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EFFECT OF ACUTE HYPOXIA ON MORTALITY AND SEX RATIO OF THE EMBRYO

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The action of acute hypoxia on embryogenesis and its effect on the sex ratio in the progeny was studied in mice. Mice on the 7th-10th days of pregnancy were kept for 3 h daily under hypoxic conditions at an atmospheric pressure of 230 mm Hg. The mice were killed on the 18th day of pregnancy, when the gonads of the embryos were examined. The sex ratio in the progeny was determined as the ratio of the number of males to the number of females. Investigation showed that acute hypoxia on the 7th-10th day of pregnancy, while leading to high mortality among the adult animals, did not significantly affect embryonic development. No selective death of mouse embryos of either sex was found in the experimental and control groups of animals.

KEY WORDS: hypoxia; embryonic mortality; sex ratio.

Several investigations into the effect of hypoxia on embryogenesis have been published [2, 3, 7, 8]. Attention has been concentrated in particular on the study of the action of chronic hypoxia on fetal development [1, 6]. It has been shown, for instance, that chronic maternal hypoxia disturbs the development and causes death of the embryos. However, the effect of acute hypoxia on embryogenesis has received much less study.

The object of this investigation was to study the action of acute hypoxia on embryogenesis in mice. The opportunity was also taken to analyze the effect of acute hypoxia on the sex ratio in the progeny, in view of earlier reports that spontaneous embryonic mortality is unrelated to the sex of the embryo [4, 5].

EXPERIMENTAL METHOD

Experiments were carried out on C3H inbred mice from the Rappolovo nursery. After mating the day of discovery of a vaginal plug was taken as the first day of pregnancy. On the 7th-10th days of pregnancy the mice were kept for 3 h under hypoxic conditions at an atmospheric pressure of 230 mm Hg. The mice were killed on the 18th day of pregnancy. The embryos were removed from the uterus, laparotomy was performed, and the gonads were examined with the MBS-1 microscope. The sex ratio in the progeny was determined as the ratio of the number of males to the number of females.

Altogether 166 pregnant animals (86 experimental and 80 control) were used. The sex of 789 embryos (309 experimental and 480 control) was determined.

EXPERIMENTAL RESULTS

Keeping the pregnant mice for 3 h under hypoxic conditions at 230 mm Hg led to high mortality among the adult animals. Of the 86 experimental mice 35 (40.6%) died.

However, as Table 1 shows, acute hypoxia did not significantly increase the embryonic mortality compared with the control (19.7% and 14.4% of embryos died in the experimental and control series respectively). Meanwhile, analysis of mortality among the embryos on individual days of pregnancy showed that in the case of exposure to hypoxia on the 9th day the embryonic mortality was higher than in the control (27.1% in the

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TABLE 1. Postimplantation Mortality among Mouse Embryos on 18th Day of Development

Group of animals	Time of experiment (day of pregnancy)	Number of pregnant mice	Number of implanted embryos	Number of embryos dying after implantation	
				absolute	%
Experimental	7-th	14	115	18	14,7±3,3
	8-th	10	66	13	19,6±4,7
	9-th	15	107	29	27,1±4,2
	10-th	12	97	16	16,4±2,68
Total		51	385	76	19,7±2,02
Control		80	561	81	14,4±1,48

TABLE 2. Sex Distribution of Mouse Embryos

Group of animals	Number of embryos		Sex ratio (M:F)	χ^2	P
	M	F			
Experimental	164	145	113,1:100	0,58	>0,2
Control	234	246	95,1:100	0,3	>0,5

experimental and 14.4% in the control series). However, the living embryos were equal in size to the control and no morphological abnormalities could be found in the experimental embryos.

The study of the sex distribution of the embryos (Table 2) showed that in the experimental mice in the initial period of organogenesis the sex ratio of the embryos was 1:1 ($\chi^2 = 0.5$; $P > 0.2$), i.e., the same as in the mice of the control group.

The investigation thus showed that acute hypoxia in mice on the 7th-10th day of pregnancy, although causing considerable mortality among adult animals, does not significantly affect embryonic development. By contrast with the action of acute short-term hypoxia, chronic hypoxia produced by keeping the animals for a long time in a pressure chamber at an atmospheric pressure of 550 mm Hg gives a well-marked embryotoxic effect [1]. High embryonic mortality was observed in experiments on guinea pigs exposed to acute anoxia after compression of the uterine vessels [3], a factor with more powerful action than the conditions of the present experiments.

Analysis of the embryonic mortality and sex ratio in the experimental and control groups showed absence of selective mortality among mouse embryos of either sex in the course of embryogenesis and during exposure to acute hypoxia.

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