

Working together to eliminate cyanide poisoning, konzo, tropical ataxic neuropathy (TAN) and  
neurolethyrism



# CCCDN

## News

Cassava Cyanide Diseases & Neurolethyrism Network

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#### Editorial

Fifty year ago, a paper in Biochemistry by Professor S.L.N. Rao described the chemical structure of a new amino acid occurring abundantly in the seeds of *Lathyrus sativus* and believed to be the cause of the human crippling disease neurolethyrism. Since then this non-protein amino acid  $\beta$ -N-oxalyl-L- $\alpha$ ,  $\beta$ -diaminopropionic acid or  $\beta$ -ODAP, has been the focus of numerous studies in a great variety of disciplines including toxicology, neurology, biochemistry, agronomy and plant biotechnology.

On page 2, the author of that memorable paper explains his present views on this compound. Efforts by the research community to prevent neurolethyrism and cassava cyanide diseases have helped in the understanding and prevention of these crippling diseases of the poor, but as yet there is more reason to increase the efforts than to retire.

In Kinshasa, D.R. Congo, a "Global Advocacy Workshop" on brain development, neurology and child development was organized by IBRO – ARC on 4-5 September. The workshop included a symposium on konzo where the present situation in D.R. Congo and elsewhere was highlighted. A report on this workshop is included in this issue. On the neurolethyrism front, a Danish church sponsored workshop was organized in Bahir Dar, Ethiopia on 4-6 October.

A detailed report on this event can be found online: <http://reliefweb.int/report/ethiopia/ethiopia-grass-pea-blessing-and-curse>. In this report a preliminary survey is mentioned that identified some 800 affected individuals.

Five year after the Ghent workshop 'Konzo and neurolethyrism: similarities and dissimilarities between those crippling neurodegenerative diseases of the poor', an evaluation of progress and developments and a confrontation of ideas would be opportune. Attempts for obtaining financing for a September 2015 workshop in Ghent are being made and suggestions are welcome. Progress and planning will be communicated when available.

Wishing you all a peaceful and successful year 2015.

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### 50 years of ODAP and *Lathyrus* research: Some random thoughts

The saga of *Lathyrus* research over the last 50 years was marked by two very significant developments, firstly the discovery of ODAP and its toxicity to certain animals and the realization that it is only the excessive consumption of *Lathyrus* as a staple over a period of time especially during drought and famine that is responsible for Neurolathyrism. In India in particular the disease has almost disappeared and in the recent past only Ethiopia and Bangladesh had some episodes. Recent surveys from India suggest that wherever *Lathyrus* has been consumed as part of a normal diet there are no cases of neurolathyrism<sup>1</sup>. As a yesteryears *Lathyrus* researcher I consider it relevant to highlight through this dedicated newsletter some of my random thoughts on new directives that could in the near future remove the unfortunate stigma that is attached to *Lathyrus sativus* and establish its deserved status as a food legume.

**ODAP toxicity:** A clear cut toxicity of ODAP following either oral or intraperitoneal administration is seen only in two laboratory animals, the day old chick and the C57BL/6 black mice<sup>2</sup>. A primate model developed quite early in the sixties employed (after failure with oral feeding regimens) a rather unorthodox intrathecal administration of ODAP to demonstrate toxicity<sup>3</sup>. Other studies with primates, goats, horses have been reported claiming toxicity but these have generally employed very complex feeding and/or treatment regimens and might be difficult to reproduce. A rat model of neurolathyrism has also been produced by repeated injection of ODAP to neonatal rats<sup>4</sup>. While the C57BL black mice are susceptible to ODAP toxicity the BALB/C (Albino) mice are not susceptible but become susceptible if pretreated with tyrosine. This led to the discovery of inhibition of TAT activity (Tyrosine amino transferase) by ODAP<sup>2</sup>. This observation may hold the real key to several unanswered questions relating to human neurolathyrism. Over the years, the effect of ODAP has been examined on a variety of biochemical parameters but the only effect that is significant and that can be reproduced in any laboratory is the stereospecific inhibition of TAT activity by L-ODAP but not by D-ODAP. Any mechanism of toxicity proposed for ODAP should in fact take this into consideration. The glutamate receptor interactions by ODAP for instance is a very basic biochemical mechanism and cannot be different between two strains of mice and the same argument also holds good for reports on the inhibition of NADH dehydrogenase by ODAP<sup>5</sup>. The one primary biochemical difference between the two mice strains is that black mice have a predominant melanin (catecholamines) pathway. This could

be the reason why white mice become susceptible to ODAP following pretreatment with tyrosine due to activation of the melanin pathway as a consequence of inhibition of TAT. Several intermediates in the Tyrosine- melanin pathway (neuromelanins) have in fact been implicated in certain neurological manifestations<sup>6</sup>. Since the melanin pathway is activated by exposure to sunlight there is a strong possibility that exposure to sunlight such as during hard physical labor in field conditions, inhibition of TAT by ODAP may result in activation of minor catecholamine pathways due to increased levels of tyrosine. This could well be the reason why women are less prone to *Lathyrus* toxicity. Another puzzling feature of ODAP toxicity to animals and humans relates to the *inVivo* metabolism of ODAP. Administered or ingested ODAP is excreted mostly unchanged (not metabolized) in all laboratory animals, but in humans however, it is nearly quantitatively degraded<sup>7</sup>. The steps involved in this process need to be identified and this raises a spectre of doubt whether such a mechanism could be absent or deficient in susceptible individuals.

One of the hypotheses concerning toxicity of ODAP relates to its non NMDA receptor interactions (AMPA receptors) unlike BMAA (beta-methylaminoalanine), the cycad neurotoxin. However, some studies suggest that ODAP interactions at glutamate receptors may be mostly nonspecific<sup>8</sup>. A recent report shows that BMAA fed *Drosophila* develop severe locomotor disability and eventual death while ODAP feeding had no effect on locomotor capabilities although both compounds behaved as NMDA agonists<sup>9</sup>.

During the last few years ODAP has also taken a new avatar under the name Dencichine which has been discovered in the traditional Chinese medicinal herb *Panax notoginseng* and other species of *Panax*<sup>10</sup>. Many of the medicinal properties of Ginseng are now attributed to Dencichine (= ODAP) and few patents utilising its hemostatic properties have already appeared. Band aid strips and toothpastes containing ODAP are now commercially available in China<sup>11,12</sup>. Other properties of ODAP in areas like wound healing, neurogenesis, angiogenesis and hypoxia are in the patents pipeline. ODAP though born as a neurotoxin is gradually making headways into human health.

Homoarginine, the first unusual amino acid to be discovered in *Lathyrus* had almost been ignored since it was nontoxic. Homoarginine is now recognized as a normal metabolite in humans and its importance in the cardiovascular system is growing<sup>13, 14</sup>. This should make *Lathyrus* a prized legume as a functional food and creates a totally new avenue into *Lathyrus* research as it could become the only known dietary source (nutraceutical !) of this amino acid.

## References

- <sup>1</sup>Arjun L. Khandare, J.J Babu,, M. Ankulu, N. Aparna, Amol Shirfule & G. Shankar Rao. Grass pea consumption & present scenario of neuropathy in Maharashtra State of India. *Indian J Med Res* 140, July 2014, pp 96-101.
- <sup>2</sup>Shasi Vardhan K, Pratap Rudra M.P. and S.L.N. Rao Inhibition of tyrosine aminotransferase by ODAP the *L. sativus* neurotoxin. *J. Neurochem.*1997, 68, 2477-2485,
- <sup>3</sup>S.L.N. Rao, P.S.Sarma, K.S. Mani, T.R. Raghunatha Rao and S. Sriramachari. Experimental neuropathy in monkeys. *Nature.* 1967, 214, 610,
- <sup>4</sup>Kusama-Eguchi, K., Y. Yamazaki, T. Ueda, A. Suda, Y. Hirayama, F. Ikegami, K Watanabe, M. May, F. Lambein and T. Kusama. Hind-limb paraparesis in a rat model for neuropathy associated with apoptosis and an impaired vascular endothelial growth factor system in the spinal cord. *J Comp Neurol.* 2010,518 (6): 928-942.
- <sup>5</sup>Sriram K, Shankar SK, Boyd MR, Ravindranath V. Oxidation and loss of mitochondrial complex I precede excitatory amino acid-mediated neurodegeneration. *J Neurosci* 1998; 18 : 10287-96
- <sup>6</sup>Linan Chen, Yunmin Ding, Barbara Cagniard, Amber D. Van Laar, Amanda Mortimer, Wanhao Chi, Teresa G. Hastings, Un Jung Kang, and Xiaoxi Zhuang. Unregulated Cytosolic Dopamine Causes Neurodegeneration Associated with Oxidative Stress in Mice. *The Journal of Neuroscience*, January 9, 2008; 28(2):425– 433 • 425
- <sup>7</sup>Pratap Rudra.M.P, Raghuvver Singh.M, Junaid.M.A, Jyothi.P and RAO S.L.N. Metabolism of dietary ODAP in humans may be responsible for the low incidence of Neuropathy. *Clin.Biochem*, 2004, 37, 318-322
- <sup>8</sup> Renu K Jain, Mohammed A Junaid and S.L.N. Rao Receptor interactions of  $\beta$ -N-oxalyl-L- $\alpha$ , $\beta$ -diaminopropionic acid, the *Lathyrus sativus* Putative Excitotoxin with synaptic membranes. *Neurochem Res*, 1998. 23, 1193-1198.
- <sup>9</sup>J. H. Koenig , Joy J. Goto , Kazuo Ikeda. Novel NMDA receptor-specific desensitization/inactivation produced by ingestion of the neurotoxins,  $\beta$ -N-methylamino-L-alanine(BMAA) or  $\beta$ -N-oxalylamino-L-alanine (BOAA/ $\beta$ -ODAP), *Comp.Biochem.Physiol, Part C* 20015,167. 43–50.
- <sup>10</sup>Yu-Haey Kuo, Fumio Ikegami, Fernand Lambein. Neuroactive and other free amino acids in seed and young plants of *Panax ginseng*. *Phytochemistry*, 2003,62, 1087-1091.
- <sup>11</sup>United States Patent. Compositions and methods for treating hemorrhagic condition. June 2011, 20110160307,
- <sup>12</sup>Gang Zhao, Fourth Military Medical University. Notoginseng factor in preparing medicine for treating neurodegenerative diseases drugs Application of dencichine in preparation of medicament for treating neurodegenerative diseases. China Patent, July 2012, CN 102579418A,
- <sup>13</sup>Valtonen P, Laitinen T, Lyyra-Laitinen T, Raitakari OT, Juonala M, Viikari JS, et al. Serum L-homoarginine concentration is elevated during normal pregnancy and is related to flow-mediated vasodilatation. *Circ J*, 2008; 72: 1879-84.
- <sup>14</sup>Drechsler C, Meinitzer A, Pilz S, Krane V, Tomaschitz A, Ritz E, et al. Homoarginine, heart failure and sudden cardiac death in haemodialysis patients. *Eur J Heart Fail* 2011; 33:852-9

S.L.N. RAO

## Eating raw peeled cassava roots is dangerous because they contain poisonous cyanide

Raw, peeled cassava roots are eaten in Eastern, Central and West Africa<sup>1-3</sup> and it is reported<sup>1</sup> that some people enjoy chewing a fresh cassava root like a carrot. This contrasts with the situation in the South Pacific, Indonesia and other Asian countries where peeled, cassava roots are always cooked before consumption, such as by boiling, steaming or frying.<sup>4,5</sup> The reason for the difference in behaviour is a perception in Asia and the South Pacific that cassava roots contain a poison that is removed by cooking, whereas this information is not known by the people of tropical Africa.<sup>3</sup> Another problem is that sometimes there is biased reporting of cassava as Africa's staple food, without any mention of the fact that it contains poisonous cyanide.<sup>6</sup>

Analyses of many different cassava varieties have shown that the inside (parenchyma) of the cassava root has a wide range of cyanide contents from 1-1500 ppm.<sup>5, 7</sup> Sweet cassava is at the low end of this range, and the cyanide-containing compound (linamarin) which is bitter, causes high cyanide cassava to taste bitter. The practice of eating fresh, raw cassava roots is sometimes considered to be permissible, providing that the roots are from sweet cassava. This idea is incorrect, because very few of the sweet cassava varieties have a cyanide content of < 10 ppm, 7-10 the WHO safe level for cassava flour.<sup>11</sup> In a time of drought when cyanide levels are 2-4 times higher than normal<sup>5, 12</sup> and konzo occurs in Mozambique and Tanzania, there would be no roots with cyanide levels of < 10 ppm.

In Tanzania and Nigeria both sweet and bitter cassava are consumed raw by farmers, who do not understand that cassava contains toxic cyanogens.<sup>3</sup> The mean total cyanide content (recalculated from a dry weight to a fresh weight basis) of roots eaten raw in Tanzania was 22 ppm and in Nigeria was 42 ppm,<sup>3</sup> which are 2–4 times the WHO safe level. Clearly, the intake of cyanide from eating freshly peeled raw cassava roots greatly increases the total cyanide load, and could contribute to the occurrence of cassava cyanide diseases such as konzo and tropical ataxic neuropathy (TAN).<sup>13</sup>

## Recommendation.

It is recommended that the practice of eating raw, peeled cassava roots should be very strongly discouraged and that people should be educated to understand that there is a poisonous cyanogen present in cassava.

## References

- <sup>1</sup>Nweke FI, Bokanga M. (1994) Importance of cassava processing for production in sub-Saharan Africa. *Acta Hort.* 375, 401412.
- <sup>2</sup>Ministry of Health Mozambique (1984) Mantakassa: an epidemic of spastic paraparesis associated with chronic cyanide intoxication in a cassava staple area of Mozambique. 2. Nutritional factors and hydrocyanic acid content of cassava products. *Bull. WHO* 62, 485-492.
- <sup>3</sup>Oluwole OSA, Onabolu AO, Mtunda K, Mlingi N.(2007) Characterization of cassava varieties in Nigeria and Tanzania, and farmer's perception of toxicity of cassava. *J.Food Comp. Anal.* 20, 559-567.

<sup>4</sup>Nambisan B. (1994) Evaluation of the effect of various processing techniques on cyanogen content reduction in cassava. *Acta Hort.* 375, 193-201.

<sup>5</sup>Cardoso AP, Mirione E, Ernesto M, Massaza F, Cliff J, Haque MR, Bradbury JH. (2005) Processing of cassava roots to remove cyanogens. *J. Food Comp. Anal.* 18, 451-460.

<sup>6</sup>Madamombe I. (2006) Is cassava Africa's new staple food? *Africa Renewal* 20, 13.

<sup>7</sup>Bokanga M. (1994) Distribution of cyanogenic potential in cassava germplasm. *Acta Hort.* 375, 117-123.

<sup>8</sup>Bradbury JH, Egan SV, Lynch MJ. (1991) Analysis of cyanide in cassava using acid hydrolysis of cyanogenic glucosides. *J. Sci. Food Agric.* 55, 277-290.

<sup>9</sup>Djazuli M, Bradbury JH. (1999) Cyanogen content of cassava roots and flour in Indonesia. *Food Chem.* 65, 523-525.

<sup>10</sup>Ernesto M, Cardoso AP, Cliff J, Bradbury JH. (2000) Cyanogens in cassava flour and roots and urinary thiocyanate concentration in Mozambique. *J. Food Comp. Anal.* 13, 1-12.

<sup>11</sup>FAO/WHO (1991) Joint FAO/WHO Food Standards Programme. *Codex Alimentarius Commission XII, Suppl. 4*, Rome, FAO 1991.

<sup>12</sup>Bokanga M, Ekanayake IJ, Dixon AGO, Porto CM. (1994) Genotype-environment interactions for cyanogenic potential in cassava. *Acta Hort.* 375, 131-139.

<sup>13</sup>Nhassico D, Muquingue H, Cliff J, Cumbana A, Bradbury JH. (2008) Rising African cassava production, diseases due to high cyanide intake and control measures. *J. Sci. Food Agric.* 88, 2043-2049.

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### **Three decades of toxic cyanide management in Uganda. Doing and communicating more with minimal strategies. Has it been possible?**

#### **Summary**

For over three decades, documented reports of cyanide poisoning and cyanide-based deaths, caused by eating cassava, has been commonplace in media, health centres and village communities in Uganda. The first documented case of cyanide deaths associated with poisonous cassava in Uganda was reported as early as 1990s and most recently cases have been reported for isolated districts. While there have been many reported cases year in and year out, not many Ugandans believe that the problems of cyanide poisoning could be beyond merely eating poisonous cassava. Time and again the manufacturing industry has been reluctant to use cassava, and/or hide it in products without a mention, due to fear of residual cyanide in products. But what could be the reality? This editorial gives an insight into decades of cassava cyanide management strategies. Socio-economic and other underlying factors of cyanide effects, cassava toxicity and alternative possible sources, direct and indirect consequences and community indigenous knowledge and taught efforts

to avoid cyanide poisoning exposure in Uganda are described. Finally, cyanide reduction recommendations for the global community are discussed.

#### **Introduction**

Cassava (*Manihot esculenta* Crantz) is the second most important crop in Uganda after banana, grown mainly in East, North and Eastern regions and a staple food for an estimated  $\geq 20$  million. Uganda produces an estimated 5.2 million tons or 14% of world production and is ranked third producer after Mozambique (100 million tons) and Tanzania (5.4 million tons) and in yield per ha (120,184) second to Mozambique (128,205) in Eastern Africa (FAOSTAT, 2013). It can be estimated that approximately 60% of the population of 35 million get exposed to the dangers of cyanide intoxication. Nevertheless, the importance of cassava as a food source in Uganda is bound to increase due to the recent devastating effects of Banana Bacterial Wilt (BBW) which has ravaged more than 70% of plantains in major producing areas of Western and Central Uganda. Furthermore, combined with its high yields and survival in marginal conditions, some cultivars are taken as emergency foods in times of hunger and are gaining importance as industrial raw material, Cassava has the potential to surpass bananas in the near future in Uganda. The implication of this is that more people will likely get exposed to poisonous cyanide if drastic preventive measures are not put in place.

#### **Major types of cassava varieties in Uganda**

In Uganda, sweet and bitter cassava cultivars are concurrently cultivated mostly by communities who entirely depend on them as sources of food and income. The sweet cultivars consist of both local and improved varieties while the bitter ones are solely local traditional cultivars which have been grown since their introduction into Uganda between 1862 and 1875<sup>7</sup>. Cassava cyanide contents, its toxicity and effects in humans and animals are documented<sup>2, 6</sup>. All the above can vary from region to region due to differences in climatic, socio-economic and political environments. Nonetheless, sweet (improved and local) cultivars contain between 1 and 100 ppm total cyanogens (TC) while their bitter counterparts can go to 2,000 ppm TC, depending on the region and time of harvest. The main bitter cultivars grown on a large scale per region include Tongolo/Karangwa (Northern, North Eastern and Mid-Western), Rutuga (Western) and Migyera (Eastern).

The National Agricultural Research Organisation (NARO), a national institution mandated to conduct agricultural research in Uganda, has over the years, encouraged and facilitated communities to adopt her improved varieties. However, in some areas and regions, farmers prefer traditional bitter cultivars to improved varieties due to their unique traits such as long storability in the soil, disease and pest resistance or tolerance, root qualities (high yield and white flour) and commercial value for the production of alcohol and flour. Due to high TC of bitter cultivars, they require processing, and the poor

traditional processing methods (mentioned later), in addition to periods of hunger, unscrupulous traders and illiteracy among the communities, are responsible for increased cyanide poisoning among the population.

### **Importance of poisonous cassava in Uganda**

As indicated earlier, poisonous cassava cultivar types are region-specific, conceivably due to the socio-cultural and economic values attached to these cultivars. There are several individual poisonous cultivars in small villages, districts and regions of Uganda but of importance here are three cultivars, viz: - Rutuga, Tongolo/Karangwa and Migyera cultivars which have stood the test of time. At harvest, both Rutuga and Tongolo have been found to contain between 700 and 2,000 ppm total cyanogens while the moderately bitter Migyera can contain between 100 and 150 ppm, subject to the time and season of harvesting the roots. The high values (2000 ppm) are usually obtained during the dry season and when young plants were uprooted less than 18 months after planting.

Rutuga variety is exclusively grown and consumed in Western Uganda. Its mealy nature and ability to blend well with millet, makes Rutuga the best choice of the majority of Western Ugandan populations. Together with sweet and improved varieties, Rutuga is blended with finger millet flour to make ready-to-eat bread and at times freshly steamed or boiled and eaten with milk as a breakfast snack.

Unlike Rutuga which is planted mainly for home consumption, Tongolo/Karangwa serves the dual role of home consumption and trade. Its purity of starch, high yielding roots, ability to tolerate water-stress conditions and fast drying also contribute to the popularity of Tongolo in the semi-arid areas of Northern, Eastern, North-western and mid-western Uganda. In these areas, Tongolo is not eaten fresh but has to be processed and used for crude ethanol production and other fermented products. Recently, its unique properties have found application in flexible and transparent films as potential material for industrial food packaging (Tumwesigye, unpublished information).

Migyera, also known as Nigeria or Nase 3, is a preferred cultivar of mainly Eastern Uganda. Its moderate total cyanogens, semi-arid tolerance, high yield (9-16 tons/ha on-farm and 25-40 tons/ha research station), high dry matter content (up to 40% fresh weight) makes it a cultivar of choice in these areas and easily finds application as home consumed and as fermentable local brew in almost 100% household. Moreover, it's a staple crop and the majority derive their livelihoods from it.

### **Common and potential sources of cyanide exposure in Uganda**

Cultural, social and economic factors have directly and indirectly contributed to high potential risk sources of cyanide exposure in Uganda. The communities have been exposed to cyanide poisoning in different ways, either through eating, drinking and breathing vapours during processing.

### **Raw poisonous Cassava**

Bitter Cassava roots uprooted prematurely and either eaten raw or inadequately cooked is more toxic than the sweet ones and have been the major cause of death of media-reported cases in Uganda. Particularly, children aged between 5-13 years have been exposed when left to roam uncontrolled around cassava fields.

As pointed out earlier, Ugandan fresh roots and leaves of toxic cassava contain up to 2,000 ppm of the toxin. This is very high considering that the person can be safe when the cyanide level in the body is less than 0.5 – 3.5 ppm per body weight. A child weighing 15 kg is 20 times more affected than a 60 kg adult when exposed. According to Nhassico *et al* for an adult person of 60kg the lethal dose ranges between 30 -210 mg hydrogen cyanide, corresponding to 60-420 g of the most toxic cassava roots. Important to note here is that the lethality can be higher when other incomplete hydrolysed forms such as cyanohydrins successfully reach the human gut. It could be for this reason that toxic cassava has been able to cause deaths within hours in Uganda without necessarily leading to many disabilities and abnormalities of tropic ataxic neuropathy (TAN) and KONZO, which occur after prolonged exposure to sublethal doses, in more than three decades now.

### **Geo-economic and political factors**

By virtue of its agronomic and commercial pros, cassava (including bitter cultivars) is widespread as a staple food of specific communities in Eastern, Northern and mid-Western Uganda. In these regions, homesteads depend on cassava for both lunch and supper meals, and because of the feeding monotony, the communities are highly exposed to cyanide. Also, cultural factors, lack of time for adequate cassava dish preparations, compounded by other stresses, households resort to feeding on cassava because it can be kept for a reasonable number of days before it spoils.

The increasing numbers of unregulated promotional programs have also been a factor for cyanide frequencies in communities. Most of the development-related activities by Non-Governmental Organisations (NGOs) have been targeted on cassava growing and consuming communities. As a result and perhaps due to errors and emphasis on quantity-oriented promotions (how many people have been reached with developmental materials), little or no attention is paid to small but vital details like cultivar type and proper processing methods. It's for this reason that some toxic varieties find their way into communities.

Famine, social turmoil, illiteracy and abrupt increases in cassava lucrative markets are by far the greatest factors contributing to cyanide toxicity exposure by rural communities in the last one or two decades. During famine periods, bitter varieties seem to be the answer to community food needs. Moreover, in these communities bitter cassava is a reserve famine food. During these periods of high cassava demand, crafty middle traders and even the community's desire to raise household income,

trade bitter cassava (as fresh roots, flour and baked products) to the illiterate and unsuspecting consumers. Also, when the demand of cassava and its products within the region increased, most households sold-off sweet and safe cassava, and were left with the option of consuming bitter varieties.

#### **Small and Medium processing centres**

A fast expanding source of cyanide poisoning exposure is the rapid increase in cassava processing for both home consumption and sale. A high market of cassava chips and the concurrent trade of cassava flour will likely become the greatest source of cyanide exposure. Both poisonous and sweet cassava is processed in Uganda. Bitter cultivars are transformed into local brew using the traditional methods of heap and aqua-pond fermentations. Because bitter cassava peels contain approximately 80% of root cyanide, they are usually discarded, and their accumulation has an impact on the environment. These rudimentary and inadequate processing methods are principal causes of human cyanide exposure.

Sweet varieties are normally regarded as safe but long term exposure and accumulated quantities can be potential hazards to humans and animals. In Uganda, the numbers of small and medium-scale processing enterprises have mushroomed in the past decade, resulting in the spread of small local units. Because of the poor processing equipment and gear employed, the risk of cyanide exposure has been increasing. Small and medium scale processors and traders inhaling dust arising from poorly ventilated processing and storage houses and transport vehicles are more vulnerable to cyanide toxicity. Women and children, often involved in processing of cassava, have been at a higher risk of cyanide exposure.

#### **Natural plants**

When cyanide poisoning is mentioned, minds are directly tuned to cassava, especially to wild or domesticated bitter varieties. However, it should not be forgotten that other local natural plants and herbs e.g. eucalyptus, apple seeds, mango seeds and arrow grass (for animals) can also be additional sources of cyanide poisoning. When their leaves, seeds and stems are chopped, squeezed or chewed, cyanide is released. The unregulated big number of herbalists and drug supplement processors on the market may put at risk patients visiting their clinics.

#### **Other possible sources**

Imported construction materials, household items (socks, sweaters, kitchen utensils and cabinets); plastic tents can be potential sources of cyanide poisoning. In Uganda, the uncontrolled burning of these materials, compounded by limited disposal information, could contribute to release of reasonable amounts of cyanide dangerous to health. Smoke inhalation, including cigarette smoke, can be a possible cause of cyanide poisoning among household owners and commercial building's

fire fighters.

#### **Cultural, socio-economic and environmental consequences of cyanide exposure**

Rural and peri-urban exposure to cyanide poisoning has had cultural, socio-economic and environmental origin.

#### **The hunger factor**

Seasonal occurrences of hunger account for cyanide-associated consequences which are attributed to easy access of toxic cassava cultivars by many communities. In Uganda, incidences of chronic cyanide poisoning have been reported and attributed to scarcity of food in communities which consistently depend on a diet containing large quantities of cyanogenic cassava. To date, no death has been reported to have arisen due to other dietary sources.

The recent increases in food theft assisted by high demand of cassava in the region and high incidences of cassava brown streak disease (CBSD) presented a serious threat to sustainable production, leading to food scarcity, and causing local famines. In these situations, people have resorted to cultivating poisonous cassava due to the inherent theft-repellent status of these cultivars. However, due to traditional preparation styles of steaming, and cutting short the processing steps, the derived food remains dangerous to their health. Besides, most of the time poisonous cassava is served with other dishes like fish, beef and other sauces. The cyanide toxicity associated with hunger could also be responsible for the severities and fast-acting death among the population due to high malnutrition levels among women and children.

Whenever illnesses occur after eating such dishes, local traditional beliefs overrule the truth, with illnesses being attributed to the secondary accompanying dishes rather than the poisonous cassava. There have been poor and uncoordinated reports of deaths due to cyanide poisoning. The situation has also been compounded by poor information flow between the extension and local communities. Nevertheless, in the absence of official reports on the incidence of cassava-related deaths in Uganda, a number of media reports clearly indicate the occurrence. For example, New vision publications reported the death of seven and critical illness of others (2002) and eight (2009) people caused by eating poisonous cassava in Nakapiripirit, Acholi, Teso and Lango regions. Other reported deaths included 3-year old boy, his two siblings and his father (By Uganda Radio Network-URN, 2002).

#### **Household poverty**

In instances of food crises, particularly in cassava consuming communities, there is a tendency for subsistence farmers to sell off their cassava produce (chips and flour) from sweet varieties for income earnings. This results in acute food shortages leading to options, either to depend on inadequately processed cassava or food relief. In most cases, the population demands exceed food relief and communities have no option but to resort



on poisonous cassava with toxic consequences.

### **Traditional processing methods**

Heap fermentation and pond water soaking are traditional processing methods practiced by close to 100% of households engaged in poisonous cassava business in Eastern, Northern, North-western and mid-western regions. Regardless of the availability of improved processing methods, small-scale processors in these regions insist on fermentation process for both sweet and bitter varieties. Several reasons are advanced including the ability of the method to impart an attractive aroma due to alcohol and acid formations. However, due to high cyanogens in poisonous cassava, the peels are discarded in the environment during heap fermentation. Together with pond fermentation, the methods contribute huge quantities of biotoxins in the environment. Of particular interest is the contamination of streams and other water reservoirs in which communities derive their daily water for domestic and public uses.

### **Other underlying factors**

Lack of simple and handy cyanide confirmatory tests meant that people used guess work methods to know which cultivars are safe. This, particularly during starvation periods, would expose the hungry masses to cyanide poisoning. Absence of alternative foods and limited high nutrient foods rich in protein aggravated the exposure risk of many locals. It's important to mention that zones within the country are so divided, it would be hard for nutritious foods to be transported between in order to avert hunger.

By and large, there have been consistent unreported cyanide poisoning-related deaths since early 90s. Nonetheless, isolated reports point to massive food shortages and starvation causing 3,000 deaths in the eastern and northern districts of Uganda (NARO & Univ. Greenwich, 2004). Reports of disintegrated polygamous families and forced early marriages of young girls in exchange for cassava were evidences in which families gave away safe cassava and resorted to more poisonous one with no market value.

### **Local communities' indigenous knowledge and coping strategies for management of cyanide toxicity**

A number of indigenous cyanide management methods have been used since the introduction of cassava in Uganda. Smallholders are knowledgeable about the potential risks of using poisonous cassava and have developed appropriate techniques for detoxifying the roots before consumption. Indisputably, these techniques have protected many rural areas from cyanide poisoning. The type of methods used greatly varies and are usually location-specific and also depends on cultural beliefs.

In western Uganda, a simple method of harvesting, peeling, slicing, drying, milling, roasting and blending with finger (Millet: Cassava flour, 4:10) ensured that high cyanogens. Rutuga and Kalinga varieties were safe for consumption. The advantage

of this method is that, in addition to processing method, the concentration of cassava cyanogens in the composite flour is diluted and the balance of essential amino acids is better, resulting in a safer diet. The disadvantage is that not many people, particularly children, can guess the intention of adding small quantities of finger millet in cassava. Apart from making the resulting bread palatable, it has an indirect way of detoxifying the poisonous cassava. However, children take it for granted and eat raw uncooked roots which are highly toxic particularly when the plants are less than 12 months old.

By contrast, detoxifying Tongolo or Karangwa in northern, North-western and mid-western regions requires a different approach: wet and dry fermentation. The wet (pond water) fermentation involves harvesting roots, submerging them in streams or ponds until they become soft in not less than a week. This is followed by either crushing into small particle before they are dried. However, pond fermentations generate huge undesirable sour colored chips/flour, solid residues and liquid wastewaters, which may pose a major disposal problem to the environment. Detoxifying this cultivar through processing has received attention<sup>3</sup> by its ability to detoxify and also to reduce the disposal of toxic materials in the environment.

Heap fermentation of poisonous cassava entails peeling, heaping, covering and fermenting roots for 5-7 days until mould grows. Later the mould is scraped off, roots dried and used later as flour. Heap fermentation in Uganda has been adequately described<sup>4</sup> and is still practiced today. The disadvantage of this method is that a lot of solid residues in form of peels containing cyanide are generated into the environment.

Like Tongolo, Migyera/Nase 3 is detoxified by turning the roots into bread and local brews. These processes are elaborate, involving fermentation and roasting. In Uganda, many small and medium processors are involved leading to increased exposure of cyanide poisoning, mainly through inhaling powder dust and vapour during drying, milling and roasting.

Indigenous local treatments also exist in particular cases where an individual is suspected to have been exposed to cassava cyanide poisoning. Although activated charcoal is medically recommended in poison circumstances, smallholders use wood charcoal and other herbs to induce vomiting as the first aid preventive measure. In most cases, where the toxicity is not severe, individuals have recovered while in other cases exposed persons die due to lack of transport means to reach the nearest health centres.

Generally, the above methods reduce total cyanogens by almost 80%. However, the issue here is that sometimes the whole processes are not followed to completion and this leads to cyanide exposure either directly to humans or indirectly in the environment.

## Existing approaches for management of cyanide toxicity in Uganda

Several strategies have been deployed to reduce the sources and consequences of cyanide exposure in cassava dependent communities. These strategies include, but are not limited to, i) development of high-yielding and low-cyanide cassava varieties through breeding programs; ii) development of improved processing technologies and detoxifying methods; iii) community nutritional awareness advocacy and campaigns by National Agricultural Research Organisation (NARO), NGOs and development partners; and iv) health care treatments.

Since early 1990s, when deaths resulting from eating poisoning cassava started receiving media coverage, a number of improved low cyanide varieties have been bred and promoted. These varieties have cyanide contents between < 10 and 100 ppm. These varieties are responsible for the gradual reduction of cyanide in cassava germplasm from as high as 2,000 and 300 ppm to current 700-900 and < 10 -100 ppm in bitter and sweet varieties, respectively. The mechanism of gradual cyanide degradation in the last two decades in the Uganda cassava germplasm is not yet known or studied. However, recent laboratory total cyanogen analyses point to an overall reduction.

The successful breeding programs have been concurrently followed by development of improved processing equipment, methodologies and processing for reduction of cyanide in cassava roots. Using the new technologies, safe dry products (chips, flour, starch) with cyanide contents <10 ppm have been obtained both in research stations and on farm. Together, with simplified cyanide assay techniques<sup>1</sup>, community nutritional awareness and development of national and regional standards, cyanide poisoning by cassava and cassava products has been adequately managed for some communities in Uganda. Below are the excerpts of awareness messages for cyanide prevention and care.



*Be careful: Roasted or fresh cassava on highways can be poisonous. Avoid buying and eating cassava if you are not sure of the source*

However, with existing strategies, improved technologies and advocacy unable to completely reduce and/or eliminate cyanide related poisoning and deaths, new approaches are desired in order to supplement the existing strategies.

## Novel approaches to reduce negative effects of cyanide in communities

Although low cyanogen cassava breeding, adequate processing procedure and proper nutrition hold potential, novel methodologies and approaches are needed not only to safeguard the safety of humans and animals but also to reduce the environmental impact of poisonous cassava derived by-products. Improved cassava and processing methods need to be rapidly promoted to communities in order to prevent cyanide poisoning. This has been and is still a challenge amidst continued use of local poisonous cultivars notwithstanding the availability of improved cultivars.

In Uganda, the current option proposed, and which can be widely applied globally, is deployment of improved downstream processing of whole cassava root. Unpublished data show that simultaneous release, recovery and cyanogenesis (intrinsic) of cassava starch, flour and other derivatives using the whole root has the potential to directly and indirectly solve the numerous advocacy challenges of zero toxic poisoning among the populace.

In several laboratory experiments, Tongolo poisonous root total cyanogens (a combination of hydrogen cyanide, cyanohydrins and non-glucose cyanide) were reduced by 99-100% using low-cost intrinsic procedures (Fig 1). This is good news because there is no need to force subsistence smallholders and small and medium processors to switch from traditional poisonous cassava to improved varieties. This novel approach, away from the conventional methods of fermentation and promotion of less poisonous varieties is a strategy that can offer novel inexpensive safe products to smallholders and medium processors and at the same ensures clean environment by utilizing the peels. Moreover, this novel approach can offer other added advantages of reducing the amount of energy needed to obtain water for processing, reduction in waste disposal and legislation costs and ensure sustainable systems.

### **BE INFORMED: POISONOUS CASSAVA CAN KILL**

The adage that '**prevention is better than cure**' is the best option to reduce exposure to cyanide poisoning.

Poisonous cassava roots can be made safe using a simple preparation procedure: wash fresh roots with clean water; peel, scrape off outer skin then rewash; crush/grate (pulp) or slice/chip (smaller size chips); squeeze out juice (pulp); ferment (heaping or soaking in water 3-5 days); roast; cool; sieve and pack. Cassava leaves can be pounded, washed 3 times with clean water and boiled/steamed or mixed with other sauces. They can also be dried, milled into powder.

**DON'T BOIL, ROAST AND EAT POISONOUS CASSAVA.**



## Conclusion

Cyanide poisoning continues to cause unrivalled deaths in spite of available improved processing technologies and wide coverage and public advocacy.

Potential and lethal non-cassava sources of cyanide poisoning obscure to the public are real and demand attention for successful and sustainable cyanide reduction programs.

New approaches of cyanide management, taking into consideration the reduction of cyanide at low costs while protecting the environment and concurrently uplifting the socio-economic status of vulnerable communities, create sustainable systems and necessitate expeditious and precise action.

Most importantly, as cyanide poisoning continues to pose health problems, particularly to those who eat cassava and the increasing urban health concerns, measures to control cyanide poisoning can best be managed by establishing a network of experts from different fields for sustainability.

EAT CASSAVA with fish, lean meat, turkey, eggs, soybeans, milk, yoghurt, liver or cheese in your daily meals. Foods with proteins and VitB12 have the potential to reduce some of the toxic effects of cyanide.

It can be very difficult to know that someone has been exposed to cyanide. It is always best, as the first option, to seek medical advice as fast as possible. Initial first aid can be given to people far away from health facilities (especially those who have eaten cassava) by putting the person in fresh air or giving activated charcoal. However, it is advisable not to force a person to vomit. This can result into second-hand infection. Unlike other poisons, cyanide quickly enters the body cells and vomiting may not help especially when a person breathes in a high dose.

Treatment for life-threatening cyanide poisoning is possible (at health centres) using cyanide antidote kit and other treatments as per medical prescriptions. Visit the health centre if you know have eaten poisonous cassava

## References

- <sup>1</sup>Bradbury, M. G., Egan, S. V. & Bradbury, J. H. (1999). Determination of all forms of cyanogens in cassava roots and cassava products using picrate paper kits. *J. Sci. Food Agric.*, 79, 593-601.
- <sup>2</sup>Burns, A. E., Bradbury, J. H., Cavagnaro, T. R. and Gleadow, R. M. (2012). Total cyanide content of cassava food products in Australia. *Journal of Food Composition and Analysis*, 25, 79–82.
- <sup>3</sup>Cardoso, A. P., Mirione, E., Ernesto, M., Massaza F., Cliff, J., Haque, M. R. and Bradbury, J. H. (2005). Processing of cassava roots to remove cyanogens. *Short Communication. Journal of Food Composition and Analysis*, 18, 451–460.
- <sup>4</sup>Essers, A. J., Ebong, C., van der Grift, R.M., Nout, M.J., Otim-Nape, W. and Rosling, H. (1995). Reducing cassava toxicity by heap-fermentation in Uganda. *Int J Food Sci Nutr*, 46(2):125-36.
- <sup>5</sup>Hamel, J. (2011). A Review of Acute Cyanide Poisoning With a Treatment Update. *Critical Care Nurse*, American Association of Critical-Care Nurses, The InnoVision Group Public. Columbia, 12p. [www.cconline.org](http://www.cconline.org)
- <sup>6</sup>Nhassico, D., Muquingue, H., Cliff, J., Cumbana, A. and Bradbury, J. H. (2008). Rising African cassava production, diseases due to high cyanide intake and control measures. Review. *J Sci Food Agric*, 88, 2043–2049
- <sup>7</sup>Otim-Nape, G. W., Bua, A., Ssemakula, G., Acola, G., Baguma, Y., Ogwal, S. and R. (1998). Van der Grift, Cassava development in Uganda: A country case study towards a global cassava development strategy. In: FAO, 2005. A review of cassava in Africa with country case studies on Nigeria, Ghana, the United Republic of Tanzania, Uganda and Benin. *Proceedings of the Validation Forum on the Global Cassava Development Strategy, volume 2*. FAO Corporate Document Repository. [www.fao.org](http://www.fao.org)

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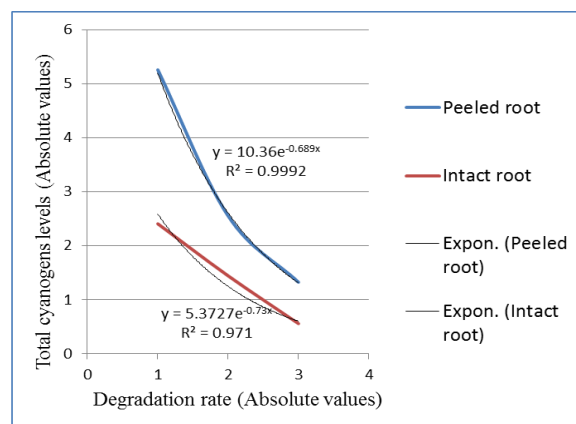
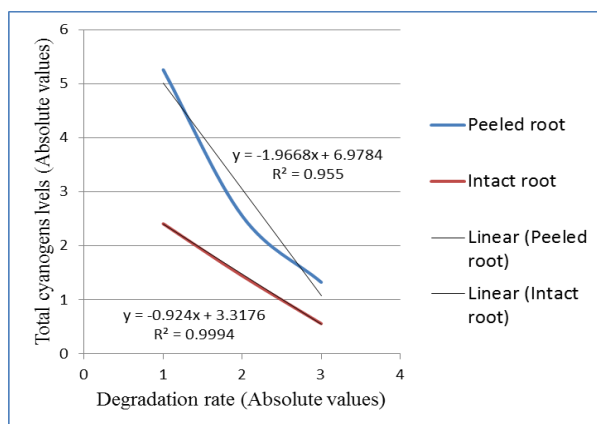


Fig.1 Cyanide reduction potential of intrinsic method linear (a) and exponential relationship graphs

### Consumption of cassava leaves among farmers from three geographical regions of Ghana

Cassava leaves are affordable and sustainable source of vitamin A<sup>1, 2</sup>, given their high  $\beta$ -carotene content of 20,803  $\mu\text{g}/100\text{g}$  dry weight<sup>3</sup>. They are also rich in protein (7.4 g/100g) and other micronutrients like iron (5.6 mg/100g) and zinc (5.0 mg/100g)<sup>3</sup>. A major constraint to their consumption is the presence of cyanogenic glycosides, however, with adequate processing, the cyanogens could be reduced to safe residual levels, and still conserve these key nutrients<sup>4</sup>.

A cross sectional survey was conducted in 3 geographical regions of Ghana on cassava leaf consumption. These were the southern belt (Asuansi, Abakrampa and Nyamedom), middle belt (Wenchi, Subinso No. 2 and Asuano) and the northern belt (Damongo, Sori No.2 and Congo village), as illustrated in Fig. 1 with their ecological factors. Depending on the number of cassava farm holders in those regions<sup>5</sup>, 37, 42 and 22 farmers from the south, middle and the north respectively were interviewed on cassava leaf consumption and processing methods employed. The results showed that consumption of cassava leaves depends significantly on geographical location ( $p$ -value=0.001), with most consumers residing in the northern belt (81.8%). Only 31.4% and 39.0% of the respondents in the south and the middle belts respectively were consuming cassava leaves. In southern and middle Ghana, reasons for not consuming cassava leaves were predominantly;

Ignorance of their edibility and preparation methods. Among the consumers, notably the northerners, cassava leaves were best harvested at 6 months old for good quality (greenness and softness) and quantity of leaves. Moreover, at periods when there vegetables, cassava leaves were harvested for vegetable, thus reinforcing cassava as drought, famine and war crop in a changing world<sup>6</sup>. were hardly other leafy. Three processing methods were identified namely, i) Boiling ii) Chopping followed by boiling iii) Pounding followed by boiling. The first two were exclusive to southern and middle Ghana, with about 97% southern belt respondents processing cassava leaves by "chopping followed by boiling". The northern belt solely processed cassava leaves by "pounding followed by boiling", before consumption. According to the majority of the northerners (63%), they had traditionally been taught to pound and boil cassava leaves before consumption. They did not know the rationale behind these processing techniques. It was evident that, as opposed to northern Ghana, the southern and the middle parts of Ghana were neither familiar with cassava leaves consumption nor knowledgeable about the adequate techniques needed to prepare cassava leaves for consumption. From literature, "pounding followed by boiling" reduces cyanide drastically to a residual level of 2.2%<sup>7</sup> compared to "chopping followed by boiling" which brings the residual cyanide to a moderate level of 14.5%<sup>8</sup>. By pounding, the enzyme linamarase is freed from its cellular compartments and brought into contact with the cyanogenic

glycosides, which are also found in different compartments, for enzymatic catalysis<sup>9</sup>. As a result, much of the hydrocyanic acid is removed compared to "chopping followed by boiling" and "boiling" alone.

In conclusion, respondents from northern Ghana widely consume cassava leaves and employ adequate processing techniques to reduce the hydrocyanide content before consumption, as opposed to their counterparts from southern and middle Ghana. People who are familiar with cassava (leaves) have the right kind of knowledge in handling it<sup>10</sup>. Education on adequate processing of cassava leaves for consumption in Ghana will be most useful if targeted especially to the southern and the middle belts.

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### References

1. Siqueira EMA, Arruda SF, Vargas RM, Souza EMT. (2007)  $\beta$ -carotene from cassava (*Manihot esculenta* Crantz) leaves improves vitamin A status in rats. *Comparative Biochemistry and Physiology Part C* **146**: 235-240.
2. Stadlmayr B, Charrondiere UR, Addy P, Samb B, Enujiugha VN, Bayli RG, Fagbohoun EG, Smith IF, Thiam I, Burlingame B. (2010) Composition of selected foods from West Africa. Food and Agriculture Organization, Rome. pp 13-14.
3. Takyi EEK.. (1999) Children's consumption of dark green, leafy vegetables with added fat enhances serum retinol. *J. Nutr.* **129**: 1549-1554.
4. Bradburry JH, Denton IC. (2011) Mild methods of processing cassava leaves to remove cyanogens and conserve key nutrients. *Food Chemistry* **127**: 1755-1759
5. SRID-MoFA (2008) Statistical Research and Information Directorate, Ministry of Food and Agriculture-Ghana.
6. Burns A, Gleadow R, Cliff J, Zacarias A, Cavagnaro T. (2010) Cassava: The drought, war and famine crop in a changing world. *Sustainability* **2**: 3572-3607.
7. Ngudi DD, Kuo Y-H, Lambien F. (2003) Cassava cyanogens and free amino acids in raw and cooked leaves. *J Sci Food Agric* **41**: 1193-1197.
8. Nambisan B. (1994). Evaluation of the effects of various processing techniques on cyanogen content reduction in cassava. *Acta Horti* **375**:193-201.
9. Whyte WLB, McMahon JM, Sayre RT. (1994) Regulation of cyanogenesis in cassava. Cited: Montagnac JA, Davis CR, Tanumihardjo SA. (2009) *Comprehensive Review in Food Science and Food safety* **8**: 17-27.
10. Asiedu R. (2009) Cited: MediaGlobal. Saunders A. Ed. In Depth: Cassava's link to iodine deficiency requires further study. Retrieved from: <http://www.mediaglobal.org/article/2009-01-15/in-depth-cassavas-link-to-iodine-deficiency-requires-further-study-on-22-04-11>

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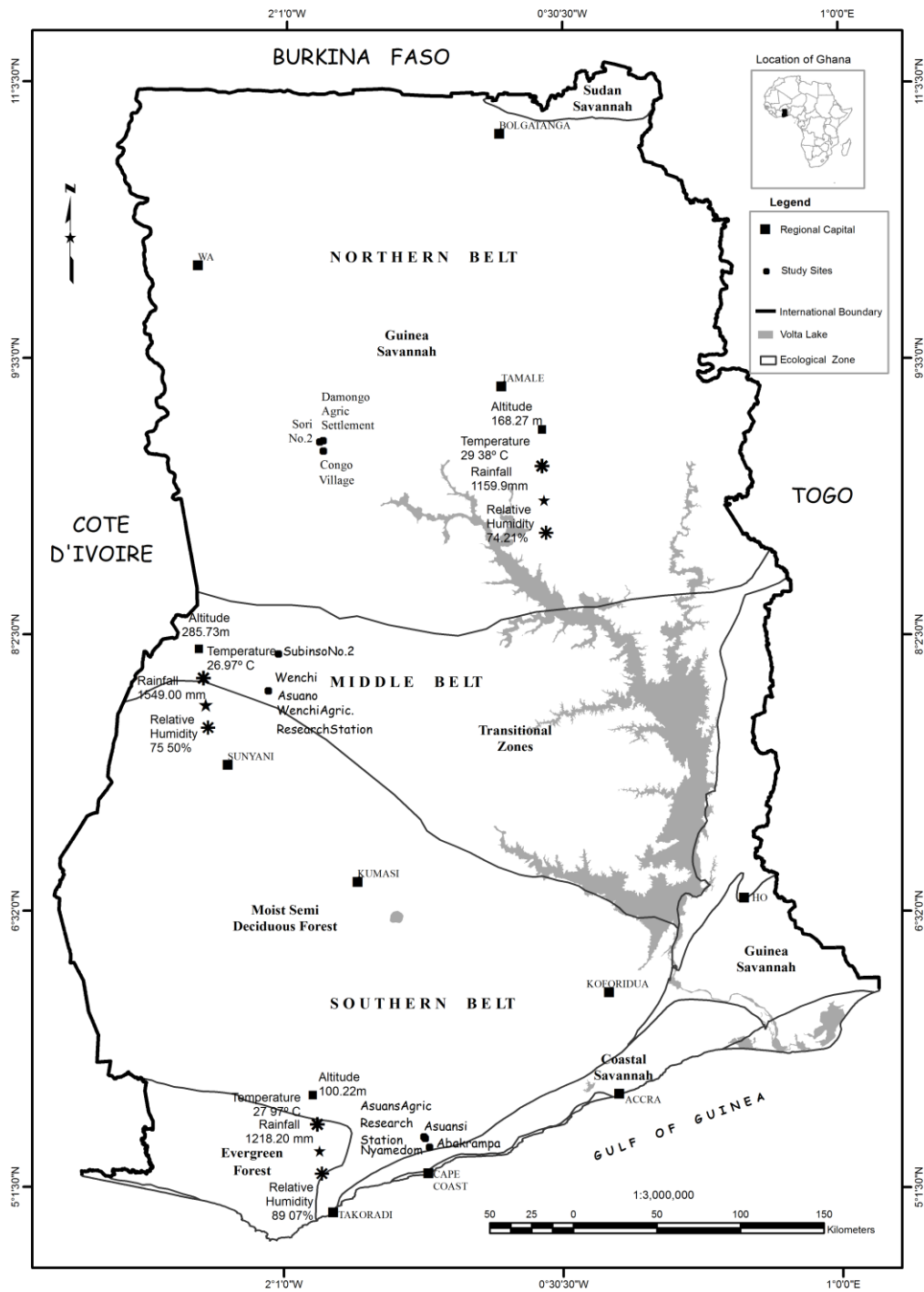


Fig 1 SURVEYED AREAS IN THE SOUTH, MIDDLE AND NORTHERN GHANA

**KONZO: The IBRO Africa Regional Committee (ARC) organizes its first Global Advocacy Workshop for Neuroscience in Kinshasa**

A Special Symposium on Konzo dealing with Recent Advances, Innovations and Civic Engagement took place on September 5 during the first Global Advocacy Workshop for Neuroscience of the International Brain Research Organization/ Africa Regional Committee (ARC) held in Kinshasa, Democratic Republic of Congo (DRC).

The keynote speech was given by Dr Jean Pierre Banea (Director of PRONANUT/ DR Congo) on "a century of konzo in Congo-Kinshasa: Myth or reality?" The aim of this keynote was to show the itinerary of konzo since its first occurrence in DRC till to date, by presenting views of the Ministry of Public Health in DRC. This presentation was outlined in four points: Brief history of konzo in the scientific literature, konzo as a public health problem in DRC and to conclude: is konzo a myth or a reality? The recognition of konzo as a distinct entity with a clear and simple set of diagnosis criteria makes konzo a reality and opens a way for research strategies and interventions to avoid konzo in affected areas. Many aspects of the disease still remain unknown and need further deep studies for clarification. Konzo is also an indicator of poor conditions of life.

After the keynote, a panel dealt with cassava, nutritional toxicity and epidemiology of childhood neurodevelopmental disabilities in Africa. Dr Michael Boivin from Michigan State University, USA talk about the neuropsychological effects of konzo in three learning objectives: Neurological symptoms of konzo, a neuromotor disease from toxic cassava, neuropsychology sequelae from konzo and ecological causes and interventions for konzo. The conclusion of this presentation was the findings of a pervasive subclinical neurocognitive effect in children with konzo. This provided the first evidence that researchers should be aware of that a motor proficiency examination can effectively characterize konzo severity.

Dr Delphin Diasolua Ngudi from Ghent University, Belgium outlined his presentation entitled: Konzo and neuroathyrim, nutritional challenges in three subchapters: epidemiology of konzo and neuroathyrim (two fraternal twin like diseases), a case study on neuroathyrim animal model from oxidative stress and networking by the CCDNN Newsletter. Konzo & Neuroathyrim should be considered and viewed as "Crippling hidden hunger" and thus they are CHRONIC NUTRITIONAL DEFICIENCIES. From the case study based on the article of Kusama Eguchi et al (2009) about hind-limb paraparesis in rat model for neuroathyrim associated with apoptosis and an impaired vascular endothelial growth factor system in the spinal cord. Participants learned that after creating stress to rat pups by separation from their mothers, followed by the subcutaneous L- $\beta$ -ODAP treatment, resulting in 4.6-fold higher incidence of the paraparesis compared with unstressed controls, they proposed a novel pathological process of motor neuron death induced by peripheral  $\beta$ -ODAP. To finish, an

advocacy of the CCDNN newsletter which is received by around 700 members worldwide was done. Participants were invited to join the network and to share short articles of their researches.

Next, Dr Ester Agbor from University Dschang, Cameroon provided a preliminary insight into the awareness and knowledge of konzo and TAN among rural women specifically in Andom village located in the East Region of Cameroon. The non-existence of rural public's awareness and knowledge towards konzo/TAN clearly suggest that there is an urgent need for health education in cassava producing/consuming communities in order to raise awareness/knowledge. Increasing awareness and knowledge of konzo/TAN will encourage consumers of cassava products to practice post-harvest processing techniques that eliminate cyanide, the major causative factor of konzo/TAN.

To close the first panel of the symposium, Dr Julie Cliff discussed about the occurrence and the prevention of konzo in Mozambique. She pointed out the recrudescence of konzo with the three large epidemics encountered in 1981, 1993 and 2005, respectively and the concerns which are the danger of further epidemics (unequal development, increasing rural poverty, neglect of agriculture and climate change) and long term impact of chronic cyanide intoxication. To conclude, she proposed diet diversification, process improvement and agricultural development as solutions to fight against konzo.

A second panel of the symposium discussed FAO agriculture projects and other food technology innovations (improved cassava dishes and nutraceuticals) in favor of konzo affected population.

Recommendation were made at the end of the symposium: All participants recognized that we need a multisectoral effort to eliminate konzo. Socioeconomic development of affected areas is the big challenge to deal with for the elimination of konzo. Sharing research findings can help to keep attention of decision makers and sponsors.

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