Acute poisoning from the consumption of cyanogenic plants has been the subject of many reports and is a well-established phenomenon. There is however a marked difference between chronic and acute conditions of toxicity which is usually fatal. The presence of SCN in cassava is therefore of great nutritional significance and a recognition of its poisonous potential has resulted in the evolution of various methods of processing, aimed at considerable alleviation of its toxicity, in all tropical areas where its use as food and feed is predominant. Drying appears to be one of the most effective methods of reducing toxicity, but even in the dry state, there still appears to exist the danger of cumulative effects of ingestion of residual quantities of cyanide, with resultant chronic poisoning. The minute amounts of thiocyanate normally present in urine, blood and saliva however show that there is an intrinsic cyanide metabolism to thiocyanates, in which form it is eliminated. The body’s principal metabolic pathway for cyanide detoxification is by reaction with thiocyanate to form thiocyanate and cyanate, the reaction being catalysed by rhodanase, which is a sulphur transferring enzyme (Lang, 1933). A second pathway is the reaction of cyanide with mercaptopyruvate, catalysed by mercaptopyruvate-cyanide sulphur transferase, to form thiocyanate and pyruvic acid (Fiedler and Wood, 1956) in the presence of adequate amounts of cysteine as a sulphur donor. On the other hand, coenzyme A may react more directly with cyanide to produce 2-imino-4-thiazoline carboxylic acid which is excreted as such in the urine (Wood and Cockey, 1956).  2-imino-4-thiazoline carboxylic acid is however unusual compared with other pathways of detoxification. The speed for a sulphur source in the detoxification of cyanide by rhodanase pathway is evident (Kansyoy and Edmondson, 1970) and the possible role of sulphur amino acids as sources of labile sulphur for cyanide detoxification leads itself to considerable experimental variables. Kaiser and Consor (1970) found that methionine supplementation of cassava diets improved performance and resulted in an increased urinary thiocyanate excretion by rats. Voegelin et al. (1952) showed that cysteine protected animals from minimal lethal doses of cyanide.

Since the involvement of the sulphur amino acids in cyanide detoxification centres around the provision of sulphur for thiocyanate formation, it was considered desirable from a production standpoint to investigate the effect of other sources of dietary sulphur, with a view to increasing growth rate on the relative involvement of methionine in the correction of dietary protein deficiency in growth and in cyanide detoxification.

Twelve growing male pure-bred Yorkshire pigs with an average body weight of 13.46-5.05 kg and a mean initial weight of 33.7% were fed dried bitter cassava (containing 0.5% C and 0.3% on hydrolysis with added linoleic) based diets alone or supplemented with either 0.2% methionine, 0.76% Na₂S.4H₂O or 0.4% elemental sulphur. The pigs were individually housed in metabolic cages and were fed in two intakes daily. Deionised water was offered. Urine and blood samples were analysed daily for a period of 14 days. Supplementation with any of the above sources had no effect on serum thiocyanates (0.04, 0.07, 0.39 and 0.76 mg/100mg) or urinary nitrogen excretion (0.46, 0.52, 0.52, 0.52 and 0.52 mg/day). The addition of sodium thiocyanate significantly (P < 0.05) increased urinary excretion of thiocyanate (10.31, 17.53, 23.56, 20.76 mg/kg feed), and sulphur (5.14, 5.53, 17.54, 7.05 mg/kg feed). Elemental sulphur (0.2%) significantly increased sulphur excretion, but caused an insignificant rise in urinary thiocyanate excretion.

Cameron, et al. (1970) reported that there was no direct relationship between plasma thiocyanate and methionine supplementation of rat diets. It does appear that the physiological implication of the existence of a dynamic equilibrium between cyanide and thiocyanate (Boulden and Forster, 1955; Forster and Boulden, 1953-1954) would be a direct metabolic control of thiocyanate production in the body, preventing its accumulation beyond a threshold level. The lack of response in urinary nitrogen excretion suggested that supplementation did not interfere with nitrogen metabolism and also that no direct metabolic relations between the urinary nitrogen and the detoxification process were probable. The higher urinary thiocyanate excretion on the sodium thiocyanate supplemented diet was in line with expectation. The enzymatic reaction in the body’s principal metabolic pathway for cyanide detoxification is supposed to proceed in the following manner (Lang, 1933).

[Chemical reaction]

\[ \text{SCN} + \text{Na}_2\text{S} + \text{H}_2\text{O} \rightarrow \text{Na}_2\text{SO}_3 + \text{H}_2\text{S} \]

The results therefore suggested that the sodium thiocyanate probably supplied a relatively available sulphur source for cyanide detoxification. Thus the availability of thiocyanate might be a rate limiting step in vivo cyanide detoxification and, although the level of thiocyanate in the experimental diets was not extremely elevated, at higher levels of cyanide ingestion, supplemental thiocyanate might have a greater effect on urinary thiocyanate excretion. The slight increase in urinary thiocyanate excretion observed in the methionine supplementation suggested that this sulphur source might be metabolically important in the detoxification process. The higher urinary sulphur excretion on the thiocyanate supplemented diet was probably due to better handling of the source, or might even be associated with the total level of thiocyanate in the urine.

Conclusions: Drying of cassava appears to reduce the level of cyanide and residual glucoside to a point where it can be safely handled by the pig without any apparent side effects. It would appear that one method of reducing overall effects of cyanide intoxication could be alleviated by the use of supplemental sulphur sources such as sodium thiocyanate.