periods of increased *Melosira* abundance suggest periods of relatively low lake level and increased duration and effect of spring circulation and summer turbulence in Elk Lake.

The general climatic model that has been developed for the midwestern United States, largely from studies of past vegetation, is one of gradual increases in warmth and dryness from about 8500 years ago to maximums about 7000 years ago, and then gradual decreases to more or less present conditions about 4000 years ago (*1*). In contrast, Bryson *et al.* (*10*) argued that Holocene climate was punctuated by rapid transitions between numerous dry and moist periods, each lasting hundreds of years.

The data from the Elk Lake core indicate that between about 8500 and 4000 years ago, climate drier than today's did indeed result in expansion of prairie conditions (for example, increase in grasses and sagebrush in the drainage basin, decrease in decomposition of soil materials, and increase in turbidity and salinity of the lake). During the drier climate of the prairie period, Elk Lake may have resembled the shallow, eutrophic, prairie lakes that exist today south and west of Elk Lake in Minnesota and the Dakotas.

Several sediment components show the long-term shift to prairie conditions, but they also show that the prairie period was punctuated with shorter-term cyclic variations in climatic conditions, with abrupt transitions. These shorter-term cyclic variations are best seen in those variables that monitor changes in the drainage basin, such as sodium (Fig. 4), and in those variables that monitor short-term changes in the lake, such as dolomite and *Melosira*. There is evidence, therefore, for both the gradual climate changes of Wright (*1*) and the shorter-term, abrupt climate changes of Bryson *et al.* (*10*).

The response of paleoclimatic indicators to the beginning and end of the prairie period are not synchronous within the Elk Lake core. *Artemisia* percentages increase as early as 8800 years ago, preceding the influx of clastic material (measured by varve thickness and percentage of sodium) by at least several hundred years. Similarly, the decline in *Artemisia* began about 4500 years ago, although the influx of clastic material, and thus higher rates of erosion, persisted until 3800 years ago. This nonsynchroneity may be explained by different responses of the components described here to environmental conditions on different spatial scales. Changes in weathering, erosion, lake chemistry, and diatom composition are responsive to changes in conditions in the drainage basin or in the lake. On the other hand, pollen data from a lake the size of Elk Lake probably reflect changes in vegetation over a much larger area (*11*). Therefore, whereas *Artemisia* percentages provide a record of regional expansion and contraction of prairie in northwestern Minnesota, other components record the development of open vegetation, unstable slopes, and lower lake levels.

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References and Notes

1. H. E. Wright, Jr., *Quat. Res. (N.Y.)* **6**, 581 (1976).
2. W. S. Cooper, *Geol. Soc. Am. Bull.* **69**, 941 (1958).
3. J. McAndrews, *Torrey Bot. Club Mem. 22* (1966).
4. D. Stark, *Arch. Hydrobiol. Suppl.* **50**, 208 (1976).
5. F. J. H. Mackereth, *Philos. Trans. R. Soc. London Ser. B* **250**, 165 (1963).
6. W. E. Dean and E. Gorham, *Limnol. Oceanogr.* **21**, 259 (1976); in *Quantitative Techniques for the Analysis of Sediments*, D. F. Merriam, Ed. (Pergamon, New York, 1976), p. 61.
7. K. Kelts and K. J. Hsu, in *Lakes-Chemistry, Geology, and Physics*, A. Lerman, Ed. (Springer-Verlag, New York, 1979), p. 295; W. E. Dean, *Soc. Econ. Paleontol. Mineral. Spec. Publ.* **31**, 213 (1981).
8. E. Gorham, W. E. Dean, J. E. Sanger, *Limnol. Oceanogr.* **28**, 287 (1983).
9. W. G. Lund, *Bot. Zh. (Leningrad)* **51**, 176 (1966).
10. R. A. Bryson, D. A. Baerreis, W. M. Wendland, in *Pleistocene and Recent Environments of the Central Great Plains*, W. Dort, Jr., and J. K. Jones, Jr., Eds., (Univ. of Kansas Press, Lawrence, 1970), p. 53.
11. G. L. Jacobson, Jr., and R. H. W. Bradshaw, *Quat. Res. (N.Y.)* **16**, 80 (1981).
12. We thank T. C. Winter, E. J. Cushing, and D. P. Adam for their helpful reviews. Supported by the U.S. Geological Survey Climate Program.

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A *trans*-Acting Product Needed for P Factor Transposition in *Drosophila*

Abstract. *A transposable genetic element of the P family in* Drosophila melanogaster *was found to be unstable in the presence of other P elements but stable in their absence. A sensitive assay for P transpositional activity is provided by the* sn^w *allele, a defective P insert in the* singed bristle *locus which becomes hypermutable only in the presence of complete elements. This measure of activity was highly correlated with a type of female sterility normally associated with P activity. There was no cross-reactivity with transposase from another hybrid dysgenesis-causing element (the I factor).*

A distinguishing feature of transposable genetic elements of the P family in *Drosophila melanogaster* is their ability to enter a state of high transpositional activity. This behavior is triggered by a cellular condition known as the M cytotype and is responsible for the hybrid dysgenesis syndrome of germ line abnormalities as described previously (*1–4*). O'Hare and Rubin (*5*) describe two classes of P elements: (i) the complete elements, which are 2.9 kilobases (kb) with short inverted terminal repeats, and (ii) defective elements, which have deletions of various lengths internal to the repeats.

This report concerns a genetic assay for P transpositional activity which has provided evidence that complete P elements produce a *trans*-acting product (transposase) needed for transposition whereas defective elements transpose only when this product is provided for them. This method has already been used to detect and characterize transposase-producing P factors (*6, 7*) that were introduced into embryos by microinjection. [The terms "P factor" and "P element" are used synonymously in this report (*8*).]

A P element insertion mutation at the X-linked *singed bristle* locus was recovered in the progeny of a hybrid dysgenic male (*9*). It occurred on an X chromosome that was originally derived from the strain π_2 but had acquired a complex chromosomal rearrangement (*10*). This chromosome also had many other P elements in addition to those at or near the *singed bristle* locus. In the M cytotype, sn^w mutates at high frequencies to sn^e, a more extreme *singed* phenotype, and to sn^+, a state nearly equivalent to wild type (*11*). The two alternative mutational events correspond to the excision of one or the other of two contiguous defective P elements inserted in reverse orientation into the *singed* gene (*12*). When the original sn^w-containing chromosome was crossed into the M cytotype, the mutation rate of sn^w was sufficiently great (20 to 60 percent for sn^e and sn^+ combined) that it could be used to detect the M

cytotype in individual flies. In the P cytotype (the alternative state, where P elements are relatively quiescent) sn^w was nearly stable (*13*).

The first indication of a transposase occurred when the sn^w mutation was separated from other P elements in the genome. This was done by a four-generation crossing procedure in which the autosomes were replaced by chromosomes derived from an M strain (a strain free of P factors and having the M cytotype) and the X chromosomal regions flanking the *singed* gene were replaced by meiotic recombination with a multiply marked M-strain X chromosome. The latter step required a double recombination event to separate sn^w from the complex rearrangement that surrounded it (*10*).

The resulting strain, designated sn^w(M), had the M cytotype, as was expected after removing all autosomal and most X-linked P elements (*13, 14*), but the hypermutability normally expected of sn^w in the M cytotype had vanished. Neither sn^e nor sn^+ appeared among 5829 sn^w progeny from 118 independent crosses. This was surprising since the original sn^w chromosome would have produced 1000 to 3000 mutations in this many tests.

One possible explanation was that sn^w hypermutability required the presence of other P elements. This hypothesis was tested by crossing sn^w(M) females to males whose autosomes were heterozygous for marked M chromosomes and P chromosomes derived from the π_2 strain. The presence or absence of sn^w hypermutability in the germlines of the resulting four phenotypic classes of sons indicated the effect of each of the π_2 major autosomes. The results (Fig. 1) showed that either of the major autosomes of π_2 restored sn^w to its hypermutable condition, and sn^w was nearly stable in those sons that did not receive major π_2 autosomes. The small fourth chromosome was not monitored in this experiment and might be the explanation for the single sn^e mutation in Fig. 1d. Similar results (not shown) were obtained with sn^w(M)/π_2 recombinant X chromosomes, indicating that parts of the π_2 X chromosome have the same effect on sn^w as the autosomes. Finally, these experiments were repeated but with a different and unrelated P strain known as 8.31.15. The results were fully equivalent to those in Fig. 1 except that chromosome 3 was slightly more effective in activating sn^w than chromosome 2, whereas the reverse was true for π_2. The two strains probably differ in their genomic distributions of P factors. Therefore, sn^w hypermutability requires a *trans*-acting product (transposase) that can only be provided by factors on P-containing chromosomes, presumably the complete P factors themselves. Furthermore, the P insertion at sn^w must be a nonautonomous element defective for production of this transposase.

This interpretation has been confirmed by experiments in which complete P factor DNA was seen to activate sn^w hypermutability after being injected into sn^w(M) embryos and integrated into the genome (*6*). The transposase gene needed to activate sn^w hypermutability has been further characterized by directed mutagenesis experiments (*7*). At least four sites spanning the length of the complete P element were shown to be essential. A second gene, a putative repressor, whose action is counter to that of the transposase can be postulated from the existence of the P cytotype (*14*) where transpositional activity is apparently repressed, but the repressor gene has not yet been identified.

The requirement of the P transposase for sn^w hypermutability is distinct from the requirement for the M cytotype. This difference was demonstrated by crossing males from the sn^w(M) stock to females from a compound-X stock with the P cytotype (*15*). The resulting sons were genetically identical to the hypermutable males tested in Fig. 1a, but they had the P cytotype (*13, 14*). These males produced no sn^e or sn^+ progeny among 902 examined from ten replicate crosses. Thus, neither the presence of P factors nor the M cytotype is sufficient by itself for sn^w hypermutability. Both are necessary.

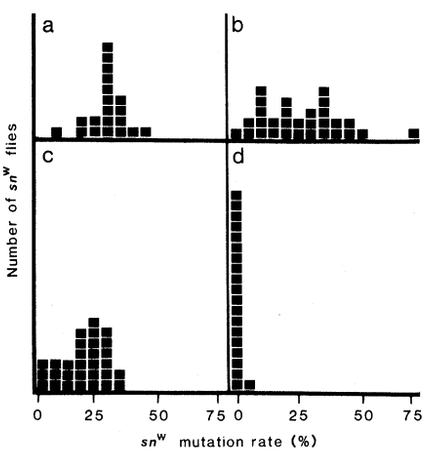

Fig. 1. The ability of the two major autosomes of π_2 to cause sn^w hypermutability. Females from the π_2 strain were mated to *bw;st* males to yield nondysgenic F_1 males. The recessive eye color mutants, *bw* (brown) and *st* (scarlet) (*23*), were markers for the second and third chromosomes, respectively. These F_1 males were crossed to sn^w(M); *bw*; *st* females to produce dysgenic F_2 males in each of four eye-color classes depending on which paternal chromosomes were received. Hypermutability of sn^w was measured for each male by crossing to compound-X females and scoring the male progeny. Each block represents the total sn^w mutation rate (sn^e and sn^+ combined) of a single male with the indicated π_2 chromosomes. (a) Chromosomes 2 and 3 of π_2; (b) chromosome 2 only; (c) chromosome 3 only; (d) neither π_2 chromosome.

A series of experiments involving only M strains was designed to determine whether hypermutability could be restored to sn^w(M) in the absence of P factors. For each experiment, I will indicate the size of the experiment and the results in the shorthand form (m/n; k) where m is the number of mutations (combining sn^e and sn^+), n is the total number of progeny in which such mutational events could have been observed, and k is the number of sn^w flies that were individually testcrossed to obtain the total of n progeny. Thus, each experiment involved k independent trials. For comparison, the results in Fig. 1, a to d, could be summarized by (271/994; 20), (178/1005; 30), (272/889; 30) and (1/1000; 20), respectively.

The effects of sex and parental derivation of sn^w(M) were jointly assessed by counting progeny from sn^w matroclinous females (0/1659; 20), patroclinous females (0/1315; 20), matroclinous males (0/1239; 14), and patroclinous males (0/1239; 19). These negative results indicate that restoration of hypermutability is not related to sex or parental derivation.

Some studies (*16*) have demonstrated that the meiotic mutant *mei*41, acts to enhance at least some dysgenic traits in the M cytotype. However, placing a *mei*41 allele onto the sn^w(M) chromosome did not cause hypermutability in the absence of P factors (0/362; 5).

Certain M strains, referred to as pseudo-M strains (*17*), carry a large number of transposable elements that have sequence homology to P elements, but possess the M cytotype. To determine whether these elements can produce the P transposase, males from a pseudo-M strain were crossed to homozygous sn^w(M) females, and the resulting sons were testcrossed to measure the stability of sn^w. The results (0/229; 15) suggest that these elements produce no P transposase.

There is a parallel system of hybrid dysgenesis in *D. melanogaster* known as the I-R system (*2, 18*) in which putative transposable elements, I factors, become transpositionally active in certain classes of hybrid females. In this system, sn^w(M) is an I strain. Female progeny from both reciprocal crosses between

sn^w(M) and the R strain se_{F8}, were tested for sn^w hypermutability and also for the kind of sterility that is diagnostic of I-R dysgenesis. The sterility tests confirmed that I-R dysgenesis occurred in the *se*/sn^w females (404/608 = 66.4 percent embryo lethality) and not in the reciprocal sn^w/*se* females (19/320 = 4.9 percent embryo lethality). However, neither group displayed sn^w hypermutability: (0/580; 15) and (0/445; 10) respectively. Thus, there is no evidence that the sn^w P element can cross-react with a transposase produced by I factors.

Considerable quantitative variability is known to exist among P strains in their potential to cause gonadal dysgenic (GD) sterility, one of the traits associated with hybrid dysgenesis and characterized by agametic morphology. Strains like π_2 cause nearly 100 percent GD sterility under appropriate conditions, while Q strains (*19*) cause no detectable GD sterility, but do have the P cytotype. Most P strains lie somewhere along the continuum between these two extremes. Since all these strains have the P cytotype, this variability might be explained by differences in the transposase function of the P elements rather than the repressor. To test this hypothesis, a set of 12 P strains representing the entire range of GD sterility potential was selected and tested for both the ability to activate sn^w(M) hypermutability and the potential to cause GD sterility. In each case, only autosomes and Y chromosomes of the P strains were tested. The results (Fig. 2) indicate a very close correlation between the transposase function as measured by sn^w(M) activation, and the potential for GD sterility. One strain in Fig. 2 (designated 78.74) was clearly an outlier and is under further investigation. A reasonable interpretation of this experiment is that GD sterility and sn^w hypermutability are both caused by the P transposase, and that P strains vary widely in the amount or kind of transposase they produce.

For each of the 12 strains in this experiment, in situ hybridization was used to estimate the number of autosomal P elements (*20*). The values obtained were highly variable among different strains (ranging from 7 to 38). However, there was no statistically significant correlation between the number of autosomal P elements, which include both complete and defective elements, and the frequency of GD sterility (Kendall's $\tau = -0.23$, $P = 0.15$) or the mutability of sn^w ($\tau = -0.06$, $P = 0.39$). This contrasts with the high correlation ($\tau = 0.779$, $P = 0.00023$) between GD sterility and sn^w mutability. We infer that the variability in transposase function comes from intrinsic differences among P elements (complete versus defective, for example) or from differences in their chromosomal positions.

The reason for a link between transposase and GD sterility is not clear, but one possibility is that a high frequency of dominant cell–lethal mutations, perhaps caused by chromosome breakage, occurs in the germ cells when transposase is present. The germline would be eliminated early in development, and GD sterility could result. However, as argued elsewhere (*3, 21*), the sterility frequencies and temperature sensitivity curves for GD sterility differ quantitatively from expectations under this hypothesis. Therefore, it seems likely that a more specific temperature-dependent interaction between transposase and the presumptive germ cells leads to failure of gonadal development.

In conclusion, transpositionally autonomous and nonautonomous P elements both exist and can be distinguished using the sn^w assay procedure. The P family of elements is thus analogous to *Ac* and *Ds* controlling element systems in maize and combinations of elements in prokaryotes (*22*). The P transposase is probably responsible for GD sterility in the hybrid dysgenesis syndrome. The hypermutability of sn^w is specific to the P transposase, and is not affected by activity in the I-R dysgenesis system.

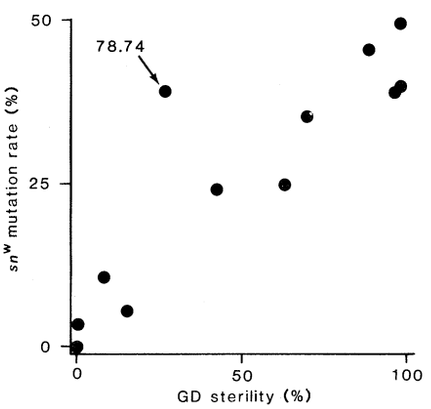

Fig. 2. The correlation between GD sterility and sn^w hypermutability. Males from each of 12 P strains were crossed to sn^w(M) females to obtain sons whose sn^w mutability was determined as in previous experiments (see legend to Fig. 1.). An average of 598 sons of sn^w males were scored per strain. The same males were also mated to compound-X M strain females to yield daughters to be tested for GD sterility (*21*). Approximately 138 such F_1 females were tested per strain. A compound-X M strain was used in these crosses to keep the paternal genome of the dysgenic females identical to that of the males being tested for sn^w mutability. The P and Q strains used were from a set of inbred wild-derived stocks (*24*). The names of the stocks used are (from left to right in the figure): 78.38, ν_6, 8.30.25, 7.29.11, 78.74, 7.29.26, 78.55, 8.31.12, 78.61, 8.31.15, π_2, and 78.100. The number of autosomal P elements could not be determined precisely by in situ hybridization because of variability between slides. However, our estimates are: 38, 10, 24, 13, 16, 34, 14, 27, 7, 20, 22, and 11, respectively.

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References and Notes

1. M. G. Kidwell, J. F. Kidwell, J. A. Sved, *Genetics* **36**, 813 (1977).
2. J. C. Bregliano and M. G. Kidwell, in *Mobile Genetic Elements*, J. A. Shapiro, Ed. (Academic Press, New York, 1983), pp. 363–410.
3. W. R. Engels, *Annu. Rev. Genet.* **17**, 315 (1983).
4. P. M. Bingham, M. G. Kidwell, G. M. Rubin, *Cell* **29**, 995 (1982).
5. K. O'Hare and G. M. Rubin, *ibid.* **34**, 25 (1983).
6. A. C. Spradling and G. M. Rubin, *Science* **218**, 341 (1982).
7. R. E. Karess and G. M. Rubin, *Cell* **38**, 135 (1984).
8. Bingham *et al.* (*4*) distinguish between the terms "P factor" and "P element" depending upon whether it has been identified by genetic effects or DNA homology, respectively. Some ambiguity in this terminology is indicated by the present results which show that even nonautonomous elements such as those at sn^w can be identified genetically. A more useful distinction is that of autonomous versus nonautonomous transposition.
9. W. R. Engels, *Proc. Natl. Acad. Sci. U.S.A.* **76**, 4011 (1979).
10. W. R. Engels and C. R. Preston, *Cell* **26**, 421 (1981).
11. The allele previously called sn^+ is actually distinguishable from the wild type in heterozygotes with the sn^{x2} allele; unpublished results. In addition, H. Roiha, K. O'Hare, and G. Rubin (personal communication) have shown that sn^+ retains a P element in the *singed* gene.
12. H. Roiha, K. O'Hare, G. Rubin, personal communication.
13. W. R. Engels, *Genetics* **98**, 565 (1981).
14. The rules of inheritance of cytotype have been described previously [W. R. Engels, *Genet. Res. Camb.* **33**, 219 (1979), and (*13*)]. The system involves aspects of both chromosomal and extrachromosomal heredity. The P cytotype is thought to indicate the presence of an element-encoded repressor of transposition.
15. The stock C(1)DX,yf π_2 was described in (*13*). See also (*23*) for genetic terminology. Crosses to compound X females result in males with patroclinous X chromosomes.
16. B. Slatko, S. Hanlon, R. Woodruff, *Drosophila Inform. Serv.* **59**, 115 (1983).
17. The pseudo-M strain used here was *Muller-5 (Birmingham)* as described in (*4*).
18. J. C. Bregliano *et al.*, *Science* **207**, 606 (1980); A. Bucheton, R. Paro, H. Sang, A. Pelisson, D. Finnegan, *Cell* **38**, 153 (1984).
19. M. G. Kidwell, *Genet. Res.* **33**, 105 (1979). The properties of Q strains are also discussed in W. R. Engels and C. R. Preston, *Drosophila Inform. Serv.* **56**, 35 (1981); M. G. Kidwell, *Genetics* **98**, 275 (1981); M. J. Simmons, J. D. Raymond, T. P. Culbert, T. R. Laverty, *Genetics* **107**, 49.
20. Labeled RNA was hybridized to salivary gland chromosomes and analyzed by autoradiography. The probes homologous to P sequences and procedures are described in W. R. Engels and C. R. Preston [*Genetics* **107**, 657 (1984)] and (*5*).
21. W. R. Engels and C. R. Preston, *Genetics* **92**, 161 (1979).
22. J. Shapiro, Ed., *Mobile Genetic Elements* (Academic Press, New York, 1983).
23. D. L. Lindsley and E. H. Grell, *Carnegie Inst. Washington Publ.* 6 (1968).
24. W. R. Engels and C. R. Preston, *Genetics* **95**, 111 (1980).

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