tion of potassium in the mechanism generating the cholinergic IPSP and ACh potential is minimal, so that a fall in potassium permeability after administration of copper sulphate cannot by itself explain the present results. Furthermore, the absence of action of copper sulphate on neurones with non-cholinergic IPSPs which depend on potassium excludes an effect on potassium permeability. It may be then concluded that, as in the case of frog skin, copper sulphate in dilute concentrations markedly reduces the permeability of the neuronal membrane to chloride. The effect seems to be similar for the fluxes of chloride in both directions, because the hyperpolarizing and the depolarizing effects of ACh are reduced in the same proportion. This indicates that the effect of copper sulphate is not achieved through a modification of the chloride pump which probably exists in these cells[2].

According to the Goldman–Hodgkin–Katz equation, copper sulphate should produce an increase in the resting potential by reducing the permeability to chloride. The fact that copper sulphate produces a diminution of the resting potential is rather puzzling, but further work is needed to elucidate this point.

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Alterations in the Urinary Excretion of Pseudouridine and Deoxycytidine in Rats induced by Glucocorticoids

One of the characteristics of corticoid action[1–5] is a marked catabolic effect on the lymphatic organs, such as thymus and spleen. This is demonstrated by the loss in weight of these organs[5,6] as well as by the decrease in the nucleic acid content[7], and by the diminution of incorporation of the precursors of nucleic acids after administration of glucocorticoids[8].

Degradation of nucleic acids is accompanied by an increased excretion of their catabolic products (pseudouridine and deoxycytidine) in urine[9–11]; this has been proved by the effect of ionizing radiation on the organism. The object of the present work was to ascertain whether the catabolic effect of corticoids on the nucleic acids in the spleen results in a similar degradation pathway.

Infantile male rats of the Wistar-Konárovice strain, weighing 55–65 g, in groups of five, were kept in metabolic cages and fed on a standard diet. Urine was collected every 24 h. Pseudouridine and deoxycytidine were determined in the urine using a method previously described[10]. Splenectomy was performed under ether anaesthesia 4 days before the administration of cortisol. In both splenectomized and normal animals cortisol was given in a dose of 2 mg/100 g of body weight for 3 consecutive days.

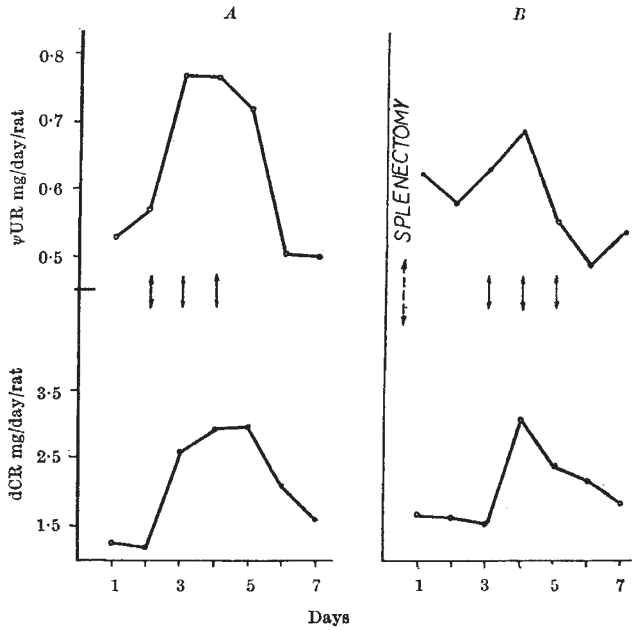

Fig. 1. The concentration of deoxycytidine (dCR) and pseudouridine (ψUR) in the urine of eusplenic (*A*) and splenectomized (*B*) rats after administration of cortisol.

In normal rats cortisol produced a marked increase in the excretion of both catabolites 24 h after the first administration. This increase was maintained after the repeated administration of the steroid but ceased rapidly after the last dose (Fig. 1*A*). In splenectomized animals the course of the urinary excretion of these catabolites differed considerably after the administration of cortisol, in comparison with the findings in the normal animals. In this experiment the increase in their content occurred after the first dose only; repeated administration did not produce any effect (Fig. 1*B*).

On the basis of these experiments it may be assumed that the mode of catabolic degradation of nucleic acids is similar after both ionizing radiation[9–11] and glucocorticoid action in that in both cases degradation of nucleic acids takes place predominantly in the spleen.

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