International Scholars Journals

Frontiers of Agriculture and Food Technology Vol. 1 (1), pp. 001-004, December, 2011. Available online at www.internationalscholarsjournals.org © International Scholars Journals

Full Length Research Paper

Assessment of cyanide overload in cassava consuming populations of Nigeria and the cyanide content of some cassava based foods

OKAFOR, P. N.

Department of Chemical Sciences (Biochemical Unit). Federal University of Agriculture, Umudike, P.M.B. 7267, Umuahia, Abia State, Nigeria. Email: pnokafor@yahoo.com.

Accepted 22 July, 2011

The cyanide overload of 40 cassava (gari) processing workers, 25 frequent consumers of cassava foods and 20 cigarette smokers (all Nigerians) were assessed using a new simple kit method for determination of thiocyanate (cyanide metabolite) in urine. The mean thiocyanate measured in the urine of three groups of cassava processing workers were 9.48±2.8, 8.70±2.23, and 7.46±2.57 ppm. The mean thiocyanate measured in the urine of frequent cassava consumers and cigarette smokers were 3.57±0.6 and 13.99±13.81 ppm, respectively. The urine samples of 15% of the processing workers measured ≥10 ppm SCN (172µmol/L), while two urine samples from the smokers measured 30, and 44 ppm SCN. The cyanide contents of some cassava based foods commonly consumed among Nigerians were determined and ranged from 0.68± 0.31 to 0.88±0.21 for total cyanide and 0.14±0.03 to 0.52±01 mgCN$^-$ 100g^{-1} DM for free cyanide. The toxicological implications of these findings are discussed.

Key words: Cyanide overload, thiocyanate, simple kit method, cassava foods.

INTRODUCTION

Cassava (Manihot esculenta crantz) is one of the most important food crops in the tropical countries and is probably the most widely distributed major human food crop with high content of cyanogenic glycosides (Osuntokun, 1980; Osuntokun and Monekosso, 1969). High cyanide intake from the consumption of insufficiently processed cassava has been advanced as a possible aetiologic factor in some diseases such as “Konzo” or upper motorneuron diseases (Tylleskar et al; 1992), iodine deficiency disorder (Ermans et al; 1983) and tropical ataxic neuropathy (Osuntokun, 1981).

Akinrele (1986) reported that large scale cassava processing could be hazardous, not by consuming residual cyanide in food, but the discharge of hydrocyanic acid into the air. In this context, the hydrocyanic acid contamination of the atmospheric air (Okafor and Maduagwu, 2000) and natural water sources (Okafor et al., 2001) in areas near large scale “gari” processing as well as possible occupational exposures of humans to cyanide poisoning during large scale cassava processing (Okafor et al., 2002) have been reported. Because cyanide is converted in the body to thiocyanate, determination of thiocyanate content of urine (Bradbury and Holloway, 1988; Casadei et al., 1990; Banea-Mayambu; et al., 1997) can be used to check the cyanogens overload of populations related to intake of cassava roots and products. A simple kit method for determination of thiocyanate in urine which could be used to monitor the cyanide overload in cassava consuming populations was developed by Bradbury and Haque (1999). The use of this simple kit method to assess the exposures of cassava processors (mainly gari processors), consumers of cassava foods and cigarette smokers in Nigeria to hydrocyanic acid is hereby reported.

MATERIALS AND METHODS

Materials

Kit D1 for thiocyanate determination in urine was supplied by Howard J. Bradbury of Australian National University, Canberra.

Table 1. Urinary thiocyanate excretion, serum total protein and albumin levels of gari processing workers, frequent cassava consumers and cigarette smokers in Nigeria.

Subjects	Mean urinary SCN (ppm)	Mean serum albumin (g/l)	Mean total plasma protein (g/L)
Gari Processing workers			
Group I	9.48±2.8	37±2.21	68.9±7.1
Group II	8.70±2.23	-	-
Group III	7.46±2.57	38.0±1.3	69.0±3.5
Group IV	3.94±1.13*b	40.0±0.5	68.0±2.1
Group V	2.28±1.05*b	-	-
Cassava consumers	3.57±0.06	40.0±5.1	69.0±4.7
Cigarette smokers	13.99±13.81	38.0±2.0	67.0±5.2

*b: Urinary thiocyanate levels of two groups of gari processing workers measured after returning from days off, because of sickness.

Cassava Food Products

The cassava food materials; lafun, cassava flakes, gari and fofo listed in Table 2 were purchased from Ogbomoso, and Owelli-Agwu Markets in Nigeria.

Determination of cyanide content of samples

Total cyanide was determined by phosphoric acid extraction, hydrolysis of cyanogenic glycosides with linamarase from cassava, followed by colorimetric determination of cyanide. Free cyanide was determined in a similar way but without the use of linamarase (Cooke, 1978; Ikediobi et al., 1980).

Subjects and Procedures

This study was carried out among 85 volunteers; 40 gari processing workers, 25 frequent cassava consumers and 20 male students of Federal University of Agriculture, Umudike who are cigarette smokers. The cassava processing workers were divided into five groups. Groups I to III were processing workers from different cassava processing centres and groups IV and V were workers returning back to work after few days off. The cassava processing workers were women, who are non-smokers, ages 24-50 years and were apparently healthy. About 5-15 metric tons of cassava is processed per working day at each centres where they work. Urine samples from the individuals were collected in clean plastic containers for thiocyanate determination. Venous blood samples from the subjects were also collected into 5 ml vacutainer tubes, for total plasma proteins and serum albumin. Blood and urine samples were collected between the hours of 5.00 to 7.00 p.m. after they have been exposed to hydrocyanic acid discharged into the environment during processing.

Thiocyanate (SCN) determination

Urine thiocyanate was determined using a simple kit method developed and supplied by J. H. Bradbury. The method was based on the quantitative oxidation of thiocyanate in acid permanganate at room temperature in a closed vial with liberation of HCN which reacts with a picrate paper.

Total protein and albumin determination

Total protein was determined by the biuret method as described by Layne (1957) and serum albumin by dye-binding (Bromo Cresol Green) method (Dumas et al., 1971).

RESULTS AND DISCUSSION

The results of this study using the simple kit method showed high exposures to hydrocyanic acid among cassava processing workers and cigarette smokers in Nigeria. The mean thiocyanate measured in the urine of frequent cassava consumers, three groups of cassava processing workers and cigarette smokers were 3.57±0.06 ppm (62 µmol/L), 7.46±2.57 to 9.48±2.8 ppm (129 to 166 µmol/L), and 13.99±13.81ppm (241 µmol/L) respectively (see Table 1). The urine sample of 15% of the processing workers measured ≥ 10ppm (172 µmol/L), while two urine samples from cigarette smokers measured 30 and 44ppm. From the criterion of urine (thiocyanate remains the most useful chemical biomarker for dietary cyanide intake) cyanide overload results mainly from gari processing rather than ingestion of cassava foods (see Table 1). There was statistically significant difference ($p<0.05$) between the mean thiocyanate excretion of the processors and the consumers. Thus gari processing is the highest source of cyanide exposures of humans among Nigerian communities dependent on cassava as their major staple. The high level of urine thiocyanate results from inhalation and skin absorption of hydrocyanic acid during processing as well as ingestion of cassava foods. Absorbed from the skin or inhaled during roasting of gari or from cigarette smoking, hydrogen cyanide readily passes the lungs into the blood stream and is converted to SCN in the liver and kidneys. Determination of the later compound in urine has been used to indicate cyanide overload (Bradbury and Holloway, 1988; Casadei et al.,

1990; Banea-Mayambu et al., 1997).

Most of the cassava processing workers who are mainly women complained of dizziness, headache, pains and other symptoms, consistent with signs of sublethal acute cyanide intoxication . Many of them take off a lot of time due to sickness which could be attributed to inhaling hydrocyanic acid gas. One of the problems at hand now, is how to estimate quantitatively, the amount of hydrogen cyanide inhaled or consumed by gari processors as well as the amounts released into the environments. Preliminary works have been done on this (Akinrele, 1986; Okafor and Maduagwu, 2000; Okafor et al., 2001, 2002) but these studies are not conclusive.

Most of the cassava food products were analyzed for their cyanide contents. The results are shown on Table 2. Although, all of them are cyanogenic (Table 2), the amounts of cyanide produced, may be considered low, since, only plants that accumulate more than 200 mg CN equivalent per 100 g fresh weigh are considered to be dangerous (Kingsbury, 1964). The mean HCN found in the cassava products ranged from 0.14±0.03 – 0.88±0.21 $mgCN^{-}100g^{-1}$ DM. (Table 2). These results demonstrate that cassava food products (especially gari) are safe for human consumption and therefore their production and consumption should be encouraged.

Table 2. Cyanide contents of some cassava foods.

Processed cassava foods	Total cyanide (mg/100 g)	Free CN^{-1} (mg/100 g)
White gari	0.74±0.41	0.52±0.1
Red gari	0.68±0.31	0.47±0.02
Cassava flakes (Abacha)	0.82±0.32	0.82±0.32
Lafun	0.94±0.10	0.14±0.03
Fofo	0.88±0.21	0.21±0.02

Our calculated potential cyanide intake from daily consumption of about 750 g cassava products per individual, aided by questionnaire administration is 3.7-4.7 mg CN/24h per person or 148- 188 µmol CN/24 h per person. With potential increases from other materials (Table 2) the cyanide intake may reach 155-210 µmol/24 h for 60 kg adult. This is supported by the fact that, in a population with cassava roots as their main staple, a basic daily energy need of 1500 kcal can be satisfied with 500 g dry weight cassava roots products. Cyanides intake of 3.7-4.7 mg/24 h per person is far below the lethal dose range for humans of HCN intake by mouth, 0.5-3.5 mg kg^{-1} body weight for a 60 kg adult which amount to 30-210 mg HCN (Montgomery, 1980; Solomonson, 1981; Halstorm and Moller, 1945). No evidence suggest acute adverse effect of cyanide exposure rates below 5 mg (200 µmol) cyanide per 24 h (Tyllekar et al.,1992). The potential cyanide intake reported in this work falls within the Codex Alimentarius safe level of 10 mg HCN equivalent per kg for cassava products.

High urinary thiocyanate excretion was also observed among cigarette smokers. Two of the urine samples from the smokers measured 30 and 44 ppm SCN. This is in agreement with earlier report that one of the major sources for the inhalation of hydrocyanic acid affecting man is tobacco smoke (Richert et al., 1980)

Our findings at Ogbomoso, Oyo State and Owelli-Agwu, Enugu State Nigeria were consistent with earlier report (Dufour, 1989) that strict adherence to effective processing methods enable populations in tropic to use roots from bitter cultivars with high linamarin without adverse effect (Tylleskar et al., 1992). However, cyanide poison free situation may be reversed if processing techniques are not followed or if the feeding habit of individuals is altered for worse. Cyanide poisoning from cassava can only result when food preparation is carelessly done or is almost non-existent and people begin to consume insufficiently processed foods. Hence, iodine deficiency and/or low protein intake could be the major problems of the communities where chronic cassava cyanide diseases were reported in Nigeria in recent times. When the diet is low in protein in cassava consuming population, chronic cyanide poisoning often results. The total protein and serum albumin of these subjects were within the values for normal health individuals (Table 1). Thus, indicating adequate protein intake as a fall in serum albumin level could be a pointer to low protein intake. In this context, plasma proteins (especially albumin) are known to be involved in cyanide detoxification via its convention to thiocyanate. The amounts of sulphur needed to detoxify ingested cyanide of cassava is very small compared with the daily intake of sulphur containing amino acids and therefore cannot affect levels of protein energy malnutrition (Bradbury and Holloway, 1988).

In conclusion, cynanide overload, in cassava consuming populations of Nigeria results mainly from cassava processing rather than ingestion of cassava foods.

ACKNOWLEDGMENT

The authors wish to thank Dr. Howard J. Bradbury of Australian National University, Canberra for supplying (for free) the Kit DI for determination of thiocyanate in urine, used in this work.

REFERENCES

Akinrele IA (1986). Hydrocyanic acid hazard during large scale cassava processing. Trop. Sci. 26: 59 –65.

Banea–Mayambu JP, Tylleskar T, Gitebo N, Matadi N, Gebre-Medhin M, Rosling H (1997). Geographical and regional association between

linamarin and cyanid exposure form cassava and the upper motor neuron disease konzo in former zaira. Trop. Med. Int. Health 2:1143-1151.

Broadbury JH, Haque MR (1999). Simple method for determination of thiocyanate in urine. Clin. Chem. 45:9: 1459-1464.

Bradbury JH, Holloway WD (1988). Chemistry of tropical roots: significance for nutrition and agriculture in the Pacific. Canberra, Australia: Australian Centre for International Agricultural Research, 1988:102.

Casadei E, Cliff J, Neves J (1990). Surrveillance of urinary thiocyanate after epidemic spastic paraparesis in Mozambique. J. Trop. Med. Hyg. 93:257-61.

Codex Alimentarius Commission (1988). Report on 8th session of the codex coordinating committee for Africa. Cairo: FAO/WHO, Rome, Italy.

Cooke RD (1978). An enzymatic assay for total cyanide content of cassava (manihot esculenta Crantz) J. Sci. Food Agric. 29: 345-52.

Dufour DL (1989). Effectiveness of cassava detoxification techniques, used by indigenous peoples in Northwest Amazonia. Internciencai, 14:88-91.

*Doumas BT, Waston WA, Biggs HG (1971). Albumin standards and the measurements of serum albumin with Bromo Cresel Green. Clin. Chim Acta 31:87

Ermans AM, Bourdoux P, Klint Heart J, Lagasse R, Luvivila K, Mafuta M, Thilly LH, Delange F (1983). Role of cassava in the etiology of endemic goitre and cretinism. In cassava toxicity and thyroid. Research and public health issues. Proceedings of a workshop held in Ottawa, Canada, 31st May-2nd June (1982) ed. Delange, F. and Ahiluwalia, R. IDRC-207e, 9-16 Ottawa, Canada.

*Halstrom F, Moller KD (1945). Acta Pharmacol. Toxicol. 1: 18.

Ikediobi OC, Onyia GO, Eluwa CE (1980). A rapid and inexpensive enzymatic assay for total cyanide in cassava (Manihot esculenta Cranz) and cassava products. Agric. Biol. Chem. (Japan) 44 (12): 2803-2809.

Kingsbury JM (1964). Poisonous plants of the U.S and Canada. Prentice Hall, Englewood Cliffs, New Jersey.

Layne E (1957). Spectrophotometric and turbidimetric methods for measuring proteins. In Colowick, S.P and Kaplan, N.O. (eds): Methods I Enzymology, vol. III. New York Academic Press, p. 447.

Montgomery RD (1980). Cyanogens. In: Liener I.E ed. Toxic constituents of plant foodstuffs New York, Academic Press.149-160.

*Okafor PN, Maduagwu EN (2000). Cyanide contamination of atmospheric air during large scale “gari” processing and the toxicity effects of such cyanide equivalent on rat. Afr. J. Biomed. Res 3-19.

*Okafor PN, Okoronkwo CO, Alaneme FO, Maduagwu EN (2001). Cyanide contamination of natural water sources during large scale cassava processing Afr. J. Biomed. Res. 4:25-27.

Okafor PN, Okoronkwo, C. O.; and Maduagwu, E. N. (2002) Occupational and dietary exposures of humans to cyanide poisoning from large scale cassava processing and ingestion of cassava food. Food Chem. Toxicol. 40: 1001-1005.

Osuntokun, B. O.; Monekoso, G. L. Wilson J. (1969). Relationship of a degenerative tropical reuropathy to diet, report of a field study. Br. Med. J. 1:547-550.

Osuntokun BO (1980). A degenerative neuropathy with blindness and chronic cyanide intoxication of dietary origin. The evidence in Nigerians. In Toxicology in the Tropics. Ed. RL Smith, EA Bababunmi, pp, 16-79. London: Taylor.

Osuntokun BO (1981). Cassava diet, chronic cyanide intoxication and a degenerative neuropathy in Nigeria. Ph.D thesis, Ibadan, Nigeria.