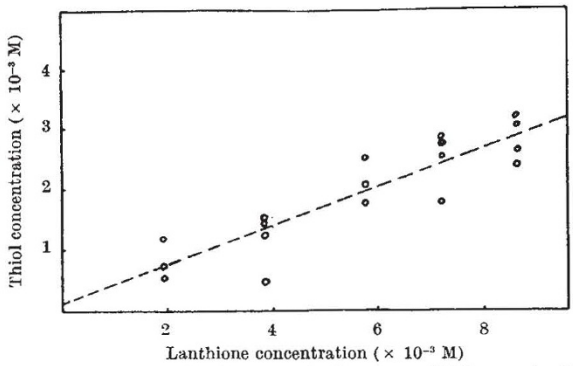

Fig. 1. Original lanthionine concentration versus thiol concentration found after boiling a mixture of lanthionine, cyanogen bromide and sodium cyanide for 10 min

Dann *et al.*[6] showed that lanthionine is attacked by alkali with the formation of thiazolidine derivatives. In our experiments the solution rapidly turned alkaline as the hydrochloric acid was converted into HCN and expelled by boiling. The resulting high *p*H of the solution, due to residual NaCN, could well be responsible for the conversion of some thio-ether into the heterocyclic compound.

We conclude that under our reaction conditions conversion of thio-ether to sulphydryl by a boiling mixture of CNBr and NaCN, as claimed by Sullivan and Folk, does occur. The quantitative value of the method is, however, impaired by the formation of 2-iminothiazolidine-4-carboxylic acid, which may be due either to cyclization of some of the liberated cysteine by the cyanogen bromide or to direct attack of alkali on the thio-ether.

N. J. J. VAN RENSBURG

South African Wool Textile Research Institute,
Grahamstown,
South Africa.

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BIOCHEMISTRY

Sarcoplasmic Proteins in Pale, Exudative Pig Muscles

AMONG the histological aspects of the so-called white muscle disease or 'Muskeldegeneration' (pale, exudative muscle)[1] is the presence of dark bands which cross the muscle fibres when these are seen in longitudinal section[2,3]. Bendall and Wismer-Pedersen[3] presented evidence that these bands consisted of denatured sarcoplasmic protein. The denatured proteins appeared to be precipitated on to the myofibrils under the conditions of relatively low *p*H and high temperature, which obtain during post-mortem glycolysis in this disorder. It has recently been shown, using starch-gel electrophoresis, that various glycolytic enzymes, including creatine kinase, are among the sarcoplasmic proteins which precipitate in exudative pork[4]. These proteins remain soluble if the temperature during post-mortem glycolysis is relatively low or, irrespective of temperatures up to 37° C, if the ultimate *p*H is high.

It would have been inferred from these findings that no bands of denatured sarcoplasmic proteins would form in muscle of high ultimate *p*H, but that in muscle of abnormally low ultimate *p*H (ref. 2) such bands would have been especially marked. In the course of our present investigation, however, we have observed: (*a*) bands apparently of denatured sarcoplasmic protein in longitudinal sections of muscle fibres of ultimate *p*H 6·94; (*b*) the relative freedom from such bands in sections from muscle of ultimate *p*H 4·74, compared with sections from muscle of ultimate *p*H 5·40. Recent work by Scopes[6] on the solubility of sarcoplasmic proteins under various *p*H/temperature combinations affords an explanation of these anomalies, and also supports the view that the bands are in fact of sarcoplasmic origin. He found that, whereas a high ultimate *p*H appreciably protects sarcoplasmic proteins against precipitation at temperatures up to 37° C, denaturation occurs almost independently of *p*H at 45° C. It is known that the temperature of pig muscles may rise above 37°/38° C immediately *post mortem*[3], and in such cases it would appear that bands of precipitated sarcoplasmic proteins might form even although the *p*H is about 7·0. In this connexion it is of interest that the holding of pigs at 45° C for a period before slaughter causes a pale, exudative musculature to develop[7].

Scopes[6] also found that sarcoplasmic proteins were more soluble at *p*H 4·7 than at *p*H 5·4, although they were denatured to a greater extent at the former value; and it has been found that the percentage of sarcoplasmic protein which is extractable (at the *p*H of the muscle) attains a minimum when the ultimate *p*H is about 5·3 (ref. 8). These findings could explain the prevalence of bands in muscles having an ultimate *p*H of 5·4 and their relative absence in muscles of abnormally low ultimate *p*H.

R. A. LAWRIE
I. F. PENNY
R. K. SCOPES
C. A. VOYLE

Low Temperature Research Station,
Downing Street,
Cambridge.

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Conversion of DPNH Dehydrogenase to DPNH-cytochrome Reductase by Thiourea

INVESTIGATIONS in this laboratory have shown that the various DPNH-cytochrome reductase preparations from heart and liver mitochondria described in the literature are fragments of the respiratory chain-linked DPNH dehydrogenase produced by the extraction method (acid-ethanol-heat or thermal degradation) used in the isolation of cytochrome reductases[1–5]. By the action of these agents as well as various proteolytic enzymes, urea and under a number of other experimental conditions the purified dehydrogenase is also degraded to lower molecular weight cytochrome-reducing fragments. This conversion involves major changes in many of the molecular and catalytic properties of the enzyme of which the emergence of cytochrome (and DCIP) reductase activity is but one feature. The observations on thermal and proteolytic degradation have been confirmed and extended by Kaniuga[7,8], who has independently discovered the urea-catalysed transformation of the enzyme. Acid-ethanol degraded DPNH dehydrogenase is not distinguishable from the preparations of Mahler *et al.*[9], de Bernard[10], and Mackler[11], while the thermally degraded enzyme is in every respect identical with the preparation isolated by King and Howard[12] of which the major component is the Mahler enzyme[9]. Although these investigations have directed attention to the fact that DPNH dehydrogenase is unstable under a variety of conditions[13] and readily breaks down to fragments with very different