the cells took place, autospore formation might have taken place. Light may not therefore have been the cause of autospore formation after the 18–19 h time lag from the beginning of the light period. The addition of fresh nutrients can cause "physiological shock" which induces a lag phase which can be followed by the formation of autospores. Fig. 2 of ref. 1 shows an eight-fold increase of cell numbers in *Scenedesmus* which appears to confirm autospore formation. But this increase could have been caused by added nutrients as well as photoperiodic effects. We believe that the experiment should be repeated without dilution at the end of the dark period in order to confirm that light was the controlling factor of autospore formation.

We have subjected the marine diatom, *Skeletonema costatum* (Grev.) Cl., to three light intensities simulating surface, 10 and 50 m light conditions in the oceans and three photoperiods (9 h light–15 h dark, 12 h light–12 h dark and 16 h light–8 h dark). After a minimum of three cycles of light and dark, we did not observe synchronous cell division in any of the photoperiods used (unpublished work of W. A. G.). We therefore believe that species that undergo simple cell division to two new cells should be examined, as well as those forming autospores, before photoperiod alone is concluded to cause synchronous cell division. Jorgensen[3] claimed that photoperiod caused synchronous cell division in *Skeletonema*; we did not observe it in culture conditions similar to his.

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Received September 5, 1967.

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Obtaining Synchronous Cultures of Algae

We agree with Glooschenko and Curl, in the preceding communication, that species which produce autospores may have a mechanism of cell division different from those species like diatoms and dinoflagellates, which always form two cells. We believe, however, that cell division is always triggered only after growth and maturation of the cells. We chose autotrophic species and cultured them in a simple mineral medium, so that for growth and maturation they needed a light period. Full maturation—replication of DNA—can, as we saw, take place in the dark. After this period of DNA replication the autospores are formed, but they do not grow in the dark. They accumulate and start growing synchronously in the next light period.

Only in optimum conditions of light, temperature and culture medium do all the cells mature together and complete their life cycle in the shortest possible time. When such conditions are not available, partially synchronized cultures appear; these are synchronized in two or more groups[1]. We observed this in cultures at low temperatures or low light intensities. Our intention was to develop a simple culture apparatus while obtaining the most favourable external conditions for growth[2]. A method based on light–dark changes and daily dilutions of the culture to a standard cell number with fresh medium was adopted from Kuhl and Lorenzen[3]. Dilution is necessary to replace the spent culture medium, and, furthermore, it prevents unfavourable light conditions from developing in the culture with an increase in cell density. This method is only used when permanent synchronization is required. Complete synchronization can be achieved without dilution, and this is the case after inoculation during the first three cycles of light and dark. We have found many times that dilution can be carried out at any time during the light–dark cycle without disturbing the life cycle or the division time of the cells. But it should be noted that dilution during the light period abruptly changes the light conditions in the culture and this may injure the cells if no precautions are taken[4].

There may be special problems involved in obtaining completely synchronized cultures of diatoms or dinoflagellates. These species are often to some extent heterotrophic, so that their optimum growth conditions are usually not as easily understood as those of most unicellular green algae. Glooschenko and Curl give no details of their method and conditions of culture, but it may be pointed out that even in natural conditions a partial synchrony of cell division is observed for dinoflagellates[5], and periodicity of cellular divisions induced by light–dark changes has been observed in cultures of diatoms[6,7].

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Received November 8, 1967.

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Gene Equipoise and Pathogenicity

Flor[1] suggested that parasitic relationships have a specific genetic basis, and that a gene-for-gene relationship exists between host and parasite. Person[2,3] analysed this relationship and showed it to be valid in several host–parasite systems. Since 1959 numerous reports have appeared which support this validation.

The importance of nutrition in host–parasite relationships has been reported in many studies[4–7]. Apple varieties have been rendered resistant to *Venturia inaequalis* by the injection of phenylalanine[8]. Tryptophan increased the indole acetic acid and so is involved in the formation of galls by *Ustilago zeae* (*maydis*)[9]. Similar functions for this amino-acid have been suggested by other workers[10,11]. Several amino-acids have also been found to be active against *P. graminis*[12], and prominent amino-acid changes occurred after infection of wheat by this fungus[13]. Sempio reported a difference in infectivity depending on the sugars in the nutrient solutions which were used[14]. Keitt and associates studied the nutritional aspects of host–parasite relationships using auxotrophic mutants of *Venturia inaequalis*[15,16]. Lewis[17] suggested nutrition as the basis of pathogenicity in his nutritional or balance hypothesis; Garber suggested the hypothesis of nutrition inhibition in host–parasite relationships[18].

In this investigation auxotrophs of *Ustilago hordei* (Pers.) Lagerhans were used to investigate the nutritional basis of the host–parasite relationship. Dikaryons were produced from auxotrophic lines of the parasite and these were used to infect several varieties of barley. Table 1 summarizes the results from a number of these infections. The pathogenicity varied widely from one dikaryon to another on the same variety of barley, and from one variety to another when the same dikaryon was used. The first pathogenicity index P_1 enables the pathogenicity