The P-M Hybrid Dysgenesis Cline in Eastern Australian *Drosophila melanogaster*: Discrete P, Q and M Regions Are Nearly Contiguous

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ABSTRACT

The dramatic latitudinal cline in P-M hybrid dysgenesis characteristics along the east coast of Australia is not smooth. Tests of recent collections of *Drosophila melanogaster* from the southeastern coast define the previously described cline as comprising three discrete, apparently contiguous regions of P, Q and M phenotypes, respectively. Northern populations from Cairns (16.9°SLat) to Ourimbah (33.4°SLat) are phenotypically P; populations from Wollongong (34.4°SLat) to Eden (37.1°SLat) are Q; and populations from Genoa (37.5°SLat) to Cygnet (43.2°SLat) are M. The decline in P activity from northern Queensland (55–60% gonadal dysgenesis (GD) in cross A) to mid-New South Wales (20–30% GD in cross A) is gradual; proceeding south, there then is a sharp drop to Q populations (<10% GD in crosses A and A*). This drop in P activity occurs in only 150 km, across the urban and suburban area of Sydney. Q populations are then found south to Eden, but Genoa, only about 50 km further southeast, is clearly M (48% GD in cross A*), as are two populations further south. The two discontinuities in the P-M cline do not correspond to obvious climatic differences along the coast, nor to obvious barriers to dispersal of *D. melanogaster*. The cline has apparently not moved between 1983 and 1985–1986.

EASTERN Australian populations of *Drosophila melanogaster* vary dramatically in their characteristics in the P-M system of hybrid dysgenesis, and the variation is clinal with latitude (BOUSSY 1987). This strong clinal pattern contrasts with a worldwide pattern of near qualitative homogeneity within continental areas, but large differences between such areas (ENGELS and PRESTON 1980; ANXOLABÉHÈRE, NOUAUD and PERIQUET 1982; KIDWELL, FRYDRYK and NOVY 1983; TAKADA *et al.* 1983; ANXOLABÉHÈRE *et al.* 1984; YAMAMOTO, HIHARA and WATANABE 1984; ANXOLABÉHÈRE *et al.* 1985; KIDWELL and NOVY 1985; S. ISHIWA, personal communication).

P-M hybrid dysgenesis (KIDWELL, KIDWELL and SVED 1977) is a collection of phenomena which are caused by or correlate with the insertion and/or excision, often at high rate, of transposable elements called *P* elements in the genome of *D. melanogaster* (BINGHAM, KIDWELL and RUBIN 1982; O'HARE 1985). The phenomena include gonadal dysgenesis in F_1 individuals reared at high temperature, embryonic lethality of F_2 eggs, recombination in males (which does not normally occur in *D. melanogaster*), chromosomal transmission ratio distortion, chromosomal breaks and rearrangements, and mutations, which are often unstable [see reviews by BREGLIANO and KIDWELL (1983) and ENGELS (1983)]. The phenomena are due to events that occur at high frequency in the germ line of F_1 individuals derived from crosses between females of "M" strains (M for "maternal" contribution) and males of "P" strains (P for "paternal" contribution), but usually not at all or only at very low rates in the reciprocal cross or within strains (KIDWELL, KIDWELL and SVED 1977; PRESTON and ENGELS 1984).

Flies from a natural population are tested for their characteristics in the P-M system of hybrid dysgenesis by making reference crosses under defined conditions to well-characterized tester strains and scoring one or more of the typical manifestations of the system (*e.g.*, KIDWELL, FRYDRYK and NOVY 1983). Surveys of wild flies, then, usually entail two reference crosses, cross A (males are crossed to females of a reference M strain) to evaluate P activity potential (KIDWELL, KIDWELL and SVED 1977), and cross A* (females are crossed to males of a reference P strain) to evaluate susceptibility to P activity (ENGELS and PRESTON 1980). Gonadal dysgenesis (GD) in F_1 females is the trait most commonly evaluated. Populations with a high proportion of dysgenic female offspring from cross A and a low proportion from cross A* are said to have P phenotype; populations with a low proportion in both tests are said to have neutral or Q phenotype; and populations with a low proportion of cross A GD but with high cross A* GD are said to have M phenotype (KIDWELL, FRYDRYK and NOVY 1983). P-

M properties of flies collected from nature actually occur along a continuum from strong P to weak P to Q to weak M to strong M, so the use of any of these descriptors is a qualitative label only.

All wild M populations that have been sampled in the last 10 yr have been found to contain nonautonomous *P* elements, which seem to be partly deleted versions of the complete 2907 base *P* element sequence (BINGHAM, KIDWELL and RUBIN 1982; O'HARE and RUBIN 1983; TODO *et al.* 1984; ANXOLABÉHÈRE *et al.* 1985; SAKOYAMA *et al.* 1985; I. A. BOUSSY, M. J. HEALY and J. G. OAKESHOTT, unpublished data). At least some of these strains may lack complete *P* elements entirely. Such populations and lines are called M′ or pseudo M, while lines completely lacking any *P* element-hybridizing DNA are called true M (ENGELS 1984; KIDWELL 1985). Note that such designations are phenotypic *and* genotypic designations. Lines collected before the mid-1950s in North America, the mid-1960s in France, and 1970 in the Soviet Union and kept in the laboratory since then were nearly all true M in recent tests [a few early lines that were found to contain *P* elements had probably been contaminated by P flies during their stays in the laboratory (D. ANXOLABÉHÈRE, M. G. KIDWELL and G. PERIQUET, personal communication)]. KIDWELL (1983, 1986) has interpreted this temporal pattern to mean that *P* elements were only introduced to, or only became common in, *D. melanogaster* since about 1950 [but see ENGELS (1986)].

Recently collected samples of populations from North America, South America, and sub-Saharan Africa have been determined to be virtually all phenotypically P to Q (ENGELS and PRESTON 1980; KIDWELL, FRYDRYK and NOVY 1983; ANXOLABÉHÈRE *et al.* 1984; KIDWELL and NOVY 1985). Populations in France have been shown to be generally Q; in the rest of Europe, northern Africa, and across Asia, M populations have been generally found (ANXOLABÉHÈRE, NOUAUD and PERIQUET 1982; ANXOLABÉHÈRE *et al.* 1984; ANXOLABÉHÈRE *et al.* 1985). In Japan, weak P or Q populations have been described, although some populations have been found to be M (TAKADA *et al.* 1983; YAMAMOTO, HIHARA and WATANABE 1984; S. ISHIWA, personal communication).

BOUSSY (1987), testing six collections made in 1983, showed a dramatic cline in P-M characteristics from north to south along the east coast of Australia, spanning 2900 km from 16.9°SLat to 43.2°SLat. The three northern populations sampled were found to be distinctly P, a Q population was found at Bateman's Bay (35.7°SLat), and the two southernmost populations were clearly M. There was much variability within and between individual lines from each locality. The mere existence of the cline was not enough to distinguish between hypotheses about its origination and subsequent dynamics. One conclusion of the study was that more samples were needed in the P-to-M transition region, between 30.3°SLat and 37.6°SLat, to determine the detailed structure of the cline and to evaluate its temporal stability or pattern of change, if any. This paper presents the results of cross A and A* GD tests on lines from 15 localities, from Coff's Harbour (30.3°SLat) in the north to Cann River (37.6°SLat) in the south, which span the P-to-M transition zone identified in the earlier study. The lines were collected and tested in 1985 and 1986. Comparisons are made with data from the 1983 collections from Coff's Harbour, Bateman's Bay, and Cann River (BOUSSY 1987). The current data corroborate the existence of the cline, define it as consisting of three discrete, apparently contiguous regions of P, Q and M phenotypes respectively, and show no apparent changes in the cline since 1983.

MATERIALS AND METHODS

Localities, lines and culture conditions: Localities were chosen close to the coast to avoid effects of local inland weather patterns. Populations from the localities were represented as collections of isofemale lines (each line started from a single wild-caught female). The localities (from north to south), numbers of lines tested, and details of the collections are as follows (NSW: New South Wales; Vic: Victoria): Coff's Harbour (NSW, 30.3°SLat), 20 lines, 21 February 1985, collection within 100 m of the site of the 1983 collection (BOUSSY 1987); Kempsey (NSW, 31.1°SLat), 20 lines, 20 February 1985; Taree (NSW, 31.9°SLat), 20 lines, 19–22 February 1985; Medowie (NSW, 32.8°SLat), 20 lines, 19–22 February 1985; Ourimbah (NSW, 33.4°SLat), 20 lines, 18 February 1985; Wollongong (NSW, 34.4°SLat), 5 lines, March 1986; Jasper Valley Winery, near Berry (NSW, 34.8°SLat), 5 lines, March 1986; Ulladulla (NSW, 35.4°SLat), 5 lines, March 1986; Bateman's Bay (NSW, 35.7°SLat), 5 lines, March 1986, same site as 1983 collection (BOUSSY 1987); Moruya (NSW, 35.9°SLat), 5 lines, March 1986; Narooma (NSW, 36.2°SLat), 5 lines, March 1986; Bega (NSW, 36.7°SLat), 5 lines, March 1986; Eden (NSW, 37.1°SLat), 5 lines, March 1986; Genoa (Vic, 37.5°SLat), 5 lines, March 1986; Cann River (Vic, 37.6°SLat), 19 lines, March 1986, same site as 1983 collection (BOUSSY 1987). The 1985 collections were made by I. A. BOUSSY and A. S.-F. CHONG; the 1986 collections were made by P. R. ANDERSON. All lines were kept at 20–23° in 30-ml vials on a standard cornmeal-sugar-yeast-agar medium. The mean census size in each vial was about 30. The tests of the 1985 collections were initiated between 20 June and 15 July 1985, and tests of the 1986 collections were initiated between 27 June and 27 July 1986.

Tester stocks and experimental procedures: Gonadal dysgenesis at 29° in reference crosses A and A* was used to assay for P activity and for susceptibility to P activity, respectively. The tester strains used were: Harwich-w, a strong P subline of the standard P strain Harwich, bearing a mutant at the *white* (*w*) locus; and Canton-S-red, a subline of the true M strain Canton-S, bearing an eye-color mutation on the second chromosome (locus not identified). The mutants in both these strains occurred spontaneously, and the strains were isolated by M. G. KIDWELL. Their properties in th P-M system do not differ obviously from the well-char-

acterized stocks from which they were derived.

Both the 1985 and 1986 collections were tested within 4 months of their establishment (less than eight generations). Flies from a line were assayed individually. Virgin females were held before use for 3–7 days after eclosion. For cross A tests, each male was crossed with five Canton-S-red virgins; for cross A* tests, each female was crossed with five Harwich-w males. Each cross was made at 29° in a vial with medium. Adults were left in the vials at 29° for 2 or 3 (cross A) or 3 or 4 (cross A*) days, then discarded. On the 14th day, emerged F_1 flies of both sexes were transferred to a fresh vial with medium and live yeast at room temperature and allowed to mature for 3 or 4 days before dissection of females. An ovary was scored as nondysgenic if even one ovariole was developed (ENGELS and PRESTON 1979; SCHAEFFER, KIDWELL and FAUSTO-STERLING 1979). All F_1 females were dissected and scored; a score was used only in calculations if ten or more F_1 females were produced in the cross. For each isofemale line, five or more reference crosses were set up for cross A and for cross A*. For the 1985 tests, the mean number of crosses scored per line in the cross A tests was 4.8, and in the cross A* tests, it was 4.7; the mean number of F_1 females scored per cross was 39.9. For the 1986 tests, the mean number of crosses scored per line was 4.4 for cross A tests, and 3.8 for cross A* tests; the mean number of F_1 females scored per cross was 56.8.

As controls, in tests of both the 1985 and 1986 lines, crosses were made of Canton-S-red females and Harwich-w males. In addition, in tests of the 1985 lines the reciprocal crosses were also made (Harwich-w × Canton-S-red), and in tests of the 1986 lines the intrastrain sterilities of Canton-S-red and of each wild line were tested. The 1985 control crosses were made between five virgin females and single males, while the 1986 controls were made by mass crosses of five virgin females and five males. The mean number of F_1 females dissected and scored per control cross for the 1985 and 1986 tests was 43.8. All control crosses were done concurrently with the experimental crosses.

For each cross and intrastrain mating producing ten or more F_1 females, the proportion of dysgenic females (with one or both ovaries undeveloped) was recorded. Since this measure (BOUSSY 1987) weights unilaterally and bilaterally dysgenic females equally, it generally gives a somewhat higher value than the proportion of undeveloped ovaries, used in some studies (*e.g.*, ANXOLABÉHÈRE *et al.* 1984; KIDWELL 1985; KIDWELL and NOVY 1985), or the proportion of completely infertile F_1 females (probably roughly equivalent to the proportion of bilaterally dysgenic females) used in others (*e.g.*, ENGELS and PRESTON 1980; KIDWELL, NOVY and FEELEY 1981). Unweighted means of results from all dysgenesis tests were calculated to yield a cross A and a cross A* datum for each line, and unweighted means of those means were calculated to yield overall averages for each population for crosses A and A*.

RESULTS AND DISCUSSION

Figure 1 shows the mean percent gonadal dysgenesis for each isofemale line from each of the 15 localities sampled, together with comparable data from the 1983 collections from three of the localities [the latter from BOUSSY (1987)]. The data are presented as points on graphs of cross A *vs.* cross A* sterilities (YAMAMOTO, HIHARA and WATANABE 1984). The coastline of southeastern Australia is also presented, to indicate the relative location of each population. Lines from the northern New South Wales coast as far south as Ourimbah (33.4°SLat) vary from neutral (Q) to moderately strong P within each population. Lines from the southern New South Wales coast, from Wollongong (34.4°SLat) to Eden (37.1°SLat), are generally Q, with relatively little variability. Lines from two populations in eastern Victoria [Genoa (37.5°SLat) and Cann River (37.6°SLat)] vary from Q to moderately strong M. The data from 1983 for Coff's Harbour, Bateman's Bay and Cann River differ in detail from those of 1985 and 1986, but the qualitative results are the same for 1983 and 1985–1986 (P, Q and M, respectively) for all three populations.

Patterns within populations: It appears that the following general rules hold: (1) lines from populations whose central tendency is P all tend to have low susceptibility to P activity (strong regulatory ability), but can have differing amounts of P activity; (2) lines from populations whose central tendency is Q all tend to have low P activity and low susceptibility; and (3) lines from populations whose central tendency is M all tend to have low P activity, but can have differing susceptibilities to P activity. Although it is feasible that a line might have high P activity and high susceptibility to P activity at the same time, such seems to be seldom the case in nature; KIDWELL (1986) states that such a condition can exist transiently in the laboratory, but that such strains tend to evolve to a P, Q or M state, or to go extinct. Some of the lines from the 1983 collections which showed both elevated P activity and elevated susceptibility were later shown to have changed to a state like the norm of the population from which they came (I. A. BOUSSY, unpublished data), suggesting that such atypical states may occur transiently in the wild as well.

That wild-caught flies or lines tend to be clearly P, Q or M has been shown in several studies including the present one. As is apparent from Figure 1, most lines in this study are close to one or the other axis (or both) on a graph of cross A *vs.* cross A* GD. Among reports on individual flies from wild populations or large, recently captured cage populations, the proportion of flies with cross A *vs.* cross A* test scores more than 10% away from either axis is low. ENGELS and PRESTON (1980) found no cross A* sterility in a P population, and SIMMONS (1986) found about 11% of individuals tested with greater than 10% cross A* sterility in three P populations. ANXOLABÉHÈRE, NOUAUD and PERIQUET (1982) and OHISHI, TAKANASHI and ISHIWA CHIGUSA (1982) found no P activity among individuals they tested from M populations. Among all studies that tested isofemale lines in crosses A and A*, the overall proportion of lines that had scores that were not within 10% of one or the other axis is only 56/620 = 9.0% (YAMAMOTO, HIHARA and

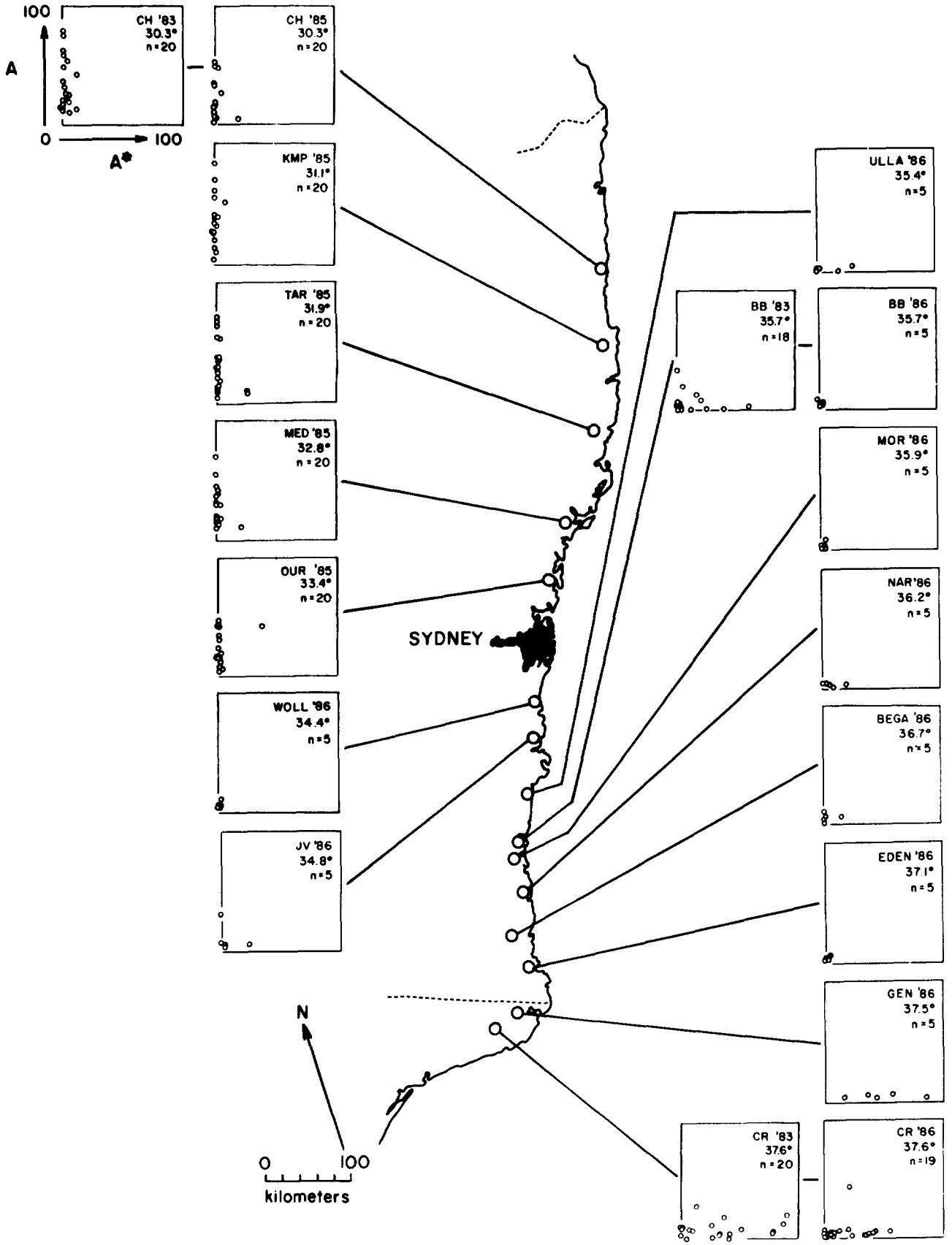

FIGURE 1.—Graphs of the cross A (vertical axis) and cross A* (horizontal axis) mean gonadal dysgenesis scores (%) for each isofemale line tested from each of 18 collections. The locality from which the collection was made is indicated on a map of the southeastern coast of Australia. The data for the three 1983 collections are from BOUSSY (1987). Each graph is labeled with the abbreviation of its collection locality and the year of collection, the latitude of the locality, and the number of isofemale lines tested. Abbreviations are: CH, Coff's Harbour; KMP: Kempsey; TAR: Taree; MED: Medowie; OUR: Ourimbah; WOLL: Wollongong; JV: Jasper Valley; ULLA: Ulladulla; BB: Bateman's Bay; MOR: Moruya; NAR: Narooma; BEGA: Bega; EDEN: Eden; GEN: Genoa; CR: Cann River.

TABLE 1

Gonadal dysgenesis scores of Australian populations from 3 yr of collections and tests

Locality[a]	°SLat	Year	No. of lines tested	Mean % ± SE		
				Cross A	Cross A*	Intrastrain
Cairns, Qld	16.9	1983[b]	16	54.5 ± 5.1	11.1 ± 4.1	
Rockhampton, Qld	23.4	1983[b]	20	60.6 ± 5.2	5.4 ± 1.8	
Coff's Harbour, NSW	30.3	1983[b]	20	34.7 ± 4.8	2.7 ± 0.8	
		1985	20	19.0 ± 3.6	1.6 ± 0.1	
Kempsey, NSW	31.1	1985	20	34.6 ± 4.9	0.8 ± 0.5	
Taree, NSW	31.9	1985	20	31.1 ± 4.7	2.7 ± 1.7	
Medowie, NSW	32.8	1985	20	31.2 ± 3.7	1.8 ± 1.1	
Ourimbah, NSW	33.4	1985	20	21.3 ± 3.5	6.4 ± 2.7	
Wollongong, NSW	34.4	1986	5	6.5 ± 1.0	0.9 ± 0.5	0.5 ± 0.5
Jasper Valley, NSW	34.8	1986	5	8.2 ± 5.4	5.5 ± 4.6	4.7 ± 5.7
Ulladulla, NSW	35.4	1986	5	1.5 ± 0.7	10.2 ± 6.2	3.3 ± 1.7
Bateman's Bay, NSW	35.7	1983[b]	18	6.4 ± 2.0	9.3 ± 4.0	
		1986	5	3.7 ± 1.1	1.8 ± 0.6	5.2 ± 3.7
Moruya, NSW	35.9	1986	5	2.5 ± 0.9	2.4 ± 0.6	1.5 ± 1.5
Narooma, NSW	36.2	1986	5	1.4 ± 0.6	8.5 ± 3.4	2.5 ± 1.1
Bega, NSW	36.7	1986	5	3.8 ± 1.7	3.8 ± 3.6	2.9 ± 1.2
Eden, NSW	37.1	1986	5	2.8 ± 0.8	1.2 ± 0.5	2.4 ± 1.6
Genoa, Vic	37.5	1986	5	3.5 ± 0.8	48.2 ± 11.7	4.9 ± 4.0
Cann River, Vic	37.6	1983[b]	20	8.8 ± 1.5	33.0 ± 6.7	
		1986	19	4.1 ± 2.2	16.5 ± 4.0	3.1 ± 1.5
Cygnet, Tas	43.2	1983[b]	20	3.4 ± 0.5	46.7 ± 6.5	

[a] Qld: Queensland; NSW, New South Wales; Vic: Victoria, Tas: Tasmania.
[b] Data from BOUSSY (1987); Bateman's Bay lines collected in 1983, tested in 1985.

WATANABE 1984; HIHARA, HISAMATSU and HIROTA 1985; KIDWELL and NOVY 1985; and BOUSSY 1987 and the current study, using a 15% criterion, to compensate for the different measure of GD sterility used). Thus the designation of populations as phenotypically P, Q or M (YAMAMOTO, HIHARA and WATANABE 1984; KIDWELL 1986) seems robust. It should be noted that all the above studies, including the present one, found large variability within the P class for P activity and the M class for susceptibility to P activity, as is apparently typical for wild populations (BOUSSY 1987).

Corroboration of clinal pattern of P-M hybrid dysgenesis in eastern Australia: The 1983 collections of six populations tested and reported by BOUSSY (1987) seemed to show a dramatic latitudinal cline in P-M characteristics, from P in the north, to Q at Bateman's Bay, to M in the south. There was a possibility, however, that the perceived pattern was the result of fortuitous sampling, and that the true pattern was not clinal. Table 1 presents the mean GD scores in cross A and cross A* tests for each locality tested to date [including data from BOUSSY (1987)], together with intrastrain tests for some populations. Table 2 shows control cross results for each set of tests, including data inadvertently omitted from BOUSSY (1987). Figure 2 plots the data of Table 1 for each collection against the latitudes of the localities of collection. The current results, as shown in Figure 2, corroborate the original interpretation of a latitudinal cline in P-M characteristics, and establish that the cline is nearly monotonic over space.

The 1985 collections were made in an attempt to better define the P to M transition zone of the cline. The region from Coff's Harbour to Ourimbah was chosen to span the mouth of the Hunter River, since a bottle population of combined lines originally collected in 1974 from six wineries in the Hunter Valley (≈32.5°SLat, about 50 km inland from the mouth of the Hunter River) had been shown to be Q (SVED 1976; KIDWELL, FRYDRYK and NOVY 1983). The result that the Coff's Harbour to Ourimbah populations are weak to moderate P is not necessarily inconsistent with the Q phenotype of the Hunter Valley sample. It is clear from Figure 1 that some essentially P populations, especially Coff's Harbour and Ourimbah, contain Q lines. The Hunter Valley strain had originally been collected as six lines from different Hunter Valley wineries; the lines were subsequently combined into one bottle population (SVED 1976). The lines may have simply been Q samples from weak P populations. It is also possible that the Hunter Valley strain lost much of its P activity while maintained in the laboratory [as has been shown to sometimes occur (ENGELS and PRESTON 1980)]. According to SVED (1976), the stock was capable of generating male recombination and high egg lethality in test crosses, and so had some P characteristics (as is typical for at least Q lines from P populations (ENGELS and PRESTON 1981; GREEN 1984)).

TABLE 2

Gonadal dysgenesis in control crosses

Test year	Control cross[a]	Mean % ± SE (no. of crosses; mean no. of dissections per cross)	Control cross[a]	Mean % ± SE (no. of crosses; mean no. of dissections per cross)	Control cross[a]	Mean % ± SE (no. of crosses; mean no. of dissections per cross)
1983[b]	CS Intra	0.0 ± 0.0 (5; 15.0)	CS × H	100.0 ± 0.0 (13; 28.5)	H × CS	3.7 ± 3.7 (6; 19.0)
	H Intra	2.8 ± 2.2 (11; 24.9)				
1985[c]			CSr × Hw	100.0 ± 0.0 (9; 61.0)	Hw × CSr	1.1 ± 0.9 (10; 49.4)
1985			CSr × Hw	100.0 ± 0.0 (9; 52.4)	Hw × CSr	1.4 ± 0.9 (10; 38.9)
1986	CSr Intra	6.7 ± 4.7 (5; 28.8)	CSr × Hw	100.0 ± 0.0 (23; 45.9)		

[a] Female × male; CS: Canton-S; H: Harwich; CSr: Canton-S-red; Hw: Harwich-w; Intra: intrastrain crosses.
[b] Controls for 1983 crosses reported in BOUSSY (1987).
[c] Controls for 1985 crosses reported in BOUSSY (1987).

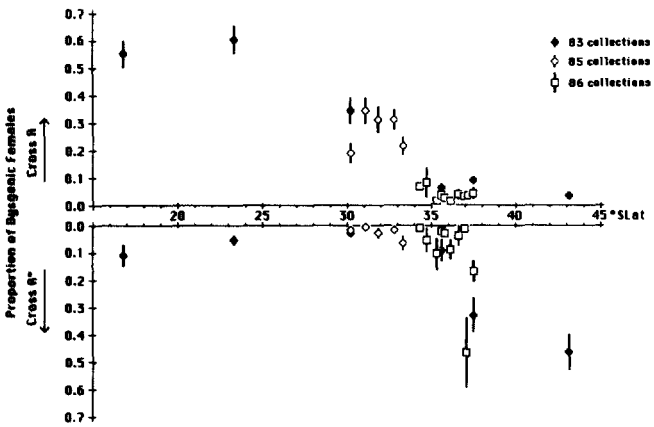

FIGURE 2.—Means of cross A and cross A* GD scores for all populations studied in eastern Australia, plotted against latitude of collection locality, with cross A scores reading above the horizontal axis and cross A* scores reading down from the horizontal axis. The three sets of tests are designated by black diamonds for 1983 collections [from BOUSSY (1987)], open diamonds for 1985 collections, and open squares for 1986 collections. Vertical lines through symbols indicate plus or minus one standard error.

A partial motivation for the 1986 collections was to determine the validity of the finding that the 1983 collection from Bateman's Bay (35.7°SLat) was Q (BOUSSY 1987). The 1983 collection (18 isofemale lines) had been kept in the laboratory for about 45 generations before being tested. The results of the 1986 collections fully vindicate the original finding. Not only is the Bateman's Bay population Q, but so are the other seven populations sampled along the southern New South Wales coast, from Wollongong (34.4°SLat) to Eden (37.1°SLat).

The two collections south of Eden, at Genoa (37.5°SLat) and Cann River (37.6°SLat), are unequivocally M.

Temporal changes are not evident: The 1985 samples from Coff's Harbour have a mean P activity distinctly lower than that of the 1983 collection (Mann-Whitney U, $P = 0.004$), and the 1986 samples from Cann River have a mean susceptibility to P activity lower than that of the 1983 collection (Mann-Whitney U, $P < 0.001$). It is difficult to interpret the biological significance of these data, because the testing of the collections was done at different times, in different laboratories and with different reference strains; gonadal dysgenesis is known to be quite sensitive to temperature of rearing of the F_1 flies, for instance, and this could have been different enough to account for some of the apparent temporal changes. There is no difference between 1983 and 1986 scores for Bateman's Bay in cross A (Mann-Whitney U, $P = 0.368$) or cross A* (Mann-Whitney U, $P = 1.000$). Overall, there is no clear indication that the cline moved in the two to three year period between collections. The Coff's Harbour region was found to be still P, the Bateman's Bay region still Q, and the Cann River region still M.

Discontinuities in the clinal pattern: Ignoring the temporal discrepancy of the P activity scores from Coff's Harbour and the susceptibility scores from Cann River, the overall pattern of P-M characteristics is clearly clinal from north to south. Northern New South Wales populations are weak to moderate P as far south as Ourimbah, then a dramatic drop in P activity takes place, so that Wollongong is clearly Q. The change in P activity between Ourimbah and Wollongong occurs over only 150 km, which is quite a short distance considering the extreme long-distance migration potential of *D. melanogaster* (COYNE *et al.* 1982). The change is further remarkable in that it occurs over a region that is largely urban and suburban; Sydney is a sprawling metropolis which must offer sufficient suitable resources to support a spatially continuous population of *D. melanogaster*. One would have expected that migration within this region would have led to homogeneity for P-M characteristics. The urban ecology of *D. melanogaster* is not well known, however, and careful sampling in this area is clearly needed to clearly describe the distribution in this region.

South from Wollongong to Eden the populations are essentially uniformly Q, but only about 50 km separate Eden from Genoa, which is clearly M. This

Q to M change is fully as dramatic as the P to Q change further north, and may occur over a much shorter distance. Detailed sampling is needed in this region to define the Q to M transition.

Hypothesis about the cline: BOUSSY (1987) has proposed and discussed various hypotheses about the formation of the cline. His arguments can be summarized as follows:

1. The coastal latitudinal gradient of temperature might be acting as a selective agent to form the P-M cline. Given certain assumptions, models of transposable element population dynamics have shown that selection against individuals carrying large numbers of elements per genome can "regulate" the proportion of individuals carrying elements (UYENOYAMA 1985) or the mean number of elements per genome (CHARLESWORTH and CHARLESWORTH 1983). Since gonadal dysgenesis rises dramatically in dysgenic flies reared above 25° (ENGELS and PRESTON 1979; SCHAEFER, KIDWELL and FAUSTO-STERLING 1979), high temperatures could act to select for individuals with low P activity. However the clinal pattern, of P populations in the hot, tropical north and M populations in the cool, temperate south, is in the wrong direction for such an explanation to apply.

2. The P-M cline coincides with clines in frequencies of ADH (and other isozyme) alleles and of cosmopolitan inversions, suggesting that historical introductions could explain all three kinds of clines. However other observations strongly suggest that the different clines do not have a common cause. First, linkage disequilibrium exists between certain allozymes and inversions (MUKAI and YOELKER 1977; KNIBB 1983), suggesting that these inversions are of unique origin rather than repeatedly created. Second, rare endemic inversions do not show a latitudinal pattern in Australia [KNIBB, OAKESHOTT and GIBSON (1981); I. A. BOUSSY, unpublished data]. These two observations suggest that P-M dysgenesis is not generating either cosmopolitan or unique endemic inversions, and so is not a cause of the inversion cline. VOELKER *et al.* (1978) and KNIBB (1983) have shown that allozyme clines still exist when data are corrected for linked inversions, suggesting that they are independent. Finally, the northern hemisphere clines in allozyme frequencies (OAKESHOTT *et al.* 1982) and cosmopolitan inversion frequencies (KNIBB, OAKESHOTT and GIBSON 1981) which mirror those in Australia, in the absence of any P-M clines similar to the Australian one, suggest independence of the P-M cline from the other two in Australia. Furthermore, simple introductions (taking *P* elements to be simple infective features of the *D. melanogaster* genome, without consideration of their complex genomic dynamics) should not be able to generate certain features of the P-M cline, such as Q and pseudo M populations (BOUSSY 1987).

A consideration of various aspects of the genomic dynamics of *P* elements appears to be necessary in order to model the clinal pattern.

3. The observed cline might be explained by a model involving a consideration of *P* element genomic dynamics, including insertion, excision, suppression of transposition, and degeneration of autonomous elements to nonautonomous forms. The only published model of transposable element dynamics that takes into account a degenerative process is that of KAPLAN, DARDEN and LANGLEY (1985). Their model describes the evolution of a single population. Briefly, it maintains that upon introduction of elements into a finite population, the active, complete elements very quickly increase in frequency to their self-regulated number. Concomitantly, the degenerative process begins converting complete to deleted elements. A quasi-equilibrium state is reached, in which the proportion of complete elements declines only slowly. Eventually, the degenerative process leads inexorably to the loss of complete elements from the population. This model can be taken to be a reasonable description of the dynamics of a *P* element invasion. With many assumptions about the relationship between P-M genotype and phenotype, it predicts a progression from true M to P to Q to pseudo M in a population undergoing such an evolution. However it does not address the issue of multipopulational dynamics of such an element, and as such is not directly applicable to the clinal situation in eastern Australia.

More data are needed: Preliminary data (I. A. BOUSSY, M. J. HEALY and J. G. OAKESHOTT, unpublished data) have shown a strong correlation between the numbers of potentially full-sized *P* elements per genome and the P or M characteristics of the line tested (BOUSSY 1987). More work is clearly needed to understand the relationship between *P* element genotypes and P-M phenotypes, especially in natural populations. Several studies addressing these questions are currently in progress in our laboratory.

The eastern Australian clinal pattern cannot be fully explained with our current knowledge of *P* element population dynamics, and the hypotheses that can be proposed are limited by the currently inadequate information about *P* element biology (BOUSSY 1987). More details of the cline are needed also, especially to define the P-to-Q transition region around Sydney and the Q-to-M transition region between Eden and Genoa. Temporal data for the overall cline, establishing either the stability of the cline or its rate and direction of movement, are needed in order to evaluate possible hypotheses about its causality (BOUSSY 1987). KIDWELL (1986) has speculated that many of the properties of the P-M system of hybrid dysgenesis that make it unique may be due to the *P* element's having only recently invaded *D. melanogaster*. If this

is so, then the large differences seen in P-M characteristics between continental areas (KIDWELL, FRYDRYK and NOVY 1983; ANXOLABÉHÈRE *et al.* 1984), the shallow Q-to-M clines from France across Asia and from France to Spain (ANXOLABÉHÈRE *et al.* 1985; D. ANXOLABÉHÈRE and G. PERIQUET, personal communication), and the dramatic P-to-M cline in eastern Australia may all be transient phenomena. These massive natural experiments may offer unique opportunities to study the dynamics of a transposable element only shortly after its invasion of a species.

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