

8. M. Kawakami, E. Terasawa, and T. Ibuki, *Neuroendocrinology*, 6, 30 (1970).
9. K. Krnjevic, in: *Methods of Neurochemistry* (ed. by R. Fried), Dekker, New York (1971), p.130.
10. K. Kubo, R. A. Gorski, and M. Kawakami, *Neuroendocrinology*, 18, 176 (1975).
11. S. E. Monroe, R. W. Rebar, V. L. Gay, et al., *Endocrinology*, 85, 720 (1969).
12. E. R. Smith, C. Y. Bowers, and J. M. Davidson, *Endocrinology*, 93, 756 (1973).
13. K. Taya and M. Igarashi, *Endocrinol. Jpn.*, 20, 199 (1973).
14. K. Yoshinaga, R. A. Hawkins, and J. F. Stocker, *Endocrinology*, 85, 103 (1969).

THE SEX RATIO IN INBRED STRAINS OF MICE

L. D. Udalova

UDC 612.6.07:612.6.06

A study of the sex ratio in mice of inbred strains CBA and C3H and of the connection between the postimplantation embryonic mortality of mice of these strains and the sex distribution of the embryos showed that the embryonic sex ratio of these lines of mice obeys the 1:1 distribution. Data in the literature and the writer's own observations suggest that genetic differences between mice of inbred strains have no significant effect on the sex ratio of the progeny. The postimplantation embryonic mortality in C3H mice is greater than that of CBA mice (14.4 and 9.3% respectively). However, the presence of a balanced sex ratio in the mice of these strains is evidence of absence of selective death of embryos of either sex during embryogenesis.

KEY WORDS: *sex ratio; embryonic mortality; inbred strains of mice.*

The investigation of the mechanism controlling the sex ratio in progeny is of great interest. Information in the literature on the effect of genetic differences between mice of different strains on the sex distribution in the progeny is highly contradictory. Some workers have found that genetic differences between inbred strains have no marked effect on the sex ratio [4], whereas others consider that their effect is insignificant and they point to an unbalanced sex distribution of fetuses of the mice of certain strains [3, 6].

The object of this investigation was to study the sex ratio in mice of inbred strains CBA and C3H and to analyze the connection between the postimplantation embryonic mortality of mice of these strains with the sex distribution of the embryos.

EXPERIMENTAL METHOD

Mice of strains CBA and C3H obtained from the Rappolovo nursery were studied. Females were crossed with males of the same strain and the day of discovery of a vaginal plug was taken as the first day of pregnancy. The females were killed on the 18th day of pregnancy. Embryos at the 18th day of development were removed from the uterine cavity and, after laparotomy, their gonads were examined with the MBS-1 microscope. The ratio between the number of males and the number of females was determined.

EXPERIMENTAL RESULTS

The postimplantation embryonic mortality was determined from the 18th day of pregnancy. As Table 1 shows, the postimplantation mortality of the C3H embryos was higher than that of the CBA embryos.

A study of the sex distribution of the embryos (Table 2) showed that both in the C3H and the CBA mice the sex ratio of the embryos obeyed the 1:1 distribution.

Department of Embryology, Institute of Experimental Medicine, Academy of Medical Sciences of the USSR, Leningrad. (Presented by Academician of the Academy of Medical Sciences of the USSR M. A. Petrov-Maslakov.) Translated from *Byulleten' Éksperimental'noi Biologii i Meditsiny*, Vol. 83, No. 2, pp. 223-224, February, 1977. Original article submitted August 9, 1976.

This material is protected by copyright registered in the name of Plenum Publishing Corporation, 227 West 17th Street, New York, N. Y. 10011. No part of this publication may be reproduced, stored in a retrieval system, or transmitted, in any form or by any means, electronic, mechanical, photocopying, microfilming, recording or otherwise, without written permission of the publisher. A copy of this article is available from the publisher for \$7.50.

TABLE 1. Postimplantation Mortality of Mouse Embryos at the 18th Day of Development

Strain of mice	Number of pregnant mice	Number of implanted embryos	Number of embryos dying after implantation	
			absolute	% ($M \pm m$)
C3H	80	561	81	14,4 \pm 1,48
CBA	94	674	63	9,3 \pm 1,11

TABLE 2. Sex Distribution of Mouse Embryos at the 18th Day of Development

Strain of mice	Number of embryos	Sex ratio	χ^2	P
C3H	234♂ : 246♀	95,1 : 100	0,3	>0,5
CBA	300♂ : 311♀	96,4 : 100	0,19	>0,5

The higher postimplantation mortality in the C3H mice than in the CBA mice thus did not disturb the sex ratio of the progeny, evidence that in the mice of these strains selective death of embryos during embryogenesis does not take place in the embryos of either sex.

The results of this investigation confirm earlier results indicating that embryonic mortality does not affect the sex ratio of the progeny of laboratory mice [2, 5, 7]. A sex ratio of 1:1 has been found for mice of many inbred strains [4, 6]. However, an unbalanced sex distribution has been observed among the fetuses of some inbred strains of mice. For instance, there are more females than males in the progeny of mice of strains A, C57BR/ed, and LAC, and more males than females in the progeny of the CE mice [3]. A sex ratio differing from the 1:1 distribution has also been observed in the progeny of C58/J mice [6]. Equally contradictory results have been obtained from hybrid mice. Some workers consider that the sex ratio of hybrids corresponds to the 1:1 distribution [1], whereas others find different variations in the sex ratio which are probably more closely connected with the genetic characteristics of the hybrid mice than in mice of inbred strains [4].

An unbalanced sex ratio, it must be noted, has been observed in mice of several strains whereas, according to data in the literature and to the writer's own observations, in mice of a substantial majority of inbred strains the embryonic sex distribution conforms to the 1:1 ratio. These findings suggest that the genetic differences between mice of inbred strains have no significant effect on the sex ratio in the progeny.

LITERATURE CITED

1. V. P. Kryshkina, Byull. Éksp. Biol. Med., No. 11, 118 (1972).
2. L. D. Udalova, Byull. Éksp. Biol. Med., No. 2, 234 (1976).
3. M. J. Cook and A. Vlcek, Nature, 191, 89 (1961).
4. A. Howard, A. McLaren, D. Michie, et al., J. Genet., 53, 200 (1955).
5. M. H. Kaufman, J. Reprod. Fertil., 35, 67 (1973).
6. G. Schlager and T. H. Roderick, J. Hered., 59, 363 (1968).
7. A. D. Vickers, J. Reprod. Fertil., 20, 63 (1969).