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# The geographical distribution of *P-M* hybrid dysgenesis in *Drosophila melanogaster*

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## Summary

In *Drosophila melanogaster* the syndrome of germline abnormalities generated in the *P-M* system is caused by transposable elements known as the *P* element family. The frequency of gonadal dysgenesis, *GD* sterility characteristic of the *P-M* system, was estimated in 120 populations, collected in 1980-1983 from arround the world, in order to determine the present distribution of this system of hybrid dysgenesis. Marked geographical differences appear between these populations. In North America most of them possess individuals of the *P* and the *Q* type whereas the *M* type is absent or present at only very low frequencies. A similar pattern has been found in central Africa, whereas the *P* type is practically absent in North Africa, Europe and Asia. In these regions another pattern exists. In France the *Q* type is very frequent and the *M* type of low frequency, whereas *M* becomes very common going to the east of Yugoslavia and Tunisia towards India, China and Japan. Hypotheses on the evolution of the *P-M* system in natural populations polymorphic for the *P* elements will be discussed.

*Key words : Transposable elements, populations, polymorphism, evolution.*

## Résumé

*Répartition géographique du système* P-M *de dysgénésie des hybrides chez* Drosophila melanogaster

Chez *Drosophila melanogaster* la dysgénésie des hybrides due aux éléments transposables de la famille *P* est un syndrome d'anomalies génétiques incluant une stérilité thermodépendante et un fort taux de mutation. Afin de déterminer la distribution de ce système parmi les populations mondiales de drosophiles, un ensemble de 120 souches capturées entre 1980 et 1983 a été étudié pour ses potentialités de stérilité. En Amérique du Nord la plupart des populations possède des individus de type *P* ou *Q* tandis que le type *M* est

pratiquement absent. Une répartition similaire a été observée en Afrique centrale. Dans les autres régions (Afrique du Nord, Europe et Asie) une distribution différente est observée, dans laquelle le type *P* est pratiquement absent. Le type *Q* très fréquent en France se rencontre moins souvent vers l'est, tandis que le type *M* assez rare en France se rencontre très fréquemment de la Yougoslavie au Japon. Les hypothèses de l'évolution du système *P-M* dans des populations naturelles polymorphes pour les éléments *P* sont discutées.

*Mots clés : Eléments transposables, populations, polymorphisme, évolution.*

## I. Introduction

The interactions of the *P-M* system of hybrid dysgenesis, which are manifested in certain interstrain hybrids, result in a number of correlated aberrant genetic traits such as high frequencies of gonadal sterility (*GD* sterility) male recombination and mutation (KIDWELL *et al.*, 1977). In the *P-M* system three types of individuals, *P*, *Q* and *M*, have been described on the basis of their cross effect properties. Hybrids between *P* males and *M* females show dysgenic traits that are reduced or absent in the reciprocal hybrids. *Q* individuals do not exhibit *GD* sterility in any cross combinations but produce mutation and male recombination in crosses with *M* females. All *P* and *Q* strains so far examined carry 30-50 copies of the *P* family of elements (BINGHAM *et al.*, 1982 ; RUBIN *et al.*, 1982). *Q* individuals are thought to be a subset of the *P* element family which appear to lack sterility potentiality while retaining mutator activity and other *P* element functions (ENGELS, 1981 ; PÉRIQUET *et al.*, 1981 ; RUBIN *et al.*, 1982). Conversely, all-long-established laboratory *M* strains examined (except one), completely lacked homology with the *P* element family. *P* elements are subject to destabilisation in the maternally derived cellular state of a *M* strain (*M* cytotype) but are quasi-stable within a *P* or a *Q* cellular state (*P* cytotype) (ENGELS, 1979).

Although much previous research on transposable elements has been on their molecular properties, little is known about the population genetics of such sequences. The purpose of this report is to present the results of an extensive survey of actual *D. melanogaster* populations with respect to their dysgenic potential and to discuss hypotheses of the evolution of the *P-M* system.

## II. Materials and methods

120 strains derived from diverse localities around the world were determined with respect to their *GD* sterility potential. Wherever possible each strain was derived from a large number (over 30) of recently collected (1980-1983) individuals. They were kept in standard laboratory conditions by mass culture of about 500 individuals and normally analysed during their first five generations following capture. For each strain two crosses were routinely made with the same *P* and *M* reference lines. Thirty individuals of the population under test were mass mated as follows :

Cross A : Canton-S *(M)* ♀♀ × ♂♂ under test.

Cross A* : ♀♀ under test × ♂♂ Harwich *(P)*.

Dissection of 50 $F_1$ females allowed an estimation of the frequency of dysgenic ovaries (*GD* sterility criterion). Cross A provided a measure of the activity of *P* factors in males, and *P* strains are not expected to produce more than trivial (5 p. 100) levels of *GD* sterility in cross A*. Cross A* distinguishes between *M* cytotype (> 5 p. 100 *GD* sterility) and *P* cytotype (< 5 p. 100 *GD* sterility). *Q* strains are defined as those which produce less than 5 p. 100 of *GD* sterility both in crosses A and A*. Moreover, potentiality for intrastrain sterility was tested in each *M* strain in order to avoid confusion between *GD* sterility and maternally inherited sterility of character. Such as *grandchildless* (THIERRY-MEIG, 1976) or *atrophie gonadique* (PERIQUET, 1980). The frequencies of *GD* sterility were estimated using the method of KIDWELL *et al.* (1981).

## III. Results and discussion

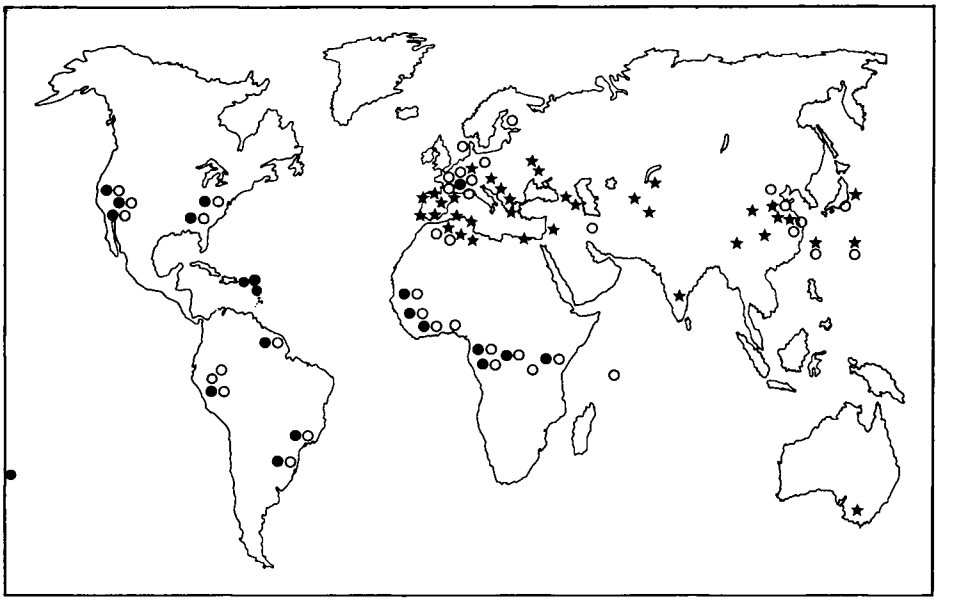

FIG. 1

*Geographical distribution of world-wide collected strains according to their potential for the* P-M *system.* P (●), Q (○) *and* M (*) *strains.*

*Distribution du système* P-M *de dysgénésie des hybrides. Souches* P (●), Q (○) *et* M (*).

The data (fig. 1 and tabl. 1) show marked geographical differences in the present distribution of the *P-M* system. In North America most of the strains show *P* activity and have levels of induced *GD* sterility which fluctuate around an average value of 15 p. 100. According to the technique used here (mass characterisation), this suggests that natural populations are polymorphic for *P* and *Q* types as has been previously demonstrated by ENGELS & PRESTON (1980) in a natural population from Madison

and as can be shown here in the Concord iso-female lines. No *M* strains have been identifield in the present study which agrees with the fact that *M* strains have been found very rarely in modern U.S. populations (KIDWELL, 1983). In our study, *P* strains have also been found in South America. The other main area where *P* strains has been found is Central Africa from Senegal to Kenya. In these regions *M* strains also appear rare and the observation of a relatively high level of intra sterility in one Cotonou strain does not allow its classification as an *M* strain.

TABLE 1

*Results of wild-type strains tested in the* P-M *system by their frequencies of* GD *sterility and intra-strain sterility.*

*Caractérisation des souches sauvages dans le système* P-M, *par leurs fréquences de stérilité* GD *et de stérilité intra-souche.*

| Strains, year of capture | | % of dysgenic activity | | |
|---|---|---|---|---|
| | | *P* | *M* | *Intra-strain* |
| *Americas* | | | | |
| U.S.A. | | | | |
| Winters | 1982-A | 5.0 | 0.0 | 0.0 |
| Winters | 1982-B | 21.0 | 0.0 | 2.0 |
| Winters | 1982-C | 12.0 | 0.0 | 0.0 |
| El Rio | 1982 | 12.0 | 0.0 | — |
| Death Valley | 1982 | 27.0 | 0.0 | 0.0 |
| Raleigh | 1982 | 20.0 | 0.0 | — |
| Concord | 1982-1* | 8.0 | 1.0 | — |
| Concord | 1982-2* | 24.0 | 0.0 | — |
| Concord | 1982-3* | 9.0 | 0.0 | — |
| Concord | 1982-4* | 6.0 | 1.0 | — |
| Concord | 1982-5* | 1.0 | 3.0 | — |
| Concord | 1982-6* | 2.0 | 2.0 | — |
| Concord | 1982-7* | 3.0 | 3.0 | — |
| Porto-Rico | 1983 | 13.0 | 3.0 | 0.0 |
| Saint-Thomas | 1983 | 14.0 | 0.0 | 0.0 |
| Guadeloupe | 1983 | 15.0 | 0.0 | 0.0 |
| Guyane | 1983 | 10.0 | 0.0 | 0.0 |
| Brazil | | | | |
| Rio | 1982 | 10.0 | 0.0 | 0.0 |
| Porto Allegre | 1982 | 8.0 | 0.0 | — |
| Peru | | | | |
| Iquitos | 1983 | 1.0 | 0.0 | 0.0 |
| Huaraz | 1983 | 9.0 | 0.0 | 0.0 |
| Huaylas | 1983 | 3.0 | 1.0 | — |

* Isofemale lines.

** Tested in 1982.

TABLE 1 *(continued)*

| Strains, year of capture | | % of dysgenic activity | | |
|---|---|---|---|---|
| | | *P* | *M* | *Intra-strain* |
| *Africa* | | | | |
| Senegal, Nianing ..... | 1979** | 19.0 | 5.0 | 3.0 |
| Sierra Leone ..... | 1983 | 14.0 | 0.0 | 0.0 |
| Ivory Coast | | | | |
| Tai ..... | 1981 | 10.0 | 2.0 | 3.5 |
| Lamto ..... | 1983 | 1.0 | 0.0 | 3.0 |
| Benin | | | | |
| Cotonou ..... | 1982-A | 0.0 | 0.0 | 0.0 |
| Cotonou ..... | 1982-B | 3.0 | 9.0 | 5.0 |
| Gagon, Makokou ..... | 1981 | 8.0 | 0.0 | 1.0 |
| Congo | | | | |
| Brazzaville ..... | 1981 | 7.0 | 0.0 | 1.0 |
| Loua ..... | 1983-A | 8.0 | 0.0 | — |
| Loua ..... | 1983-B | 8.0 | — | — |
| Loua ..... | 1983-C | 12.0 | — | — |
| Loua ..... | 1983-D | 18.0 | 0.0 | — |
| Burundi ..... | 1979 | 0.0 | 0.0 | 0.0 |
| Kenya | | | | |
| Nairobi ..... | 1982[k] | 19.0 | 0.0 | — |
| Nairobi ..... | 1982[l] | 36.0 | 0.0 | 1.0 |
| Seychelles ..... | 1981 | 1.0 | 3.0 | 1.0 |
| Algeria | | | | |
| Tlemcen ..... | 1981 | 0.0 | 0.0 | 0.0 |
| Oran ..... | 1982 | 0.0 | 5.0 | 0.0 |
| Annaba ..... | 1982 | 0.0 | 55.0 | 0.0 |
| Tunisia | | | | |
| Chebika ..... | 1982 | 0.0 | 69.0 | 0.0 |
| Gafsa ..... | 1982 | 0.0 | 89.0 | 0.0 |
| Nasr' Allah ..... | 1981 | 0.0 | 80.0 | 0.0 |
| Gabès ..... | 1982 | 0.0 | 94.0 | 0.0 |
| Egypt | | | | |
| Alexandria ..... | 1981 | 0.0 | 75.0 | 0.0 |

* Isofemale lines.
** Tested in 1982.

TABLE 1 *(continued)*

| Strains, year of capture | | % of dysgenic activity | | |
|---|---|---|---|---|
| | | *P* | *M* | *Intra-strain* |
| *France* (from West to East) | | | | |
| Non mediterranean area | | | | |
| Tours | 1982-I | 3.0 | 2.0 | 0.0 |
| Tours | 1982-II | 0.0 | 0.0 | 0.0 |
| Vierzon | 1982 | 2.0 | 0.0 | 0.0 |
| Bourges | 1982 | 1.0 | 3.0 | 0.0 |
| Ménétréol | 1982-II | 2.5 | 4.0 | 2.0 |
| Ménétréol | 1982-III | 3.0 | 0.0 | — |
| Chamballud | 1981 | 1.0 | 0.0 | 0.0 |
| Crécy | 1982 | 1.0 | 53.0 | 0.0 |
| Moulins | 1982-II | 0.0 | 0.0 | — |
| Vichy | 1982-II | 0.0 | 0.0 | 0.0 |
| Vichy | 1982-III | 0.0 | 21.0 | 0.5 |
| Les Jouberts | 1982 | 0.0 | 0.0 | — |
| Pinols | 1982-I | 0.0 | 0.0 | 1.0 |
| Pinols | 1982-II | 1.0 | 0.0 | 0.0 |
| Souillac | 1982 | 0.0 | 7.0 | — |
| Vermeil | 1982-A | 0.0 | 0.0 | — |
| Vermeil | 1982-B* | 13.0 | 4.0 | 0.5 |
| La Poujade | 1982 | 7.0 | 0.0 | — |
| Biziat | 1982-II | 0.0 | 0.0 | 0.5 |
| Givors | 1982 | 0.0 | 0.0 | 0.0 |
| Chessy | 1982 | 0.0 | 2.0 | 0.0 |
| Remoulins | 1982 | 1.0 | 0.0 | 2.0 |
| Ménerbes | 1981 | 0.0 | 0.0 | 0.0 |
| Ménerbes | 1982 | 5.0 | 3.0 | 3.5 |
| Mediterranean area | | | | |
| Montpellier | 1981 | 0.0 | 0.0 | 0.0 |
| Tautavel-cave | 1981 | 0.0 | 0.0 | 6.0 |
| Tautavel-cave | 1982 | 4.0 | 67.0 | — |
| Tautavel-village | 1981 | 1.0 | 37.0 | 2.0 |
| Tautavel-village | 1982-A | 0.0 | 0.0 | — |
| Tautavel-village | 1982-B | 0.5 | 7.5 | 4.0 |
| La Sirole | 1981 | 0.0 | 0.0 | 0.0 |
| La Sirole | 1982 | 0.0 | 0.0 | — |

* Isofemale lines.
** Tested in 1982.

TABLE 1 *(continued)*

| Strains, year of capture | | % of dysgenic activity | | |
|---|---|---|---|---|
| | | *P* | *M* | *Intra-strain* |
| *Eur-Asia* | | | | |
| Finland, Helsinki ..... | 1981 | 0.0 | 0.0 | 0.0 |
| Denmark, Copenhaguen .. | 1980** | 1.0 | 2.0 | — |
| G.F.R., Tubingen ..... | 1982 | 0.0 | 1.0 | — |
| Portugal, Algarv ..... | 1981 | 0.0 | 30.0 | 0.0 |
| Spain | | | | |
| Gerona ..... | 1980 | 0.0 | 38.0 | 3.0 |
| Gerona ..... | 1982 | 0.0 | 56.0 | — |
| Castropol ..... | 1983 | 0.0 | 74.0 | 0.0 |
| Aviles ..... | 1983 | 1.0 | 84.0 | 0.0 |
| Villaviciosa ..... | 1983 | 1.0 | 65.0 | 0.0 |
| Sevilla ..... | 1983 | 0.0 | 19.0 | 0.0 |
| Yugoslavia | | | | |
| Krsko ..... | 1982 | 0.0 | 86.0 | 0.0 |
| Divcibare ..... | 1982 | 0.0 | 73.0 | 0.0 |
| Slankamen ..... | 1982 | 0.0 | 75.0 | 0.0 |
| Greece | | | | |
| Athens ..... | 1982[e] | 0.0 | 78.0 | 4.0 |
| Athens ..... | 1982[k] | 0.0 | 70.0 | 0.0 |
| Mati ..... | 1982* | 0.0 | 76.0 | 4.0 |
| Israel | | | | |
| Jerusalem ..... | 1982-A | 0.0 | 26.0 | 0.0 |
| Jerusalem ..... | 1982-B | 0.0 | 22.0 | 0.0 |
| U.S.S.R. | | | | |
| Gomel ..... | 1981 | 0.0 | 91.0 | 0.0 |
| Uman ..... | 1981 | 3.0 | 87.0 | 2.0 |
| Ubinskaya ..... | 1982-2* | 0.0 | 74.0 | 0.0 |
| Ubinskaya ..... | 1982-6* | 2.0 | 94.0 | 0.0 |
| Tbilisi ..... | 1981 | 0.0 | 87.0 | 3.0 |
| Tachkent ..... | 1981 | 1.0 | 91.0 | 0.0 |
| Dusanbe ..... | 1982 | 0.0 | 96.0 | 6.0 |
| Alma-Ata ..... | 1981 | 0.0 | 94.0 | 0.0 |
| Iran, Teheran ..... | 1983 | 0.0 | 0.0 | 0.0 |

* Isofemale lines.

** Tested in 1982.

TABLE 1 *(continued)*

| Strains, year of capture | | % of dysgenic activity | | |
|---|---|---|---|---|
| | | *P* | *M* | *Intra-strain* |
| India, Mysore | 1982 | 0.0 | 52.0 | 1.0 |
| China - Mainland | | | | |
| Kunming | 1983 | 0.0 | 62.0 | — |
| Xian | 1983 | 0.0 | 87.0 | — |
| Changsha | 1983 | 0.0 | 86.0 | — |
| Beijing | 1983 | 1.0 | 2.0 | — |
| Jinan | 1983 | 0.0 | 22.0 | — |
| Nanjing | 1983 | 0.0 | 43.0 | — |
| Qindao | 1983 | 0.0 | 3.0 | — |
| Wuxi | 1983 | 0.0 | 18.0 | — |
| Jinhua | 1983 | 2.0 | 2.5 | — |
| Zhenjiang | 1983 | 0.0 | 0.0 | — |
| China - Taiwan | | | | |
| Tong-pu | 1981 | 2.0 | 0.0 | 3.0 |
| Taichung | 1981 | 0.0 | 28.0 | 0.0 |
| Japan | | | | |
| Otsuki | 1981-A | 0.0 | 2.0 | 0.0 |
| Otsuki | 1981-B | 0.0 | 5.0 | 0.0 |
| Australia, Melbourne | 1982 | 0.0 | 29.0 | 0.0 |
| Tahiti | 1982 | 10.0 | 0.0 | 0.0 |

* Isofemale lines.
** Tested in 1982.

In North Africa, Europe and Asia the distribution patterns change dramatically, *P* strains being almost absent and *M* strains very common. In fact, a marked difference has been found between north western Europe, best characterised by the French populations, and the rest of Europe, the mediterranean and Asia. In France most current strains are *Q* ones and the few observed *M* strains are essentially mediterranean. Only two *P* strains have been found, but in samples collected 4 kms apart, and thus probably representing the same population. In all other regions from Yugoslavia to Japan, a majority of *M* strains has been characterised, generally with a high level of *GD* sterility. However some *Q* strains have also been found and this observation suggests the existence of cytotype polymorphisms in these areas, as has been demonstrated in France, Tunisia and Japan (ANXOLABÉHÈRE *et al.*, 1982 a ; OHISHI *et al.*, 1982). Nevertheless, we have to remember that if the patterns of all these regions appear devoid of *P* strains, this situation may be due to the distribution of our sampling and that *P* sporadic types might exist as in France. Independently of the modern geographical variation KIDWELL (1983) has shown temporal trends in the distribution of strains. The frequency of *M* strains is positively correlated with labo-

ratory age and *P* strains do not appear in samples taken before 1950, but then increase rapidly in frequency.

To explain these relationships ENGELS (1981) proposed the stochastic loss hypothesis in which the temporal trends result from the stochastic loss of *P* elements when flies sampled from natural populations are maintained in the laboratory. However the presence of numerous *M* strains in the wild supports the idea that an evolutionary process is responsible for their maintenance in natural populations. In her rapid invasion hypothesis, KIDWELL (1983) proposes that before about 1950 almost all natural populations were essentially of the *M* type, the *P* element family being absent or extremely rare. About 30 years ago, *P* factors rapidely began to invade natural populations. The present distribution could then be explained by the complete invasion of most natural populations by the *P* element family, but the structure and function of individual elements would be expected to vary widely due to internal deletion of element sequences leading to *Q* or *M* strains in different areas. However, from such a chiefly random process, different patches of homogeneity can be generated from a balance between migration and random drift (JONES *et al.*, 1981) and would be expected to lead to a very heterogeneous geographical distribution rather than the grouped distribution which we have found. Moreover, naturally occurring polymorphisms for the *P* factors (ENGELS & PRESTON, 1980) and for the cytotype (ANXOLABÉHÈRE *et al.*, 1982 a) are now known.

To include these data ANXOLABÉHÈRE & PERIQUET (1983) have proposed the recurrent phases hypothesis in which most natural populations are polymorphic for the *P* family elements both in number and structure. *P* elements with different functional properties are, they propose, commonly activated or inactivated by, for example, internal recombination between elements. The process of dispersion or regression of such elements would be under the control of balancing forces such as the rate of transposition, the occurrence of dysgenic hybrids, the ratio of the different types and fluctuations of their fitnesses in different environments (RONSSERAY, 1984) and would lead to successive and recurrent periods of invasion, stabilisation and regression, not necessarily synchronous over the whole world. During those periods, *P* elements with sterility potentiality can be « inactivated » (e.g. replaced by *P* elements devoid of the sterility potentiality but not of their mutator activity), and populations polymorphic for the cytotypes (even with high level of *M* cytotype as in Nasr'Allah) can exist. In such populations the reappearence of potential sterility can be produced, by the « reactivation » or the reintroduction of active *P* elements and a new invasion phase will start again. This evolutionary hypothesis is supported by the following observations : 1) some strong *M* strains from North Africa, polymorphic for the cytotypes, have also revealed some structural homology with a cloned *P* element (ANXOLABÉHÈRE *et al.*, unpublished data) ; 2) in the U.S.A. the actual *P* factor activity of strains collected before 1970 is, average, considerably stronger than that of strains collected during the last decade (KIDWELL, 1983) ; 3) a similar but more advanced process seems to have occurred in France (ANXOLABÉHÈRE *et al.*, 1982 b) where the current predominance of wild *Q* strains follows a previous period (1963-1973) in which *P* and *M* strains would have been more frequent ; 4) the occurrence of periods of high mutability in natural populations of North America, Europe and Asia (GOLUBOVSKY, 1980 ; BERG, 1982) ; and 5) the world-wide distribution patterns described here, which are better explained by deterministic rather than only stochastic factors.

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