the presence of the toxic substance in it was confirmed by feeding tests.

Later it was found that the unknown spot, which is shown up by the ninhydrin reaction, is accompanied by another, which does not give the ninhydrin reaction, but is detectable in ultra-violet light. As an alternative to the chromatographic separation, which is extremely laborious on a large scale, it was found that the toxic substance could be precipitated by mercuric acetate, added in 25 per cent alcohol solution acidified with acetic acid. The precipitated mercury salt was suspended in water, and hydrogen sulphide was passed to precipitate the mercury. The solution was evaporated to dryness. About 20 mgm. nitrogen of the substance obtained was required to give the toxic symptoms in a ferret.

On chromatographic examination it was found that the spot which we believed to be that of the toxic substance was not now accompanied by the fluorescent material. The product has, however, varied somewhat in different preparations, probably owing to an uneven efficiency of the separations, which are not easily controlled. In some cases the two-dimensional chromatogram shows only the one spot; in others, it has been accompanied by others representing histidine, glutamine, asparagine, valine and leucine. Traces of cysteine have also been detected in some preparations. It was later found that in some of the toxic preparations all the chromatographic spots could be accounted for, so that it is likely that the toxic substance, although closely associated with glutamine, does not itself give a ninhydrin colour. This is confirmed by the fact that a similar product from a non-toxic flour also gives the same spots. A further purification on the same scale is being attempted.

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Influence of Desoxycorticosterone on Glycogen Formation and Glucose Uptake of Isolated Muscle

Isolated diaphragm muscle of rats produces glycogen from glucose especially in the presence of insulin in oxygenated Ringer's solution. This is inhibited by the addition of small doses of desoxycorticosterone[1]. If the muscle works in response to rhythmic stimulation for two hours, glycogen is newly formed and is broken down again. This is also inhibited by desoxycorticosterone[2]. The action of other steroid hormones was also studied. They had either equal effect (progesterone, testosterone) or none (corticosterone, etc.)[3].

We have now studied whether desoxycorticosterone also inhibits the glucose uptake[4] of the surviving isolated muscle.

The diaphragm of normal rats was cut into three pieces immediately after death. One served for the estimation of the starting value of glycogen content. The two other parts were each put in a vessel with 2 c.c. of Ringer's solution with 200 mgm. glucose and 1 unit of insulin per 100 c.c. The vessels were shaken in an oxygen atmosphere at 37° C. for 90 min. To one vessel 0·1 mgm. desoxycorticosterone was added (5 mgm. per 100 c.c.).

Thirty rat diaphragms were tested, and the results are given in mean values with the median error ($\sqrt{\Delta^2/n(n-1)}$). Besides the glycogen formation, the glucose content of the solution was also measured at the beginning and end of the experiment. The methods used were the same as before[1-3].

	Mean values		
	Glycogen production (30 experiments)	Glucose uptake	NH_3 produced (13 experiments)
Per 100 gm. muscle in 90 min.	272±37 mgm.	457±24 mgm.	8·0±0·6 mgm.
Same, with *addition of* 4·4, 5 or 7 mgm. per 100 c.c. desoxycorticosterone	56± 5 mgm.	200±20 mgm.	9·0±1·3 mgm.

The surviving muscle about doubled its glycogen content in this period, as in former experiments[1]. It took up glucose from the solution, but considerably more glucose disappeared than corresponds to the newly formed glycogen[4]. This occurred in each single experiment and is shown in the mean value, where 272 mgm. glycogen production compares with 457 mgm. glucose consumption.

Desoxycorticosterone totally inhibited the glycogen production; but it decreased the glucose uptake only to about one half. This value seems to correspond to the difference between the glycogen produced and the total glucose consumption of the muscle without addition of desoxycorticosterone. It might be, therefore, that the remaining glucose is metabolized, and that desoxycorticosterone inhibited only glycogen formation. However, it may also be that primarily the permeability for glucose is decreased, and this accounts for the decrease in glucose uptake and in glycogen formation. We intend to study this problem further.

In thirteen experiments, the ammonia production of the surviving muscle was estimated in the solution, using Conway's method[5]. It seemed possible that after the inhibition of glycogen formation, the muscle increased protein breakdown through desamination. No increase of ammonia production was found.

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