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

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Historical Awareness of Neurolathyrism, and Cassava Toxicity

Most people studying lathyrism are likely to have training in bio-scientific methods or in social science, yet there are considerable gaps of thinking and practice between different disciplines, e.g. from plant biologists to historians of food in human cultures. There are different approaches to what is considered 'well-founded knowledge', and how it may be established. This paper recognises such differences, and their relevance to knowledge-development in neurolathyrism, with brief comparison of cassava (manioc) and its toxicity. It also considers why a broader approach to knowledge is important, and whether historical knowledge can be made useful to people who face increased food scarcity, and are 'below the radar' of government attention.

Review articles in lathyrism often begin with a glimpse of history, citing ancient texts where *Lathyrus sativus* seems to appear, or archaeological reports of seeds found from antiquity. A recent review of South Asian research tabulates prehistoric remains of Indian pulses at 90 sites, 48 of which include *L. sativus*[1]. Some are from studies in the 1970s and 1980s, when advanced technology for identification and dating was hardly available. Across the sites where *L. sativus* was found, estimated dates extend from 2500 BC to 1600 CE. The earliest South Asian textual evidence possibly indicating lathyrism also has a wide date range. That medical text, attributed to Susruta, probably started as a collection on surgery from several centuries BC, and was expanded to its final form by the 5th century CE. The text itself, in Susruta's *Nidana-Sthana*, 2.1 (On Breath and Wind), is not very clear: "Someone who has tremors when starting forward or who seems to limp as he goes is to be known as 'pea-lame' (*kalaya-khanja*). The connections in the joints are

undone." The Sanskrit specialist translator cautiously notes that this "might conceivably" refer to lathyrism, yet *kalaya* is "normally taken to be simply the common garden pea", not the grass pea[2]. Translators having surgical rather than philological expertise identify *L. sativus* in this passage, without critical reservations[3].

Similar drawbacks occur in early European evidence of *Lathyrus sativus* and textual reports of possible lathyrism. Thirty European sites (amidst a wider tabulation) with archaeological finds of likely seeds, date back possibly to 8000 BC; yet it remains uncertain whether these were stray weeds, or cultivated *Lathyrus* species[4]. A site in Greece, early in the 4th millennium BC, shows grass pea "as frequent as pea and lentil", suggesting cultivation[5]. The Greek medical school of Hippocrates, from the 5th to 4th centuries BC, recorded that: "At Ainos, eating legumes continually, women, men, lost the power in their legs, and it persisted." Greek translit.: "*En AinO ospriophageuntes ksunechеOs, thEleiai, arsenes, skeleOn akratees egenonto, kai dieteleon*" (in *Epidemics,* book II, sect. IV: 3, with some variant readings)[6]. However, the vegetable eaten is unclear, as is the time period involved. Several authors, using this text, curiously specify "all the men and women", which would conflict with most lathyrism experience, where only a minority, mostly male, are actually lamed by prolonged heavy use of the grass pea. The Ainos event is widely cited as early evidence of *Lathyrus* spp. causing lathyrism, yet scholarly views differ, and the evidence remains uncertain[7].

Until recently, the antiquity of Cassava (*manioc*, or *yuca*) was also being introduced in Eurocentric academic works with vague mention of an Egyptian papyrus and the "poisonous properties of bitter almonds" in Dioscurides (1st century CE). Meanwhile, in Central and South America, micro-fossil studies have focused more specifically on pollen, starch grains and phytoliths, indicating "the domestication and spread of important native crops" including maize and manioc (*Manihot esculenta*, Crantz), "between 10,000 and 5000 years ago"[8]. This research field was already maturing 30 years earlier, as shown by a more demanding scrutiny of evidence in literature reviews[9]. Early Amerindian awareness of cassava toxicity is assumed from ancient evidence suggesting various processing methods such as "sun-drying, leaching followed by drying, and soaking in running water ... drying and roasting"[10]. Manuscript notes of cassava toxicity seem to have reached Europe in 1493 from the first voyage of Columbus, collated by Peter Martyr D'Anghera in *De Orbo Novo*, published informally in 1504, 1507, and formally in 1511. Peter refers to the root crop *yuca*, which the 'New World' islanders "first cut and squeeze (for it is juicy) then they pound and cook it into cakes ... they say the juice of yuca is more deadly than hemlock; if drunk, it kills on the spot {*qui epotus illico perimit*}. However the bread from the pulp, as everyone has experienced, is tasty and good for you"[11].

In Ethiopia, there is documentary evidence of popular awareness of the harmful potential of *Lathyrus sativus* in the mid-16th century CE, when the vernacular term transliterated as *gwaya* or *guaia*, having a 'breaking' or 'paralysing' sense, was given as equivalent for the Arabic *gilban, djilban* etc, meaning *L. sativus* (or possibly some botanical close relative)[12]. In India, the *Ain-i-Akbari* asserted the "unwholesomeness" of *kesari*, "a pulse, resembling peas, which is eaten by the poor" in the late 16th century[13]. The same word, *kesari* or *khesari*, is used now for the commonly eaten form of *L. sativus*. It was used by Francis Buchanan, surveying in Central India in the 1810s, in the earliest English medical description of the impairment caused by lathyrism in India[14]. Yet conflicting evidence caused Buchanan not to believe that *L. sativus* was responsible. In both countries there is probably some earlier manuscript evidence linking the plant and the risk, but there is very seldom clarity of botanical identification, or of the resulting impairment. China had earlier printed work combining careful descriptions and woodcut illustrations, e.g. the *Chiu Huang Pen Ts'ao* (On Wild Food Plants for Use in Emergencies) by Chu Hsaio, published in 1406 CE, including some *Lathyrus* species, and specific directions for preparation of some plants comparable to cassava in their toxicity[15].

What use is 'history'? Historical studies provide a fuller background to 'neglected diseases', so that modern scientists may at least start with whatever is already known and perceive some of the complexities that misled or puzzled earlier thinkers. Abyssinia's earliest detailed case histories from a lathyrism epidemic, following the great famine of 1888-1892, were published in 1899 by the Russian neurologist Friedrich Holzinger but, for lack of historians pursuing them, they went missing for a century, and remain uncited in modern scientific literature[16]. Maybe this was an 'own goal' by anglophone scientists who imagined that anything worth reading must be published in English; also Holzinger's brief title, "On lathyrism", shows no link to Ethiopia (then known as Abyssinia).

More remarkable is the disappearance of a detailed scientific and anthropological study on lathyrism in Central India by the experienced Indian Medical Service officer, Andrew Buchanan, commissioned by the [British] Government of India

and delivered in 1904[17]. Major Buchanan made a close study of earlier and current literature in five languages, and of district officers' reports responding to a specially commissioned lathyrism survey. He toured rural areas for three months, sampling villages with varying levels of lathyrism. He cross-questioned families, getting beyond the point where they merely gave the answer they thought the British officer wanted, and going deeper into the linguistic tangles of what foods they had actually eaten, how they were cooked, and what theories the villagers held on the causes of lameness. Buchanan organised and analysed the agricultural data, the times and seasons, village responses and scientific evidence, and the various hypotheses and complexities, offering reasons for his conclusions. Not all were correct, but his methodology, reasoning, and scepticism were appropriate for the complexities. His report was welcomed in the medical press, yet there was no apparent official follow-up. Soon, this "very important and comprehensive" report was lamented as "so little known, so difficult to procure"[18]. For a century, the British Library catalogue failed to show the report under its author's name, listing it only in a large collection of government reports on "other diseases". Finally, in a recent review of 244 documents on *Lathyrus sativus*, Buchanan's report is cited for four different reasons, including the curious point that many sufferers from neurolathyrism seemed well nourished, strong and hard-working[19]. There is still a dearth of rigorous observation of progressive neurolathyrism and penetrating enquiry into the dietary details. In this case, Buchanan's field observations and reasoning, from a time when the technology was primitive, still suggest why more complex hypotheses are needed to elucidate puzzles that confronted earlier scientists and remain unresolved.

Politicians everywhere are inundated with vast and complex problems lacking any simple solution. Some of these floods seem to have been imported along with 'western civilisation', and hardly belong to the 'southern' countries. To gain decision-makers' attention, it is useful to be able to prove that *Lathyrus* species have a long history within the country, showing some good features as well as bad. Still better, if one can show that indigenous knowledge and skills have historically been used to counter the problems of lathyrism, in appropriate, low-cost ways. An attractive solution may be one that can be explained comprehensibly to people without science degrees; and where the historical heritage is blended with technical innovation, giving not only 'invisible' benefits (i.e. rural people no longer being crippled, as they learn safer preparation of the least toxic varieties), but also positive gains (a readily available fodder crop, and a good famine safety-net when combined with feasible dietary supplements). Building the case requires well-founded knowledge from many sides.

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Acute cyanide poisoning from cassava: is it still common?

Acute cyanide poisoning from cassava causes nausea, vomiting, dizziness, weakness, headache, abdominal pain, diarrhea, sometimes collapse, and occasionally death.

In 2010, a literature review was carried out as part of an exercise to measure incidence and prevalence rates of cassava-related disease for the Food borne Disease Burden Reference Group (FERG) of the World Health Organization (WHO). Two additional reports located in 2012 are included.

Acute cyanide poisoning usually goes unreported because it may be common, is mostly non-fatal and often occurs in geographically remote areas. The information that follows is certainly incomplete, and we would be grateful if readers could send additional information.

Reports are ordered by geographical location:

Asia, South-East

Sporadic incidents of acute cyanide poisoning from cassava have been reported in the medical literature, often in the past. For example, in Malaysia, cases were reported in children in 1978 and 1992[1, 2], and in Thailand in 1999[3]. In 1987, a Vietnamese physician reported that hospitals could potentially see several cases of acute poisoning each year[4].

Two more recent reports suggest that poisoning still occurs. For example, in the Philippines, a report in 2005 mentioned poisoning from cassava, particularly among the indigenous people of Mindanao, due to hunger and drought[5]. In Thailand, the Ministry of Public Health reported several hundred cases of poisoning between 2000 and 2005 in their epidemiological bulletin (http://epid.moph.go.th/fact/Cassava.htm. This site is no longer available).

Latin America

In Latin America, Venezuela, is the only country in the Americas to have reported cases of cyanide poisoning in the medical literature, back in 1992[6].

Oceania

In 1990, Brian[7] reported a case of suspected poisoning in the Papua New Guinea highlands, and noted that cyanide poisoning from cassava occurred there. The first undated edition of the PNG paediatric textbook mentions cyanide poisoning from cassava[8]. More recently, John Vince, Professor of Paediatrics at the University of Papua New Guinea, noted that cases of cassava poisoning are rare, but are often fatal (email communication, 2010).

Sub-Saharan Africa

The first written report of acute poisoning from cassava comes from the slave trader Tippo Tip[9]. Individuals accompanying Emir Pasha's expeditions in the late 19th century also reported poisoning from cassava[10].

Central

Reports of acute cyanide poisoning do not feature in the extensive medical literature on cassava and disease in the DRC. In 2006, Metre[11] recounted that cyanide intoxication was widely reported in South Kivu Province, mainly in the rural areas, and that severe and deadly intoxications affected the elderly and children.

East and Southern

In East Africa, Mlingi[10] cited anecdotal and other reports of lethal cases of poisoning from cassava in both Tanzania and Kenya between the 1960s and 1980s.

In a survey in southern **Ethiopia**, published in 1998, Abuye et al.[12] found that 50% of individuals consuming cassava reported symptoms of poisoning. Acute cyanide intoxication from a cassava meal in children was also reported in another survey[13].

In **Kenya**, fatal cases were reported in 2002 during a famine in Butere/Mumias District; the cassava was believed to have originated in Uganda[14]. In 2008, Nungo[15] noted media-reported cases of cassava poisoning and sometimes deaths in western Kenya.

In **Malawi**, a study reported in 2000 found that 36% of interviewees had seen or heard of others in the community who had suffered acute poisoning[16].

In **Mozambique**, symptoms of acute poisoning have been frequently reported during konzo epidemics[17, 18]. In a survey in a previously konzo-affected community during a drought, 53% of the interviewees complained of symptoms of acute poisoning in the past two weeks (unpublished data in possession of the author).

Three outbreaks of acute intoxication were recorded in non-cassava staple areas of Mozambique in the 1980s and 1990s, all associated with the consumption of newly introduced bitter cassava during drought[19, 20].

In **Namibia**, a 2012 news report cited a suspected fatal case of cyanide poisoning from cassava in Ruacana Constituency. Six children and three adults were affected[21].

In 2000, in Kivu Region, **Rwanda** at least 20 refugees were reported to have died from cassava poisoning[22].

In **Tanzania**, in 1988, an extensive outbreak of acute intoxication from cassava occurred in southern Tanzania, during a prolonged drought [23].

When studying cassava consumption and goitre, Mlingi et al.[24] found that 14% of women stated that they had experienced dizziness after eating meals associated with cassava consumption. Later, in an investigation of a konzo outbreak in 2002, Assey and Mtunda[25] noted that acute intoxication occurred during the rainy season when no other supplementary food was available.

In an old report from **Zimbabwe**, a retrospective study of food poisoning admissions to urban referral hospitals between 1980 and 1989 found five cases of cassava poisoning [26].

West

Acute cyanide poisoning from cassava was common in West Africa in the early part of the 20th century[27]. In 1994, Osuntokun[28] stated that deaths after consuming cassava were still frequently reported in Nigeria. Also in 1994, Akintonwa et al. [9] noted that from 1989 onwards, cases of acute poisoning from cassava were commonly reported in the news media. They also reported two fatal cases admitted to, and other non-fatal cases in Lagos and Epe. In the latter area, the population was aware of acute cassava toxicity, which occurred occasionally and seasonally in children. In a survey of paediatric admissions for poisoning at the University of Maiduguri Teaching Hospital in north-eastern Nigeria from 1984 to 2003, 19 cases (16.8 %) resulted from cassava poisoning[29].

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International conference on "Recent Trends in *Lathyrus sativus* Research" (Hyderabad, India, November 8-9, 2012).

Lathyrus sativus (grass pea) came to the limelight in India again due to the severe shortage of pulses production. Increased burden on import made the policy makers to retrospect and pay attention to this neglected crop in the Indian Subcontinent. This pulse has been cultivated and consumed by millions of people without suffering from neurolathyrism. It is often considered by the farmers as a lifesaver crop[1] or as insurance crop[2]. It is also a cheap source of protein for human and animal consumption[2]. Grass pea is a high yielding, drought tolerant legume crop containing 31% protein, 41% carbohydrate, 17% total dietary fiber[3,4]. The protein content of grass pea seeds which is higher than the average percentage of protein content (21–25%) in other legume seeds[5]. Homoarginine present in *L. sativus* seed[6] can produce nitric oxide in the body and may help in the prevention of cardiovascular disorders (CVD) caused by endothelial dysfunction.

Neurolathyrism occurs when *L. sativus* is consumed as the sole staple for extended periods, such as during severe drought and famine as occurred in the recent Ethiopian famine in 1996–1997[7]. Recurrence of famines and droughts has declined in India in recent past and this can be noted as one of the major reasons for the decrease in neurolathyrism cases in the Indian Subcontinent.

Keeping in view all the beneficial values of grass pea and to promote better usage of grass pea, an international conference on "***Recent Trends in Lathyrus sativus Research"*** was conducted at the National Institute of Nutrition (NIN), Hyderabad on November 8-9, 2012.

The two days conference encompassed four sessions and 32 presentations including 4 plenary lectures by eminent people in various areas of *Lathyrus* research and 7 posters covering topics on agronomy, molecular studies, animal studies and future of the *Lathyrus* pulse.

Dr. Fernand Lambein from Ghent University in his key note address said that the etiology of neurolathyrism has been oversimplified and several factors such as oxidative stress, mineral content, the total diet, and deficiency of essential amino acids have been neglected. He also mentioned that recent epidemiological and pharmacological studies indicated the importance of essential amino acids and oxidative stress in the incidence of neurolathyrism, and proposed strategies for prevention.

Dr. S.L.N. Rao from Osmania University (Retd-professor, Biochemistry Department) delivered a lecture on the paradigm shifts in our understanding and study of neurolathyrism and the potential role of homoarginine in preventing CVD. Dr. Arjun Khandare, organizing secretary of the conference, delivered a lecture on the present status of neurolathyrism and on the consumption pattern of grass pea in India.

Dr. Marijke Van Moorhem from Ghent University presented her findings on the ability of β-ODAP to cross the blood brain barrier and triggering oxidative stress.

Dr. Shantilal Kothari, social activist who has been front runner in leading the nation-wide movement to lift the ban, has given a lecture on the nutritional value of grass pea and its availability at low cost.

The 'International Conference on Recent Trends in *Lathyrus sativus* Research' culminated with a panel discussion on the future directions related to the pulse, its production, consumption and research in India.

Dr. Kalagam Polasa, Director in charge, NIN chaired and moderated the session. Speaking on the occasion Dr. Fernand Lambein said that there is no doubt that *Lathyrus sativus* has positive advantages from agronomic viewpoint, however he explained that the research has focused too much on β-ODAP while the deficiency of sulfur amino acids methionine and cysteine was overlooked. He said in the Indian scenario, lifting the ban on the pulse without accompanying measures could wrongly be understood by the people to mean that everything is safe and good with the pulse and its consumption. He also stressed that people who are hungry (especially in drought hit areas) should never consume it as staple.

Responding to few concerns about the safety of soy as compared to Khesari dal, Dr. Polasa said that the concern about soy is mainly of intolerance or allergenicity, whereas it is toxicity in the case of Khesari dal.

Dr. Shiv Kumar Agrawal (ICARDA, Syria) in his remarks said that pulse production in India after independence has almost remained the same. At the time of Independence, he said that the per capita consumption was about 60-65 g/day, which has now come down to 30-35 g/day. He said that in the context that its consumption and production is already low, there should not be much concern about it. When there is some evidence on nutritional benefits of the pulse and β-ODAP is the only problem we should try to manage this and help it detoxify and use the pulse for the benefit of the poor. The Discussion ended with a vote of thanks by Dr. Khandare.

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Neurolathyrism in Bidar and Medak districts of South India

A cross sectional survey of 59 villages with a total population of 89,913 on the banks of the river Manjira in Medak district of Andhra Pradesh and Bidar district of Karnataka in South India revealed 29 cases of neurolathyrism spread over 21 villages. The cases were all in stage 1 (no stick) or stage 2 (one stick). The disease occurred sporadically between 1964 and 1990. Dietary history of the affected respondents was recorded which showed that at the period of survey their *Lathyrus* intake was between 2.4 and 6.8 *g/cu/day (Group1)* compared to an intake *of 1.2-10.8 g/cu/day* in non-lathyrism (group ll) families (Table 1).

The cultivation and consumption of *Lathyrus* decreased during the last two decades as a consequence of a ban on sale of *L. sativus* under the prevention of food adulteration Act (PFAA). At the time of survey, the price of *Lathyrus* seeds (25 rupees/kg) was considerably higher than the price of rice (5 rupees/kg). No new cases occurred after 1990.

Table 1: Average *Lathyrus* intake in group I (neurolathyrism) and group ll (no neurolathyrism) cases.

Group average lathyrus intake	male		female		total	
(g/cu/day)	n	%	n	%	n	%
(g/cu/day)						
I (lathyrism):						
No stick stage	10	37	2	100	12	41.4
One stick stage 2.4-6.8*	17	63	0	-	17	58.6
Total	27	100	2	-	29	100
II (No lathyrism) 1.2-10.8**	-	-	-	-	-	-

* Age group from 7 to 17 years, low intake due to cases in families
** Age group from 1 to 17 years
Cu: consumption unit containing the daily caloric requirement, calculated according to age, gender and physiological status.

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Residual Cyanide content In Cassava Product of India

Tapioca\Cassava (*Manihot esculanta* Crantz. Euphorbiaceae) which is highly cyanogenic, is the third major food of the tropics after rice and maize. The cyanogenic potential of cassava flour may cause deleterious health effects and is a serious public health problem[1].

Cyanide is lethal to humans at a dose rate 0.5-3.5 mg HCN\Kg body weight. The human body can detoxify up to 100 mg of cyanide per 24 hours via conversion to thio-cynate[2].

Apart from rare fatalities due to consumption of Cassava there is strong evidence that several neurological disorders such as tropic ataxic neuropathy and epidemic spastic paraparesis are caused by long time chronic ingestion of sublethal

Different Types of Sago Brands

doses of cyanide from Cassava[3, 4].

Table 1: Residual Cyanide Content in Different Brands of Cassava Products from India.

Sr. No.	Name of Sago Brand	Treatment	Test	Amount of HCN in ppm
1.	Cow Brand	Sago powder + D.W. Sago powder + D.W.+ C.E.	+ve +ve	10 10
2.	Darshan Brand	Sago powder + D.W. Sago powder + D.W.+ C.E.	+ve +ve	10 10
3.	Sacha moti Brand	Sago powder + D.W. Sago powder + D.W.+ C.E.	+ve +ve	10 10
4.	Vara Lakshmi Brand	Sago powder + D.W. Sago powder + D.W.+ C.E.	+ve +ve	10 10
5.	Shiv Shankar Brand	Sago powder + D.W. Sago powder + D.W.+ C.E	+ve +ve	10 10
6.	Nylon Brand	Sago powder + D.W. Sago powder + D.W.+ C.E.	+ve +ve	10 10

D.W. - Distilled Water
C.E - Cuscuta Extract

Nowadays Tapioca is extensively cultivated in the southern part of India to produce an edible product known as sago (sabudana). During the processing to obtain this product most of the cyanide is removed. However, the possibility of residual cyanogen in cassava products can not be ignored[5]. The processing of cassava is more or less efficient in eliminating maximum cyanide content from the finished product[4], but it is observed that some small amounts of cyanide still remain in the final finished product. Different brands of SAGO or SABUDANA, finished commercial products of cassava were examined for the presence of cyanide with the help of a field kit developed and provided by Bradbury et al [6] .

The results indicate that none of the commercial brands of SAGO in India examined contains more than 10 to 15 ppm of cyanide. This is within the range of approximately 100mg of cyanide the human body can detoxify in 24 hours via it's conversion into thiocyanide[1]. The cassava products if wetted for about 5 hours show remarkable reduction in cyanide content[6]. In India the processed SAGO or SABUDANA is soaked at least for 2 hours before use, hence seems to be quiet safe for consumption unless utilized in large quantity.

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