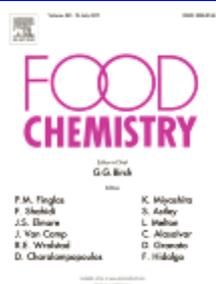




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# Fate of ingested linamarin in malnourished rats

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## Abstract

Pure linamarin at a dose level of 30 g per 100 g body weight was administered in food to a group of Wistar rats maintained on vitamin B<sub>2</sub>-deficient, sufficient and excess diets for 5 weeks and to another group of kwashiorkor rats. Free and total cyanide, intact linamarin and thiocyanate levels were estimated in urine and faeces obtained at 0-, 24-, 48- and 72-h periods and in blood samples obtained in the seventy-second hour after the drug had been administered. There was no detectable cyanide or intact linamarin in the faecal samples. Vitamin B<sub>2</sub>-sufficient and excess groups of rats excreted higher total and free cyanide than the respective vitamin B<sub>2</sub>-deficient groups. Most of the linamarin was degraded after the first 24h. The rate of breakdown of the glucoside within the first 24 h was slowest for the zero and half normal vitamin B<sub>2</sub> status, respectively, as evidenced by its appearance in large quantities in the urine. The kwashiorkor rats, on the other hand, excreted less thiocyanate than the

controls. In addition, their control group excreted most of the thiocyanate ( $\text{SCN}^-$ ) in the first 24 h whilst the kwashiorkor rats excreted theirs in the first 48 h. Dietary protein deficiency prolongs the time of metabolism and hence increases the toxicity of cyanogenic glycoside in the body. It is also suggested that excessive exposure of malnourished humans to cyanide could be a contributory factor in the rampant cases of tropical ataxic neuropathy (TAN).