Rat intestinal kinin-forming enzyme thus appears to be distinct from trypsin, plasmin, plasma kallikrein and rat pancreatic kallikrein.

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Inhibition by spinal transection of renin release from ischaemic rat kidneys

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The re-establishment of renal blood flow after ischaemia of 4 h duration causes a pressor response in rats anaesthetized with Nembutal (50 mg/kg i.p.). This response is related to the release of renin from the ischaemic kidney and is totally abolished by transection of the spinal cord at any level rostral to thoracic 1 (Hayden & Targett, 1971). These qualitative studies suggested that less renin was released by the ischaemic kidney after spinal transection than by the kidney of an intact preparation.

We have compared the plasma renin activities of arterial blood samples taken before, and 3 min after, re-establishment of the circulation from spinal and intact rats in which the left kidney pedicle had been totally occluded for 4 h (Hayden & Targett, 1971). In the spinal rat preparations the cord was cut between cervical 2 and cervical 3. The mean B.P. of the anaesthetized rats was 80–120 mmHg and 40–60 mmHg in the spinal rats. Plasma renin activity was estimated by the method of McKenzie, Ryan & Lee (1967).

The results of these experiments are given in Table 1.

TABLE 1. *Plasma renin activity of rat arterial blood samples taken before and after re-establishment of blood flow through an ischaemic kidney*

Sample	Plasma renin activity ± S.E.M.†
Before re-establishment of renal blood flow in intact rat	13·4 ± 2·9 (n=11)
3 min after re-establishment of blood flow in intact rat	35·7 ± 4·5 (n=7)
3 min after re-establishment of blood flow in spinal rat	18·6 ± 5·5 (n=8)

†Expressed as ng angiotensin II formed by 0·025 ml plasma incubated at 42° C for 6 h in the presence of an excess of renin substrate.

These results show that a central mechanism is concerned in the release of renin from an ischaemic kidney under these conditions and that the failure of the spinal

preparation to show a pressor response is not solely a result of abolishing the centrally mediated effects of angiotensin II.

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Intracellular enzymes in local lymph during homograft rejection

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The concentrations of some intracellular enzymes as well as that of protein increases in the lymph draining a rabbit hind limb after the limb has been subjected to thermal or chemical injury, but the nature of the enzyme pattern depends upon the degree of cellular injury (Lewis, 1969; Boyles, Lewis & Westcott, 1970). In the present experiments a different kind of injury has been investigated in a similar way—the immunological response to skin homotransplantation.

Rabbits were anaesthetized with pentobarbitone sodium (40 mg/kg) and a cannula inserted into the main femoral lymphatic of the right hind limb as described by Lewis (1969). The operation was carried out under aseptic conditions and the rabbit allowed to recover. Six to eight full thickness skin grafts were made on the right hind limb between the knee and ankle. For autografts the grafts were transplanted to the opposite side of the right limb of the same animal; for homografts they were exchanged between Norfolk and New Zealand White rabbits.

In the supernatant of the lymph obtained after centrifugation, the activities of four of the six enzymes examined—cathepsin, acid phosphatase, glutamic pyruvic transaminase (GPT), and glutamic oxalacetic transaminase (GOP) increased during the first 5 days after the grafting of autografts or homografts. These increases probably result from the non-specific injury of transplantation. With autografts the increases then subsided, but with homografts they increased further during, and immediately after, rejection when there was an even greater increase in the activities of the other two enzymes examined—LDH and β-glucuronidase. In the cell pellets obtained after centrifugation of the lymph the activities of the six enzymes did not increase during the first 5 days although subsequently they all increased with homografts but not with autografts. Since there were no concomitant increases in the lymphocyte counts, some of the lymphocytes must have become activated during the time leading up to rejection in order to contain higher enzyme activities. It has already been shown that a lymphatic connexion between the graft itself and the host tissue is not a prerequisite of homograft rejection and that such a connexion is not usually established under the present experimental conditions (Jasani & Lewis, 1970). It is concluded, therefore, that the increases in enzyme activities of the lymph collected during and after rejection result from 'activated lymphocytes' which infiltrate the graft bed and functional tissue and subsequently undergo necrosis.