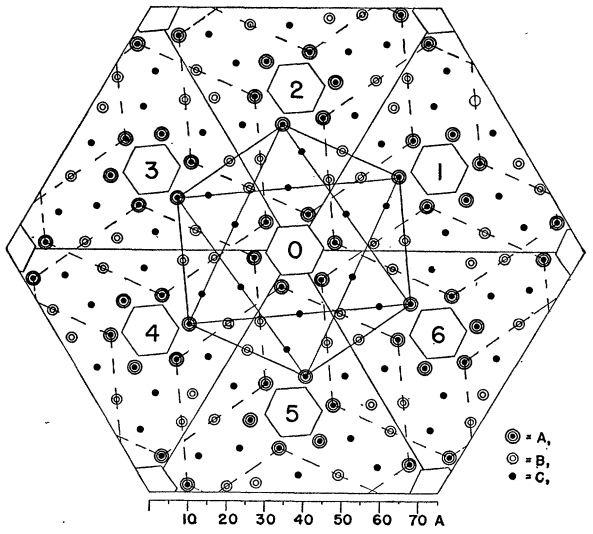

points of the C_2 we obtain Patterson-Harker peaks for a molecule at 0 which lie at the corners and midpoints of the sides of the octahedron $2l$ with center at 0. The figure shows the projection on the c-plane of these 18 peaks giving a hexagon with center at 0, with side length 33.9 A. There are six at its corners, six at the midpoints of its sides and six at the midpoints of lines joining alternate corners; indicated for convenience as A, B and C, respectively.

We now notice that Crowfoot's c-plane projection also gives 18 peaks per molecule, reproduced in Fig. 1, which fall into a pleasing pattern of hexad, triad and dyad sets. Superposing the C_2 hexagon on this diagram, we turn this hexagon about its center, through an increasing angle α until any of its points falls upon a Crowfoot peak. We find with $\alpha = 6°$ that all A peaks fit on A peaks, B peaks on B peaks and C peaks on C peaks, as shown in Fig. 1.

This procedure allocates to the molecule at 0 one A peak in each of the hexad sets surrounding the points 1,2,3,4,5,6; the most remote B peak of each neighboring triad set; the near C peak of each neighboring dyad set. Drawing corresponding hexagons around other molecules all the A, B and C peaks are filled in. The six nearest A and B peaks around O are contributed, one each, by the molecules associated with the positions 1,2,3,4,5,6, and none of them by the molecule at 0.

So far the details of the skeleton and the positions of the side chains attached to the C_2 molecules have been left out of account. Nevertheless, the 18 peaks per molecule in Crowfoot's c-plane projection are given in the correct positions, on the assumption that there are concentrations of atoms at the six slits.

The full investigations will shortly be published. They show the direct relation between the diagrams obtained from the C_2 structure and all the diagrams given by Crowfoot and include also an account of the geometric method of interpreting Patterson-Harker diagrams in general, both for molecules and for mega-molecules.

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l-GLYCERIC ALDEHYDE AND TUMOR METABOLISM

IT was reported some years ago that dl-glyceric aldehyde inhibits glycolysis of tumor cells.[1] Under *anaerobic* conditions glycolysis is inhibited about 75 per cent. by 10^{-3} M. and almost completely stopped by 1.5×10^{-3} M. This applies to all malignant tumors, *i.e.*, to human and animal tumors, to spontaneous tumors and tumors transplanted or produced by carcinogenic substances, to sarcoma as well as to carcinoma, whether filterable or non-filterable. The *aerobic* glycolysis of tumors is less sensitive to glyceric aldehyde; 4×10^{-3} M. are required for an inhibition of about 75 per cent.

Some months ago J. Needham and H. Lehmann[2] found that d-glyceric aldehyde does not affect glycolysis of embryos. Nor does it inhibit glycolysis of tumors, as we have shown in a previous communication.[3] The effect of dl-glyceric aldehyde thus seems to be due to the l-form, and therefore it was of interest to examine the action of this isomeride.

H. O. L. Fischer and E. Baer recently succeeded in preparing pure l-glyceric aldehyde.[4] With this substance experiments were carried out on tumor slices, and it was found that l-glyceric aldehyde is about twice as effective as the racemic form. A slight inhibition (about 25 per cent.) of the anaerobic glycolysis of tumors is obtained with concentrations as low as 3×10^{-4} M., glycolysis is inhibited about 75 per cent. by 5×10^{-4} M. l-glyceric aldehyde, and almost completely stopped by 7.5×10^{-4} M. (see Table 1).

TABLE 1
ANAEROBIC GLYCOLYSIS OF RAT SARCOMA 39

l-Glyceric Aldehyde M/litre	$\frac{N_2}{Q}$ $\frac{}{CO_2}$ (second half hour)
.....	37.7
3×10^{-4}	27.2
5×10^{-4}	8.8
7.5×10^{-4}	3.7

Under aerobic conditions 2×10^{-3} M. l-glyceric aldehyde inhibits glycolysis about 75 per cent.

[1] B. Mendel, *Klin. Woch.*, 8: 169, 1929.
[2] J. Needham and H. Lehmann, *Biochem. Jour.*, 31: 1913, 1937.
[3] B. Mendel, F. Strelitz and D. Mundell, *Nature*, 141: 288, 1938.
[4] E. Baer and H. O. L. Fischer, SCIENCE, July 29, p. 108.

The rotation of an aqueous solution of l-glyceric aldehyde, if kept in the ice-box, changes in a week's time from $[\alpha]_D^{20°} - 14$ to about -7. Dr. Baer has been able to restore the original optical activity of such a solution by evaporating it and heating the residue to 55–60° C. *in vacuo* for 2 hours. We found that the decrease in optical rotation is not accompanied by any noticeable change in the biological activity: both solutions whether $[\alpha]_D^{20°}$ was -7 or -14 were found to inhibit glycolysis to the same degree when used in equal concentrations.

As dl-glyceric aldehyde (0.5×10^{-2} M.) is known to inhibit respiration of brain cells, experiments were carried out to decide whether this effect is due to the l- or to the d-component. It was found that the inhibition is brought about mainly by l-glyceric aldehyde, whereas the d-form has only a slight effect on respiration. (See figure).

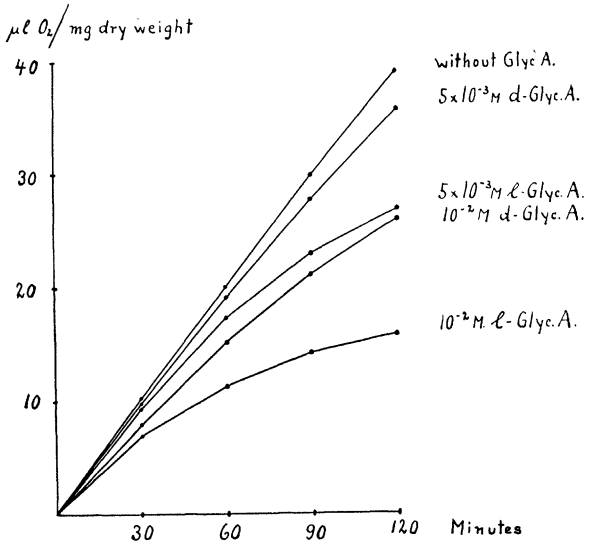

FIG. 1. Effect of d- and l-glyceric aldehyde on respiration of rat brain (grey matter) Ringer Phosphate, pH = 7.4.

The respiration of tumors is less sensitive to glyceric aldehyde than the respiration of brain, and the differences between the two optical forms were found to be less marked in experiments on sarcoma 39.

d- and l-glyceric aldehyde are fermented at the same velocity by slices of rat liver under anaerobic conditions.

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A THEORY OF CORTICO-ADRENAL AND POST-PITUITARY INFLUENCE ON THE KIDNEY

THE possibility of an important relationship between the adrenal cortex and the posterior pituitary lobe in regard to their actions on the kidney has been discussed in previous papers.[1,2,3] We have recently carried out experiments which confirm our earlier work and give fairly clear indication of the mode of action of the hormones concerned. Tests were made of responses of female opossums to orally administered water, salt and urea, and the influence of cortico-adrenal extract and post-pituitary solution (Squibb) was determined. Both normal and adrenalectomized opossums were utilized, and more than three hundred separate experiments were made in this study.

The results are given in summary in Table 1. It

TABLE 1

EFFECT OF CORTICO-ADRENAL EXTRACT AND POST-PITUITARY SOLUTION (SQUIBB) ON RENAL SECRETION

Fluid administered†	Hormone administered	Normal opossums			Adrenalectomized opossums		
		Average water output, cc.*	Concentration in mgm./cc.		Average water output, cc.*	Concentration in mgm./cc.	
			Chlorides	Urea		Chlorides	Urea
Water	none	16.4	0.21	4.51	8.1	0.32	6.66
	cortico-adrenal extract	17.6	0.32	3.58	16.9	0.30	4.91
	post-pituitary solution	5.7	6.50	18.2	3.0	1.72	25.0
NaCl (0.9 %)	none	13.2	7.12	6.51	5.2	7.50	10.2
	cortico-adrenal extract	22.0	6.37	4.18	12.2	4.58	7.60
	post-pituitary solution	17.0	10.0	6.77	8.8	11.1	10.3
NaCl (0.9 %) plus Urea (2 %)	none	10.0	7.60	40.6	8.8	4.20	38.6
	cortico-adrenal extract	15.2	6.31	27.2	13.3	3.31	33.3
	post-pituitary solution	14.8	8.51	26.5	...	...	...

† 20 cc./kilo body weight/2 hours.
* Each figure represents an average of 12–21 metabolic runs. Water excretion on basis of cc/kilo body weight/2 hours.

will be observed that in every case the action of cortico-adrenal extract on chloride excretion (per cc of urine) by the kidney is the direct opposite of that of the post-pituitary effect. Further, in every case the elimination of water under the influence of adrenal extract is greater than that under post-pituitary solution. It would appear that the latter hormone acts to bring

1 H. Silvette, *Am. Jour. Physiol.*, 117: 405, 1937.
2 H. Silvette and S. W. Britton, *Am. Jour. Physiol.*, 121: 528, 1938.
3 H. Silvette, *Proc. Am. Physiol. Soc.*, fiftieth annual meeting, Baltimore, 1938, p. 188.