

# matters arising

## Algal sexuality

CAVALIER-SMITH<sup>1</sup> raises many interesting points on the origin of the eukaryotic cell and its organelles. I find myself in general agreement with him and with others<sup>2-4</sup> who have provided an alternative to the widely held theory that eukaryotic organelles were originally acquired by endosymbiosis. I wish to comment, however, on one aspect of his evolutionary scheme (see ref. 1, Fig. 5), namely, the assertion that sex is absent in the Dinophyceae, Euglenophyceae, and Cryptophyceae. In fact, there have been several reports<sup>5-7</sup> of sexuality in the Dinophyceae. Beam and Himes<sup>8</sup> have observed pairing between cells of *Cryptocodinium cohnii* and their fusion into a single cell, followed by nuclear fusion. Furthermore, those workers<sup>8</sup> and others<sup>9</sup> have demonstrated genetic recombination in *C. cohnii*. Although there has been no such conclusive demonstration of sexuality in the Euglenophyceae, it is necessary to approach this negative evidence cautiously—especially since there is evidence of a meiotic process in two genera of this group<sup>10,11</sup>. One can only repeat Leedale's careful conclusion that "It seems probable that there is no sexuality in the majority of euglenoids, but it remains a possibility that the process does occur as a rare phenomenon; this can never be disproved". Finally, there is a dearth of information on the Cryptophyceae, and consequently it is premature to conclude that sexuality is absent in that group.

These observations do not detract from Cavalier-Smith's main arguments. If anything they strengthen his evolutionary scheme since, if sex does exist in the Dinophyceae, Euglenophyceae, and Cryptophyceae, one could move those groups from their isolated position on Fig. 5 of ref. 1 to a position among the other algae and speculate on their interrelationships. (For example, how are the Dinophyceae and Cryptophyceae related to other algae whose chloroplasts contain chlorophylls *a* and *c*?)

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<sup>1</sup> Cavalier-Smith, T., *Nature*, 256, 463-468 (1975).

<sup>2</sup> Allsopp, A., *New Phytol.*, 68, 591-612 (1969).

<sup>3</sup> Raff, R. A., and Mahler, H. R., *Science*, 177, 575-582 (1972).

<sup>4</sup> Uzzell, T., and Spolsky, C., *Am. Sci.*, 62, 334-343 (1974).

<sup>5</sup> von Stosch, H. A., *Naturwissenschaften*, 52, 112-113 (1965); *Br. phycol. J.*, 8, 105-134 (1973).

<sup>6</sup> Vien, C., *C.r. hebdom. Séanc. Acad. Sci., Paris*, D264, 1006-1008 (1967).

<sup>7</sup> Zingmark, R. G., *J. Phycol.*, 6, 122-126 (1970).

<sup>8</sup> Beam, C. A., and Himes, M., *Nature*, 250, 435-436 (1974).

<sup>9</sup> Tuttle, R. C., and Loeblich, A. R., *Science*, 185, 1061-1062 (1974).

<sup>10</sup> Krichenbauer, H., *Archs Protistenk.*, 90, 88-122 (1937).

<sup>11</sup> Leedale, G. F., *Archs Mikrobiol.*, 42, 237-245 (1962).

<sup>12</sup> Leedale, G. F., in *The Biology of Euglena* (edit. by Buetow, D. E.), 185-242 (Academic, New York and London, 1968).

## Irradiation and DNA breaks

JOHANSEN and Boye<sup>1</sup> reported their findings on the controversial question of whether the initial number of breaks induced by ionising radiation in DNA in oxic and anoxic conditions are identical, or whether less breaks occur in nitrogen than in oxygen. Their conclusion supports the latter.

They found<sup>1</sup> the difference in the yield of breaks in cellular DNA when *Escherichia coli* was irradiated in oxic and anoxic atmospheres and to minimise rapid repair<sup>2</sup> as far as possible, the time from irradiation to lysis of the cells was, according to those authors, "a fraction of a second". They assumed that cellular repair enzymes would not rejoin breaks in this short time and on the basis of some theoretical extrapolations on the correlation between the yield of breaks at a given dose and the rate of rejoining by cellular enzymes, they believed that the differences in yield of breaks due to rapid repair was an erroneous conclusion. They assumed that rejoining of breaks would not have occurred and if so, that the initial yield of breaks in oxic irradiation was at least three times more than that in anoxic conditions. I wish here to analyse the status of this controversy and I disagree with Johansen and Boye<sup>1</sup>.

The subject has long been a matter of debate and Dean *et al.*<sup>3</sup> were the first to suggest that anoxic breaks were subject to rapid enzymatic repair, showing that in experimental conditions which inhibited rejoining of strand breaks, nearly identical yields were obtained following oxic and anoxic irradiation.

The isolation of a DNA polymerase I mutant<sup>4</sup> and the study of the kinetics of single-strand break repair in *polA1* mutant<sup>2</sup> led to the recognition of a rapid repair system occurring in *E. coli*. The work on cellular DNA involved complications because repair probably

works very fast, rejoining breaks during irradiation and up to the moment of lysis. Town *et al.*<sup>5</sup> attempted to overcome the problem by preinactivating the cells at 52 °C for 10 min before irradiation and they obtained identical yields of breaks. Heat treatment, however, induces single-strand breaks in DNA<sup>6</sup> and release of sulphhydryl compounds in the medium<sup>7</sup>.

Another approach<sup>7</sup> was to irradiate bacteriophage  $\lambda$  extracellularly in oxic and anoxic atmospheres. Contrary to the situation for cellular DNA, any change in phage DNA would thus remain unmodified and beyond the influence of cellular repair as long as the DNA was not injected into a host. DNA could safely be extracted and layered on an alkaline sucrose gradient<sup>7</sup>. Identical yields of DNA breaks were obtained in oxic and anoxic extracellular irradiation<sup>7</sup>. When  $\lambda$  DNA was irradiated intracellularly in a *polA1* host, the decrease in molecular weight of  $\lambda$  DNA was identical in both oxic and anoxic conditions and equalled that obtained after extracellular irradiation. Intracellular anoxic irradiation of  $\lambda$  DNA to the same dose in wild-type bacteria, yielded no breaks<sup>7</sup>. This demonstrates that there is no difference in the initial number of breaks and that a rapid repair system, involving DNA polymerase I, rejoins breaks very rapidly. Similarly, there are data<sup>8,9</sup> on the equal number of breaks in oxic and anoxic conditions with phage T3 and T7 irradiated extracellularly.

Wild-type bacteria were injected for 10 min with  $\lambda$  DNA which had been irradiated extracellularly with 50 krad in oxic and anoxic conditions.  $\lambda$  DNA was immediately analysed for single-strand breaks.  $\lambda$  DNA irradiated anoxically had no breaks, but a few could be seen in DNA irradiated in oxygen. Since the initial yields of breaks were identical<sup>7</sup>, the difference in molecular weight after infection is attributable to cellular repair.

Sulphydryl compounds may have a function in the radiation sensitivity of *E. coli*<sup>1</sup> but I feel that the experiments described (ref. 7 and above), supported by other results<sup>2,5,8,9</sup> should leave no doubt that: (1) initially the number of DNA breaks are identical in oxic and anoxic irradiation and (2) rapid repair, involving DNA polymerase I and probably ligase, does exist in cells and preferentially rejoins anoxic-type breaks. This could explain the differ-