A latitudinal cline in *P–M* gonadal dysgenesis potential in Australian *Drosophila melanogaster* populations

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Summary

Isofemale lines of *Drosophila melanogaster* from six localities along the east coast of Australia, spanning 2900 km and 26 degrees of latitude, were assayed for their gonadal dysgenesis characteristics in the *P–M* system of hybrid dysgenesis. A strong clinal pattern with latitude was discovered. From north to south, the first two populations were typical strong *P* populations, and the next population was moderate *P*. The next population to the south was neutral (*Q*), with some weak *P* and weak *M* characteristics. The two southernmost populations were typical *M* populations. Much variance in *P* activity in *P* populations and in susceptibility to *P* activity in *M* populations was detected among isofemale lines. This clinal pattern with latitude of the *P–M* system is paralleled by similar clinal patterns for frequencies of common cosmopolitan inversions and of certain allozymes in Australia. A model of introductions of flies with different characteristics in the north and south could account for the *P–M* clinal pattern, but cannot account for an intermediate *Q* population, nor establish the inversion and isoenzyme clines at the same time. Current models of transposable element population dynamics are limited to single population dynamics, and are therefore inadequate for these clinal data.

1. Introduction

P elements are mobile genetic elements in *Drosophila melanogaster* that may transpose and replicate at high frequency under certain conditions in germline cells. Such transposition causes or correlates with some or all of a suite of phenomena, collectively termed '*P–M* hybrid dysgenesis' since the necessary conditions were originally detected in crosses between *M* strain females (*M* for 'maternal') and *P* strain males (*P* for 'paternal'), but not in the reciprocal crosses (Kidwell, Kidwell & Sved, 1977). The phenomena include temperature dependent gonadal dysgenesis, embryonic lethality in F2 eggs, recombination in males, transmission ratio distortion, chromosomal breaks and rearrangements, and mutations, which are often unstable (Bregliano & Kidwell, 1983; Engels, 1983; Kidwell, 1984). Full-sized, intact *P* elements (O'Hare & Rubin, 1983) are competent or 'autonomous' (Engels, 1984) in that they can mediate their own transposition and that of non-autonomous *P* elements. The latter generally seem to be internally deleted versions of autonomous *P* elements. The degeneration process that forms deleted elements may be linked to transposition, since it can be relatively rapid under

dysgenic conditions (Voelker *et al.* 1984; Daniels *et al.* 1985*a*; Daniels, Strausbaugh & Armstrong, 1985*b*).

The presence of autonomous *P* elements may be required for suppression of transposition, although recent results cast some doubt on this (Sakoyama *et al.* 1985; H. M. Robertson & W. R. Engels, personal communication). The transposition-suppressing state is at least partly maternally inherited and is called '*P* cytotype' (Engels & Preston, 1979); its absence is called '*M* cytotype'. The two conditions are extremes between which are found different degrees of susceptibility to *P* activity (Kidwell, 1985). It is apparent that active *P* elements can be 'infective', replicatively transposing to different chromosomes or different regions of the same chromosome (Kidwell, Novy & Feeley, 1981) in both *M* and *P* cytotypes, albeit at much lower rates in *P* cytotype (Preston & Engels, 1984; Engels, 1985). At the population level, major areas that are not understood include the conditions under which *P* elements can invade a population, their population dynamics in an invaded population, their genomic and populational distributions, and their effects on a population both as potentially deleterious factors and as sources of genetic variation (Engels, 1985; MacKay, 1985; Kidwell, 1986).

P–M hybrid dysgenesis was initially detected in crosses between certain wild strains and laboratory

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strains (Kidwell *et al.* 1977). The differences between them suggested that there might be variation in *P–M* characteristics among populations in the wild. Surveys using reference crosses to define *P–M* characteristics have in fact provided a picture of great interpopulation variation around the world (see next paragraph). Reference cross A is a cross of males of a line to females of a tester *M* strain; cross A* is a cross of females of a line to males of a tester *P* strain (Engels & Preston, 1980). Lines are characterized as '*P*' if they cause high gonadal dysgenesis in cross A and low gonadal dysgenesis in cross A*; as neutral or '*Q*' if both crosses show very low dysgenesis; and as '*M*' if cross A shows low but cross A* shows high gonadal dysgenesis (Kidwell, Frydryk & Novy, 1983). Molecular analysis has shown that phenotypically *M* strains are in fact of two types: some laboratory strains completely lack any *P* element-hybridizing DNA, and are called *true M*; all current wild *M* strains that have been tested and some laboratory strains have *P* element-hybridizing DNA (Bingham, Kidwell & Rubin, 1982; Todo *et al.* 1984; Anxolabéhère *et al.* 1985; unpublished data), and are called *M'* or *pseudo M* (Kidwell, 1985). Populations are characterized as *P*, *Q*, or *M* if most of the individuals they comprise are *P*, *Q*, or *M* respectively.

In North America, South America and subSaharan Africa, virtually all examined populations are *P* to *Q* (Engels & Preston, 1980; Kidwell *et al.* 1983; Anxolabéhère *et al.* 1984; Kidwell & Novy, 1985). *Q* populations predominate in France and *M* populations predominate in southern and eastern Europe, northern Africa and across Asia (Anxolabéhère, Nouaud & Périquet, 1982; Anxolabéhère *et al.* 1984, 1985). In Japan, *P* or *Q* populations predominate, although some populations are *M* (Takada *et al.* 1983; Yamamoto, Hihara & Watanabe, 1984; S. Ishiwa, personal communication). Sparse data from India, southeast Asia, and Micronesia show *Q* to *M* populations (S. Ishiwa, personal communication). Considerable variation is typically found within populations, so that the above designations qualitatively describe central tendency only.

Prior to this study, Australia had not been well surveyed for *P–M* hybrid dysgenesis. A few populations, from southern latitudes, had been shown to be *Q* or *M* (Kidwell *et al.* 1983; Anxolabéhère *et al.* 1985), and one strong *P* line was known (Angus & Raisbeck, 1979). A preliminary study by M. M. Green and J. B. Gibson (personal communication) showed a moderate degree of male recombination (*MR*) in tests of extracted chromosome lines from Townsville, Queensland (19° S lat., on the east coast of Australia), and no *MR* activity of extracted chromosome lines from Huonville, Tasmania (43° S lat.). These results suggested that a north–south survey might show variation in *P–M* characteristics.

Accordingly, populations from six localities spanning 2900 km and 26 degrees of latitude along the east coast of Australia were surveyed. Gonadal dysgenesis in reference crosses was used as an assay of *P–M* characteristics. This paper reports the results of the survey, which show a dramatic latitudinal cline in *P–M* hybrid dysgenesis potential, and discusses hypotheses of its cause.

2. Materials and methods

(i) *Localities and tested lines*

Localities for this study were chosen close to the eastern coast of Australia, all at elevations of less than 150 m, to avoid effects of local inland weather patterns. Six localities were chosen, spaced from 225 to 875 km apart, and spanning 25 degrees of latitude, from tropical northern Queensland to cool temperate southern Tasmania. *D. melanogaster* populations from the localities were represented as collections of lines, each line started from a single wild-caught female.

The localities, numbers of isofemale lines tested, and details of collection are as follows: CAIRNS, Queensland (16·9° S lat.; 16 lines; collected 25 November 1982, by P. R. Anderson; under mango tree, on dropped fruit); ROCKHAMPTON, Queensland (23·4° S lat.; 20 lines; collected 4 December 1982; by P. R. Anderson; banana bait traps in suburban locality); COFF'S HARBOUR, New South Wales (30·3° S lat.; 20 lines; collected 21 March 1983, by I. A. B. and S. Easteal; in banana plantation, on discarded fruit); BATEMAN'S BAY, New South Wales (35·7° S lat.; 18 lines; collected 21 March 1983, by P. R. Anderson; banana bait traps in suburban locality); CANN RIVER, Victoria (37·6° S lat.; 20 lines; collected 21 March 1983, by P. R. Anderson; banana bait traps in suburban locality); CYGNET, Tasmania (43·2° S lat.; 20 lines; collected 27 April 1983, by A. V. Wilks and J. B. Gibson; at cider factory, on crates of apples). Each isofemale line was maintained in the laboratory in 30 ml vials on a standard cornmeal-sucrose-yeast-agar medium.

(ii) *Method of testing and scoring*

Gonadal dysgenesis in reference crosses was chosen as the most expeditious assay technique for *P* activity and for susceptibility to *P* activity. The lines from Bateman's Bay were assayed in 1985 (about 45 generations after establishment); the others were tested in 1983 (4–10 generations after establishment). The tester strains used were: HARWICH, a strong *P* strain with 30–50 *P* elements per haploid genome; and CANTON-S, a strong *M* strain completely lacking *P* elements (*true M* (Kidwell, 1985)). The properties of these strains in the *P–M* system have been well-characterized (Bingham *et al.* 1982).

Two reference crosses were done with each line to characterize its *P* activity (measured as gonadal dysgenesis in cross A) and its degree of susceptibility to

P activity (measured as gonadal dysgenesis in cross A*) (Engels & Preston, 1980). Flies from the line being tested were assayed individually. Virgin females were held for three to seven days after eclosion before crossing. For Cross A tests, each male was crossed with five Canton-S virgins; for Cross A* tests, each female was crossed with five Harwich males. Each cross was made at 29 °C in a vial with medium. Adults were left in the vials for three (cross A) or four (cross A*) days, then discarded. On the 14th day, emerged F₁'s of both sexes were transferred to a fresh medium vial at 23 °C with live yeast and allowed to mature for four days before dissection of females to assay gonadal dysgenesis. An ovary was scored as non-dysgenic if an egg in even one ovariole was developed (Engels & Preston, 1979; Schaefer, Kidwell & Fausto-Sterling, 1979). A test was only recorded if ten or more F_1 females were produced; all F_1 females produced were scored. For each isofemale line, reference crosses were performed until at least three individual males had been scored in cross A and at least three individual females in cross A*. The mean number of crosses scored per line in the cross A tests was 3·6, in the cross A* tests, 5·1; the mean number of F_1 females scored per cross was 26.

Each cross was scored for the proportion of dysgenic females (with one or both ovaries undeveloped). This measure gives a somewhat higher value from the same raw data than the proportion of undeveloped ovaries, used in some studies (e.g. Anxolabéhère *et al.* 1984; Kidwell, 1985; Kidwell & Novy, 1985), or the proportion of completely infertile F_1 females (probably equivalent to the proportion of bilaterally dysgenic females) used by others (e.g. Engels & Preston, 1980; Kidwell *et al.* 1981). Unweighted means and standard errors of the cross A and cross A* scores were calculated for each line; the line means were averaged and standard errors calculated to yield cross A and cross A* means for each population.

3. Results

(i) *Population means*

Since there are no conventions for defining the phenotypes *P*, *Q*, and *M* using the different ways of evalu-

ating sterility, a line will be arbitrarily called *P* if it has > 10% dysgenic females in cross A and < 10% in cross A*; *Q* if it has < 10% dysgenesis in both reference crosses; and *M* if it has < 10% dysgenesis in cross A and > 10% dysgenesis in cross A*. The means of the Cross A and Cross A* tests for the six populations tested are presented in Table 1. The three northern populations differ dramatically from the two southern ones in their *P* activity potential and in their susceptibility to *P* activity. The mean *P* activity values of 55% for the Cairns population and 61% for the Rockhampton population define them as typical *P* populations; the mean *P* activity value of the Coff's Harbour population, 35%, is much less, but still moderate *P*. The low degrees of susceptibility for all three populations class them as having *P* cytotype. Bateman's Bay is, on average, neutral or *Q*. The two southernmost populations show little *P* activity (9% for Cann River, 3% for Cygnet) and show moderate degrees of susceptibility to *P* activity (33% for Cann River, 47% for Cygnet), and are thus *M*. The changes over space in *P* activity and in susceptibility both appear to be clinal with latitude. The changes occur reciprocally, as is expected from the known properties of the *P–M* system.

(ii) *Isofemale line means and variances*

The data presented in Table 1 and above are means of means, and they mask interesting variation within and among isofemale lines from the populations under consideration. In Fig. 1 are presented graphs for each locality of mean *P* activity score and mean susceptibility to *P* activity for each isofemale line. Each graph presents the data for one locality; the graphs are presented with a map of the east coast of Australia. The general clinal pattern from north to south of the population means, described above, is seen to be due to a general trend of, firstly, lowering *P* activity among lines with increasing latitude to 36° S lat., then increasing susceptibility to *P* activity among lines south of 36° S lat. It is immediately apparent that there is much variance among isofemale lines within each of the populations. The Rockhampton population has some

Table 1. *Mean dysgenesis among lines for each population in reference crosses A and A**

Locality	S lat. (degrees)	Number of lines tested	Cross A dysgenesis (*P* activity) mean ± s.e. (%)[a]	Cross A* dysgenesis (susceptibility) mean ± s.e. (%)[a]
Cairns	16·9	16	54·5 ± 5·1	11·1 ± 4·1
Rockhampton	23·4	20	60·6 ± 5·2	5·4 ± 1·8
Coff's Harbour	30·3	20	34·7 ± 4·8	2·7 ± 0·8
Bateman's Bay	35·7	18	6·4 ± 2·0	9·3 ± 4·0
Cann River	37·6	20	8·8 ± 1·5	33·0 ± 6·7
Cygnet	43·2	20	3·4 ± 0·5	46·7 ± 6·5

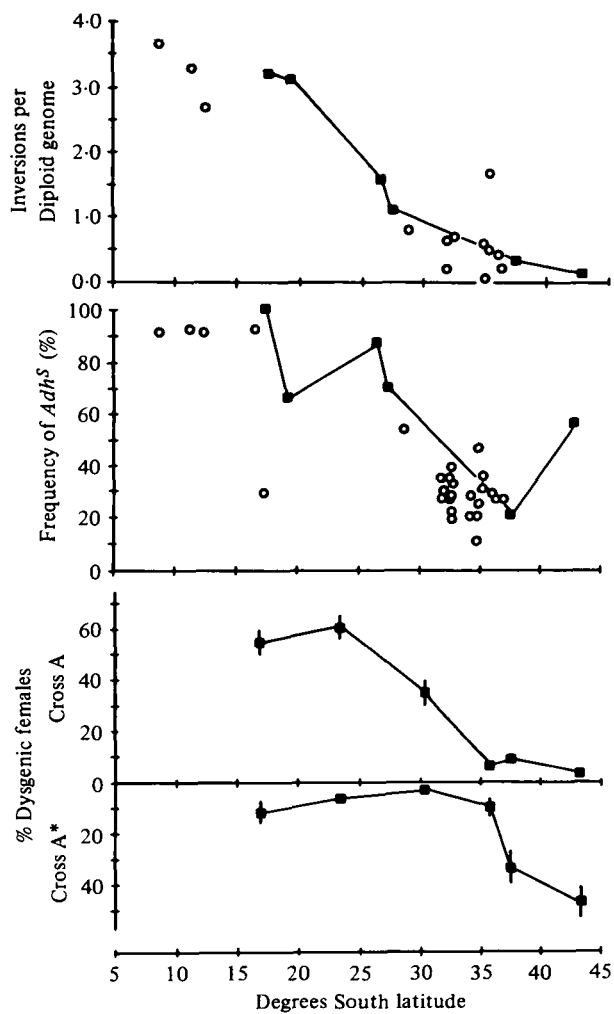[a] Unweighted means, ± standard error, of the individual isofemale line means.

Fig. 1. Graphs of means of P activity (proportion dysgenic females in cross A, vertical axes) and susceptibility to P activity (cross A*, horizontal axes), with standard errors, for each isofemale line in the six populations tested, displayed next to a map of the east coast of Australia. The names of localities, their latitudes (°S lat.), and the number of lines tested from that locality (n) are displayed in each graph.

lines with P activity values of nearly 100%, and others of only around 15%; Coff's Harbour has one line with a P activity value of 85%, and others of less than 10%. Half (9/18) of the lines from Bateman's Bay are virtually completely neutral or Q (no sterility in either cross A or A*), but among the rest one line shows 34% P activity, and another has 61% susceptibility to P activity. Among the lines from Cann River, nearly half have P cytotype (less than 10% susceptibility), but some show nearly 100% susceptibility; lines from Cygnet are more uniformly spread along the susceptibility axis, with values from 0% to more than 95%. It should also be noted that individual flies tested showed much within-line variation. The large standard error brackets around some of the means of lines in Fig. 1 reflect the large variance among individuals in these lines (as well as the small numbers of individuals tested per line). Such variance is, in fact, the

norm for natural populations, as P populations have been found to comprise individuals or isofemale lines with P to Q characteristics, and M populations manifest a range of Q to M traits (Engels & Preston, 1980; Anxolabéhère *et al.* 1982; Yamamoto *et al.* 1984; Kidwell & Novy, 1985; S. Ishiwa, personal communication).

4. Discussion

(i) *Dysgenesis is due to the* P–M *system*

The formal possibility exists that the results described above are not due to the *P–M* system of hybrid dysgenesis but to some other interaction between the wild lines and the tester stocks. Based on Southern blot analysis of genomic DNA digested with the restriction enzyme *Acc* 1, using different P element probes, preliminary results show that lines from all five populations have many P element-hybridizing DNA sequences present in their genomes. However lines from the three northernmost populations have many full-sized P elements per genome (or potentially full-sized; based on the presence of a 2·36 kb *Acc* 1 fragment), whereas those from the two southernmost populations have none or only a few full-sized elements (unpublished data). Thus the 'M' lines in the southern populations are correctly classified as M' or *pseudo M* (Kidwell, 1985). These results are consistent with other studies that show correlations between inferred numbers of full-sized P elements per genome and the P activity and susceptibility to P activity of lines from natural populations (Todo *et al.* 1984; Anxolabéhère *et al.* 1985). I tentatively conclude that gonadal dysgenesis in the reference crosses may be, at least to a first approximation, a good indicator of the relative number of full-sized P elements, and that the gonadal dysgenesis observed in the reference crosses of the present study is in fact the result of *P–M* hybrid dysgenesis.

(ii) *Assumptions about the cline and* P *element biology*

D. melanogaster was present in Australia as early as 1894 (Bock & Parsons, 1983), and was probably widespread by at least 1923 (Malloch, 1923). It seems likely that P elements were not present in at least most *D. melanogaster* populations around the world prior to about 1950 (Kidwell, 1983; but see (Engels, 1986)), since they are not found in most lines collected before the mid-1950s in North America, the mid-1960s in France, and 1970 in the Soviet Union (D. Anxolabéhère, M. G. Kidwell and G. Periquet, personal communication; a few early lines with P element homology are most easily interpreted as having been contaminated during their time in the laboratory). It therefore seems likely that, prior to at least 1950, *D. melanogaster* was present in Australia as *true M* popu-

lations (completely lacking *P* elements), and probably with a geographical distribution essentially like the present one. Thus hypotheses about the *P–M* cline must establish it within a 35-year period, and over an area with *true M* flies already present.

An alternative assumption, that *D. melanogaster* populations in Australia were *M'* prior to a recent (i.e. since 1950) introduction of full-sized *P* elements, begs the question of the origin of the *M'* strains. Since early strains from the rest of the world seem to have been *true M* (as noted above), such an assumption suggests an Australian origin of the *M'* populations. This is merely a longer term version of the assumption of establishment of the present pattern within the last 35 years.

(iii) *Hypotheses*

(a) *'Varying selection with latitude' hypotheses.* Models have shown that selection against individuals with large numbers of transposable elements per genome could lead to 'regulation' of the numbers of elements per genome (Charlesworth & Charlesworth, 1983). High temperatures could select for individuals with low *P* activity, since gonadal dysgenesis rises dramatically in dysgenic flies reared above 25 °C (Engels & Preston, 1979; Schaefer *et al.* 1979). Indeed, mixes of *P* and *M* strains maintained in the laboratory at 27 °C resulted in *P* populations with reduced *P* activity potential compared to controls at lower temperature (Kiyasu & Kidwell, 1984), consistent with the predictions of a selection model (Charlesworth & Charlesworth, 1983).

Air temperature (mean summer and winter maxima and minima) varies approximately linearly with latitude along the east coast of Australia. February (summer) mean maxima and minima are approximately 32° and 25 °C for Cairns, Queensland (16·9° S lat.) and 21° and 12 °C for Hobart, Tasmania (42·8° S lat., near Cygnet); and July (winter) mean maxima and minima are 25° and 16 °C for Cairns and 12° and 5 °C for Hobart (data from Commonwealth Bureau of Meteorology). If food sources such as rotting fruit are close to or above ambient air temperatures, then critical temperatures for the *P–M* system are certainly exceeded in the north of Australia, where a fly might complete its entire life above 25 °C; these temperatures are rarely even achieved in the extreme south. Disregarding the role of susceptibility in determining the degree of gonadal dysgenesis, one might therefore hypothesize that gonadal dysgenesis (or, possibly, some other subtler deleterious effect of transposition) in the hot northern populations would select against *P* activity, resulting in lower levels of *P* activity potential in the north relative to populations in more temperate climes. Since the clinal pattern is obviously in the other direction, however, such an hypothesis can be dismissed. Even if the assumption that larval habitat achieves the same temperatures as

ambient air is not made, larval habitat temperature in the south is extremely unlikely to be higher than in the north, so a direct effect of latitudinal temperature cannot have caused the cline by the selective mechanism demonstrated in the laboratory experiments cited above.

Other, subtler, selective processes could vary with latitude. It is conceivable that some aspect of the genetic background of the host flies varies clinally, leading to the clinal pattern in *P–M* characteristics. Likewise, transposition and degeneration rates might intrinsically vary with latitude in a way that could establish a cline. No data are available at present for any such variability.

(b) *Other clines and 'introductions' hypotheses.* The *P–M* cline is in fact the *third* latitudinal cline found in *D. melanogaster* in Australia. Knibb and co-workers have shown a dramatic cline in frequencies of the common cosmopolitan inversions with latitude (Knibb, Oakeshott & Gibson, 1981), and Oakeshott, Anderson and co-workers have documented clines in allozyme frequencies for several enzymes including alcohol dehydrogenase (ADH; EC 1.1.1.1) (Oakeshott *et al.* 1982). Fig. 2 shows graphs of mean numbers of the common cosmopolitan inversions per diploid genome, frequencies of Adh^S, and *P–M* gonadal dysgenesis in crosses A and A*, plotted against latitude for populations from the east coast of Australia. While the variation is large in all three data sets, the clinal patterns are dramatic and occur over the same latitudes.

P–M hybrid dysgenesis has probably not generated the cline in cosmopolitan inversions, since linkage disequilibria between particular inversions and alleles at linked loci are found. In particular, $In(2L)t$ is in marked gametic disequilibrium with Adh^S and $Gpdh^F$ (Mukai & Voelker, 1977; Knibb, 1983); indeed, among 80 $In(2L)t$-carrying gametes, Knibb (1983) found only five carrying Adh^F, and the inversion involved may well have been $In(2L)Cy$, a rare cosmopolitan inversion that is difficult to distinguish from $In(2L)t$ (W. R. Knibb, personal communication). Such linkage argues strongly for a unique origin of $In(2L)t$, rather than on-going *de novo* creation at an appreciable rate; the same is probably true for the other cosmopolitan inversions. Mettler, Voelker & Mukai (1977) suggested that 'unique' inversions might be 'associated with the syndrome termed "hybrid dysgenesis".' In Australia there is no obvious association of unique or 'endemic' inversions with the clinal *P–M* hybrid dysgenesis pattern; the frequencies of endemic inversions are low and similar (about 5% on average) in all populations examined (Knibb *et al.* 1981; unpublished data). It must be noted that the latitudinally clinal pattern of Adh^S frequency in Australia is only marginally affected by removal of $In(2L)t$-Adh^S data (Knibb, 1983) as was shown for the weaker but similar clines in North America (Voelker *et al.* 1978), so that the clines in

P–M hybrid dysgenesis, cosmopolitan inversion frequencies, and *Adh*^{S} frequencies seem to be independent.

The remarkable concordance of these three sorts of genetic variation suggests an introduction hypothesis as a causal explanation of the *P–M* cline. Introductions from two areas, with different characteristics in the *P–M* system, with different *Adh* allele frequencies, and with different frequencies of the cosmopolitan inversions, could have generated the patterns seen today if the introductions were made into the northern and southern regions respectively and there were no indigenous flies. However, as argued above, *D. melanogaster* was probably distributed in Australia essentially as it is now prior to the earliest probable introduction of *P* element-bearing flies. Active *P* elements can spread infectively through populations, but inversions, allozymes, and defective *P* elements (in the absence of autonomous elements) cannot. It therefore seems unlikely that any simple introduction pattern could simultaneously effect the three clinal patterns shown in Fig. 2; the *P–M* clinal pattern was probably secondarily imposed on pre-existing clines in isozyme and inversion frequencies.

A 'two *P* introductions with degeneration' hypothesis could establish a *P–M* clinal pattern. A first introduction could have converted all Australian *true M* populations to the *P* state; then the degenerative process that produces deleted elements could have changed them all to *M'*. [It should be noted that this latter supposition, regarded as unlikely on the grounds of parsimony by Engels in the context of discussion of the Recent Invasion hypothesis (Engels, 1986), must be accepted as a possible scenario, since the degenerative process is known to occur (Voelker *et al.* 1984; Daniels *et al.* 1985*a*, *b*).] A later reinvasion of *P* elements into the northern part of Australia would generate *P* populations and a *P*-to-*M'* transition zone moving southward. The clinal pattern at the *P*-to-*M'* encounter zone would depend on the interaction between the diffusion rate of chromosomes into a population, the genomic transposition rate of the *P* elements into the *M'* genomes, and the precise relationship between numbers and types of *P* elements and the individual and population phenotypes. Such a model might generate a rapid switch of the phenotype of a population from *M'* to *P* as *P* elements invade, rather than an intermediate *Q* population as is found at Bateman's Bay. Since invasion or chromosomal contamination experiments have not been done with *M'* lines, no experimental data directly address this question. However one can argue that if an introduction lead to a conversion of *M'* to *Q*, rather than to *P*, then invasion would not occur, as introduced *P* elements would only generate a *Q* population in which they could not spread further due to suppression of transposition by *P* cytotype. A *Q* population would act as a barrier to further spread. Since *P* populations are found over a large area in Australia, invasion dy-
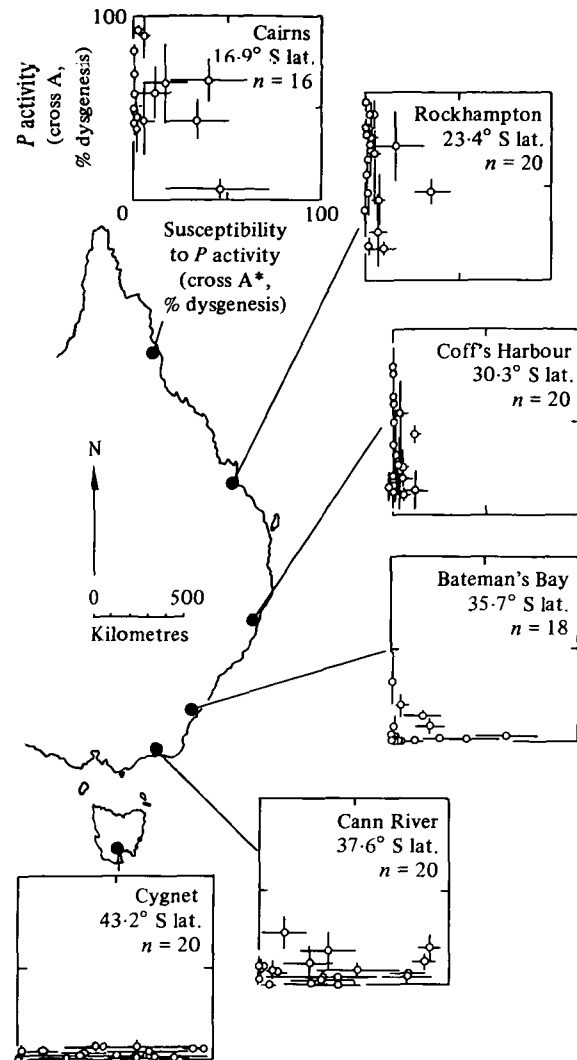

Fig. 2. Mean number of common cosmopolitan inversions per diploid genome (*ex* Knibb, Oakeshott & Gibson, 1981), frequency of the *Adh*^{S} allele (*ex* Oakeshott *et al.* 1982), and mean *P* activity and susceptibility to *P* activity (percent dysgenic females in crosses A and A*; data from Table 1) all plotted against degrees south latitude (°S lat.). Data for populations of *D. melanogaster* from the east coast of Australia are shown as connected dark squares; data from inland and elsewhere (including data from New Guinea, 8·8° S lat.) are shown as unconnected open circles.

namics must not conform to such a model. The Bateman's Bay lines were only assayed after some 45 generations in the laboratory. It is possible that their characteristics had changed from weak *P* to *Q* in that period (although preliminary new results suggest that this is not the case (unpublished data)). Therefore the 'two *P* introductions with degeneration' hypothesis cannot be critically evaluated without more data on *P* element biology and details of the temporal and spatial nature of the cline.

(iv) *General considerations*

Kaplan, Darden & Langley (1985) have published the only model of the population dynamics of trans-

posable elements that takes into account a degenerative process that generates deleted, non-autonomous elements. The degenerative process, replicative transposition, excision, and self-regulation of numbers of elements per genome govern the dynamics of their model. Briefly, upon introduction of elements into a finite population, the active, complete elements increase in frequency very quickly to their self-regulated number, then the degenerative process leads inexorably (but possibly very slowly) to the loss of complete elements. This latter state can be a sort of quasi-equilibrium, with a nearly constant average proportion of complete elements and total number of elements per genome for a long period of time. The model is limited to the dynamics of an element introduced into a single finite population, and as such is not directly applicable to the $P–M$ clinal pattern in eastern Australia. It is important for demonstrating the power of the degenerative process in controlling the quasi-equilibrium state in which a population would find itself after invasion was over. However models of the interpopulational dynamics of transposable elements are clearly needed before the distributions of P elements in nature can be understood.

More data are needed also, both to define the nature of the cline between Coff's Harbour and Cann River, especially near Bateman's Bay, and to determine whether the cline is stationary or moving. Kidwell (1986) has argued that the current status of the $P–M$ system in natural populations may be a transient condition of invasion of a species by a mobile element. If so, the clinal pattern in eastern Australia may be transient as well, and there is a certain urgency to sampling it. The dramatic juxtaposition of P and M populations within potential migration distances of the flies (Coyne *et al.* 1982) has not been described anywhere else in the world, and seems a unique opportunity to examine the active population dynamics of P elements in wild populations.

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