It might be expected that such pronounced changes in the wide-spread lymphatic system would result in definite and marked changes in the blood leucocyte picture, particularly as regards the lymphocytic elements. Such a change has been demonstrated experimentally in the mouse, rat and dog.[5, 6] Injection of ACTH or adrenal cortical extracts into these forms has resulted in the production of an absolute lymphopenia.

The mechanism of production of this lymphopenia has been of sufficient interest to stimulate the following experimental approach. Single doses of pure ACTH (1–6 mg dose levels) were injected (by subcutaneous and intraperitoneal routes) into normal and adrenalectomized adult female rats of the Long-Evans strain. The hormone (in amounts of 6 mg per cc) was dissolved in a phosphate buffer solution of pH 7.4. To measure the flow of thoracic duct lymphocytes into the blood stream, the common lymph sac, into which empties the thoracic duct in the rat, was cannulated[7] intermittently, before and after injection of the various hormone preparations. The animals were anesthetized by intraperitoneal injection of a 1 per cent. solution of sodium pentobarbital. Leucocyte counts were carried out on thoracic duct lymph specimens at intervals of 30 to 60 minutes for a number of hours preceding and following administration of ACTH.[8]

The results of the experiments obtained to date justify the following conclusions. Administration of ACTH by either the subcutaneous or intraperitoneal route produced a rapid decrease in the number of lymphocytes in the thoracic duct lymph. This reduction occurred within 15 to 30 minutes, was usually more than a 50 per cent. reduction in the pre-existing cell count, and persisted in most rats for the duration of the experiment (4 to 10 hours). Administration of ACTH to the adrenalectomized animal did not produce a comparable effect. Subcutaneous injection of pure growth[9] and lactogenic[10] in similar dose levels failed to imitate the action of ACTH. The buffer solution and the anesthetic agent employed produced no demonstrable effect on the lymphocyte level of the thoracic duct lymph. There were no changes in the rate of flow of the lymph which could account for changes in the lymphocyte level in the thoracic duct.

It is apparent from the results of these experiments that stimulation of the adrenal cortex by the administration of ACTH produces a rapid and persisting fall in the number of lymphocytes entering the blood stream, probably as the result of decreased outpouring of these cells from the thymus and lymph nodes. This change can, it is felt, account in a large measure for the lymphopenia of absolute proportions in the blood stream of the rat following injection of ACTH. Further experiments must clarify the fate of the lymphocyte circulating in the blood stream. A direct experiment to demonstrate the effect of the adrenal cortex on lymphocyte production would consist in the collection of lymph from the isolated lymph node with intact artery and vein in the living animal before and after administration of ACTH or adrenal cortical extract. It will be of further intense interest to determine which of the substances present in the adrenal cortex are responsible for this rather dramatic effect on the lymphocyte level of thoracic duct lymph. The technique of measuring the fall in lymphocytes in thoracic duct lymph may further prove to be a sensitive indicator for the assay of ACTH or adrenal cortical substances.

It may be generalized that the lymphocyte level of the circulating blood is under direct adrenal cortical control. Such a generalization should divert attention to the role of the anterior pituitary and adrenal cortex in various disease states accompanied by hitherto inexplicable relative and absolute lymphopenias or lymphocytosis. To the knowledge of the writers, this is the first direct experimental demonstration using pure anterior pituitary hormones of the control of entry of a white blood cell into the circulating blood stream.

This is a preliminary communication.

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SUSCEPTIBILITY OF HAMSTERS TO CLOSTRIDIUM CHAUVEI

IN experimental work with *Cl. chauvei* where clinical infection is necessary, guinea pigs are generally employed. In comparison to cattle where the basic infection occurs naturally, an immensely larger dose of the infecting agent, on a basis of body weight, is necessary. Berg[1] notes that guinea pigs are comparatively insusceptible to blackleg and quotes Nitta's statement that it required as much culture to kill guinea pigs as calves. He also states that Graub and Zschokke found it took twice as much to kill guinea pigs as calves and sheep.

Considerable work with one strain of *Cl. chauvei* disclosed that 0.3 cc of a 48-hour Hibler culture injected

[1] W. N. Berg., *Jour. Am. Vet. Med. Asn.*, 607–622, 1922.

intramuscularly would uniformly kill adult guinea pigs in 24 to 48 hours. 0.25 cc either failed to kill or was fatal only after 4 or 5 days. Differences in body weight had no apparent effect on these amounts.

Inasmuch as hamsters are finding increasing use as laboratory animals because of their greater susceptibility to many infections, it was decided to compare their susceptibility to that of guinea pigs. Recently weaned pigs were employed so their weight would be closer to that of the young adult hamsters, although the possibility of some passive immunity from the sow would have to be considered. However, within two weeks of weaning their susceptibility has been found identical to that of adults.

TABLE 1

Amount*	Weight	cc Kg	Inoculation route	Results
	GUINEA PIGS			
0.3 cc	200.5 gms	1.49	Axilla	Dead in 24 hrs.
0.25 "	195.5 "	1.27	"	Dead in 48 hrs.
0.2 "	143.5 "	1.39	"	Survived
0.15 "	219.5 "	0.68	"	"
0.5 "	209.5 "	2.38	Orally	"
	HAMSTERS			
0.2 cc	71. gms	2.81	Thigh	Dead in 48 hrs.
0.1 "	55. "	1.81	"	" " " "
0.05 "	49.5 "	1.01	"	" " " "
.01 "	51.5 "	0.19	"	Survived
.001 "	61.5 "	0.01	"	"
0.3 "	50.5 "	5.94	Orally	"

* 48 hr. Hibler culture *Cl. chauvei.*

Table 1 again indicates that the M. L. D. for guinea pigs can not be stated in terms of body weight but exists as an animal unit. Hamsters were found to be more susceptible to *Cl. chauvei* infection than guinea pigs, succumbing to one fifth the lethal dose required for guinea pigs on an animal unit basis.

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THE GERMICIDAL EFFICIENCY OF EMULSEPT AND OF CHLORINE IN WASHING DIRTY EGGS[1]

RESULTS of previous experiments performed in this laboratory on the germicidal efficiency of one of the new types of quaternary ammonium cationic compounds, namely emulsept, have been reported by us.[2] At the same time Zagaevsky and Lutikova published data on the germicidal efficiency of chlorine in disinfecting egg shells.[3] This present paper concerns the comparative values of chlorine and emulsept as germicides in washing dirty eggs.

A. K. Epstein *et al.* have indicated that emulsept, which is the lauric acid ester of colamino formyl methyl pyridinium chloride, has the low toxicity index of 0.6[4] and that the compound, in addition to being a good detergent, is colorless, odorless and non-irritating to the skin.[5]

EXPERIMENTAL METHODS

Eggs used in our experiments were candled out at the receiving station as medium to heavy dirty eggs, the shell surfaces of which were covered with soil, chicken excreta, feathers, straw and egg meat.

Two types of controls were used for later washings with emulsept and chlorine. First, 72 dozen heavy dirty eggs were washed in 6 liters of tap water in 6 groups of 12 dozen each. After washing each 12 dozen eggs the number of organisms present in the wash water was determined by plate counts. Second, from each group, 6 eggs (2 from the top, middle and bottom of the pail) were transferred to 600 ml of sterile water. The jars were rotated rapidly in order to swirl the water over the egg-shell surfaces. This water was then plated, incubated at 30° C. for 3 days and the number of colonies of microorganisms counted. The counts from these plates gave an indication of the number of organisms carried over on each egg shell from the dirty wash water into the rinse water. Seven trials of each series were made for both methods and the results were averaged to be used as controls.

When the eggs were washed with the disinfectants the same procedures were followed with the exception

TABLE 1

COMPARISON OF GERMICIDAL EFFICIENCY OF EMULSEPT AND CHLORINE IN WASHING DIRTY EGGS

Germicide used	Successive groups of 12 dozen eggs each	Percentage of microorganisms not killed by germicide in relation to the number of microorganisms present when no disinfectant was used		Average concentration of germicide remaining
		In rinse water	In wash water	
Emulsept 0.04 per cent.	1	0.26	0.24	*
	2	1.12	0.90	
	3	3.52	3.13	
	4	3.73	6.97	
	5	8.00	33.00	
	6	17.58	36.65	
Chlorine 100 ppm	1	20.28	12.70	4 ppm
	2	13.80	10.54	0.4
	3	40.80	52.45	0.0

* When these experiments were conducted no method had been devised to measure the amount of emulsept remaining in the wash water.

[1] Preliminary report.

[2] V. Penniston and L. R. Hedrick, *U. S. Egg and Poultry Mag.*, 50: 26, 1944.

[3] J. S. Zagaevsky and P. O. Lutikova, *U. S. Egg and Poultry Mag.*, 50: 17, 1944.

[4] A. K. Epstein, B. R. Harris and M. Katzman, *Proc. Soc. Exp. Biol. and Med.*, 53: 238, 1943.

[5] A. K. Epstein, B. R. Harris, M. Katzman and S. Epstein, *Oil and Soap*, 20: 171, 1943.