TABLE III. Effect of Meso-Inositol on Multiplication of HeLa Cells.

Exp.*	Medium†	Avg No. of cells × 10^3/tube at following concentrations of inositol: 0	.25 μM	1 μM	4 μM	16 μM	Dialysate 20%
1	Human A	0 (3+)‡		11 ± 6 (2+)			103 ± 69 (0)
	Horse L	2 (")		60 ± 13 (+)			203 ± 40 (0)
2	Human W	0 (")					101 ± 17 (0)
3	Human A	22 ± 2 (3+)	50 ± 9 (2+)	272 ± 28 (0)	280 ± 20 (0)	284 ± 32 (0)	217 ± 43 (0)
	Horse L	35 ± 17 (3+)	131 ± 27 (+)	275 ± 36 (0)	292 ± 20 (0)	191 ± 13 (0)	281 ± 31 (0)

* Inoculi: 15,000, 13,000 and 27,700 cells/tube in Exp. 1, 2 and 3 respectively.
† & ‡ See footnotes to Table II.

teins(10).§ The extent to which complex forms of inositol are utilized by human cells is a matter for future study.

Summary. A concentration of meso-inositol of at least 4 μM was necessary for maximum multiplication of human conjunctival cells grown *in vitro*. Inositol was found to be essential for multiplication of HeLa cells.

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§ In our experiments analysis of human and horse sera before and after 48 hours of dialysis showed little alteration in cholesterol content.

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Lipid Mobilizer (LM) from Posterior Pituitary of Hogs.* (23206)

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Injection of plasma or plasma dialyzate, obtained from animals administered cortisone or subjected to stress, induced hyperlipemia in intact, adrenalectomized or hypophysectomized rats previously fasted and sensitized by exposure to potential hepatotoxins(1-4). The dialyzate and highly potent material crystallized from it differed from other hyperlipemia inducing agents in not requiring the presence of either the adrenals or pituitary for its action(2-4). These studies suggested that LM was released from the pituitary gland. The data in this paper establish the presence of a

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TABLE I Effect 1 mg Dialyzate of Hog Posterior Pituitary Lobes/kg IV on Plasma Lipid Components of Various Species.

Species	Time in hours post injection															
	0			1			2			4			6		24	
	CH	FA	LP	CH	FA	LP	CH	FA	LP	CH	FA	LP	CH	FA	CH	FA	LP
Intact rats (10/hr)	62	104	5	196	212	9	262	303	10	302	353	12	348	392	184	212	8
Hypophysectomized rats (20/hr)	58	92	5				242	303	11								
Adrenalectomized rats (20/hr)	64	88	5				258	312	11								
Mice (10/hr)	52	95	5	195	208	8	312	340	12	402	386	15	440	490	120	212	8
Guinea pigs (10/hr)	85	102	5	186	195	9	245	260	10	322	330	11	385	402	84	92	6
Rabbits (12/sample)	62	90	5	190	262	9	240	304	11	320	386	13	360	412	75	142	8
Dogs (6/sample)	120	160	6	290	340	11	380	400	18	412	400	19	402	440	196	220	9

CH = Total plasma cholesterol; FA = Total fatty acids; LP = Lipid phosphorus. All values in mg %.

lipid mobilizing factor in the posterior pituitary of hogs.

Materials and methods. Lyophilized anterior and posterior lobes from hogs were prepared by Armour Laboratories. Duplicate preliminary studies demonstrated that LM was present in physiological salt solution (PSS) extracts of posterior pituitary but not in those of anterior pituitary or rat gastrocnemius muscle. A lot of posterior pituitary extract was then prepared as follows utilizing conditions that assured absence of bacterial and pyrogen contamination. Ten g finely diced lyophilized posterior pituitary was macerated with 3 g alcohol washed sea sand and addition of 100 ml PSS in small aliquots. The suspension was centrifuged 2 hours (5°C) at 4000 rpm and the withdrawn supernatant diluted to 500 ml with PSS was passed through an ultra-fine porcelain bacteriological filter (5°C). 100 ml of filtrate was dialyzed in a sealed container through Viscose No-Jax® casing against 400 ml deionized water for 24 hours at 5°C and the dialyzate was freeze dried for 18 hours. A 0.1% solution of the dry material (Wy-1323) was filtered bacteriologically and was sterile and non-pyrogenic. All animals were sensitized, fasted, injected and bled for lipid analyses as previously described(2-4). Adrenalectomized rats were maintained on 0.9% NaCl in the drinking water, and hypophysectomized rats received supplements of oranges and white bread. Operated animals were used 10-14 days after surgery.

Results: Injection of Pitressin®, Pitocin®, muscle extract, anterior lobe extract, and posterior lobe extract into groups of 20 hypophysectomized rats resulted in hyperlipemia only in the group administered posterior lobe extract. Dialyzate (0.65 mg) equivalent to 5 mg of posterior lobe produced at least a 2-fold increase in total plasma cholesterol, total fatty acids and lipid phosphorus in 60 intact rats, 20 hypophysectomized rats, 60 mice, 60 guinea pigs, 12 rabbits and 6 dogs. The hyperlipemia action of 1 mg of dialyzate/kg in a variety of species is demonstrated in Table I. The dose response curve obtained in 40 intact rats with another batch of dialyzate established a potency of

0.2 mg of dialyzate kg to produce a 2-fold elevation of plasma cholesterol.

Discussion. Our previous work established that the pituitary was necessary for release of LM. The data presented in this paper establish that hog posterior pituitary dialyzate contained LM activity comparable to that of plasma dialyzate. Other points of similarity between plasma LM and posterior pituitary LM were observed in groups of 10 rats administered 1 mg/dialyzate kg. The hyperlipemia occurring in either group 45 minutes later was not abolished by administration of 400 U of heparin kg. *In vitro* anti-clearing activity of pituitary LM was identical with the activity previously reported for plasma LM(1,2). Exact identity of pituitary LM with plasma LM remains to be established.

Summary and conclusions. 1. Dialyzate of hog posterior pituitary lobe induced hyperlipemia in rats, mice, guinea pigs, dogs and rabbits. 2. The LM activity was indistinguishable from that of plasma dialyzate from cortisonized animals. 3. Neither Pitressin® nor Pitocin® had LM activity. 4. Hog anterior pituitary extracts had no LM activity. 5. LM appears to be a hitherto undescribed hormone either elaborated or stored in the posterior pituitary.

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Inhibition of Histamine-Stimulated Secretion by Topical Application of Antihistaminics to the Canine Mucosa.* (23207)

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Failure of antihistaminics to inhibit histamine-stimulated gastric acid secretion is generally accepted. The extensive literature supporting this generalization has been carefully summarized by Robertson and Grossman(1). The gastric receptors for histamine, therefore, appear to present a puzzling exception to the almost universal blocking action of antihistaminics on histamine receptors in other organs. These authors, however, also have collected some fragments of evidence which indicate that high concentrations of antihistaminic compounds, present locally in the gastric mucosa, may block its secretory response to histamine. Wood(2) was able to block histamine-stimulated acid secretion in anesthetized cats by retrograde injection of phenergan, neoantergan, and antistin into the ligated splenic artery, so that these compounds reached the gastric mucosa in high concentration. A chemical analogue of phenergan without antihistaminic activity (R.P. 3300) was without effect. In their own experiments, Robertson and Grossman irrigated canine pouches with high concentrations of several antihistaminics (25 and 50 mg/ml of neoantergan; 6.25, 12.5, and 25 mg/ml of pyribenzamine; 12.5 and 25 mg/ml of benadryl; and 50 mg/ml each of trimeton and chlortrimeton) and thereby inhibited the secretion of acid stimulated by histamine. However, these authors believed that, strictly speaking, their results were not "antihistaminic" but rather a direct irritant effect on the gastric glands, since 1) these compounds inhibited acid secretion induced by the parasympathomimetic drug, urecholine, as well; and 2) these compounds stimulated flow of a mucoid liquid from the pouches, which was frequently bloody. The concentrations of drug which

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