Thus two reactions, the decomposition and the formation of cysteine and cystine derivatives, occur more readily in the case of peptides. To these may be added a longer known third reaction fulfilling the same conditions—the racemization of peptides[4,5] by alkali. A further extension of the principle will now be considered.

Is "Oxidative Deamination" a β-Oxidation?

Oxidative deamination[6] of amino-acids to α-keto acids under biological conditions is an accepted concept. So is, to a considerable degree, the intermediate formation of a "dehydro"-amino-acid, which is, however, formulated as $RCH:C(NH_2)CO_2H$ (A) or as $RCH_2C(:NH)CO_2H$ (B), according to the tastes of the particular author concerned. The two types are tautomers. Type B is almost surely involved in the hydrolysis; but whether A or B is originally formed in the oxidation is a matter of much interest.

Some theoretical aspects of a reaction leading to the initial formation of Type A will now be discussed. In the first place, such a reaction could conveniently be regarded as a special case of β-oxidation; and such a view-point allows the drawing of certain conclusions which, it seems to the writer, may eventually allow a decision to be reached.

It is clear that the "oxidation" of hydrogen, even when it involves merely the transfer to an acceptor other than oxygen, involves the approach of the hydrogen atoms concerned to a more positive character. But the hydrogen of least positive character, and therefore that most subject to oxidative attack, is located on the β-carbon atom, due to the effect of the carboxyl group. This effect, while in the case of a free amino-acid relatively feeble, is still very real; and it should favor the formation of a dehydrogenation product of Type A.

But two types of modification of the amino-acid structure should favor such β-activation quite strongly. These are just those modifications which have been reported as affecting so strikingly the reactivity of the sulfur-carbon bond in cystine derivatives; and excellent examples would be, peptide formation on both the amino- and carboxy- groups. In other words, *if* oxidative deamination is essentially a β-oxidation, and occurs through a derivative of Type A, it can be predicted that it will occur most readily in substances which have at least the complexity of tripeptides, and that in these the middle member of the tripeptide chain will be the most susceptible to dehydrogenation.

So far as the writer knows, no clear-cut distinction on this basis has been made as yet, either theoretically or experimentally. A variety of "model" experiments in deamination have recently been reported in which charcoal, with or without air, or substances of quinoid type, have been used to induce the reaction. These experiments, which can not here be cited in detail, show glycine to be attacked with exceptional ease. This is certainly not β-oxidation; but neither is it clear that the type of deamination used in the body is here represented. On the other hand, Krebs[7] has managed to demonstrate oxidative deamination of the usually assumed type most convincingly, since he has also isolated derivatives of the expected α-keto acids; but the kidney tissue with which he induced the reaction contained such a complex of proteolytic enzymes that it has not yet been possible to decide whether or not peptides are attacked more rapidly than the simple amino-acids. It is perhaps a modification of his procedure which will settle the question.

The main idea of this portion of the present discussion is definitely this: In so far as oxidative deamination under more or less biological conditions attacks peptides more rapidly than simple amino-acids, and the intermediate members of peptide chains rather than the terminal members, in just that measure it will appear probable that the first stage of oxidative deamination is a β-oxidation, leading to an initial product of Type A.

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