methæmoglobin and oxyhæmoglobin can be demonstrated simply by shaking these solutions with chloroform, when only the former is denatured and is precipitated. When the water molecule bound to iron in methæmoglobin is replaced by fluoride or azide ions, however, the pigment acquires resistance to chloroform treatment. These results are exactly parallel to those observed in the desiccation experiments, which demonstrated an increased resistance of hæmoglobin and methæmoglobin to denaturation when their iron-bound water molecules are replaced by oxygen, fluoride or azide.

The reactions taking place during the drying of hæmoglobin and methæmoglobin, although perfectly reversible, appear to be of a more complex nature than the simple polymerization envisaged by Haurowitz[2], and to involve reversible changes in the architecture of the whole hæmoprotein molecule.

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Transaminases for Pyridoxamine and Purines

Braunstein's[1] demonstration of the transaminase reaction as a mode of formation of amino-acids has been broadened recently to include a wide variety of transaminases in bacteria[2], animal tissues[3] and plants[4]. In the present communication, we wish to report the formation of compounds other than amino-acids by the transaminase reaction.

Vacuum-dried cells of *Escherichia coli*, strains *B* and Crooks's, were used as sources of enzyme. Extracts were prepared by grinding cells with an equal weight of 500-mesh 'Carborundum', the cell debris removed by centrifugation and the clear, cell-free supernatant used directly, or after fractionation by precipitation with ammonium sulphate, followed by protamine treatment.

Table 1. Transaminases for Pyridoxamine, Adenine, Guanine and Cytosine

Reaction volume, 1 ml., containing:

M Phosphate buffer, *p*H 8·3	0·1 ml.
Alpha ketoglutarate	25 μM
Amino-donor	25 μM
Pyridoxal phosphate, calcium salt	10 γ
Cell extract	approx. 15 mgm. cells

Incubated 60 min., 37° C. Reaction stopped with 0·1 ml. 100 per cent trichloroacetic acid, 0·02 ml. transferred to paper and chromatogram developed with phenol/water (80/20). The μM glutamic acid (R_F = 0·36) formed per ml. were estimated from the chromatograms by comparison with the size and density of spots shown by known amounts of glutamic acid

Enzyme and substrate	μM Glutamate formed/ml. from amino-donors				
	No addition	Pyridoxamine	Adenine	Guanine	Cytosine
Extract	0·5	0·5	0·5	0·5	0·5
Extract, α-ketoglutarate	0·5	8	6	6	6
Extract, α-ketoglutarate, pyridoxal phosphate	0·5	8	6	6	6

The occurrence of a series of transaminases in these cell extracts is indicated by the results shown in Table 1. These preparations catalyse glutamate formation from alpha ketoglutarate in the presence of pyridoxamine, adenine, guanine and cytosine as amino-donors. The presence of ammonia does not lead to glutamate formation, thus indicating a transamination reaction. As shown in Table 2, the transfer of the amino-group of pyridoxamine to alpha ketoglutarate is dependent on the presence of the 'transaminase', pyridoxamine, alpha ketoglutarate and pyridoxal phosphate. The reversal of this reaction has been shown to occur with glutamic acid as amino-donor and pyridoxal as amino-acceptor.

Table 2. Pyridoxal Phosphate Requirement of Pyridoxamine Transaminase

Reaction volume, 1 ml. All samples contained 0·1 ml. *M* phosphate buffer, *p*H 8·3, enzyme, and additions as indicated. Substrate and coenzyme concentrations as in Table 1

Additions	μM Glutamate formed/ml.		
	Dried cells, 15 mgm.	Cell-free extract, approx. 15 mgm. dried cells	0·23–0·3 saturated ammon. sulphate fraction, approx. 15 mgm. dried cells
None	2	0·5	0
α-Ketoglutarate	2	2	0
α-Ketoglutarate, pyridoxamine	8	6	0
α-Ketoglutarate, pyridoxamine, pyridoxal phosphate	8	8	8

These observations suggest a mechanism of formation and interconversion of the purines and pyrimidines, and a route from pyridoxamine to pyridoxal. The importance of the transaminase reaction as a general mechanism of amino-transfer in living organisms is suggested.

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Formation and Breakdown of Pentose Phosphates by Liver Fractions

It has previously been shown in this laboratory[1] that at least two liver factors are necessary for the oxidation of D-ribose-5-phosphate by ammonium sulphate precipitated fractions of horse or rat liver, one being an aldolase and the other a dehydrogenase specific for coenzyme II. Ribose-5-phosphate is oxidized most rapidly by a combination of liver fractions *A* (0·4–0·5 saturation with ammonium sulphate) and *B* (0·5–0·6 saturation), the former being richest in glucose-6-phosphate dehydrogenase and the latter most active in splitting ribose-5-phosphate, as judged by the disappearance of pentose using the orcinol method[2]. The breakdown of ribose-5-phosphate, unlike the oxidation, is independent of coenzyme II and oxygen and never proceeds beyond 66–72 per cent disappearance of pentose.

Ketopentose phosphate formation has been detected by the cysteine–carbazole[3] and orcinol[2]