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## APPENDIX. Continued.

Species	Provisioning rate	Source
<i>Uria aalge</i>	146	Birkhead 1977; Harris & Wanless 1986; Birkhead & Nettleship 1987
<i>U. lomvia</i>	214	Gaston & Nettleship 1981; Birkhead & Nettleship 1987
<i>Fratercula arctica</i>	250	Harris & Wanless 1986; Barrett et al. 1987
<i>Aegolius funereus</i>	1280	Korpimäki 1981
<i>Apus apus</i>	688	Lack & Lack 1951; Lack & Owen 1955; Pellantova 1981
<i>Cypsiurus balasiensis</i>	32	Hails & Turner 1984
<i>Ceryle rudis</i>	534	Reyer & Westerterp 1985
<i>Dryocopus martius</i>	6472	Pynnönen 1939
<i>Hirundo tahitica</i>	25	Bryant & Hails 1983
<i>Delichon urbica</i>	515	Bryant & Gardiner 1979; Bryant & Westerterp 1983
<i>Progne subis</i>	1035	Walsh 1978
<i>Sturnus vulgaris</i>	8036	Dunnet 1955; Tinbergen 1981; Westerterp et al. 1982
<i>Corvus frugilegus</i>	2015	Feare et al. 1974
<i>Parus major</i>	913	Royama 1966; Eguchi 1979
<i>Pa. varius</i>	469	Eguchi 1979
<i>Certhia familiaris</i>	120	Kuitunen & Suhonen 1989
<i>Menura novaehollandiae</i>	412	Lill 1986

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### TRANSPOSON ABUNDANCE IN SEXUAL AND ASEQUAL POPULATIONS OF *CHLAMYDOMONAS REINHARDTII*

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Transposons are ubiquitous in the nuclear genomes of eukaryotes (Berg and Howe 1989). Suggested roles for transposable elements have included the regulation of structural genes (McClintock 1956) and the generation of genetic or chromosomal variation (Nevers and Saedler 1977). Alternatively, they have been hypothesized to be molecular parasites, spreading and persisting despite a deleterious effect on the fit-

ness of their host genomes (Doolittle and Sapienza 1980; Orgel and Crick 1980). The latter view of transposons is implicit in most models of their population genetics and appears sufficient to account for the observed distribution among individuals and within genomes of transposons in *Drosophila* (Charlesworth and Langley 1989).

Although the conditions in which a transposon can spread parasitically have been examined theoretically (Charlesworth and Charlesworth 1983; Charlesworth and Langley 1986; Charlesworth 1987), there has been very little experimental investigation of the degree to which these models actually describe transposon behavior. Using

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*Escherichia coli* and the bacteria transposons *Tn3* and *Tn5*, Condit (1990) obtained results consistent with a model in which even replicative transposition has little effect on transposon abundance. However, any effects of either transposition or host fitness reduction on transposon abundance were obscured by fitness differences between host strains, and Condit admitted that the experimental validation of his model was incomplete.

If transposons do reduce the fitness of their host genomes, then their spread requires genetic exchange between individuals (Cavalier-Smith 1980). Molecular parasites should thus be more successful in genomes that reproduce sexually than in those that replicate clonally (Hickey 1982), *Chlamydomonas reinhardtii* is a unicellular, heterothallic chlorophyte alga well suited to testing this prediction because it can be maintained with or without sex, and because its low doubling time permits the passage of hundreds of asexual generations or dozens of sexual cycles per year (Harris 1989). The genomes of laboratory strains of *C. reinhardtii* differ with respect to the abundance and distribution over restriction fragments of *TOC1* (Day et al. 1988) and *Gulliver* (Ferris 1989), two recently cloned transposons. We tested the expectation of reduced transposon abundance in experimental lines that were cultured asexually compared with their parental zygospores or with sexual lines.

#### MATERIALS AND METHODS

In experiment 1, crosses among two mating type plus ( $mt^+$ ) strains (CC-1010 and CC-2342) and two mating type minus ( $mt^-$ ) strains (CC-2343 and CC-1952) were performed, producing four zygotes (A, B, C, and D). These four parental spores are unrelated wild-type isolates. From each germinated zygote, one haploid spore of each mating type was randomly chosen as the parental cell for an asexual lineage. After 12 mo of culture with weekly transfer, corresponding roughly to 800 generations of vegetative reproduction, genomic DNA was extracted from liquid cultures, slot-blotted onto nylon membranes, and hybridized with fluorescein-dUTP-labeled *TOC1* or *Gulliver* DNA. A hybridized probe was detected on X-ray film as a chemiluminescent peroxidase-antifluorescein conjugate using the ECL (Amersham) detection system. Hybridization intensities were quantified by scanning laser densitometry. To correct for any variation in amounts of DNA loaded, the slot blots were stripped of probe DNA

and rehybridized with the nitrate reductase structural gene (plasmid pMN24; Fernández et al. 1989), which was detected by chemiluminescence as above. Log-transformed absorbance readings from pMN24 hybridization were subtracted from log-transformed values for transposons, and the results for parental and descendant samples were compared.

We also investigated the distribution of transposons among individuals within the  $mt^+$  and  $mt^-$  lines from zygote B. Genomic DNA was extracted from 10 spores from each line, digested with *Bam*HI, and vacuum transferred to nylon membranes that were hybridized as above with *TOC1* or *Gulliver* probes.

In experiment 2, 20 experimental lines were established from mixtures of 16  $mt^+$  and 17  $mt^-$  genotypes, which had been derived from crosses of one  $mt^+$  and four  $mt^-$  strains (Bell 1991). In ten asexual lines, only one mating type was present, precluding mating. In the other ten, both mating types were present, mating was stimulated 11 times, and sexually produced progeny were randomly selected to inoculate fresh media (da Silva and Bell 1992). After approximately 100 asexual generations and 11 episodes of sex, genomic DNA samples were obtained and compared as above.

#### RESULTS

We found no significant differences among treatments in the abundance of either transposon in either experiment. In experiment 1, both transposons were less abundant in asexual descendants than in their ancestral spores, as predicted by the parasitic DNA hypothesis. In experiment 2, *TOC1* was slightly more abundant but *Gulliver* slightly less abundant in the sexual lines than in the asexual lines (fig. 1). All differences were very small and nonsignificant ( $P > 0.1$  in two-tailed *t*-tests).

Southern blot hybridizations indicated that there are also virtually no differences among individuals within either of two populations in experiment 1. For each mating type and for both transposons, one parental spore and ten descendants gave identical hybridization patterns with the exception of one  $mt^-$  descendant that lacked one band and that had a single unique band (fig. 2). This unique fragment might have resulted either from transposition or from some type of rearrangement unrelated to transposition, such as recombination or deletion.

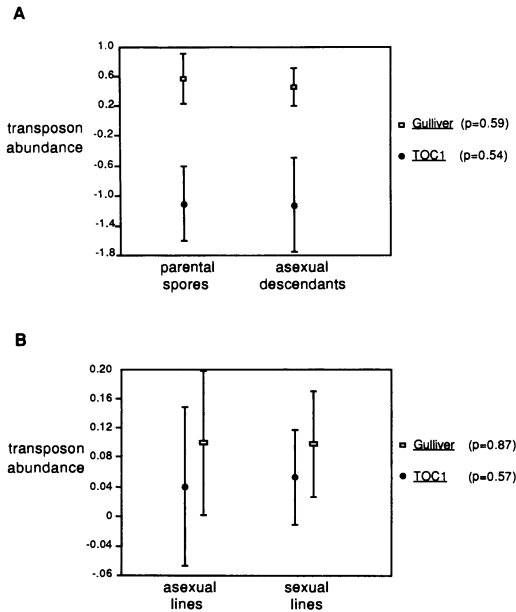


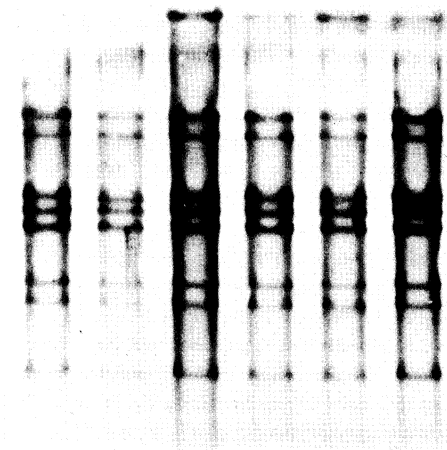
FIG. 1. Comparison of transposon abundance among treatments for experiments A and B. Error bars show one standard deviation around mean log absorbance units of X-ray film exposed to chemiluminescent slot blots, each experimental line being tested on two sets of slot blots. Values for  $P$  are from two-tailed  $t$ -tests using separate variance estimates for each treatment.

#### DISCUSSION

Transposition by *TOC1* and *Gulliver* in our experimental lines appears to have been rare, or to have had little effect on fitness, or both. At least some *TOC1* and *Gulliver* elements are capable of transposition, as they were discovered when copies transposed into, and inactivated, conventional genes that were being studied for unrelated reasons (Day et al 1988; Ferris 1989). All lines in experiment 1 and the asexual lines in B, experienced hundreds of generations of selection on rates of vegetative growth; thus, if transposition occurred and produced variation in fitness, some change in transposon abundance and distribution on *Bam*HI fragments would be expected.

The nuclear genome of *Chlamydomonas reinhardtii* may be a particularly restrictive environment for parasitic DNA. Because vegetative cells are haploid, deleterious mutations caused by transposon insertions are not masked by heterozygosity, and the opportunity for infection of new lineages is restricted to the period between gamete fusion and meiosis in the zygote. Lines of

#### Gulliver



#### TOC1

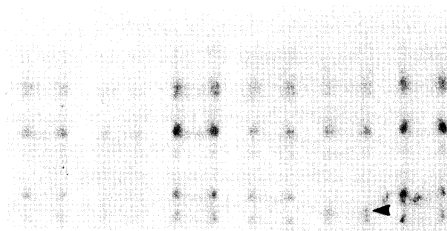


FIG. 2. Hybridization of *Gulliver* and *TOC1* to replicate Southern blots of *Bam*HI-digested genomic DNA from a parental mating type minus spore (P) and five of the ten descendant spores that were sampled. Arrowhead indicates a single unique band possibly resulting from transposition.

*Chlamydomonas* that are asexually propagated in the lab rapidly lose the ability to mate (da Silva and Bell 1992), and *C. reinhardtii* in nature is asexual to an unknown degree. Therefore, by whatever means *Gulliver* and *TOC1* initially spread among lineages of *Chlamydomonas*, they may have subsequently been domesticated by selection for minimal rates of transposition and minimal effects on fitness. Such elements would become more commensal than parasitic. Self-regulation of transposition rates (Charlesworth and Langley 1986) could explain why transposons present in the parental spores did not change in abundance.

The mutagenic potential of transposons may confer on some populations a collective fitness advantage over those lacking transposons, particularly in novel or changing environments (Biel

and Hartl 1983; Chao et al. 1983; Modi et al. 1992; Wilke and Adams 1992). However, experimental demonstrations of this effect have required that the transposons be initially present in many individuals and cannot explain their invasion of a natural population. Because all but a small fraction of the mutations caused by transposons are deleterious, the generation of adaptive variation is a hypothesis regarding the group-level effects of established transposons, not a tenable explanation of their initial spread (Charlesworth 1987).

The long-term experiments used in this study were designed to address other questions and do not allow us to test hypotheses regarding the invasion of a population by transposons. Because most or all of the individuals in our base populations already carried both *Gulliver* and *TOC1*, only transposon losses that occurred relatively frequently ( $> 10^{-3}$ ) or conferred a substantial ( $> 1\%$ ) fitness advantage would have produced differences between asexual lines and sexual or parental lines. In future work, the hypothesis that sex mediates population invasion will be tested using sexual and asexual populations in which the transposon of interest is carried by a very small fraction of individuals.

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