bromide took place in a pyrex vessel immersed in a molten metal bath. The temperatures ranged from 340° to 400° C. and were controlled by a chromel-alumel thermocouple and potentiometer system. At 340°–370° the decomposition is heterogeneous; the introduction of pyrex glass tubing leads to considerable increases in the reaction velocity. The final pressure is double the initial pressure, the products of decomposition are vinyl bromide (C_2H_3Br) and hydrogen bromide, and the reverse reaction is not appreciable at these temperatures. The reaction follows the first order equation very well.

There is no decomposition into ethylene and bromine. The calculations of Sherman and Sun lead us to believe that the decomposition into ethylene and bromine would take place at an appreciable rate at 350° with an activation energy of 50,000 calories. We have calculated an approximate activation energy for the heterogeneous reaction of about 30,000 calories.

Since the ethylene bromide molecule prefers to dissociate into vinyl bromide and hydrogen bromide, we would be inclined to look for some structural explanation of this phenomenon. The *trans* form of ethylene bromide is considered to be the more 'stable' (preferred) form, even at high temperatures[2]. This interesting theory may explain how it is that ethylene bromide can become attached to the surface in such a way as to give rise to these decomposition products. It is very different from the behaviour of ethylene iodide ($C_2H_4I_2$), where an iodine atom (or an adsorbed iodine atom) acts as a catalyst for the decomposition into ethylene and iodine[3]. Also, the molecule decomposes homogeneously into the same products. Here one of the iodine atoms in the molecule acts as a sort of 'intramolecular catalyst'. One would expect the *cis* form of ethylene iodide to be its normal condition, but there do not appear to be any definite data on this point. Recently, however, the idea that ethylene iodide dissociates directly into ethylene and iodine has been disputed[4].

Further data and experimental details will be published shortly.

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Pyruvic Acid Dehydrogenation, Vitamin B_1 and Cocarboxylase

IT had been found by Davis[1] that pyruvic acid is oxidized by the acetone preparation of *Bacterium Delbrückii*. This preparation has proved to be very useful for a closer study of pyruvic acid dehydrogenation[2].

A codehydrase could be removed from the acetone-treated lactic acid bacteria by washing with phosphate of *p*H 8. The washed preparation was activated by the addition of decoctions of animal tissues (kidney, brain, liver). The addition of vitamin B_1 was without effect. Therefore cocarboxylase was tried, which had been found by Lohmann to be a vitamin B_1 pyrophosphate[3]. A pure preparation of cocarboxylase was most kindly supplied to me by Dr. K. Lohmann, with which the following experiment was carried out.

Activator	None	Kidney decoction	20γ cocarboxylase
Oxygen consumed in 30 minutes (c.mm.)	8	137	176

The effect of cocarboxylase is surprising, because no decarboxylation to aldehyde and carbon dioxide was found with the bacteria. Pyruvic acid was broken down only by dehydrogenation to acetic acid and carbon dioxide.

With highly concentrated organ extracts, the activation found was notably higher than with cocarboxylase at saturation. Probably the additional activation is due to the presence of flavin phosphate in the concentrates. A highly purified, but not pure, preparation of flavin phosphate from heart together with cocarboxylase gave the same additional activation.

Furthermore, it was found that no dehydration takes place with the complete system in the absence of free phosphate. Phosphate is easily removed from the acetone preparation by washing with an acetate mixture of *p*H 4·7. The effect of phosphate is shown by the following experiment.

Phosphate (10^{-3} *M*. per lit.)	0	1·4	2·8	5·5
Oxygen consumed in 60 minutes (c.mm.)	9	45	79	132

Phosphopyruvic acid was found to be inactive as hydrogen donator.

In the catatorulin test of Peters[4], free vitamin B_1 activates pyruvic acid oxidation with brain tissue. But from certain experiments, Peters *et al.* concluded that after the addition of vitamin, a substance x is converted into y, y being the activator of pyruvic acid oxidation. The experiments reported here suggest that the x of Peters is vitamin itself and the y vitamin pyrophosphate, into which it is converted by the tissue.

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Crystals with Vitamin K Potency.

THE evidence for the existence of anti-hæmorrhagic vitamin (K) required by the chick for preservation of normal blood clotting time has been reviewed in a former paper[1].

This vitamin has been obtained in a crystalline fraction isolated from concentrates obtained in molecular distillation by cooling such concentrates in absolute methanol with solid carbon dioxide. The colourless crystal fraction obtained by this method was recrystallized from methanol three times by the same cooling procedure. Two such lots of crystals have been obtained.

The first lot was found capable of restoring normal blood clotting time within four days when added to the basal deficient diet given to five chicks with clotting time greater than 30 minutes. The second lot, tested by preventive assay, maintained normal

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