The exact role of the hypothalamus in the control of production of growth hormone and of corticotrophin (A.C.T.H.) is still obscure. Information derived from the effects of lesions in experimental animals and from the effects of tumours in man is equivocal. For example, Spirtos (1954) found that moderate-sized lesions of the hypothalamus (area unspecified) were followed by increased insulin sensitivity which could be countered by injections of growth hormone. Paton and Petch (1954) reported hyperglycaemia, a diabetic type of glucose-tolerance curve, and reduced sensitivity to insulin in a human subject in whom a brain tumour involving the hypothalamus (area unspecified) was discovered at necropsy. It would be valueless to attempt to explain the present findings in terms of hypothalamic function when such a conflict of evidence exists.

The interest of the present experiments lies in the fact that insulin resistance was produced in rats without destruction of pancreatic islet tissue and without the continued injection of hormones. Moreover, no direct damage was caused to the cell masses of the hypothalamus by the division of the fibre tracts leading from the hypothalamus to the brain stem. Late chromolytic changes were seen in certain cell masses which could with certainty be attributed to the section of the efferent fibres from them. Neither of the two operative procedures used in the production of insulin resistance had any demonstrable effect on insulin sensitivity. The insulin resistance which followed when both operations were done on the same rats was present 21 days after infliction of the brain lesions and 14 days after removal of the adrenal medulla. It is therefore improbable that the insulin resistance could be due to some irritative action set up by the operative procedures.

If the insulin resistance induced in these rats were due to the presence of an insulin antagonist—and the evidence is consistent with this assumption—then the possibility that insulin-resistant diabetes might arise from a cause or causes which do not involve primarily either the pancreas or the pituitary gland must receive serious consideration.

Summary

After section of the pathways from the hypothalamus to the brain stem and removal of the adrenal medulla, rats kept at an environmental temperature of 29.5° C. showed no fall in the blood-glucose level 30 minutes after the injection of insulin in a dose of 1 unit per kg. body weight. Removal of the adrenal medulla alone or section of the hypothalamic pathways produced no change in insulin sensitivity.

The resting content of glycogen in muscle (gastrocnemius) was significantly lower than normal in rats with the hypothalamic lesions and without adrenal medullary tissue. The glycogen content did not rise after the injection of glucose (1 mg. per g. body weight) or after the simultaneous injection of glucose and insulin. The glycogen content of the liver was markedly reduced (15% of normal) in the resting state. When such rats were exposed to cold for nine days they still displayed no hypoglycaemic response to the injection of insulin. The glycogen content of the liver in the resting state was reduced to 11 mg. per 100 g. of liver.

The experimental data suggest that the insulin resistance found in these rats was due to the presence of an insulin antagonist.

It is a pleasure to thank those who have made this work possible. Dr. Honor B. Fell placed laboratory facilities at my disposal, Mr. F. G. Moore and Mr. H. N. Smith made it possible to acquire most of the necessary equipment, and the Medical Research Council met part of the cost of technical assistance. I am indebted to Mr. R. D. Chambers for carrying out the major portion of the technical work.

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THE OCCURRENCE OF ELECTROCORTIN IN HUMAN URINE

BY

C. L. COPE, D.M., F.R.C.P.

AND

J. GARCIA-LLAURADO, M.D.

British Council Scholar

(From the Department of Medicine, Postgraduate Medical School of London, W.12)

The discovery by Grundy, Simpson, and Tait (1952) of a new hormone, called electrocortin,* which has the ability to increase both sodium retention and potassium excretion by the kidney, must inevitably give a new impetus to the study of the many conditions associated with sodium retention in man.

In recent years several workers have found evidence of the occurrence in certain human urines of a substance having the power to increase sodium retention by the kidney; but careful analysis of extracts of such urines by modern chromatographic methods has failed to reveal any evidence of the presence of deoxycortone, the only steroid hitherto known to possess such sodium-retaining properties in marked degree. It now seems likely that the sodium-retaining effects observed by earlier investigators were due to the presence in the extracts of this new hormone, electrocortin.

Most of the original observations suggesting the presence of a sodium-retaining principle in urine were made by Deming and Luetscher (1950). Using a method of bioassay on young adrenalectomized rats, they found that chloroform extracts of some human urines had the power, when injected, of reducing sodium excretion by the kidney. Under the conditions of their test deoxycortone acetate in doses of 5 to 25 µg. per rat had a similar effect, but larger doses of extract did not give greater sodium retention. In their earlier experiments evidence for the existence of a sodium-retaining substance in normal urine was equivocal, but a significant degree of activity was found in extracts of the urine of five out of eight patients with cardiac failure. In one patient with nephrosis a high titre of activity was also found. An extension of these observations was reported by Luetscher *et al.* (1952), who then found that the titre of sodium-retaining substance was high in the oedematous stage but fell to normal levels after diuresis with elimination of the oedema.

In 1953 Singer and Venning also found some evidence of sodium-retaining activity in extracts of nephrotic urine, and McCall and Singer (1953) found a sodium-retaining substance in eight out of nine nephrotic chil-

*The chemical formula of electrocortin has just been published by Simpson, Tait, Wettstein, *et al.* (*Experientia*, 1954, **10**, 132). It proves to be a steroid similar to corticosterone, but with an aldehyde grouping on C 18.

dren when they were gaining weight, though it was present in only one out of six when weight was stationary and the patients were no longer retaining sodium.

Chart and Shipley (1953) have claimed to detect a sodium-retaining factor excreted by normal adult males, the daily amount being equivalent in activity to 40 to 80 μg. of deoxycortone. They found that patients with hepatic cirrhosis and ascites showed a high excretion equivalent to as much as 3,162 μg. daily, although when ascites was absent excretion was normal. These workers also obtained some evidence that the active principle differed from deoxycortone itself, for they noted that it entered the aqueous phase when partitioned between benzene and water, whereas deoxycortone itself tends to enter the benzene phase freely. They suggested that it is probably associated with the so-called amorphous fraction of the adrenal cortical extract.

In 1951 Zaffaroni and Burton made a careful analysis of the steroids in adrenal cortical extract, employing their own particularly suitable and sensitive methods of paper-partition chromatography. They were able to find small amounts of deoxycortone in aqueous extracts of adrenal cortex, but could not find any trace of the hormone in chloroform extracts of human urine made at *p*H 1. Since a large part of the cortisone and hydrocortisone (compound F) content of such urines is extracted by chloroform at this *p*H it seems likely that any deoxycortone present would also be extracted in this way.

In 1952 Grundy *et al.* first published their evidence for the existence of a hitherto unrecognized hormone in the adrenal cortex causing sodium retention by the kidney. They were able to show that the new hormone was quite distinct from deoxycortone, for there was a great difference in the rates at which the two substances ran on paper chromatograms. In the toluene/propyleneglycol system of Burton, Zaffaroni, and Keutmann (1951) it ran at a rate almost exactly the same as cortisone, whereas deoxycortone ran very much faster. The activity in the cortisone zone was much greater than could be accounted for by the activity of the small amount of cortisone actually present there.

Simpson and Tait (1953) have reviewed the further intensive work they have done on this new hormone. They find that it is a reducing steroid having from 50 to 90 times the activity of deoxycortone on an equimolecular basis. The culmination of this work has been the isolation of the new hormone in the form of crystals by Simpson, Tait, Wettstein *et al.* (1953). Desaulles *et al.* (1953) have tested this purified preparation and found it to be 25 times as potent as deoxycortone on sodium retention and five times as active in increasing potassium excretion. An independent account of the properties and isolation of this hormone has been published by Mattox *et al.* (1953).

In the work presented here we have employed the assay procedure developed by Simpson and Tait for electrocortin activity to look for this hormone in human urine extracts. Our results suggest the presence of a small amount in normal urines. In addition a patient has been found who was shown on several occasions to be excreting large quantities of a substance having the special properties of electrocortin but who showed no evidence of sodium retention.

Methods

The rats used in these experiments were young of an albino strain bred in the department and weighing 60 to 80 g. Bilateral adrenalectomy was performed by the dorsal approach under ether anaesthesia. Only animals whose adrenals were removed unbroken were used. After operation the animals were fed a stock diet of "research" rat cubes and drank unrestricted 1% saline solution. Assays were carried out on the fourth day after operation. Each group of test animals consisted of five to seven rats and a control group of the same size was used on each day of testing. In preliminary experiments it was observed that variations between control groups from one week to another were greater than the differences between males and females. For this reason both male and female rats have been used in these experiments, though all animals in any one assay were of the same sex. Control and test animals were mixed in the cages to avoid variations due to the order in which the animals were handled.

In the earlier experiments survival time after withdrawal of saline was determined as evidence of completeness of adrenalectomy, but survival, implying an incomplete adrenalectomy, was so rarely observed that this was finally abandoned as a routine. The rats were allowed to continue drinking saline until the morning of the test day.

For each test four groups of five to seven rats were taken. The first group served as controls, the second group all received 10 μg. of deoxycortone, and the third and fourth groups were injected with different quantities of the extract to be tested. All injections were made up to a volume of 0.1 ml., the solvent being 20% ethanol in water. The control group received solvent alone. The injections were made at about 10 a.m. on the morning of the test. One hour later the bladders of all rats were emptied by suprapubic pressure combined with a light inhalation of ether. After this, a subcutaneous injection of 1 ml. of a mixture containing 73 μg. of Na^{24} and 295 μg. of K^{42} was made, the total radioactivity of the injected dose at the time of assay being about 60 μC. Immediately after injection each rat was placed in a separate 500-ml. beaker fitted with a raised wire mesh. All beakers had previously been cleaned with chromic acid and distilled water.

Three hours after the injection the rats were made to urinate by the method described above, all urine being collected in the beaker. Although every effort was made to get complete emptying of the bladder, this was not essential to the accuracy of the test. The beakers and wire meshes were carefully rinsed with 5 ml. of distilled water projected against the walls by means of a syringe and needle. The washing fluid was collected in a small bottle. The ratio between Na^{24} and K^{42} concentration was determined in each sample by a method essentially similar to that of Tait and Williams (1952) and Simpson and Tait (1952) which involves measurement of the intensity of radiation with and without a thin aluminium screen.

Some modifications of the detail of the method were made. Only 0.2 ml. of the diluted urine sample was used for the radioactivity measurement, since this gave a sufficient number of counts a minute, from 5,000 to 10,000. This 0.2-ml. sample was placed on a disk of thin filter paper fitted into a nickel planchette 25 mm. in diameter. With the uniform distribution of this fluid thus obtained the radioactivity count could be made without drying the sample. Direct comparison showed that there was no significant difference between this wet method of counting and the method with dried samples used by Simpson and Tait. Counts were made on each sample, both with and without a filter screen composed of six sheets of 5/1000 in. (0.13 mm.) aluminium, and using a Geiger–Müller tube, G.E.C. type Z B 7, vw. 1400.

From the ratio of unfiltered to filtered counts the relative proportions of Na^{24} and of K^{42} in the sample could be derived by reference to a calibration curve constructed from mixtures of the two isotopes in known proportions. The degree of activity of the sodium-retaining factor in the extract under test was indicated by the fall in the ratio of Na^{24} to K^{42} in the urine sample excreted after the injection,

this ratio being expressed as a percentage of the mean figure obtained for the control group on the same day.

The principle of measuring the Na/K ratio rather than the actual excretion rate of either sodium or potassium alone was introduced by Simpson and Tait, and has several important advantages. It eliminates the need to collect and measure accurately the whole urine sample excreted by each rat. To achieve this reliably Singer and Venning had to ligate the urethras of all their rats and sacrifice them to obtain the bladder urine. When only the ratio is required completeness of collection is much less important. The rats were not sacrificed in our assays, but were used again two days later for similar assays, using another method with Na^{23} and K^{39} with subsequent estimation of the Na/K ratio by means of the flame photometer. In this paper only the results obtained by the isotope method are considered. A comparative study of the results obtained by the isotope and two other methods will be published elsewhere.

Preparation of Urinary Extracts

Owing to our uncertainty about the stability of the sodium-retaining principle, the extraction procedure was deliberately made as simple as possible to minimize the risk of destruction. Twenty-four-hour samples of urine were collected without preservative. These were extracted by manual shaking with 15 volumes per 100 ml. of urine, repeated four times with fresh lots of chloroform. No acidification of the urine was done before extraction, the *p*H usually being between 6 and 7. The chloroform extract was evaporated to dryness under reduced pressure, using a water bath at 50° C. Suitable aliquots were obtained by dissolving the residue in a small known volume of fresh chloroform, dividing this into small tubes, and blowing off the chloroform with a stream of nitrogen. For injection into rats the residue was dissolved in 20% ethanol in water, the volume being so chosen that the dose to be injected into each rat was contained in 0.1 ml. solution.

All extracts were stored dry until the day of assay.

Results

Under the conditions of our experiments a linear relationship was found between the Na^{24}/K^{42} ratio, expressed as a percentage of the mean figure for the control group, and the logarithm of the dose of deoxycortone injected. This relationship was found over the range of dose from 5 to 20 μg. per rat, and is shown in Fig. 1. Below 5 μg. per rat a linear relationship was not found, the ratio rising above the control level. In this respect our observations confirm the finding of Forsyth (1953) that very small doses of deoxycortone may actually have a sodium-excreting effect. Because of this excreting effect activities equivalent to amounts of deoxycortone less than 5 μg. could not be detected. It was considered desirable, therefore, to test all extracts at two dosages in order to increase the likelihood of obtaining an optimum content of the sodium-retaining substance. In this regard it is of interest that previous workers have usually failed to establish any quantitative relationship between size of dose and degree of response. Thus Luetscher *et al.* (1952) had found that increases in dose much above the equivalent of 20 minutes' urine output by the patient did not regularly yield a proportionate increase in response. Singer and Venning have also found sodium-retaining activity in the urine from some patients with nephrosis but had failed to get a bigger reponse with a larger dose of extract.

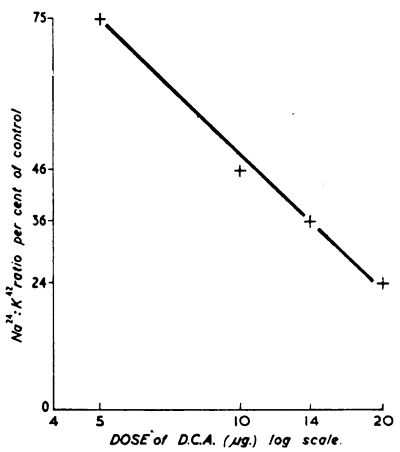

FIG. 1.—Effect of deoxycortone acetate. Linear relation between log. dose and percentage drop in Na^{24}/K^{42} ratio.

Five normal subjects have therefore been studied by injection of half-hour equivalent and of three-hour equivalent of urine extract into separate groups of six test rats. A sixth normal subject was also studied at the half-hour dose only. In four of the six subjects tested at the half-hour dose the ratio was depressed to 80% or less of the control level. An elevation of the ratio occurred in the other two (Fig. 2). This rise could have been due to a sodium-excreting effect of a very small dose of the active principle present in the extracts and comparable to the sodium-excreting effect of small doses of deoxycortone observed both by Forsyth and by ourselves. Support for this interpretation is found in the fact that both these extracts produced effective and significant depression of the ratio when given in the three-hour dose. With the other three extracts tested at both dose levels no significantly greater activity was observed at the higher dose than at the lower. Our experience of these extracts is thus the same as that of Luetscher *et al.* (1952) and of Singer and Venning (1953).

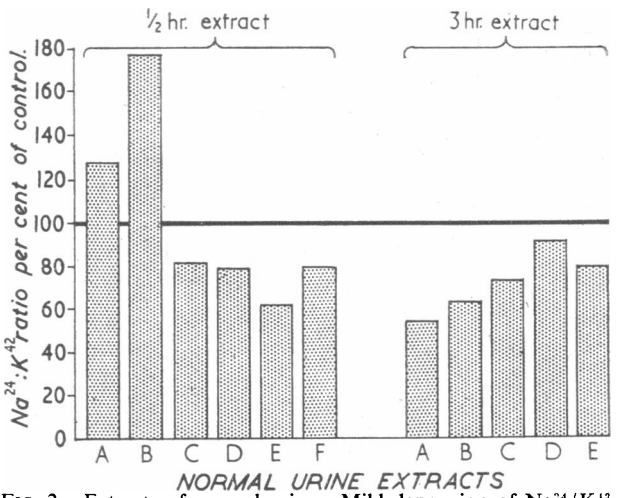

FIG. 2.—Extracts of normal urine. Mild depression of Na^{24}/K^{42} ratio.

Since the Na^{24}/K^{42} ratio can readily be depressed to a much lower figure by a sufficient dose of deoxycortone it would seem that the active principle whose effect is being observed in normal urine has very different pharmacological properties from deoxycortone itself. The results thus indicate the frequent presence of a sodium-retaining factor in chloroform extracts of normal urine, but they do not provide enough evidence on which to base an estimate of the amount present in terms of deoxycortone activity. Thus by reference to the deoxycortone calibration curve in Fig. 1, extracts C and E have respectively 4.5 and 6.5 μg. equivalents per half-hour sample. These figures correspond to 24-hour outputs of 216 and 312 μg. of activity expressed in terms of deoxycortone. On the basis of the three-hour assay, however, the same extracts showed activity indicating a daily output of active substance equivalent to only 40 and 35 μg. of deoxycortone respectively. Under such circumstances the assays are of little more than qualitative value.

They do serve, however, to show that such urinary extracts contain a principle which qualitatively alters the electrolyte excretion by the kidney. Methods of assay hitherto used have measured the effect on the rate of excretion of sodium only. Any factor in the extracts which may be toxic to the kidney and may cause a temporary impairment of renal function is likely to give a reduction in sodium excretion and thus, by a non-specific reaction, to suggest the presence of a specific sodium-

retaining factor. Any temporary impairment of renal function which may result from injection of the extracts is much less likely to affect the ratio of sodium to potassium. Changes in this ratio may be regarded, therefore, as a more reliable indication of the presence of a specific factor than will reduction in the rate of sodium excretion alone.

These results on normal urines may be contrasted with comparable assays on four patients suffering from rheumatoid arthritis. Extracts of the urine from these patients gave no indication of the presence of a sodium-retaining factor at either the half-hour or the three-hour dose. In Fig. 3 each column represents the mean effect on batches of five rats for each extract as compared with control groups of the same size.

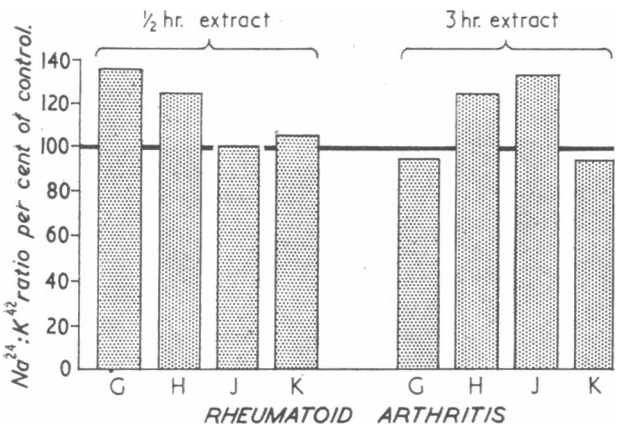

FIG. 3.—Extracts of urine from patients with rheumatoid arthritis. Failure to influence Na^{24}/K^{42} ratio.

In three patients suffering from congestive cardiac failure with gross oedema, results were less consistent. Subject P, with recurring ascites associated with severe tricuspid incompetence, showed no activity at two- or three-hour dosage. Subject F, losing oedema under mersalyl therapy, showed no activity at either half-hour or three-hour dose. On the other hand, subject M, with chronic congestive failure not improving under treatment, showed considerable sodium-retaining activity at the three-hour dose on two occasions.

These results on cardiac subjects merely confirm the findings of earlier workers that a sodium-retaining factor is present in human urine at certain times, but they do not assist in the prediction of the conditions under which it is likely to appear. Under such circumstances it is difficult to define more closely the properties of this elusive principle and to determine whether these properties coincide with those of electrocortin.

We have made several attempts to increase the output of this principle in urine to more manageable levels. The maintenance of a normal subject for a week on a diet containing less than 0.5 g. of sodium daily did not result in any detectable amount of the sodium-retaining factor in a subsequent urine extract. Similarly, the urine of a patient maintained on large doses of A.C.T.H., which was shown to have provoked a large increase in hydrocortisone output, did not show any significant activity. Thus neither of these procedures seems to lead to an increased output of the sodium-retaining factor (Fig. 4).

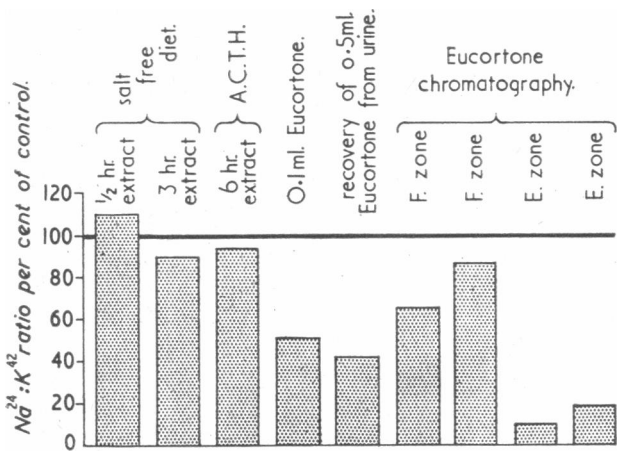

FIG. 4.—Effect of various extracts on Na^{24}/K^{42} ratio (see text). Electrocortin in "eucortone."

Detection of Electrocortin

Electrocortin can be distinguished clearly from deoxycortone by the rate of its flow on paper chromatograms. When the toluene/propylene-glycol solvent system is used electrocortin flows at almost the same rate as cortisone. While several other steroids have similar rates of flow in the chromatogram, it is very improbable that any other will combine a similar rate of flow on paper with a high degree of sodium-retaining activity. The demonstration of both these properties in an extract will thus establish the presence of electrocortin with a high degree of probability.

The most easily available source of electrocortin at present is commercial adrenal cortical extract. Using the Burroughs Wellcome preparation "eucortone," we were readily able to show that it contained strong sodium-retaining activity. When eucortone was added to urine samples and chloroform extracts were then made in the usual manner, these extracts also had strong sodium-retaining activity in our test. This activity ran in the cortisone zone. Though quantitative conclusions could not be reached it was clear that when electrocortin was added to urine a large proportion was recovered by the extraction procedure we were using (Fig. 4).

The chance discovery of a patient who proved to be excreting continually large amounts of the sodium-retaining substance in her urine has luckily enabled us to establish the approximate rate of flow of the principle on paper chromatograms and to show that this is the same as electrocortin.

The subject, Mrs. A, was a West African negress, aged 41, who was under investigation by Dr. M. D. Milne because of recurrent attacks of severe muscle weakness, which were found to be due to low serum potassium. Balance experiments conducted by Dr. Milne showed that she suffered from a very severe potassium-losing condition, apparently due to a renal tubular anomaly which was associated with a chronic pyelonephritis. Blood urea and pyelogram were normal; albuminuria varied between 1 and 3 g. per litre. At no time was there any oedema or other clinical evidence of sodium retention. Careful balance experiments showed that she was neither retaining nor losing sodium at the time of study. The full clinical history of this case with extensive electrolyte investigations is being published (Milne, 1954).

Three separate urine collections from this patient were tested and all showed strong sodium-retaining activity—much greater than we had encountered in any other subject. Depressions of the Na^{24}/K^{42} ratio with both half-hour and three-hour doses of extract are shown in Fig. 5. One of these highly active extracts, analysed for cortisone and hydrocortisone content by the chromatographic method described by Cope and Hurlock (1953), was found to contain 57 μg. of cortisone and 11 μg. of hydrocortisone a day. Thus a three-hour aliquot of this extract contained about 8 μg. of the combined hormones, a quantity much too small to influence the Na^{24}/K^{42} ratio in the tests. As may be seen from Fig. 5, an injection of 30 μg. of cortisone acetate per rat produced no detectable alteration in the ratio.

A sample of this extract equivalent to 48 hours' urine output was submitted to paper partition chromatography, using the toluene/propylene-glycol system. After a run of 72 hours most components of the extract had run off the bottom of the chromatogram, there being retained on the paper only those as polar as, or more polar than,

cortisone. Two zones were cut out from the chromatogram, one corresponding to the cortisone level (E) and the other to that of hydrocortisone (F). Both were separately eluted from the paper with methyl alcohol. The eluted fractions after evaporation of the methyl alcohol were then tested in the usual manner. The cortisone fraction showed a strong sodium-retaining activity, whereas no activity

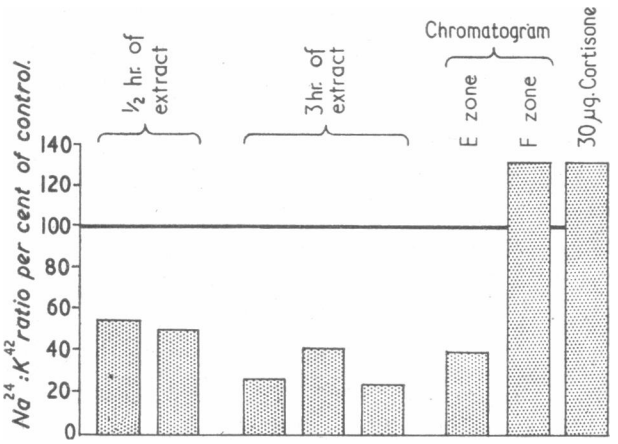

FIG. 5.—Strong electrocortin-like effect of urine extracts from Mrs. A. Activity confined to cortisone zone in the paper chromatogram.

was found in the hydrocortisone zone (Fig. 5). Thus the activity could be regarded as being due to electrocortin with a high degree of probability, the likelihood of any other substance combining a similar biological action with the same rate of flow on the chromatogram being extremely small.

Conclusions

The results obtained on normal urine extracts suggest the presence of small quantities of a substance having electrocortin-like properties, but they do not provide enough evidence on which to base any reliable estimate of the quantity present.

In contrast four urines from patients with rheumatoid arthritis all failed to show the presence of such a substance. It would be unwise to draw any conclusions from these relatively few cases, but, in view of the suggestions of Selye (1950) that disturbance of mineral corticoid production may play a part in the aetiology of rheumatoid arthritis, it is clearly of importance to determine whether such an absence of electrocortin from the urine is a regular feature of rheumatoid arthritis.

From the finding of activity in one of three patients with congestive cardiac failure examined no conclusions can be drawn. Although a high and maintained output of electrocortin by Mrs. A was demonstrated, it cannot be claimed that this throws any light on the nature of the peculiar renal and metabolic defect she was found to have. It is of great interest that in this patient there was ample proof that no sodium retention was taking place, and it must be concluded, therefore, that a high titre of electrocortin may occur in the urine in the absence of any sodium retention. Previous workers have claimed to find evidence of a sodium-retaining substance most readily in patients who were actively retaining sodium at the time. The fact that this patient was losing large amounts of potassium may, however, be of significance. It is at present quite unknown what are the relations between the level of steroid in the blood and the rate of excretion in the urine. Indirect evidence from the behaviour of hydrocortisone and allied steroids suggests that a rise in blood concentration is followed by a prompt increase in excretion. It is natural to assume, therefore, that a high output of electrocortin in the urine indicates a high concentration of this hormone in the blood, since it also is probably a very similar steroid. It is possible, however, that electrocortin may be bound to protein or even to potassium salts, so that its excretion rate is determined more by permeability changes in the kidney or by potassium output than it is by the concentration in the blood. In the present state of our knowledge of this hormone, however, any such suggestion must be purely speculative.

While this work was being prepared for publication a report appeared stating that Luetscher and Johnson (1954) have also been able to show that the sodium-retaining substance they find in the urine of nephrotic subjects runs on the paper chromatogram at a rate similar to that of cortisone. This substance also is therefore presumably electrocortin. Much work remains to be done before the significance of a high excretion of this new hormone by the human subject can be properly judged.

Summary

A substance having the power to increase sodium retention by the kidney has been found in large amount in the urine of a woman suffering from a severe potassium-losing condition. Other properties of this substance suggest that it is the adrenal hormone electrocortin.

Indications of the presence of a similar active substance in small amount in normal human urine have been found. No such active principle could be detected in four patients suffering from rheumatoid arthritis.

Our thanks are due to Dr. J. E. F. Bradley and D. G. Arnott for advice on the handling of isotopes, and to Miss Mary Standring for much technical aid.

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A medical school will form part of the Rhodesia University College at Salisbury, Southern Rhodesia, plans for which are now rapidly maturing. Last autumn the college's inaugural board visited London and discussed the college's foundation and constitution with H.M. Government, the Inter-University Council, and London University. As a result a capital grant of £1,250,000 towards the cost of its building and equipment has been promised from Colonial Development and Welfare funds, subject to the balance of the total capital cost (estimated at £2,000,000) and the annual maintenance costs (over £250,000) being found locally. The city of Salisbury has provided 458 acres as a site for the college. Land will be available near the new Salisbury Native Hospital for a medical school, and about 1,000 acres for an experimental farm. The first faculties to be established will be those of arts and science, and after that faculties of agriculture and medicine. The college will provide education for London University degrees and will be open to students of all races without discrimination. It is hoped that the college will be ready for teaching to begin early in 1956.