Zusammenfassung. Enzym-histochemische Untersuchungen weisen darauf hin, dass die sogenannten Corpora lutea praeovulationia im Ovarium von *Poecilia reticulata* keine Steroide produzieren und deswegen als Corpora atretica betrachtet werden müssen. Demgegenüber steht fest, dass in den Granulosazellen, die die Oozyten umgeben, die Enzyme 3β-Hydroxysteroid-Dehydrogenase, Glucose-6-phosphat-Dehydrogenase und einige Enzyme des Krebszyklus nachzuweisen sind. Infolgedessen können die Granulosazellen als Steroid-produzierende Zellen angesehen werden.

J. G. D. LAMBERT

Zoological Laboratory of the University, Utrecht (The Netherlands), February 25, 1966.

Evaluation of the Maternal Role in Survival of Suckling Mice[1]

In mice, GRUNEBERG has stated that, apart from malformations, suckling mortality is determined by maternal vigor[2]; however, it is not clear whether this influence is exerted pre- and/or postnatally. The present report describes experiments in which the maternal influence was tested by foster-nursing mice from strains characterized by high suckling survival to mothers from strains with a lower nursling survival, and vice versa.

Nurses were mice of the AKR/Sp, PL/Sp and C57BL/Sp strains. Non-foster-nursed and foster-nursed mice were males and females of these strains. The procedures followed have been described previously[3]. Data were analyzed using Chi square with Yates correction. Results for male and female sucklings were pooled inasmuch as no significant sex differences were found.

When mice were raised by their own mothers, survival of C57BL sucklings was higher than that of PL ($P < 0.05$) and AKR ($P < 0.001$) nurslings. Survival of PL infants was higher ($P < 0.001$) than that of AKR infants (Table).

When C57BL mice were foster-nursed, survival was decreased and was independent of the strain of origin of the foster mother (Table). When AKR mice were suckled by PL and AKR foster mothers, survival was also decreased (Table). However, when AKR mice were raised by C57BL dams and when PL infants were foster-nursed, survival was unchanged (Table). Only when AKR infants were foster-nursed did survival appear to depend on the foster mother's strain of origin and follow the same order of survival as found when these mothers raised their own progeny. While it is true that survival of AKR mice raised by AKR and PL foster mothers was lower ($P < 0.001$) than that of sucklings raised by C57BL nurses, no significant differences in survival were found when survival of mice foster-nursed by PL and AKR dams was compared, although survival of PL sucklings was higher than that of AKR mice when both were raised by their own mothers (Table). These data suggest that all the mothers transmitted potentially noxious substances in their milk and that sensitivity to these materials by sucklings was dependent on their strain of origin.

If differences in survival of foster-nursed sucklings were due only to their genotype, one would expect that the survival of mice raised by foster mothers from the same strain would be similar to that of mice raised by their own mothers. While this was the case with foster-nursed PL mice, it was not so with C57BL and AKR mice whose survival was decreased. These observations suggest the possibility that the nurslings must have become somewhat conditioned, during their in utero existence, to materials that were similar to ones that appeared later in the milk of their own mothers. Not only did these materials appear to differ from mother to mother within a given strain, but conditioning of the fetuses to them may have varied inasmuch as survival of mice raised by their own mothers was not 100%. The data suggest that maternal vigor exerted postnatally was not the only factor concerned with nursling survival, but that the strain of origin of the sucklings and non-genetic maternal influences exerted in utero must also be considered.

Although the present experiments clarify some of the factors concerned with nursling survival, they do not provide any information about the mechanism through which they operate. Recently, some observations have been made in our laboratory that contribute to the understanding of this mechanism. (1) Significant positive associations have been found between survival and thymus mitosis and between survival and thymus

Survival of foster-nursed suckling mice

Strain of mouse suckled	Foster mother	No. of litters	No. of mice at onset of nursing	Mice weaned		P Foster-nursed vs. non-foster-nursed
				No.	%	
PL	None	34	138	107	78	
	AKR	10	47	41	87	NS[a]
	PL	20	94	79	84	NS
	C57BL	23	91	76	84	NS
C57BL	None	19	104	92	88	
	AKR	38	216	96	44	< 0.001
	PL	24	112	57	51	< 0.001
	C57BL	18	93	52	56	< 0.001
AKR	None	26	152	91	60	
	AKR	6	30	5	17	< 0.001
	PL	9	54	14	26	< 0.001
	C57BL	42	213	112	53	NS

[a] NS signifies not significant.

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weight[4]. (2) Erythropoiesis has been shown to be a function of the thymus of healthy mice[5,6]. (3) An increase in dark staining cells was found in the red pulp of spleens from surviving foster-nursed C57BL female mice (decreased survival) but not in spleens from foster-nursed PL mice (unchanged survival) when compared to non-foster-nursed controls[7]. Although these cells were previously considered to be lymphocytes, recent electron microscope studies suggest that they are erythroid[8]. These observations suggest a relationship between the function of the erythropoietic system and infant survival.

Zusammenfassung. Das Überleben der von der eigenen Mutter und von stammesfremden «Ammen» gesäugten Jungen wurde untersucht an Mäusen der Stämme AKR/Sp, PL/Sp und C57BL/Sp. Die Ergebnisse zeigen, dass die genetische Herkunft der Säuglinge wie auch andere, nicht genetisch bedingte Einflüsse der Mutter auf die Jungen in utero für das Überleben der Jungen ausschlaggebend ist.

S. ALBERT, P. L. WOLF, and ROSAMOND POTTER

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Further Observations of the Angiotensin Vasopressive Effect in Rabbits

Certain sympathomimetic amines[1] and digitalis[2] seem to require the presence of catecholamines in the tissue storage sites to exert their characteristic action on the cardiovascular system. It is possible that the action of other cardiovascular agents, such as angiotensin, also depend at least in part on catecholamine activity. For this reason, a study was performed on rabbits exploring the possible role of catecholamines in vasopressor action of angiotensin.

Material and Methods. 16 male rabbits weighing between 2–4 kg were anesthetized with intraperitoneal injections of pentobarbital (35 mg/kg). The blood pressure was recorded from the common carotid artery by means of an arterial catheter connected to a suitable pressure transducer, the output of which was recorded on a Sanborn polygraph. Angiotensin was injected through a catheter in the external jugular vein. The injections were rapid and were washed in with 2 cm^3 of saline.

Doses of angiotensin (Ciba, Hypertensin)[3] producing small (10–20 mm Hg), medium (30–40 mm Hg), and large (60–80 mm Hg) elevations of mean blood pressure were determined. Since angiotensin exhibits tachyphylaxis[4], especially in high doses, care was taken in selecting an interval of injection (4–5 min) which did not show this phenomenon. The order of injections was arranged by a 3×3 latin square. Each dose-response curve required 45 min to determine and was followed immediately by a second and a third determination in the same animal. In this way the influence of time and previous injections on the reactivity to angiotensin was assessed.

To determine the response to angiotensin in a setting in which adrenergic nervous activity was reduced, β-TM 10 ([2-(26)-dimethylphenoxypropyl]trimethyl ammonium chloride)[5], an adrenergic neuronal blocking agent[6], was employed. β-TM 10 was rapidly injected into the ear vein in doses of 5 mg/kg, approximately 3–4 h before the start of the experiment. After each mean the 95% confidence limits are indicated.

Results. By a series of trials it was found that 0.1, 0.5, and 2.5 mg/kg of angiotensin produced the desired responses; namely, small, medium and large elevations of mean blood pressure (Table I). The data are summarized in Table I for each series of injections. It is evident from the data presented that there is no difference in the responses between each series. Thus 0.1, 0.5, and 2.5 mg/kg of angiotensin produced quantitatively similar increases in blood pressure in each group ($p > 0.05$). After β-TM 10 the reactivity to angiotensin was not altered. Thus the response to angiotensin (Table II) was not significantly different to the responses observed at the same doses in animals not treated with β-TM 10 (Table I).

Table I. Dose-response relationship of the angiotensin effect on blood pressure

Series of injections	Number of experiments	Average number of injections	Dose of angiotensin (mg/kg)	Average increase in blood pressure (mm Hg	CL[a])
1st	15	3	0.1	19.0	3.2
	15	3	0.5	38.6	5.0
	15	3	2.5	73.0	5.8
2nd	8	3	0.1	18.1	4.0
	8	3	0.5	36.9	9.8
	8	3	2.5	71.5	11.0
3rd	6	3	0.1	15.3	3.2
	6	3	0.5	29.0	7.0
	6	3	2.5	66.7	11.6

[a] CL = 95% confidence limit.

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