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stable short telocentric (present in 25% of the metaphases). If this is the case, our findings suggest that 1. chromosomal fission does occur in mammals, at least at the level of individual chromosomes; 2. some fission products can be highly stable; and 3. either centric fusion mechanisms involve a duplication of the centromere, or telocentric chromosomes can sometimes behave in a normal way with half centromeres.

Resumen. Los estudios cromosómicos llevados a cabo en un macho de *Presbytis entellus* sugieren que los mecanicos de fisión céntrica han intervenido en la evolución cromosómica de los mamíferos, que la fisión puede producir cromosomas telocéntricos estables, y que los mecanismos de fusión pueden acompañarse de una duplicación centromérica.

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Genetic Control over the Duration of G_1 Phase

Genes control the rate of cell reproduction, manifesting their effects in one or several cell systems. A number of mutant genes disturbing the normal rate of cell proliferation in different organs are known in the mouse. As was shown in our earlier work, the gene ocular retardation (gene symbol *or*) inhibits the mitotic activity of the retina anlage cells[1], and the gene fidget (*fi*) – that of the brain and eye-vesicle cells[2].

According to our present evidence, the mutant genes *or* and *fi* in the mouse significantly prolong the presynthetic period (G_1) of the cell cycle, resulting in the inhibition of the proliferative activity.

Material and method. We studied the parameters of cell cycle and the proliferative pool of retina anlage cells in 10-day-old embryos, homozygous for ocular retardation or fidget genes. Normal mice (+/+), with similar genetic background, were used as controls. To determine the parameters of the cell cycle, we made a single i.p. ^{3}H-thymidine injection (spec. activity 1.4 Ci/mmole) to the pregnant females (5 μCi/g of body weight). The animals were sacrificed at different periods from 30 min to 22 h. At least 2000 cells in the retinal anlage of each embryo were examined to estimate percentage of labelled mitotic figures. For term 15 embryos from 3 females were on the average analyzed. In order to determine the proliferative pool we made 3, 6 or 10 repeated ^{3}H-thymidine injections at 3-h intervals.

Deparaffined transverse sections through embryos (5 μm thick) were coated with liquid emulsion by dipping, exposed for 2 weeks at 4 °C, fixed and subjected to Mayer hematoxylin staining. The nuclei were considered labelled if they contained more than 4 silver grains.

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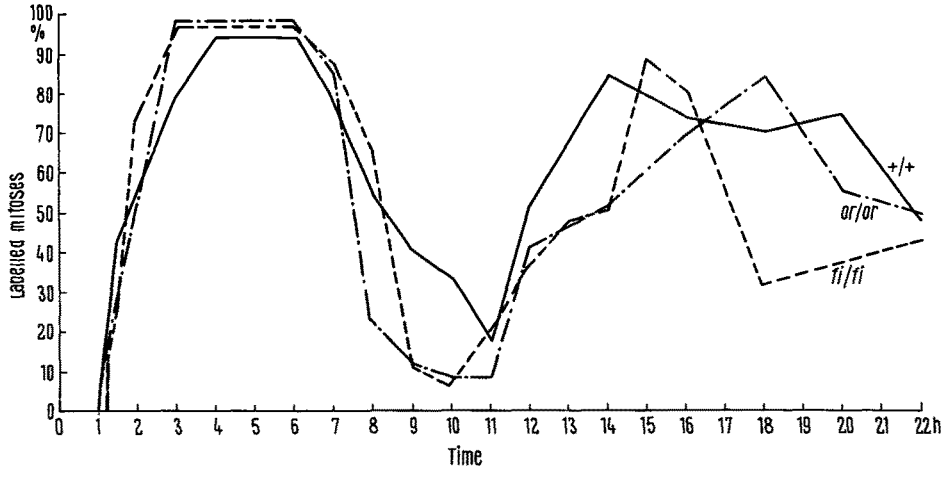

Fig. 1. Percentages of labelled mitotic figures in the retina anlage of 10-day-old +/+, *or/or* and *fi/fi* embryos after pulse labelling with ^{3}H-thymidine. Abscissa: hours; ordinate: % labelled mitoses.

Results. The data on duration of the cell cycle phases in the retinal anlage of embryos of the genotypes studied were derived from the graphic curves of the percentage of labelled mitoses by using the method of QUASTLER and SHERMAN[3] (Figure 1). The generation time (T) of the retina anlage cells shows a 1.5-fold increase in *or/or* embryos and a 1.2-fold increase in *fi/fi* embryos as compared to the norm. The total duration of G_1 and M phases in *or/or* embryos increased three times and in *fi/fi* embryos twice against the norm (Table, Figure 2). The earlier finding, that in the retina anlage of 10-day-old *or/or* and *fi/fi* embryos the mitotic index is much lower than that of +/+ embryos[1,2], leads us to suggest that changes in the length of the generation time in the mutants are due to elongation of G_1, but not M phase. The other periods of the cell cycle in the retina anlage of *or/or* and *fi/fi* embryos do not differ much from the norm.

The count of silver grains over labelled nuclei of the retina 1 h after a single ^{3}H-thymidine injection has shown slightly-increased incorporation intensity of label in *or/or* embryos as compared to the norm. Reduction of S, and more intensive incorporation of ^{3}H-thymidine into DNA, indicates some acceleration of DNA synthesis in *or/or* embryos. This is evidently a compensatory response to significant elongation of G_1 phase. In *fi/fi* embryos the intensity of ^{3}H-thymidine incorporation and the duration of S do not show any difference from those of +/+ embryos.

The duration of the cell cycle phases in the retina anlage of 10-day-old mouse embryos (h)

Genotype	t G_2	t S	t G_1 + M	T
+/+	1	$6^1/_2$	$2^1/_2$	10
or/or	$1^1/_4$	$5^2/_3$	8	15
fi/fi	1	$6^1/_2$	$4^1/_2$	12

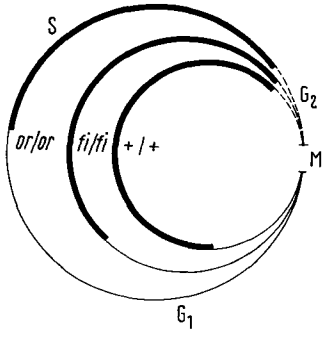

Fig. 2. The relative duration of different periods of the cell cycle in the retina anlage of 10-day-old mouse embryos of +/+, *or/or* and *fi/fi* genotypes.

The experiments with repeated ^{3}H-thymidine injections have shown that all the retina anlage cells of 10-day-old +/+, *or/or* and *fi/fi* embryos undergo proliferation. Nuclei of all the cells become labelled after 6 ^{3}H-thymidine injections.

Discussion. Thus, inhibition of the proliferative activity of the retina anlage cells by the mutant genes *or* and *fi* is caused by the elongation of the presynthetic phase of the cell cycle. Gene *or* evidently acts only in the retina anlage cells. The mitotic indices determined by us in the dorsal region of the telencephalon and ventral part of diencephalon have revealed no significant differences between 10-day-old *or/or* and +/+ embryos. Gene *fi* inhibits the proliferative cell activity in the brain and its derivative-eye vesicle. Hence, these mutant genes act in definite cell systems and disturb the synthesis of tissue-specific proteins (heterosynthesis). It has been shown for many cellular systems that the variations in the generation time are due to different length of G_1. PRESCOTT[4] suggests that there is a control point of cell reproduction at the end of the presynthetic period which, in our opinion, must be the starting-point of autosynthesis. The mutant genes *or* and *fi* changing of heterosynthesis delay the beginning of autosynthesis, bringing about the elongation of G_1 in the retina anlage cells of the mouse.

Выводы. Мутантные гены or и fi у мыши значительно удлиняют G_1 период клеток зачатка сетчатки, в результате чего происходит подавление пролиферативной активности клеток развивающейся сетчатки.

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Chromosomenaberrationen in den Wurzelspitzen von *Allium cepa* durch Aflatoxin B_1

Aflatoxin B_1, ein von *Aspergillus flavus* und verwandten Pilzarten gebildetes Mycotoxin[1], hat wegen seiner carcinogenen Eigenschaften grosses Interesse gefunden. In einer Reihe von Untersuchungen wurden Schädigungen von Zellen in Zellkulturen beobachtet, so Zerstörung von Kern und Cytoplasma bei Kälbernierenzellen[2], Hemmung der Vermehrung sowie Entstehung von Riesenzellen bei menschlichen embryonalen Lungenzellen[3,4], Wachstumshemmung und zunehmende Granulation bei HeLa- und Chang-Leberzellen[5] und Zerstörung der Nucleoli bei menschlichen Leberzellen[6,7]. Aflatoxin B_1 rief auch direkt Chromosomenaberrationen in Wurzeln von *Vicia faba*[8] und in menschlichen Leukozyten[9] sowie eine Reduktion der Mitoserate in menschlichen Lungenzellen hervor.

In der vorliegenden Arbeit wurde der Einfluss von Aflatoxin B_1 auf die Chromosomen in Zellen der Wurzelspitze von *Allium cepa* untersucht. Dieses Objekt erschien besonders interessant, weil an ihm bereits die Entstehung von Aberrationen durch Cumarin, von dem Aflatoxin B_1 chemisch abzuleiten ist, studiert worden war[11,12].

Wurzeln von *Allium cepa* wurden in verschiedenen Konzentrationen (200 µg/ml, 100 µg/ml, 10 µg/ml, 1 µg/ml) von Aflatoxin B_1 (Serva, Heidelberg) in 0.3% Dimethyl-