Genet. Res., Camb. (1991), **57**, *pp.* 227–234 *With 2 text-figures Printed in Great Britain*

Inheritance of *P*-element regulation in *Drosophila melanogaster*

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(*Received 27 April 1990 and in revised form 13 August 1990*)

Summary

The ability to repress P-element-induced gonadal dysgenesis was studied in 14 wild-type strains of *D. melanogaster* derived from populations in the central and eastern United States. Females from each of these strains had a high ability to repress gonadal dysgenesis in their daughters. Reciprocal hybrids produced by crossing each of the wild-type strains with an M strain demonstrated that repression ability was determined by a complex mixture of chromosomal and cytoplasmic factors. Cytoplasmic transmission of repression ability was observed in all 14 strains and chromosomal transmission was observed in 12 of them. Genomic Southern blots indicated that four of the strains possessed a particular type of P element, called KP, which has been proposed to account for the chromosomal transmission of repression ability. However, in this study several of the strains that lacked KP elements exhibited as much chromosomal transmission of repression ability as the strains that had KP elements, suggesting that other kinds of P elements may be involved.

1. Introduction

In *Drosophila melanogaster*, the P family of transposable elements is responsible for a syndrome of germ-line abnormalities known as hybrid dysgenesis (Kidwell *et al.* 1977; Engels, 1989). This syndrome includes sterility, aberrant recombination, chromosome-transmission-ratio distortion and high frequencies of mutation and chromosome breakage. It arises from the activation of P elements by a transposase that is encoded by the structurally complete members of the P family (Karess & Rubin, 1984; Engels, 1984; Laski *et al.* 1986; Rio *et al.* 1986). The severity of hybrid dysgenesis depends on the abundance of the transposase and also on the number of P elements present in the genome (Simmons *et al.* 1987; Rasmusson *et al.* 1990). In addition, there is a requirement for a permissive cellular condition called the M cytotype. A complementary condition, called the P cytotype, represses dysgenesis by regulating P-element activity (Engels, 1979*a*, *b*). Genetic analyses have shown that cytotype is maternally transmitted through two or more generations, but in the long run, it is determined by chromosomal factors that are correlated with the P elements themselves (Engels, 1979*a*; Kidwell, 1981). It is now widely held that certain types of P elements produce repressors that are responsible for the P cytotype (Daniels *et al.* 1987; Nitasaka *et al.* 1987; Robertson & Engels, 1989).

Early studies established a nomenclature that has been useful in describing the genetic analysis of P-element regulation (Kidwell *et al.* 1977; Engels, 1979*a*; Bingham *et al.* 1982; for a review, see Engels, 1989). M strains lack P elements in their genomes and possess the M cytotype. P strains carrying P elements and have the P cytotype. Dysgenic hybrids are produced by crossing M females with P males; however, because the P cytotype is maternally transmitted, the offspring of crosses between P females and M males are usually normal. In addition, two other categories of strains, Q and M′, have been recognized (Kidwell, 1983). Both of these carry P elements in their genomes, but they are less capable than P strains of inducing hybrid dysgenesis in crosses to true M strains (Simmons *et al.* 1985; Jongeward *et al.* 1987). Furthermore, although Q strains appear to have the P cytotype, M′ strains exhibit only a partial ability to regulate P-element activity (Kidwell, 1983, 1985; Simmons *et al.* 1987, 1990). This has led to a more generalized terminology, in which the ability to repress dysgenesis is referred to as ‘repression potential’. In this terminology, the P and M cytotypes may be considered as the extremes in a regulatory continuum. Genetic studies with some M′ and Q strains have

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https://doi.org/10.1017/S0016672300029372 Published online by Cambridge University Press

suggested that intermediate repression potential is determined mainly by additive chromosomal factors (Kidwell, 1985; Black *et al.* 1987). However, studies with other strains have suggested a complex mixture of cytoplasmic and chromosomal determination (Jackson *et al.* 1988; Simmons *et al.* 1990; Heath & Simmons, 1990).

The present experiments were initiated to investigate the inheritance of regulatory ability in a sample of P and Q strains derived from North American populations of *D. melanogaster*. Using the repression of P-element-induced sterility (gonadal dysgenesis) as an indication of P-element regulation, these experiments demonstrate a diversity of inheritance patterns, reflecting the contributions of both chromosomal and cytoplasmic factors.

2. Materials and methods

(i) Drosophila *strains*

Inbred wild-type strains: 12 strains described by Kocur *et al.* (1986) that were derived from single inseminated females captured from populations in the central and eastern United States. The populations, the year of sampling, and the derived strains are: St Paul campus of the University of Minnesota, 1978 (C-3.7, C-5.5); St Anthony Park, a subdivision of St Paul, Minnesota, 1982 (F-5, F-13); Garnavillo, Iowa, 1982 (I-1), Northeast Minneapolis, Minnesota, 1983 (N-5, N-12); Pittsburgh, Pennsylvania, 1978 (Pt-2, Pt-4); Roseville, Minnesota, 1978 (R-1); Stillwater, Minnesota, 1982 (S-2, S-7).

π_2 and ν_6: wild-type strains derived from a natural population in Madison, Wisconsin (Engels, 1979; Engels & Preston, 1981). For π_2, two sublines were available for study; one, called π_2-88, was used in both the genetic and molecular analyses, whereas the other, called π_2-89, was used only for purposes of comparison in the molecular analysis. For ν_6, only a single subline was analysed.

bw; *st*: a true M strain that is homozygous for the autosomal recessive markers *bw* (brown eyes) and *st* (scarlet eyes).

C(1)*DX*, *y f*/*Y*/sn^w; π_2; an attached-X strain in which the males carry a P-element-insertion mutation of the X-linked *singed* bristle locus (Engels, 1979; Engels, 1984). This strain has a genetic background derived from π_2 and is capable of inducing a high frequency of gonadal dysgenesis.

Sexi and Sexi.3: two wild-type strains that contain many copies of a particular incomplete P element, called KP, in their genomes (Kidwell, 1985; Black *et al.* 1987; Simmons *et al.* 1990).

(ii) *Tests of the ability to induce gonadal dysgenesis*

The ability to induce gonadal dysgenesis was tested by crossing males individually to *bw*; *st* females at 29 °C. On day 11, the progeny of each test mating were transferred to a fresh vial and aged for 2–3 days at 21 °C. Then, as many as 12 of the daughters were examined for egg production by squeezing them between glass plates. Only the daughters that did not extrude any eggs were considered to have gonadal dysgenesis.

(iii) *Tests of the ability to repress gonadal dysgenesis*

The ability to repress gonadal dysgenesis was tested by mating individual females to sn^w; π_2 males at 29 °C; the flies that were used in these tests had been reared at 21 °C. Daughters from each mating were then examined for gonadal dysgenesis as described in section 2(ii). The frequency of dysgenesis among these daughters was used as a measure of repression potential; a low frequency implied that the repression potential was high.

(iv) *Crosses to study the inheritance of repression potential*

Reciprocal mass matings were established at 21 °C between *bw*; *st* and each of the wild-type strains (the 12 inbred lines plus π_2-88 and ν_6). Matings of wild males × *bw*; *st* females were denoted cross A and matings of wild females × *bw*; *st* males were denoted cross B. The F_1 females from both sets of crosses (F_1-A and F_1-B) were then tested for the ability to repress gonadal dysgenesis in their daughters by crossing them individually to sn^w; π_2 males as described in section 2(iii).

(v) *Culture conditions*

All cultures were reared on a standard cornmeal–molasses–yeast–agar medium in vials or half-pint milk bottles. For culturing temperatures, see sections 2(ii)–(iv) above.

(vi) *Statistical analysis*

Between-group differences in repression potential were evaluated by the Mann–Whitney rank sum test.

(vii) *DNA blotting*

DNA was extracted from adult flies and digested with restriction enzymes according to the suppliers' instructions (Bethesda Research Laboratories). The DNA fragments were separated by electrophoresis in agarose gels and transferred to GeneScreen Plus membranes (Dupont) by capillary blotting. The membranes were hybridized with ^{32}P-labelled DNA probes made from the plasmid pπ25.1BWC or from fragments of the plasmic pπ25.1, both of which contain P elements (O'Hare & Rubin, 1983, and K. O'Hare, personal communication). The probes were synthesized using a

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Table 1. *Induction and repression of gonadal dysgenesis*

	Induction test			Repression test								
				Stock			F_1-A hybrids			F_1-B hybrids		
Line	N[a]	n[b]	GD ± S.E.[c]	N	n	GD ± S.E.	N	n	GD ± S.E.	N	n	GD ± S.E.
C-3.7	21	222	46·4 ± 4·5 (39·7)[d]	30	310	0	18	193	34·3 ± 7·1	30	355	0·3 ± 0·3
C-5.5	20	238	88·6 ± 4·7 (80·8)	28	315	0	28	317	86·1 ± 5·3	29	339	35·1 ± 6·2
F-5	22	88	40·1 ± 6·6 (45·5)	28	319	0·3 ± 0·3	28	336	91·1 ± 2·1	27	311	0·3 ± 0·3
F-13	24	238	10·0 ± 1·8 (11·4)	28	257	1·8 ± 1·8	29	345	99·4 ± 0·4	27	324	23·1 ± 3·9
I-1	25	245	31·9 ± 4·0 (22·9)	30	345	0	30	360	23·9 ± 4·5	29	348	0
N-5	22	207	1·2 ± 0·6 (19·6)	28	310	0·9 ± 0·5	29	333	51·3 ± 7·2	30	359	7·3 ± 3·3
N-12	25	212	96·4 ± 1·3 (68·8)	26	266	0	30	360	44·4 ± 4·6	29	341	0
Pt-2	20	206	4·7 ± 2·5 (4·2)	28	317	0	28	355	90·5 ± 3·0	27	315	0
Pt-4	19	193	20·5 ± 4·7 (46·5)	29	346	0·3 ± 0·3	29	348	45·7 ± 4·6	30	355	0
R-1	21	206	3·7 ± 1·4 (2·3)	29	348	0	30	360	96·4 ± 1·8	30	360	0
S-2	25	274	95·1 ± 1·9 (78·4)	30	345	0	29	317	94·3 ± 1·9	30	356	30·3 ± 4·9
S-7	22	201	6·8 ± 4·7 (12·2)	23	274	0	30	360	81·1 ± 3·6	30	360	25·0 ± 7·8
ν_6	25	260	0	29	305	2·6 ± 1·8	29	344	98·6 ± 0·7	30	359	23·3 ± 7·7
π_2-88	23	262	88·0 ± 4·4	29	335	0	30	360	85·0 ± 5·0	30	358	10·8 ± 5·5
bw; *st*				75	528	99·2 ± 0·5						

[a] Number of cultures.
[b] Number of daughters examined for GD sterility.
[c] Unweighted average percentage of daughters with GD sterility ± standard error.
[d] Percentage GD sterility from Kocur *et al.* (1986).

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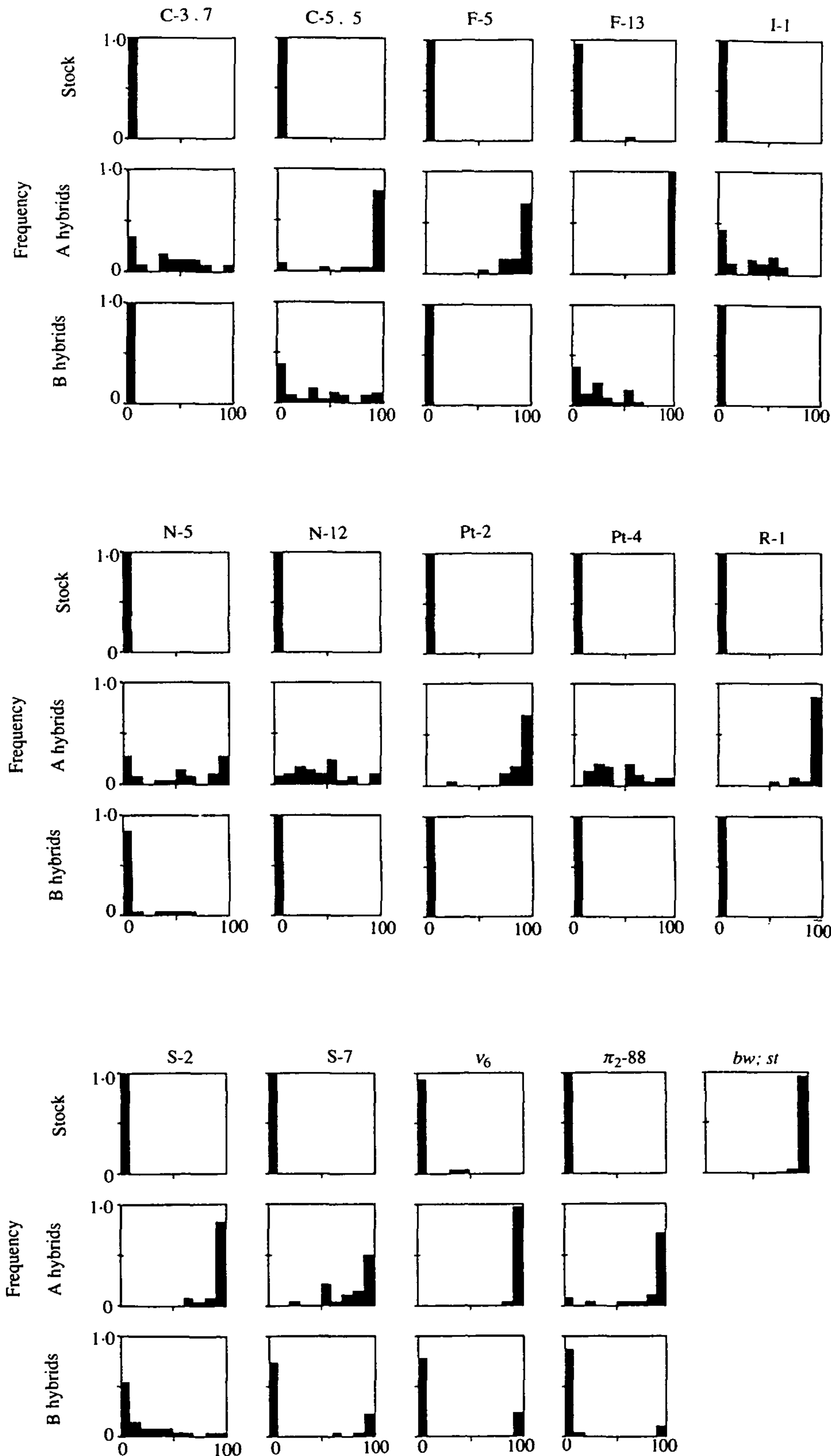

Fig. 1. Distributions of GD sterility among the daughters of females that were mated to sn^w; π_2 males. Reciprocal F_1-A and F_1-B hybrid females and stock females were used in these mating. See text for details.

random primer labelling kit (Bethesda Research Laboratories). Membranes that were used for re-hybridization were stripped of previously bound probe by shaking at 42 °C in a mild alkaline solution.

3. Results

Each of the wild-type strains was tested for the ability to induce and repress gonadal dysgenesis. As Table 1

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shows, there was considerable variation in the ability to induce dysgenesis. The males from some strains (F-13, N-5, Pt-2, R-1, S-7 and ν_6) induced very little (GD < 10%), whereas the males from other strains (C-5.5, N-12, S-2 and π_2-88) induced a great deal (GD > 88%). Although these results are qualitatively similar to those previously reported (Kocur *et al.* 1986), there are some quantitative differences that are probably due to genetic changes in the strains, as well as to sampling variation in the data. Table 1 also shows that the females from the wild-type stocks were able to repress gonadal dysgenesis in a high proportion of their daughters, suggesting that they had the P cytotype. For every line, the average frequency of dysgenic daughters was less than 3%. By contrast, more than 99% of the daughters of the M cytotype (*bw; st*) controls were dysgenic.

The inheritance of the ability to repress gonadal dysgenesis was investigated by testing F_1 hybrid females that were produced in reciprocal crosses between the wild type strains and *bw*; *st*. Each F_1 female was mated individually to dysgenesis-inducing sn^w; π_2 males to determine her ability to repress GD sterility in her daughters. Since the two types of F_1 hybrids were genetically equivalent, any differences between them in repression ability must have been due to cytoplasmic factors inherited from their mothers; this scheme therefore provides a test of cytoplasmic versus chromosomal transmission of repression potential. Table 1 and Fig. 1 present the results. Statistical analyses demonstrated that in every case, the F_1-B females had significantly more repression ability, i.e. a lower proportion of sterile daughters, than the F_1-A females. Thus, cytoplasmic factors inherited from the wild type strains must have been involved in the determination of repression potential. However, it is also clear that for most of the wild-type lines, the F_1-A hybrids had a lower proportion of sterile daughters than the controls from the *bw*; *st* stock. In some cases, the difference was very great; for example, the F_1-A hybrids from lines C-3.7, I-1, N-5, N-12 and Pt-4 had sterility frequencies less than 52%, compared to 99% for the *bw*; *st* controls. This suggests that chromosomal factors inherited from the wild-type lines also played a role in the determination of repression potential.

Figure 1 shows the frequency distributions of sterility among the daughters of the hybrid and stock females that were tested for repression potential. Although the distributions from the stock females suggest that there was a uniformly high level of repression potential among the wild-type strains, the distributions from the hybrid females indicate that the mode of inheritance varied. Some strains (C-3.7, I-1, N-5, N-12 and Pt-4) showed quite effective chromosomal transmission through the F_1-A hybrids, whereas others (F-13 and ν_6) showed essentially none. Other strains (C-5.5, F-5, Pt-2, R-1, S-2, S-7 and π_2-88) showed weak to moderate chromosomal transmission, suggesting that there was some underlying quantitative variability.

The effectiveness of cytoplasmic transmission also varied, but in this case the variation appears to have been qualitative as well as quantitative. The F_1-B hybrids of three strains (S-7, ν_6 and π_2-88) exhibited bimodal distributions of sterility among their daughters, suggesting the segregation of a cytoplasmic factor (or of a chromosomal factor necessary for the maintenance of a repressive cytoplasmic state). The F_1-B hybrids from the majority of the other strains showed complete or nearly complete repression of sterility in their daughters; however, the F_1-B hybrids from three of the strains (C-5.5, F-13 and S-2) showed quantitative variation in the ability to repress dysgenesis in their daughters. Because the F_1-A hybrids from these three strains showed little, if any, repression potential, this suggests that the cytoplasmic determinants of repression potential can produce a continuum of regulatory states (cf. Engels & Preston, 1981).

It has been proposed that a particular kind of incomplete P element, called KP, is responsible for the repression of hybrid dysgenesis in some M′ and Q strains (Black *et al.* 1987; Jackson *et al.* 1988). Genomic DNA blotting experiments were therefore performed to determine if any of the wild-type strains possessed this element. In the first experiment, DNA from each of the strains was digested with the enzyme *Dde* I, which is expected to produce a 2·18 kb fragment from within complete P elements and a 0·42 kb fragment from within KP elements. The results are presented in Fig. 2*a*. Two strains, Sexi and Sexi.3, were included as controls in this experiment since they were known to possess KP elements (Black *et al.* 1987; Simmons *et al.* 1990). A second subline of π_2, called π_2-89, was also included for comparison with the π_2-88 subline. A 420 base-pair fragment was clearly detected in the DNA from the two Sexi strains, as well as in that from four of the other strains (C-5.5, Pt-2, S-2 and π_2-88). Each of these strains was therefore reanalysed in a second experiment using DNA digested with *Ava* II. This enzyme is expected to produce 1·84 and 0·54 kb fragments from within complete P elements and a 0·63 kb fragment from within KP elements. N-12, Pt-4 and ν_6 were also included in this experiment along with the *bw*; *st*, Sexi, and Sexi.3 controls. Figure 2*b* shows that the strains C-5.5, Pt-2, S-2, π_2-88, Sexi, and Sexi.3 produced the 0·63 kb fragment, whereas N-12, ν_6 and, of course, *bw*; *st* did not. Pt-4 produced a faint smear in the vicinity of the 0·63 kb band, but this may have been due to partial degradation of the DNA. To show that the 0·63 kb fragment from C-5.5, Pt-2, S-2, π_2-88, Sexi and Sexi.3 could have been derived from KP elements, this same blot was rehybridized with a probe made from a sequence that is deleted in these elements. The results (Fig. 2*c*) show that the 0·63 kb fragment did not hybridize with the probe, strongly suggesting that the 0·63 kb fragment was from KP.

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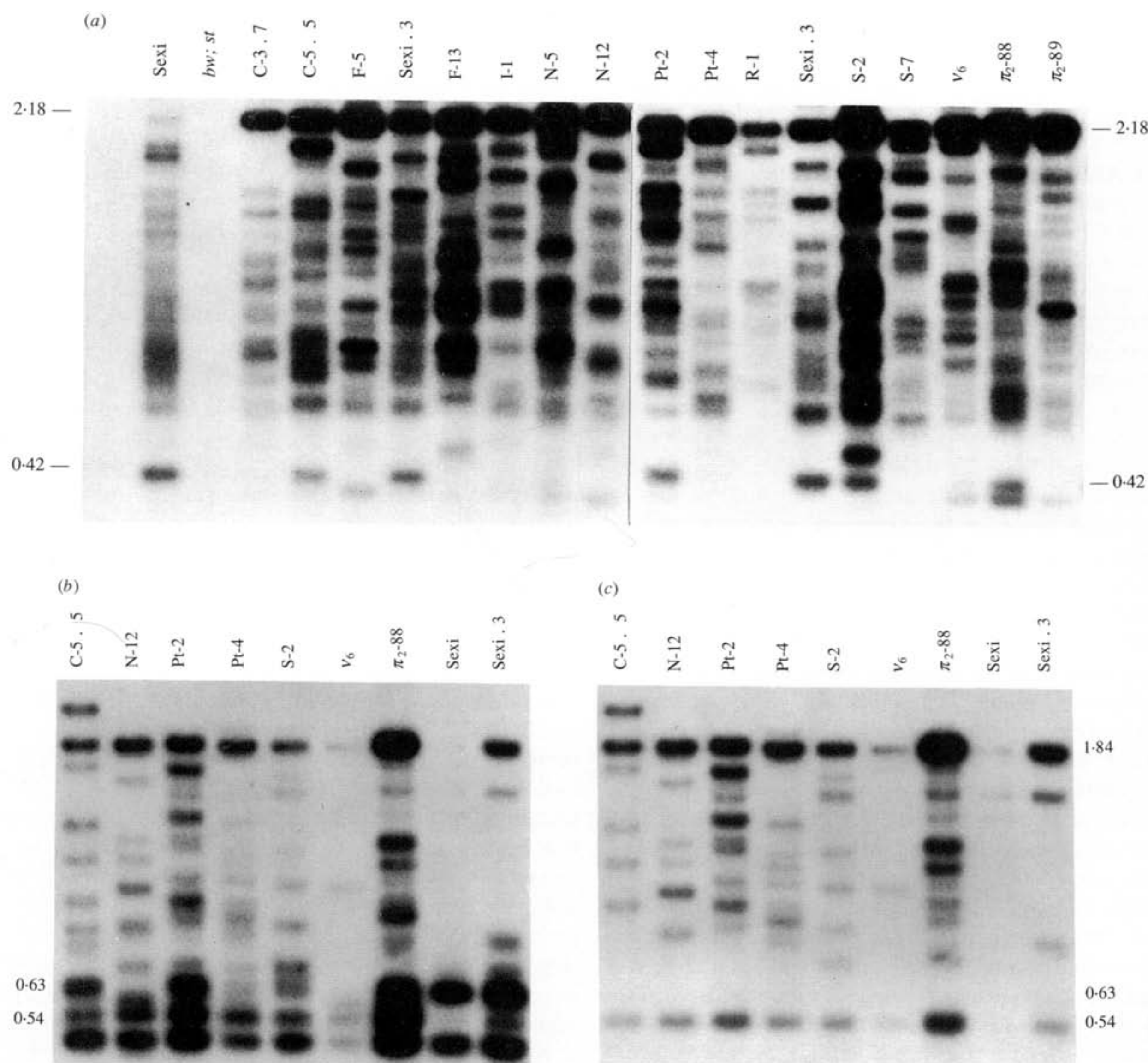

Fig. 2. (*a*) Genomic Southern analysis of *Dde* I-digested DNA from wild and control strains. After electrophoresis, the DNA was transferred to nylon membranes and hybridized with a ^{32}P-labelled DNA probe made from the plasmid pπ25.1BWC. Fragment sizes are in kilobases. (*b*) Genomic Southern analysis of *Ava* II-digested DNA from strains suspected of carrying KP elements. As in (*a*), the probe was made from pπ25.1BWC. (*c*) Genomic Southern analysis of the same membrane used in (*b*), reprobed with a ^{32}P-labelled *Hin*d III–*Sal* I fragment from pπ25.1.

Although C-5.5, Pt-2, S-2 and π_2-88 apparently possessed KP elements, other strains derived from the same populations (C-3.7, Pt-4, S-7 and π_2-89) did not. This may be due to sampling at the time the strains were established, or to evolutionary changes that occurred during laboratory culture, including the loss or acquisition of KP elements within particular strains. A strain might have acquired KP elements through the mutation of other P elements or by contamination from a KP-containing stock. This latter possibility cannot be ruled out since all the strains that were used in this study lacked visible genetic markers. Contamination may explain the presence of KP elements in the π_2-88 stock, which was kept in half-pint bottles in an incubator with many other strains, including Sexi and Sexi.3. The π_2-89 stock, which was maintained in vials in the same incubator, may represent the uncontaminated condition. It is not likely that contamination is responsible for the KP elements present in C-5.5, Pt-2 and S-2 since these strains were kept in vials in a different incubator that did not harbor any known KP-carrying strains.

4. Discussion

Previous studies have shown that there are differences in the manner in which P-element regulation is inherited (Engels, 1979*a*; Engels & Preston, 1981; Kidwell, 1981, 1983, 1985; Black *et al.* 1987; Jackson *et al.* 1988). In some strains, regulatory ability is

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transmitted maternally through two or more generations; in others, it seems to be determined by approximately additive chromosomal factors. Recent attempts to elucidate this difference have led to the proposal that there are two separate systems of P-element regulation, a maternally inherited system that is synonymous with cytotype, and a chromosomally inherited system that is based on an incomplete P element, called KP, which is found in many Q and M′ strains. Black *et al.* (1987) have postulated that this element produces a polypeptide that represses the activity of the P transposase.

The present study was carried out to examine variation in the inheritance of regulatory ability in a sample of P and Q strains, and to determine whether or not this variation could be explained by the presence or absence of KP elements. The results indicate that, although each of the strains exhibited strong regulatory ability, the pattern of inheritance varied considerably; similar results have been obtained by Jackson *et al.* (1988). All of the strains showed the influence of maternally inherited factors (P cytotype), but among them, there was evidence for quantitative and qualitative differences in the extent of this maternal transmission. The majority of the strains showed very strong maternal transmission of regulatory ability; however in some strains, the effectiveness of maternal transmission varied quantitatively. In addition, three strains seemed to show a segregation of the maternal regulatory component.

Regulatory ability was also transmitted paternally, i.e. on the chromosomes, in nearly all of the strains; only two, ν_6 and F-13, showed essentially no chromosomal transmission. However, the effectiveness of chromosomal transmission was found to vary quantitatively. Molecular analyses were performed to determine if this variation could be accounted for by KP elements. These analyses indicated that KP elements were present in four strains, C-5.5, Pt-2, S-2 and π_2-88, which all showed only marginal effectiveness in chromosomal transmission. These results are therefore not entirely consistent with the hypothesized association between KP elements and the chromosomal transmission of regulatory ability. In addition, four of the strains that lacked KP elements (C-3.7, I-1, N-5, N-12) showed considerable chromosomal transmission of regulatory ability, indicating that KP elements are not necessary for this pattern of transmission. From these and other results (Simmons *et al.* 1990), it is therefore unclear what role KP elements play in the determination of the ability to regulate P-element activity; indeed, it is entirely possible that some of the properties that have been attributed to KP elements are due to other kinds of P elements present in the genome. The quantitative and qualitative variation in regulatory ability that is observed among *Drosophila* strains, plus the diverse ways in which this ability is inherited, suggests that regulatory ability is influenced by a complex mixture of factors – possibly by many different kinds of P elements, each contributing in a way that is affected by its particular location in the genome.

Christopher F. McLarnon provided technical assistance. Research funds were provided by a grant from the National Institute of General Medical Sciences (GM40263). J.W. was supported by the Summer High School Research Program of the College of Biological Sciences, University of Minnesota.

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