshold concentration below which no toxicity is revealed. The threshold dose is proportional to the algal cell concentration.
- The appearance of a lag phase which is the first perceptible sign of toxicity. The duration of the lag phase increases with decreasing initial algal cell concentration and also increases with increasing concentrations of phenylmercuric acetate.

These results agree with previous work (KAMP NIELSEN 1971, BEN BASSAT et al. 1972 and 1975, VAIDEHI et al. 1974, DELCOURT et al. 1974) utilizing other phytoplanktonic organisms.

It must be noted, in conclusion, that the toxicity of phenylmercuric acetate is due to the binding of the molecule to a definite number of cellular sites. This presents an important problem with regard to mercurial pollution.

For indeed, if the threshold concentration of a mercurial compound

The threshold concentration for toxicity of a mercurial compound in a cell concentration between 20,000 and 100,000 cells per ml is usually higher than the mercury concentration commonly observed in continental waters. However the very low vernal algal concentrations observed naturally could mean that even the low natural mercury concentrations would exceed the toxic threshold concentration.

It would therefore be very important to study experimentally the behaviour of very low density algal populations growing in a medium containing mercurial ions in order to determine exactly the true toxicity of these compounds with respect to the phytoplanktonic organisms of the continental water.

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