THE EFFECT OF SUPPLEMENTAL SULPHUR SOURCE ON THE THIOCYANATE PRODUCTION, URINARY NITROGEN AND SULPHUR EXCRETION OF GROWING PIGS FED DRIED BITTER CASSAVA DIETS.

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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 HCN in cassava is therefore of great nutritional significance and a recognition of its poisonous potentials 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 cummulative 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 thiocyanate, in which form it is eliminated, The body's principal metabolic pathway for cyanide detoxification is by reaction with thiosulphate to form thiocyanate and sulphate, the reaction being catalysed by rhodanese, which is a sulphur transferase enzyme (Lang, 1933). A second pathway is the reaction of cyanide with 3 mercaptopyruvate, catalysed by 3-mercapto-pyruvate-cyanide sulphurtransferase to form thiocyanate and pyruvic acid (Fiedler and Wood, 1956) in the presence of adequate amounts of cystine as a sulphur donor. On the other hand, cystine may react more directly with cyanide to produce 2-imino-4-thiazoline carboxylic acid which is excreted as such in the urine (Wood and Cooley, 1956). Iminothiazolidine carboxylic acid is however small compared with other pathways of detoxification. The need for a sulphur source in the detoxification of cyanide by the rhodanese pathway is evident (Massey and Edmondson, 1970) and the possible role of sulphur amino acids as sources of labile sulphur for cyanide detoxification lends itself to considerable experimental support. Maner and Gomez (1973), found that methionine supplementation of cassava diets improved performance and resulted in an increased urinary thiocyanate excretion by rats. Voegtlin et al (1926) showed that cystine 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 throwing more light on the relative involvement of methionine in the correction of dietary protein deficiency per se and in cyanide detoxification.

Twelve growing male pure-bred Yorkshire pigs with a weight range of 31-36.50kg and a mean initial weight of 33.17kg were fed dried bitter cassava (containing 90 ppm -CN on hydrolysis with added linamarase) based diets alone or supplemented with either 0.2% methionine, 0.785% $Na_2S_2O_3.5H_2O$ or 0.2% elemental sulphur. The pigs were individually housed in metabolic cages and were fed in two instalments daily. Demineralized 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 thiocyanate (0.82, 0.75, 0.64 and 0.76 mg/100ml) or urinary nitrogen excretion (30.46, 28.45, 30.23, 33.85%). The addition of sodium thiosulphate significantly ($P<0.05$) increased urinary excretion of thiocyanate (19.31, 17.53, 23.76, 20.76 mg/kg feed), and sulphur (4.18, 5.33, 17.19, 7.05 mg/kg feed). Elemental sulphur (0.2%) significantly ($P<0.01$) increased sulphur excretion, but caused an insignificant rise in urinary thiocyanate excretion.

Calderon, et al; (1972) 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 (Goldstein and Reiders, 1953; Boxer and Rickards, 1952) could be a direct metabolic control of thiocyanate production in the blood, preventing its accumulation beyond a threehold level. The lack of differences 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 was probable. The higher urinary thiocyanate excretion on the sodium thiosulphate 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).

$$HCN + Na_2S_2O_3 \longrightarrow HSCN + Na_2SO_3$$

The results therefore suggested that the sodium thiosulphate probably supplied a readily available metabolic sulphur source for cyanide detoxification. Thus the availability of thiosulphate might be a rate limiting step in in vivo cyanide detoxification and, although the level of hydrocyanic acid in the experimental diets was not extremely elevated, at higher levels of cyanide ingestion, supplemental thiosulphate might have a greater effect on urinary thiocyanate excretion. The slight increase in urinary thiocyanate excretion observed with elemental sulphur supplementation suggested that this sulphur source might be metabolically important in the detoxification process. The higher urinary sulphur excretion on the thiosulphate 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 the fear of cumulative effects could be alleviated by the use of supplemental sulphur sources such as sodium thiosulphate.

Selected reference: Fiedler, H. and J.L. Wood 1956. J. Biol. Chem. 222:387. Goldstein F and Reiders, 1953. Am. J. Physiol. 173:287. Lang, K. 1933. Biochem Z 259:243. Maner, J.H. and G. Gomez, 1973 Int. Dev. Res. Centre. Monogr. IDRC 010e. Massey, V. and D Edmondson, 1970. J. Biol. Chem 245(24):6595. Voegtlin, C., J.M. Johnson and H.A. Dyer, 1926. J. Pharmacol and Therap. 27:467. Wood, J.L. and S.L. Cooley, 1956 J. Biol. Chem. 218:449.