Injection of DMSO caused an increased concentration in the hind limb lymph of the cytoplasmic enzymes lactic dehydrogenase and glutamic oxalacetic transaminase in cats and rabbits, and an increase in the mitochondrial enzyme glutamic pyruvic transaminase in rabbits. The concentration of the lysosomal enzymes acid phosphatase and cathepsin did not increase in the cat or rabbit lymph. This pattern of intracellular enzymes in the lymph was similar to that produced by a 60°C superficial burn, but the effect on protein concentration was different. After thermal injury, the protein invariably increased together with the lymph flow. But after DMSO, although there was a considerable increase in lymph flow, the protein fell significantly.

Injection of undiluted croton oil caused leakage of all enzymes including those from lysosomes, as well as protein and K^+ in both species. This pattern of enzymes in the lymph indicates a strong injury with cell necrosis and is comparable with that produced when the limb was frozen, causing cell break-down.

In rabbits, 50 and 75% croton oil in corn oil caused a less severe injury; all the enzymes were released, but to a lesser extent. This indicates that, like neat croton oil, the diluted solutions cause cell necrosis but affect a smaller number of cells. This is in contrast to the effect of DMSO, where the enzyme pattern indicated that as many cells were affected as with croton oil, but instead of causing cell necrosis, DMSO caused a change which allowed the escape of selected enzymes.

Histological findings were consistent with this view since, whereas after DMSO there was evidence of only vasodilatation and occasional oedema, there was gross oedema with obvious cell necrosis after croton oil.

REFERENCES

HESS, B. (1963). *Enzymes in Blood Plasma*, p. 7. New York and London: Academic Press.
LEWIS, G. P. (1967). Intracellular enzymes in local lymph as a measure of cellular injury. *J. Physiol., Lond.*, **191**, 591–607.
LEWIS, G. P. (1969). The composition of rabbit hind limb lymph after thermal injury. *J. Physiol., Lond.*, **205**, in the Press.
LEWIS, G. P. & WESTCOTT, BARBARA (1968). Intracellular enzymes in rabbit hind limb lymph after injury. *J. Physiol., Lond.*, **195**, 12–13*P*.

Turnover of pulmonary alveolar wall cells in guinea-pig and mouse after anaphylactic shock.

J. D. HENDERSON and H. SMITH* (introduced by P. GOADBY), *Dept. of Pharmacology, School of Pharmacy, The Polytechnic, Sunderland, Co. Durham.*

Experimentally induced anaphylaxis produces a different response in guinea-pig and mouse. In guinea-pig the critical organ is the lung and acute respiratory distress due to bronchiolar constriction is brought about by release within the lung of histamine, 5-hydroxytryptamine (Sanyal & West, 1958) and slow reacting substance in anaphylaxis (SRS-A) (Brocklehurst, 1953, 1955, 1960). In mouse a generalized systemic shock is observed, thought to be due to 5-hydroxytryptamine, released from enterochromaffin cells of the gastrointestinal tract.

Guinea-pigs surviving severe shock appear to recover within 24 hr, but histological studies have shown that extensive haemorrhage and pulmonary oedema are commonly found in such animals. Areas of irreversible damage consolidate and connective tissue scar formation occurs. The fate of pulmonary alveolar wall cells in areas where

recovery is possible is not known. One purpose of this investigation was to observe the alveolar cell population in these areas after anaphylaxis in the guinea-pig in terms of cytodynamics. The other purpose was to determine if cell turnover was influenced by either local mediators liberated in lung tissue or mediators produced by more generalized systemic shock. Because lung is the critical organ in guinea-pig but not in mouse, a comparison of alveolar cell populations was made between these two species.

TABLE 1. *Comparison of pulmonary alveolar cell turnover in guinea-pig and mouse after anaphylactic shock*

	Labelling index†			Mitotic index‡		
Guinea-pig	Mean	S.E.	*P*	Mean	S.E.	*P*
Control	342	23·8	—	283	27·0	—
Immediate post-shock	303	21·3	<0·30	281	20·3	<0·6
4 hours post-shock	180	16·3	<0·001	196	15·0	<0·01
36 hours post-shock	308	22·8	<0·30	215	18·8	<0·05
Mouse						
Control	484	34·3	—	400	32·3	—
Immediate post-shock	436	30·3	0·6	387	26·3	0·975
4 hours post-shock	248	22·3	<0·001	235	20·8	<0·001
36 hours post-shock	385	27·5	<0·025	280	23·5	0·005

No. of animals per group = 4; no. of sections examined per animal = 4; therefore $N = 16$

†Labelling index = No. of labelled nuclei per 10^5 cells counted in 5 μm section 1 hr after tritiated thymidine.

‡Mitotic index = No. of arrested metaphases per 10^5 nuclei counted in 5 μm section 4 hr after colchicine.

Female guinea-pigs and female mice of the same strains and age were sensitized by the intraperitoneal injection of egg albumin. Guinea-pigs were shocked by an aerosol challenge (Herxheimer, 1951). Mice were shocked by intraperitoneal injections of egg albumin. The labelling index, an index of DNA synthesis and the mitotic index were measured using the autoradiographic and colchicine techniques described by Simnett & Heppleston (1966).

In both species, alveolar cell turnover was depressed in the immediate post-shock period. Maximum depression occurred at 4 hr, with a recovery towards the pre-shock state after 36 hr (Table 1).

REFERENCES

BROCKLEHURST, W. E. (1953). Occurrence of an unidentified substance during anaphylactic shock in cavy lung. *J. Physiol., Lond.*, **120**, 16*P*.

BROCKLEHURST, W. E. (1955). Response of cavy ileum to SRS-A from lung of man and of cavy. *J. Physiol., Lond.*, **128**, 1*P*.

BROCKLEHURST, W. E. (1960). The release of histamine and formation of a slow reacting substance (SRS-A) during anaphylactic shock. *J. Physiol., Lond.*, **151**, 416–435.

HERXHEIMER, H. (1951). Repeatable microshocks of constant strength in guinea-pig anaphylaxis. *J. Physiol., Lond.*, **117**, 251–255.

SANYAL, R. K. & WEST, G. B. (1958). The relationship of histamine and 5-hydroxytryptamine to anaphylactic shock in different species. *J. Physiol., Lond.*, **144**, 525–531.

SIMNETT, J. D. & HEPPLESTON, A. G. (1966). Cell renewal in the mouse lung. *Lab. Invest.*, **15**, 1793–1801.

The effect of drugs on bovine tracheobronchial and pulmonary vascular tissue.

MAUREEN M. AITKEN* and J. SANFORD, *Department of Veterinary Pharmacology, University of Glasgow.*

Strips of trachealis muscle, segments of bronchiole and spiral strips of pulmonary artery were taken from bovine lungs within 15 min of slaughter and were suspended in