phenotypes was impossible. This phenomenon might be explained by supposing that the continuous inbreeding of the lines used in the experiment have made homozygous unknown regulatory genes, controlling the expression of the *Est-6* locus.

Beardmore and Ahmad[7] found in *D. simulans* that 3 esterase loci (one of which is tightly linked to esterase 6) influence the appearance of bands at positions corresponding to those characteristic of the *Est-6* alleles; active alleles at these loci are present in wild populations at very low frequencies. It is possible that a similar situation exists also in *D. melanogaster,* a species very closely related to *D. simulans,* even if in our case the additional cistron does not map close to the *Est-6* locus, and shows a different substrate specificity. Beardmore and Ahmad suggest that the appearance of unusual esterases may be in relation to the exposure of the wild populations of *D. simulans* to insecticides. In fact Tsakas and Krimbas[8] in *Dacus oleae,* reported evidence in favour of selection for the new enzymes. On the other hand, it must be noticed that *Est-6* is probably not involved in the physiology of the nervous system, as indirectly indicated by the data of Wright[4] and of Korochkin et al.[9]. The fact that in our case the frequency of the duplication in the original population of *D. melanogaster* is quite low and that no apparent variation in frequency has been observed in the population samples maintained in the laboratory for several generations, leads us to assume that there probably is no selective pressure acting on this duplication.

Segregation of esterase phenotypes S (slow), F (fast), and VF (very fast) in the F_2 from inter se crosses of F_1 flies from the cross S, F × VF

Phenotypes in F_2	S, F, VF	S, F	VF	S, VF	F, VF
No. of individuals	191	63	69	65	53

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A pathogenic hypothesis of malformation caused by amniocentesis[1]

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Summary. The role of amniocentesis in producing fetal malformations has been investigated in the rat. Relevant uterine contractions were observed after amniocentesis. The pathogenetic mechanism of fetal malformations is discussed on the basis of the present results.

According to literature[2,3] in rodents, malformations occur in high percentage when amniocentesis is carried out during the organogenetic stage of gestation. These malformations seem to be related to histologically observed haemorrhagic processes occurring in rat limbs after about 120 min from amniocentesis. Throughout the period of survival, no fetal haemorrhages take place when amniocentesis is performed within the amniotic sac outside the uterus[3].

On the other hand, vasopressine administration in pregnant rats provokes fetal malformations[2]. These observations support the hypothesis that alterations are due to an increase of intrauterine pressure. Furthermore it has been demonstrated experimentally[4] that needle puncture alone in amniotic sac does not give rise to pathological events; therefore, as far as fetal malformations are concerned, all the data suggest a pathogenic role of uterine volume reduction which could provoke contractions greater than those occurring during pregnancy, even when pathologic. Thus we decided to control directly in the rat the intraamniotic pressure after amniocentesis, during the same period of gestation in which, in previous experiments on the same species[4], teratogenetic effects were caused.

The experiments were carried out on 20 Wistar rats (270 g) at 15th day of gestation. The day of fecundation was established by the presence of spermatozoa in the vaginal smear. The animals were anaesthetized with ether. After laparotomy, the uterine horns were exposed and protected with warm mineral oil (37 °C). The measure of endouterine pressure was performed by means of a needle type Butterfly No. 21, introduced in the amniotic sac and connected through a pressure transducer (Sanborn Mod. 268) to a preamplifier (Sanborn 350-3000). The signals were recorded by a Tektronix oscilloscope and a Grass recording camera. The same needle was connected to a syringe by a T catheter for fluid withdrawal or introduction (0.04 ml).

During the control period, intrauterine pressure inside various sacs showed a mean value of 4 mm Hg and wave amplitude was about 3 mm Hg. The frequency of contraction was about 3/min (figure 1, A).

The pattern of wave progression shown in figure 1, A, during the control period corresponds to the variation

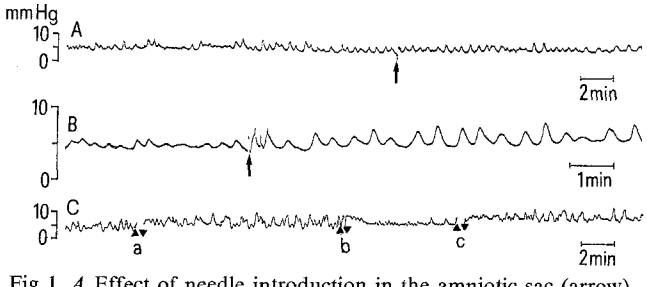

Fig. 1. *A* Effect of needle introduction in the amniotic sac (arrow) without fluid withdrawal. *B* Effect on the amniotic pressure of amniocentesis (arrow). *C* Effect of amniocentesis and of reintroduction of fluid on endouterine pressure; before a: control, a: amniocentesis (0.04 ml); b: reintroduction of the fluid (0.04 ml) withdrawn in a; c: amniocentesis. Continuous tracing. All the trials have been carried out on Wistar rats at 15th day of gestation.

frequency in the electric basic potentials, described by Kao[5] in myometers of pregnant rats in the same stage of gestation.

The simple puncture made in the amniotic sac, without withdrawing the fluid, provoked only an increase in wave frequency (up to 7/min), the mean pressure value remaining stable (figure 1, A).

After fluid withdrawal (0.04 ml) from the amniotic cavity, the record baseline fell and a series of irregular waves began, similar in frequency to those caused by simple needle introduction but about twice as high in amplitude (figure 1, B). The reintroduction of the fluid previously withdrawn brought about an immediate decrease in both frequency and amplitude of pressure waves. Further subtraction of the amniotic fluid (0.04 ml) in the same sac determined a new contraction whose amplitude and frequency waves were similar to those recorded during the first amniocentesis (figure 1, C).

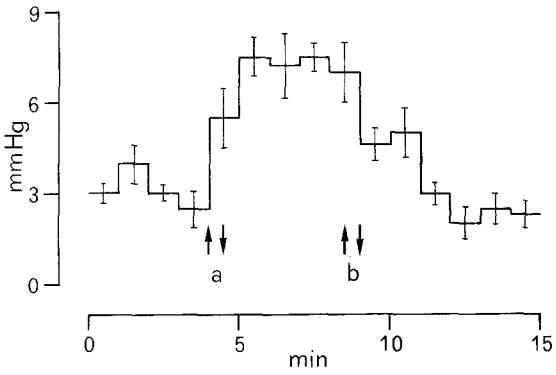

Fig. 2. Increase of endouterine pressure following amniocentesis, in Wistar rats at 15th day of gestation. Mean values of endouterine pressure for successive 1-min periods. Vertical bars give the SE of respective mean values. Between arrows, a, amniocentesis (0.04 ml); b, previously withdrawn fluid reintroduction. Abscissa: time; ordinate: endouterine pressure.

In 35 withdrawal and reintroduction tests, mean values of uterine pressure were obtained for successive 1-min periods and their SE evaluated (figure 2).

According to the results of the present research, amniocentesis induces an increase of mean endouterine pressure. This effect cannot be related to mechanical injuries, because they provoke only a transitory increase in pressure wave frequency, while an increased mean pressure value has never been observed. Admittedly, this increase in mean pressure ought to be determined by fluid withdrawing.

In previous works, fetal malformations have been observed when fluid withdrawing performed with amniocentesis was permanent. In fact, immediate reintroduction of fluid was able to eliminate the pathological effect. Needle puncture alone was also unable to induce malformations[4].

These observations support the hypothesis that fetal alterations are brought about by amniocentesis considered as volume reduction, which could provoke, through the increment of endouterine pressure, the histologically observed haemorrhagic processes and the fetal malformations. The mechanism involved in such a pathological event is not clear. The teratogenetic effect of fluid reduction as a nutritive element does not appear to play a role in this pathogenesis.

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Sex chromosome aberrations involving loss and translocation of tumor-inducing loci in *Xiphophorus*[1]

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Summary. Karyotypic changes involving a deletion and a translocation of certain sex-linked tumor-inducing loci of the platyfish, *Platypoecilus (Xiphophorus) maculatus,* have been investigated. The effects of these chromosome aberrations on tumor formation and viability of the fish are discussed.

Several well-investigated melanophore spot pattern determining loci, including Sd (spotted dorsal fin) and Sr (stripe sided body), involved in melanoma formation, have been located on the sex chromosomes of the platyfish, *Platypoecilus maculatus*[2-4]. In addition to the melanophore pattern loci, certain pterinophore determining loci are also located on the sex chromosomes. In a certain strain of platyfish, the X-chromosome carries the Sd locus and a pterinophore locus Dr (red dorsal fin), while the Y-chromosome carries Sr and another pterinophore locus Ar (red anal fin)[5]; the females are genotypically X-Dr Sd, X-Dr Sd, while the males are X-Dr Sd, Y-Ar Sr. Spot-specific melanomas develop on the fish of appropriate genotype probably due to enhanced expression of Sd (on the dorsal fin) or Sr (on the body side), following deletion or impairment of their controlling genes through selective matings or exposure to irradiation[6]. Genetic and phenotypic analysis has revealed the presence of several aberrations of the sex chromosomes of the platyfish[5], which have occurred in several different strains, and these are maintained in a balanced state in this laboratory. So far these sex chromosome abnormalities have not been cytologically characterized in terms of the size of the chromosome fragment involved and the position of the translocated material. In this report we examine the cytological nature of a deletion involving the Sd locus, and an X-Y translocation involving the Sr locus, and the genetic consequences of these sex chromosome aberrations on the tumor-potential and viability of the platyfish.

Cells for the chromosome spreads were derived from a relatively simple short-term fin explant culture method[7]. The explants were grown on a cover-glass in a culture medium containing medium 199 and calf serum (5:1, by volume), plus penicillin and streptomycin. Chromosome spreads were obtained by treating the cells, following a growth period of 48–72 h, with 0.04 mg/ml colchicine for 3 h, a 30-min treatment with a hypotonic (0.85% sodium citrate), fixation in cold methanol:acetic acid (2:1), air drying the cover-glass with the cells, a short treatment in