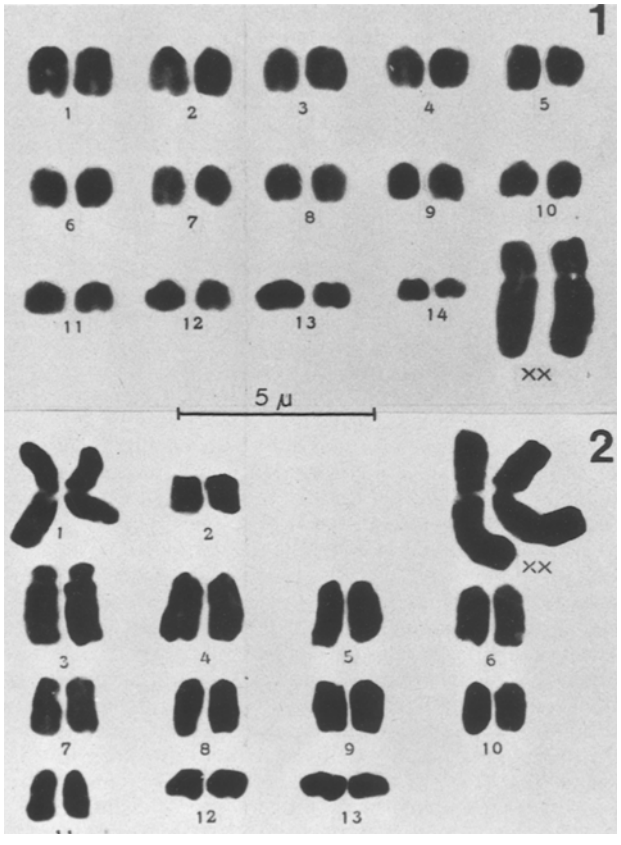

Fig. 1. Karyotype of *Apteromantis aptera* (Fuente). Female, 2n = 30.
Fig. 2. Karyotype of *Ameles decolor* (Charpentier). Female, 2n = 28.

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.

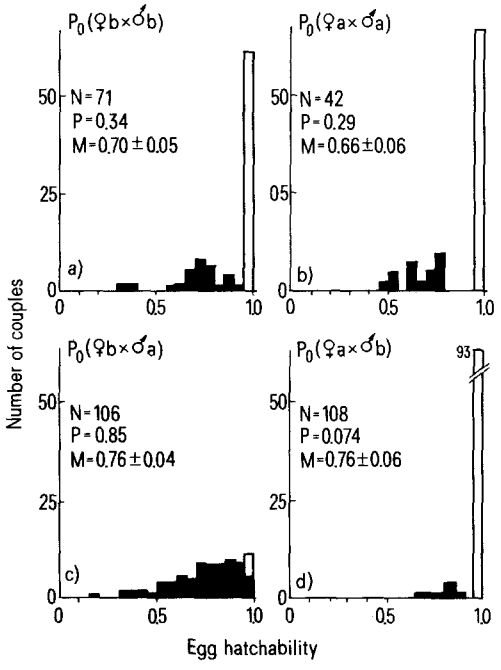
In previous work, we distinguished between brother-sister couples 'sensitive' and 'insensitive' to inbreeding[2]. Sensitive couples laid fertilized eggs with reduced hatchability due to blocking of embryogenesis or failure of the larvae to hatch. Insensitive couples laid eggs that developed normally, 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

The recrossing experiment. At first each couple was formed from a brother and sister and eggs were laid. The mates were then separated. After sperm depletion, females from sensitive (insensitive) couples were mated to new males from sensitive (insensitive) couples of the other family. Data in the last line correspond to new matings between different brothers and sisters from sensitive couples of the bb family. Hatching-to-adult survival is the proportion of adult offspring obtained from the hatched eggs.

Cross	Families from which the new mates originated		Sensitivity of the first sib couples	Remated couples		
	♀	♂		Number	Egg hatchability	Hatching-to-adult survival
1	aa	bb	Both sensitive	13	0.90	0.94
2	bb	aa	Both sensitive	13	0.92	0.93
3	aa	bb	Both insensitive	20	0.91	0.92
4	bb	aa	Both insensitive	26	0.93	0.92
5	bb	bb	Both sensitive	8	0.69	0.78



Distribution of egg hatchability for F_1 sib couples. Filled bars represent couples whose fertilized eggs had low hatchability (sensitive couples). Unfilled bars represent couples whose fertilized eggs developed normally (insensitive couples). Unfertilized eggs are not plotted since the sib couples laid eggs that were fertilized to the same extent as in controls. N: Number of couples. The number of couples is standardised to 100 in the diagrams to facilitate comparison; P: proportion of sensitive couples; M: average hatchability of fertilized eggs for sensitive couples.

b×b, each produced a F_1 family (aa and bb, respectively) showing a 1:3 ratio of sensitive to insensitive F_1 sib couples (figures a and b). Then the P_0 males were removed and their females switched (after production of fertilized eggs had ceased), forming couples a×b and b×a. 2 new F_1 families (ab and ba, respectively) resulted. Since both original P_0 pairs included 1 Is^-/Is^- and 1 Is^+/Is^- individual, either both new P_0 combinations are $Is^-/Is^- \times Is^+/Is^-$ (as in the a×a and b×b pairs), or 1 is $Is^-/Is^- \times Is^-/Is^-$ and the other $Is^+/Is^- \times Is^+/Is^-$. The 1st possibility would again lead to a pair of families with 1:3 ratios. The 2nd possibility would produce 1 family with sensitive couples only and 1 family with a 1:15 ratio, or about 0.0625 sensitive couples. This latter outcome was in fact observed (figures c and d). In the ba family (figure c), about 85% of the F_1 sib couples were sensitive; thus, 15% of the F_1 couples had offspring with a normal hatch rate. However, these couples were in fact sensitive, but with a proportion of embryonic deaths too small for normal measurement. Due to sampling error, therefore, theses couples appeared to be insensitive. The number of such 'semi-sensitive' sib couples in a family increases as the mean hatchability of the fertilized eggs laid by all the F_1 couples increases[2]. In figures a, b and d, the mean hatchability is low enough to eliminate the possibility of the occurrence of semi-sensitive couples. The proportion of sensitive couples is consequently estimated with good precision. Thus, in the ba family all couples were homozygous Is^-/Is^- although some of them did not exhibit blocking in development. The ab family (figure d) had a sensitive couple ratio of 0.074, which fits the expected value (0.0625) well. One explanation for the striking change of ratio among families from a×a, b×b and a×b, b×a P_0 couples is the single gene hypothesis under consideration.

Furthermore, if the P_0 parents of the uniformly sensitive ba family (figure c) were indeed both Is^-/Is^- homozygotes, their own offspring should be of interest: did the P_0 b ♀'s eggs hatch well and become adults? The high egg-to-adult survival (0.94–1.0) for the P_0 crosses ♀a×♂a, ♀b×♂b, ♀b×♂a, ♀a×♂b, shows that they did. This argues that the Is^-/Is^- genotype is not harmful in itself, but only when it participates in inbred matings. The P_0 parents, of course, were unrelated. Thus, the Is^-/Is^- genotype effect is best described as sensitivity to inbreeding.

To test this conclusion further, a recrossing experiment was done. F_1 flies in aa and bb families were isolated just after mating. Following sperm depletion, females from sensitive couples were mated to new males from sensitive couples of the other (unrelated) family. Flies from insensitive couples were remated similarly. As the table shows, all couples of non-related flies showed normal hatchability and offspring survival. These results might be taken as evidence for deleterious alleles at loci that differed between the 2 families, but the figures rule out that interpretation: the same gene was clearly involved in both families.

We have also confirmed the involvement of the same gene in all sensitive couples in a family. The female from each sensitive sib couple was isolated and remated with a different brother, previously a mate in another sensitive couple. As already reported[2], all new sib couples were sensitive (table, last line).

These results reveal the presence in *Drosophila melanogaster* of a gene that determines whether a female mated with her brother will lay eggs with normal or abnormal development. The natural population here studied sufferes inbreeding depression because it contained Is^- alleles at high frequency. Partial mapping in terms of chromosomal contributions has been attempted by the use of a balancer stock *(Cy/Pm; H/Sb)*. *Cy* and *Pm* are on the 2nd chromosome; *H* and *Sb* are on the 3rd. An F_1 family (wild population) having some F_1 sib couples whose eggs gave low survival was chosen. After testing egg hatchability in the F_1 sib couples, females with Is^-/Is^- genotype were separated from their mates and, once they no longer produced fertilized eggs, crossed with *Cy/Pm; H/Sb* males. Is^+/Is^-

Cy Sb F_2 flies from different sibships were intercrossed leading to four classes of offspring with phenotypes +, *Cy*, *Sb* and *Cy Sb*. In each class brothers and sisters were then mated. This scheme allowed crossing between Is^-/Is^- sibs in the *Cy* class only if the *Is* gene was located on the third chromosome, or in the *Sb* class only if the gene was on the 2nd chromosome. However, in the + class all sibs were Is^-/Is^-. As the hatchability of inbred eggs of the respective marker classes indicates (0.36 ± 0.20 for +; 0.54 ± 0.11 for *Cy*; 0.92 ± 0.07 for *Sb* sibs), inbreeding depression was associated with the 3rd chromosome.

I believe this observation reflects the action of a mutator[6,7] or controlling element[8,9]. Such elements which can change the expression of structural genes[10], perhaps by interacting with a regulatory system[11], are transposable to different loci in the genome[9,10]. Transposition[10] or chromosomal contamination[12–14] might account for normal hatchability of Is^-/Is^+ eggs bearing an induced chromosome as is the case when 1 parent possesses an Is^- allele[2]. According to this mutator hypothesis[6], disruption of genetic suppression of mutator activity through hybridisation between populations or shifts from inbreeding to outbreeding can lead to an increase in mutation frequency. These mutations or chromosomal abnormalities are then revealed by inbreeding.

Since inbreeding depression appears to be directly associated with mutator activity and related phenomena[15,16], inbred matings may play a major role in the course of evolutionary processes. My hypothesis differs from the classical view of inbreeding depression, which postulates concealed deleterious genes[17]. Of course both mechanisms could be operative.

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The karyotype of *Typhlonectes compressicauda* (Amphibia: Gymnophiona) with comments on chromosome evolution in caecilians[1]

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Summary. Typhlonectes compressicauda has a diploid number of 28. Its karyotype, when compared to that of other caecilians, suggests some discordance in the hypothesized model of chromosome reduction in the evolution of amphibian lineages.

Karyotypic information is now available for 15 species representing 3 of the 5 currently recognized families of caecilians[2–4]. The only member of the aquatic New World family Typhlonectidae karyotyped thus far is *Chthonerpeton indistinctum*[5]. This species, while having a number of derived morphological and physiological features that are associated with its aquatic habitus, also has the lowest diploid number reported for caecilians ($2n = 20$) and it lacks microchromosomes. Based on this and similar lines of evidence, it has been suggested that karyotypes provide evidence in support of the hypothesis that the general pattern of amphibian chromosome evolution is one of reduction in chromosome number (with loss of microchromosomes)[6,7]. Moreover, this reductional trend in chromosome evolution may be correlated with derived states in other features of amphibian biology[8–11]. In this report we describe the karyotype of another member of the family Typhlonectidae, *Typhlonectes compressicauda,* and reconsider the 'reduction' hypothesis of chromosomal evolution in light of new evidence presented herein.

Material and methods. 3 individuals (2 females and 1 male) of *Typhlonectes compressicauda* from Cienga Santo Tomás, Departamento Atlantico, Colombia, were karyotyped. Specimens and karyotypic preparations will be deposited in the Museum of Vertebrate Zoology, University of California, Berkeley. Animals were injected i.p. with a 0.05% colchicine solution 6 h prior to sacrifice. The best preparations were obtained from an animal that had been injected with 0.5 ml of warm yeast suspension both 48 and 24 h prior to colchicine injection[12]. Air-dried slides were prepared using gut epithelium and spleen according to the method of Patton[13] except incubation in hypotonic solution was for 1 h and centrifugation was at 700 rpm. Metaphase spreads in which most or all of the chromosomes were not overlapping were used to determine the diploid number. 17 chromosomal spreads were analyzed.

Results. Typhlonectes compressicauda has a chromosomal complement consisting of 28 biarmed elements (figure). The karyotype contains 3 groups of chromosomes: metacentrics (2 large pairs, 3 medium to small pairs); submetacentrics (5 medium-sized pairs); and subtelocentrics (4 small pairs).

Discussion. A comparison of the nonpreferentially stained karyotypes of the 2 species of typhlonectid caecilians,