Table 2. AMMONIA- AND UREA-LEVELS IN BLOOD OF RATS PROTECTED AGAINST LD_{50} DOSES OF AMMONIUM ACETATE BY THE PRIOR ADMINISTRATION OF MIXTURES OF L-ORNITHINE AND L-ASPARTIC ACID

No. of animals	Ammonium acetate	Protective mixture	Dose (m.moles/kgm.)	Blood ammonia (μmoles/ml.)		Plasma urea (μmoles/ml.)	
				Time after injection of ammonium-acetate (min.) 15	60	15	60
50	—	Phosphate buffer		0·76 ± 0·11*	0·76 ± 0·11	5·98 ± 0·73	5·98 ± 0·73
30	—	L-Ornithine + L-Aspartic acid	1·5 1·5	0·96 ± 0·12	0·71 ± 0·11	7·11 ± 0·54	7·81 ± 0·69
50	LD_{50}	Phosphate buffer		6·28 ± 0·33	2·59 ± 0·52	7·45 ± 0·76	9·87 ± 0·76
40	LD_{50}	L-Ornithine + L-Aspartic acid	0·5 0·5	3·77 ± 0·71	1·72 ± 0·72	7·66 ± 0·53	10·42 ± 1·34
50	LD_{50}	L-Ornithine + L-Aspartic acid	1·0 1·0	2·19 ± 0·39	1·39 ± 0·35	9·83 ± 0·90	12·76 ± 1·89
40	LD_{50}	L-Ornithine + L-Aspartic acid	1·5 1·5	2·38 ± 0·34	0·64 ± 0·39	10·53 ± 0·68	14·71 ± 1·81

The experimental conditions were as given in Table 1. Blood samples were drawn by cardiac puncture at the times indicated; the time of 15 min. was selected to coincide with the death of 50 per cent of the intoxicated animals. The survivors of this group were examined along with the other animals after 1 hr. Ammonia was determined in whole blood by Seligson's method (ref. 6); urea was determined in plasma by Kitamura's method (ref. 7).

* Mean values ± standard deviations.

blood ammonia. Their effect may be attributed to an enhancement of the Krebs–Henseleit mechanism of synthesis of urea.

The results of a more detailed investigation in progress will be published elsewhere.

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Chemistry of a Mucopolysaccharide produced by Guinea Pig Lymphocytes

THE guinea pig lymphocyte has long been known to contain a special inclusion body. Such lymphocytes, called Kurloff cells after their discoverer, increase greatly in number in pregnancy and were shown by Ledingham[1] to be produced in both males and females by administration of œstrogens. Marshall and Swettenham[2], by histochemical methods, have shown that the inclusion body is composed of a mucoprotein–sulphated mucopolysaccharide complex. Further work on the chemistry of a mucopolysaccharide fraction by extraction of spleens of œstrogen-treated guinea pigs gave the following results:

(1) No linked amino-acids are present in the polysaccharide fraction; (2) the electrophoretic mobility in phosphate buffer at *p*H 7 on cellulose acetate is the same as chondroitin sulphate; (3) the bulk of the amino-sugar is composed of galactosamine (Gardell's method[3]); (4) the molar ratio of hexosamine, uronic acid and sulphate is the same as chondroitin sulphate *A* or *C*, that is, if hexosamine = 1 (sulphate = 1·29, uronic acid = 1·43 and sodium = 3·43); (5) the material is susceptible to testicular hyaluronidase. Some of the technical methods used in these estimations will be described later in a separate publication.

These results confirm the conclusion of Marshall and Swettenham[2] that the inclusion body of the Kurloff cell contains a material similar to the ground-substance of cartilage or connective tissue. The formation of large amounts of such material in the pregnant animal, however, remains of unknown significance and attempts to trace the fate of this polysaccharide in the pregnant guinea pig by labelling with sulphur-35 were unsuccessful. Furthermore, it is uncertain whether the formation of such material is peculiar to guinea pig lymphocytes or whether it is only the formation of the inclusion body which is unique to this species.

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Influence of Plasma Proteins in the Control of Mitosis-rates in Generating Liver

MANY experiments have been done to elucidate mechanisms of control over growth-rates of liver, and in a comprehensive review of the regenerating liver Harkness[1] suggests that variations in the levels of plasma proteins, as shown by Glinos[2] and Gey[3], offer the most likely cause of changes in mitosis-rates after partial hepatectomy. Lately new evidence in favour of the removal of a normally present inhibitor has been offered from mouse skin[4] and from rat and guinea pig liver[5].

It was shown that adrenal corticosteroids could be the inhibitor normally present[5,6], and I now suggest control of mitoses in at least two ways: First, by a change in concentration of the plasma corticosteroids as shown by several workers[6–8] and to which normal cells are sensitive. Secondly, by a possible alteration of redox potential around each cell which has the effect of preventing corticosteroid influence on mitosis[5]. This effect has been shown by withdrawal of ascorbic acid from guinea pigs, but similar conditions could result from the output of oxidizing substances from damaged cells, such as tumours, or in wound healing where the levels of circulating corticosteroid may be high yet mitosis-rates are not inhibited.

In the experiments of Glinos[2] alterations are made in the concentrations of plasma proteins believed to

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