9 acid phosphatase allozymes (allelic isozymes) in these species might have involved 8 mutational events.

There are 2 esterase zones in *D. punjabiensis;* 3 in *D. takahashii, D. nepalensis* and *D. jambulina;* 4 in *D. malerkotliana* and *D. immigrans.* The segregating esterase phenotypes at any esterase zone do appear in the 1:2:1 proportions, suggesting monogenic control of that zone. Heterozygotes at variable esterase zone reveal only the parental bands, suggesting that esterases under their control are monomers. The enzyme phenotypes being direct representatives of genotypes, the frequencies of different *Acph/Est* alleles have been determined from the zymograms of the wild-caught individuals (figure 2). The observed heterozygosity at *Acph*-1 locus is 0.17, 0.61, 0.62 and 0.63 in *D. nepalensis, D. malerkotliana, D. punjabiensis* and *D. takahashii* respectively. The 4 esterase loci showing diallelism have heterozygotic frequencies varying from 0.42 to 0.49 and thus contribute to genetic polymorphism. The local populations of 6 *Drosophila* species show a good fit to the Hardy-Weinberg equilibrium with respect to *Acph-1* and *Est* loci, indicating that selection is not operating. Ohno[7] hypothesised that functional differences might exist between allelic variants, and such differences could be selectively important. In the present studies, 3 allelic acid phosphatases of *D. malerkotliana* have been characterized on the basis of their specific activities, V_{max}- and K_m-values, effect of metallic ions, and inhibitor sensitivity. The specific activities of 3 allelic acid phosphatases (A_1, A_2 and A_3)[5] are 60, 89 and 100 µM α-naphthol released/min/mg protein respectively. The differences in catalytic efficiency of allelic acid phosphatases (allozymes) can be argued on the basis of marked differences observed in V_{max}-values at different temperatures[6]. However, K_m-differences between allozymes are not significant[6]. One of the allelic acid phosphatase (A_3) has revealed characteristically least affinity for an inhibitor (sodium fluoride) while Mg^{++} or Mn^{++} have shown a promotory effect. It is suggested that such biochemical differences might be physiologically relevant and subject to selection.

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Chromosome complement of two species of Amelinae (Dictyoptera: Mantodea)

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Summary. The karyotypes of *Apteromantis aptera* (Fuente) and *Ameles decolor* (Charpentier) are described. Chromosomes numbers in females are 2n = 30 and 2n = 28 respectively. Sex determination mechanism in both is XO-XX.

Matthey[1], White[2,3] and Gupta[4] described in detail the karyotypes of some species from the Amelinae subfamily. At least 7 species have been studied. In *Ameles heldreichi* Br.[5] and *Ameles abjecta* Cyr.[6] intraspecific polymorphism has been found. Matthey[1] was the first to study the karyotype of *Apteromantis bolivari* Wern. This species has 2n = 29 in males: 28 acrocentric autosomes and 1 metacentric X. The author quotes as an interesting point in the analysis of this species the presence of an unusual diakinetic stage. Wahrman[5] reported an intraspecific chromosomal polymorphism in Amelinae. He found 3 karyotypic variants in *Ameles heldreichi,* which he calls type A (2n = 27 in males, having 24 acrocentric and 2 metacentric autosomes and 1 metacentric X); type B (2n = 28 in males, with 26 acrocentrics, 1 unpaired metacentric and 1 metacentric X) and type C (2n = 29 in males, all 28 autosomes being acrocentrics and 1 metacentric X). These data were confirmed in the meiotic analysis. Type A, in fact, showed in M-I 1 ring-shaped bivalent, formed by the only pair of metacentric autosomes. Type B showed, instead of it, a trivalent composed of the only unpaired metacentric autosome and 2 acrocentric homologues; and type C rod-shaped bivalents only. The 3 types had the same fundamental number. A mechanism of the Robertsonian type was proposed to explain intraspecific variation in this species. Besides, Wahrman quotes the presence, in some individuals, of a small metacentric autosome, originated by a pericentric inversion. The author did not find homozygous individuals for this structural rearrangement.

Material and methods. 6 individuals (4 males and 2 females) of *Apteromantis aptera* Fuente, an endemic species from the South of the Iberian Peninsula, were analysed. Individuals were collected in La Mala (Granada, Spain) during April and May 1977. Also 10 individuals (2 males and 8 females) of *Ameles decolor* (Charpentier) were studied. They were collected from Sierra de Cazorla (Jaen, Spain) in 1977. For classification of specimens the code of Morales Agacino[7] was used. For male meiosis analysis, testes, fixed in acetic acid-ethanol (1:3) mixture, were squashed in 1% acetic orcein. For somatic chromosome analysis ovarioles were fixed in the same fixative. Females were previously injected with 0.05% colcemid solution in isotonic saline, and material was squashed in 1% acetic orcein.

Results. The karyotype of *Apteromantis aptera* is described here for the first time (figure 1). This species displays 14 pairs of acrocentric autosomes and a pair of submetacentric X (C.I. = 2.27) in females, with FN = 30 in males. In male meiosis, 14 bivalents and 1 X univalent were observed. Meiotic analysis showed the existence of a diakinetic stage in this species. Diakinesis in another species of *Apteromantis, A. bolivari,* was previously reported by Matthey[1]. Another point to be noted is the side-arm bridge seen in approximately half of the A-II meiotic cells analysed of this species. These bridges are due to an adherence between the long arms of the sister chromatids of an X chromosome presumably by specific regions situated in them. The existence of such bridges has been reported in grasshoppers[8].

The karyotype of *Ameles decolor,* also described here for the first time (Figure 2), shows 2 pairs of metacentric chromosomes of different size (Nos 1 and 2), a pair of subacrocentric ones (No. 13) and 10 pairs of acrocentrics (Nos 4–13): It also has a pair of metacentric X-chromo-

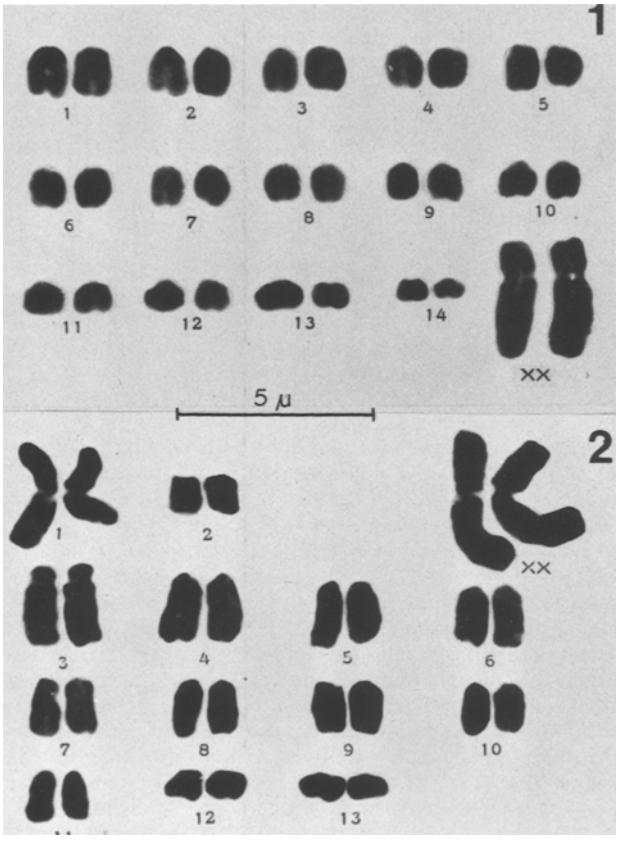

Fig. 1. Karyotype of *Apteromantis aptera* (Fuente). Female, 2n = 30.
Fig. 2. Karyotype of *Ameles decolor* (Charpentier). Female, 2n = 28.

somes. FN in this species is 34 in males, where 13 bivalents and 1 X univalent were found at meiosis. Meiosis is of the normal Mantodea type. Sex determination mechanism is XO. Diplotene and diakinesis are lacking. Transition from pachytene to M-I is immediate.

Discussion. Our observations on *Apteromantis aptera* coincide with the results obtained by Matthey[1] in *A. bolivari*. Considering that symmetrization and suppression of diplotene and diakinesis constitute fundamental trends in the evolution of mantids, the karyotypes of the species from the genus *Apteromantis* with 2n = 29 in males and FN = 30, can be regarded as the most primitive or ancestral type, from which all other karyotypes found in the Amelinae probably evolved. In this view, the karyotype of *Ameles decolor* with 2n = 27 in males and FN = 34, would have originated from the aforesaid ancester, by 2 pericentric inversions in chromosomes 2 and 3, and 1 Robertsonian translocation giving rise to chromosome 1; all rearrangements being established in the homozygous condition. This hypothesis is strongly supported by the observations of Wahrman[4] in *A. heldreichi* already referred to above, i.e. the presence of a chromosomal polymorphism in the latter species, as well as by recent observations on polymorphism in *Ameles abjecta*[6].

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An inbreeding sensitivity gene in *Drosophila melanogaster*

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Summary. Experiments are reported which tend to support the hypothesis that a single gene or gene complex may be responsible for inbreeding depression. The gene is located on the third chromosome.

I have recently interpreted certain inbreeding effects in *Drosophila* in terms of a single gene hypothesis[2]. The gene (or gene complex) affects development at different stages from fertilisation to adulthood. The effect extends to the ovaries of adult offspring, whose fecundity is reduced[3]. These offspring also show lowered overall thermogenesis, suggesting that developmental homeostasis of inbred adults does not depend exclusively on homozygosity per se[4]. Interactions between this gene and homozygosity arising from brother-sister mating have now been studied further.

In previous work, we distinguished between brother-sister couples 'sensitive' and 'insensitive' to inbreeding[2]. Sensitive couples laid fertilized eggs with reduced hatchability due to blocking of embryogenesis or failure of the larvae to hatch. Insensitive couples laid eggs that developed normally, as in unrelated crosses. Mendelian ratios were observed for this phenotypic trait of couples. This and other results[2-4] suggested that an inbreeding sensitivity gene *(Is)* with alleles Is^- and Is^+ is involved in morphogenetic events and blocks development during embryonic and larvo-pupal stages. The expression of this gene in homozygous Is^-/Is^-) embryos depends on the presence in 1 parent of an Is^+ allele, which promotes embryogenesis. Crosses between sibs of homozygous Is^-/Is^- constitution produced embryonic deaths, of a level that varied according to presence of cytoplasmic factors[2]. Homozygous embryos which survived the first critical phase continued to develop normally until the larvo-pupal period, when a number of them died.

A single (wild stock) P_o couple produces a family of F_1 siblings. The family can be characterized by the ratio between sensitive and insensitive F_1 sib couples it forms. Thus, a 1:3 ratio (0.25 sensitive couples) in a family indicates that 1 P_o parent was Is^-/Is^- and the other Is^+/Is^-, since 1 F_1 couple out of 4 with such parents would consist of 2 Is^-/Is^- individuals.

My single gene hypothesis was tested in a mate-switching experiment. *Drosophila melanogaster* were collected in the wild just before the experiment and maintained by mass culture. They were reared at 25 °C in standard axenic maize-dried yeast-agar medium[5]. 2 P_o couples, a × a and