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Gross and microscopic lesions of cyanide poisoning in a West African Dwarf goat following consumption of raw cassava root tubers and leaves: A case report

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Abstract

Ruminants have been reported to be comparatively more vulnerable to cyanide poisoning than monogastrics, due to the higher acidity and unique microbial characteristics of their gut environment. Although toxicosis and the pathophysiology of cyanide poisoning in ruminants is well known and widely reported, the specifics of its pathologies in the West African Dwarf (WAD) goat has not been properly documented. This article described the pathological changes in a WAD goat suspected of cyanide poisoning following the consumption of raw cassava root tubers and leaves. The carcass of a one-year old male WAD buck was presented at the Post Mortem Unit of the Department of Veterinary Pathology, College of Veterinary Medicine, Michael Okpara University of Agriculture, Umudike, Abia State for examination. The animal had a history of returning from the grazing fields frothing from the mouth and with a bloated abdomen and a staggering gait, before sudden death. Physical examination revealed no evidence of snake bite, trauma or swollen lymph nodes. Post mortem findings showed congestion of the lungs, liver, kidneys and the intestines. The pericardium, abdominal mesenteries and the liver capsule were all cloudy and thickened. The stomach contents included large quantities of chewed cassava root tubers and leaves. There were petechial and ecchymotic haemorrhages on the walls of the heart. Histopathology showed generalized congestion and diffuse necrosis of these organs. These pathological findings mimicked those associated with hypoxemia, which concurs with earlier reports that cyanide poisoned blood is unable to release its oxygen content to tissues, despite being saturated with oxygen.

Keywords: Cyanide poisoning; Cassava root tubers and leaves; West African Dwarf (WAD) goat, Pathological findings; Case Report.

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Introduction

Cyanide, also known as hydrocyanic acid (hydrocyanide) or prussic acid, is a compound containing the "– C≡N" group (CN^-) in its chemical structure. It is a very lethal and rapidly acting chemical poison that affects mammals (Stoltenow and Lardy, 2012), with ruminants being particularly vulnerable (Ufaysa, 2019). Cyanide poisoning commonly results from exposure to cyanide ions (CN^-) contained in cyanogenic substances. In general, monogastrics (with pH 3.0 in the stomach) have a detoxification mechanism which can prevent death when cyanide release is slow; this is absent in ruminants (with pH 7.0 in the stomach) (Oke, 1969; Cope, 2021). The greater susceptibility of ruminants to cyanide poisoning has been reported to be as a result of the rumen environment which is mildly acidic, with high water content, and with a microflora that readily converts cyanogenic glyocosides in plants to free cyanide gas (Arnold *et al*., 2024).

Cyanide is abundant in the environment, as it is found in some plants, some fumigants, soil sterilizers, fertilizers and in rodenticides (Cope, 2020). Some common plants abundant in nature and sometimes used as animal feed are natural sources of cyanide, as they are known to harbour large quantities of cyanogenic compounds. These cyanogenic compounds are called cyanogenic glycosides, and are contained in the epidermal cells of these plants with the enzymes that enable cyanide production contained in the mesophyll cells (Stoltenow and Lardy, 2012). Some common cyanogenic plants include bamboo shoots, sorghum and related species, Sudan grass and cassava. Cyanogenic glycosides on their own are generally non-toxic. However, when these plant parts containing cyanogenic glycosides are consumed, breakdown of the plant parts following maceration by mastication or by gut microflora ruptures the plant cells and allows the cyanogenic compound and the enzyme (beta-glucosidase) to combine, leading to the release of cyanide which is toxic to both humans and animals (Kwok, 2008; Stoltenow and Lardy, 2012). It has been estimated that up to 12,000 plant species produce and sequester cyanogenic glycosides (Ndubuisi and Chidiebere, 2018).

Cassava (*Manihot esculenta*), a perennial root crop native to Africa and South America contains two cyanogenic glycosides, linamarin and lotaustralin, which on enzymatic hydrolysis leads to the production of hydrogen cyanide (Cereda and Mattos, 1996; Ndubuisi and Chidiebere, 2018). It has been reported that cassava grown during drought are especially high in these cyanogenic glycosides (Ndubuisi and Chidiebere, 2018). The cyanogenic potential of cassava is a factor that has seriously guided the processing and constrained the use of cassava as food or feed for both humans and animals (Ihedioha, 2002; Ihedioha and Chineme, 2003). Despite its cyanogenic potential, cassava remains a staple in some parts of the world and as one of the highest sources of energy for humans and animals. Cassava is abundantly cultivated in Nigeria both at commercial and at subsistent levels. In fact, Nigeria remains one of the world's highest producer of cassava (FAO, 2004).

Once cyanogenic plants are consumed by ruminants and broken down, either by mastication or by the gut microbes, the cyanide released rapidly enters the blood stream and is transported throughout the body of the animal. Cyanide inhibits oxygen utilization by the body cells (Stoltenow and Lardy, 2012). Classical acute cyanide poisoning occurs when CN^- released from the cyanogenic glycosides binds to, and inhibit the ferric (Fe^{3+}) heme moeity form of mitochondrial cytochrome oxidase a_3 (cytochrome c oxidase). This inhibits the terminal enzyme in the respiratory chain halting electron transport and oxidative phosphorylation, which is essential to the synthesis of adenosine triphosphate (ATP) and the

continuation of cellular respiration (Hamel, 2011). This will cause failure of the mitochondrial electron transport chain, leading to reduction of O_2 to H_2O with the consequences being an arrest of aerobic metabolism, systemic hypoxia and death from tissue anoxia (Cope, 2021). Predictably, tissues that are heavily dependent on aerobic metabolism such as the heart and brain tissues will be most particularly affected (Cope, 2021).

Clinical signs of cyanide poisoning reported in affected animals include rapid and laboured breathing, irregular pulse rate, frothing at the mouth and/or nostrils, bloating, dilated pupils, generalized muscle tremors, staggering and eventual collapse. The mucous membranes are bright red due to the haemoglobin being saturated with oxygen (Arnold *et al*, 2024), but this become cyanotic and fade following death of the animal (Cope, 2021). These are signs generally associated with hypoxemia. In cyanide poisoning, although oxygen is present in the blood, it is not accessible to the cells. Affected animals have been reported to die within 1 – 2 hours after consuming lethal quantities of the cyanogenic plant and within 5 – 15 minutes of developing clinical signs (Ufaysa, 2019).

The lesions of acute cyanide poisoning have not been fully reported on. Gross and histologic lesions are reported to be inconsistent (Cope, 2021). Lesions described are those consistent with systemic hypoxia and tissue anoxia, and include congestions or haemorrhages of the liver, tracheal mucosa, and lungs with some froth present in the respiratory passages. There had been reports of lesions of cyanide poisoning in both humans and animals, but there are no reports in literature available to the authors on the lesions associated with cyanide poisoning in West African Dwarf (WAD) goats following consumption of cassava root tubers and leaves. The present article reported the gross and histopathologic findings in a West African Dwarf goat suspected of cyanide poisoning following the consumption of raw cassava root tubers and leaves.

Case Presentation

The carcass of a one-year old male West African Dwarf goat was presented for necropsy at the Post Mortem Unit of the Department of Veterinary Pathology, College of Veterinary Medicine, Michael Okpara University of Agriculture, Umudike, Abia State, Nigeria. The owner reported that the buck, with the rest of the flock, had gone out for a few hours to graze. When the flock returned, the buck was seen to be frothing from the mouth and had a bloated abdomen. It was also observed to be staggering in gait. The buck died shortly after.

Physical examination: The carcass was bloated (Figure 1) and appeared to be in fairly good body condition with no swollen superficial lymph nodes. There was no apparent evidence of snake bite or physical trauma. There were fleas present on the carcass.

Gross lesions: Post mortem examination revealed oedema of both lungs with severe congestion of all the lobes (Figure 2), but with no consolidations. The cut surface of the lungs was severely congested (Figure 3). The tracheal mucosa was haemorrhagic and the lumen was filled with frothy muco-catarrhal exudates (Figure 2). The pericardium of the heart was cloudy and thickened and on the walls of the heart were petechial and ecchymotic haemorrhages (Figure 2). The liver had generalized congestion and the serous capsule was cloudy and thickened (Figure 4). The kidneys were slightly rounded and showed generalized congestion (Figure 5). The spleen appeared normal but diminished in size with very sharp borders (Figure 6). The abdominal mesenteries were cloudy (Figure 6), and the stomach was bloated with gas. There was

congestion and catarrhal enteritis affecting the large and small intestines (Figure 7). When cut open, the stomach ingesta was seen to contain many pieces of chewed cassava root tubers and leaves (Figure 7).

Microscopic lesions: Microscopic examination showed interstitial and intra-alveolar oedema of the lungs as well as generalized vascular congestion (Figure 3). The liver sinusoids as well as the capsule were distended with oedema fluid (Figure 4). Some of the sinusoids were also congested while the hepatocytes showed moderate to severe necrosis (Figure 4). There was a generalized congestion of the kidney vasculature and severe necrosis of the tubules (Figure 5). Haemorrhages and mild haemosiderosis were also observed (Figure 5).

Diagnosis: A diagnosis of generalized systemic tissue hypoxia and multi-visceral congestion due to cyanide poisoning, occasioned by consumption of cyanogenic cassava root tubers and leaves was made.

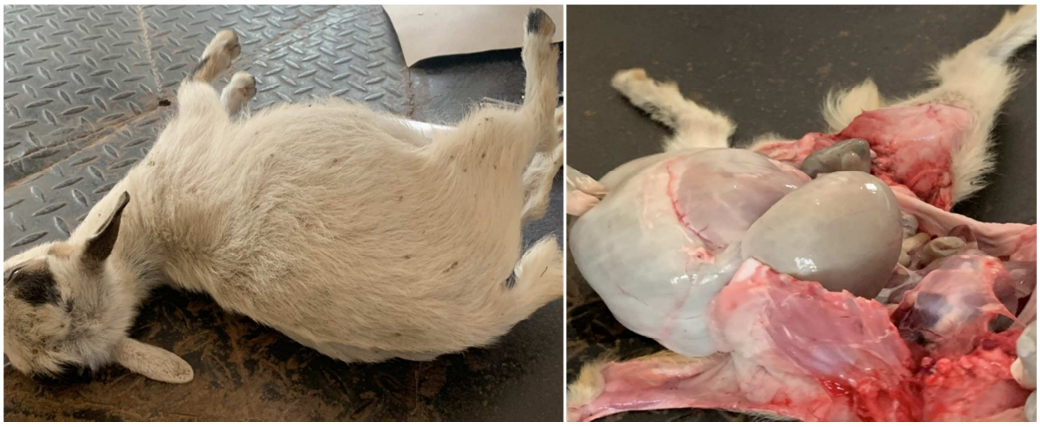

Figure 1. Carcass of a West African Dwarf (WAD) goat that died of suspected cyanide poisoning, after consuming raw cassava root tubers and leaves.

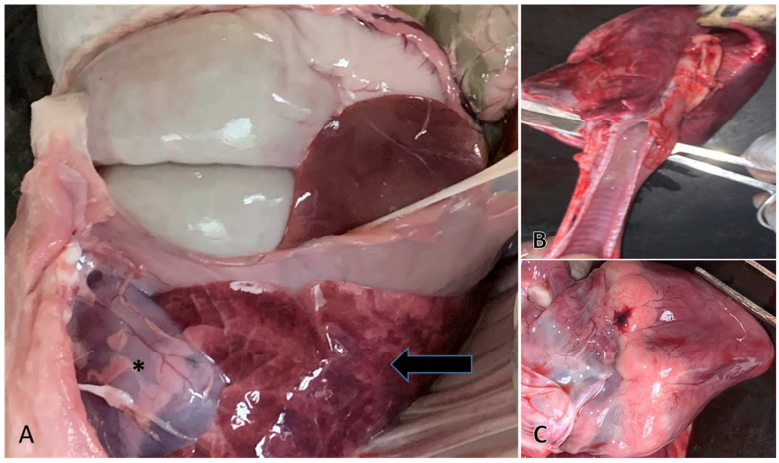

Figure 2. A – Oedema and severe congestion of all lobes of the lungs (arrow) and cloudiness of the pericardium (*) in a West African Dwarf (WAD) goat suspected of cyanide poisoning following consumption of cassava root tubers and leaves. B – The wall of the trachea was haemorrhagic and the lumen filled with catarrhal exudate. C – The walls of the heart showed petechial and ecchymotic haemorrhages.

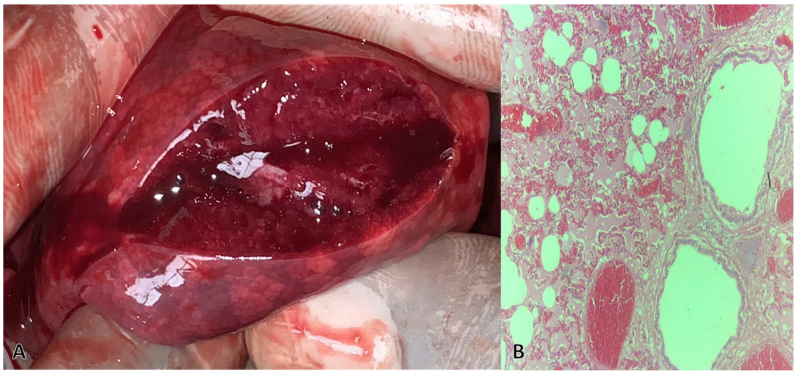

Figure 3. A – The cut surface of the lungs of a West African Dwarf goat suspected to have died of cyanide poisoning following consumption of cassava root tubers and leaves, showing severely congested and oedematous parenchyma. B – The histopathology section of the lung tissue, showing oedema with congestion (H & E, × 10).

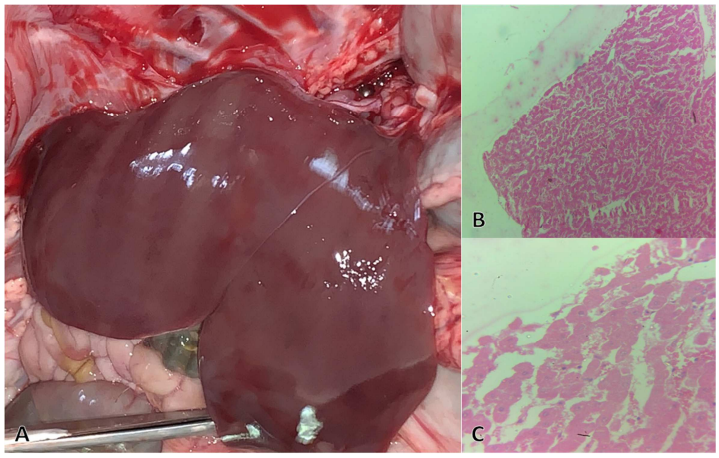

Figure 4. A – Enlarged and severely congested liver of a West African Dwarf goat suspected to have died of cyanide poisoning following consumption of cassava root tubers and leaves, with the serous capsule cloudy and thickened. B – Histopathology section of the liver showing disruption of the liver parenchyma and distension of the sinusoids and liver capsule with fluid (H & E, × 10). C – Higher magnification of the histological section of the liver, showing generalized necrosis of the hepatocytes (H & E, × 40).

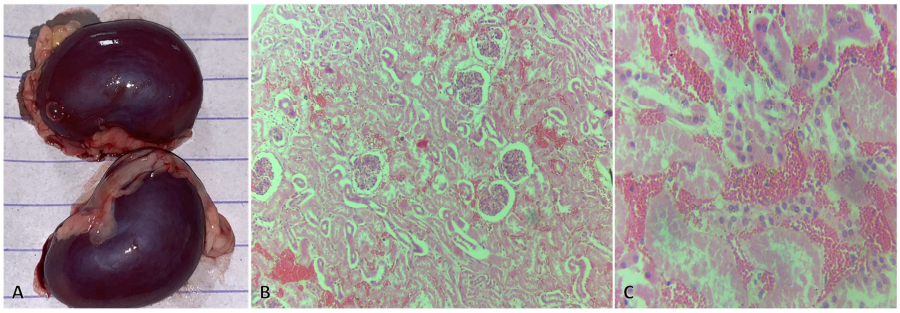

Figure 5. A – Severely congested and rounded kidneys of a West African Dwarf goat suspected to have died of cyanide poisoning following consumption of cassava root tubers and leaves. B – Histopathology section of the kidney showing generalized congestion, haemorrhages and necrosis of the renal tubules (H & E, × 10). C – Higher magnification of the kidney section, showing areas of red cell extravasation with mild hemosiderosis (H & E, × 40).

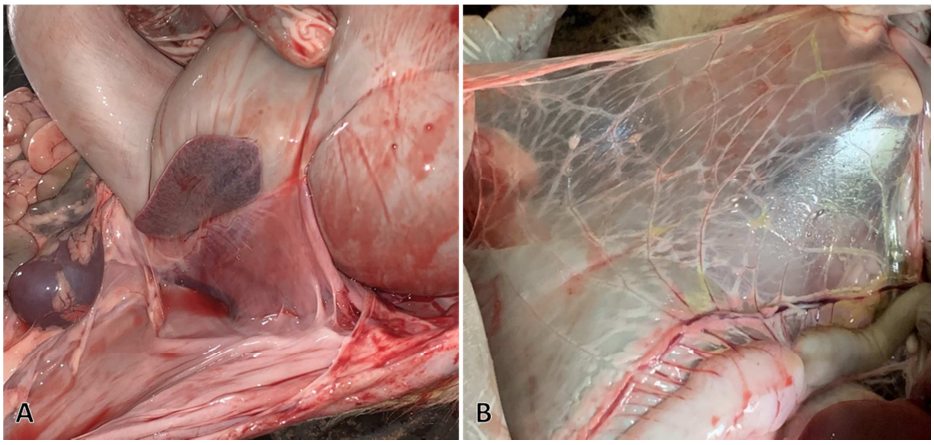

Figure 6. A – Cloudy and thickened abdominal mesenteries of a West African Dwarf goat suspected to have died of cyanide poisoning following consumption of cassava root tubers and leaves. B – The spleen was markedly decreased in size showing very sharp edges/borders.

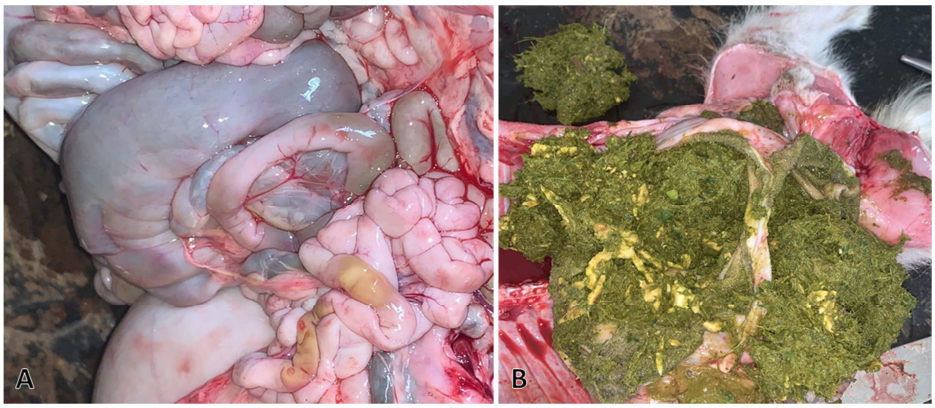

Figure 7. Enteritis and congestion of the intestines (A), with chewed pieces of cassava root tubers and leaves in the stomach ingesta (B) of a West African Dwarf goat suspected to have died of cyanide poisoning following consumption of cassava root tubers and leaves.

Discussion

In ruminants, cyanide released from cyanogenic plants can be quickly absorbed from the gastrointestinal tract into the bloodstream. In the blood, the cyanide prevents the release of oxygen from red blood cell haemoglobin to tissues (Stoltenow and Lardy, 2012). This phenomenon deprives the tissues of oxygen, leading to low oxygen tension (hypoxia) or complete lack of oxygen (anoxia) in tissues, depending on the severity. Factors that that had been reported to cause tissue hypoxia include: conditions of partial or complete blockage of arterial blood supply to an organ or part of an organ, due to thrombosis, tumours, vascular constriction etc.; conditions of very high altitudes, where the level of atmospheric oxygen taken in is very low; and conditions where the function of the heart or the lungs are compromised and are not able to effectively perform their primary functions of blood distribution (heart) or gaseous exchange (lungs) (MacIntyre, 2014; Bhutta *et al.,* 2024).

The heart and the brain, because of their very high energy demands, are the first organs to be affected when oxygen levels in blood fall (Cope, 2021). When the heart is affected, it

becomes too weak and unable to effectively pump blood into the general circulation leading to stasis of blood and consequent congestion in several organs and body parts. It is thought that this pump failure in the heart may have been responsible for the severe and generalized congestion seen in the various visceral organs of the goat in the present case under study.

Also, the fact that cyanide is rapidly absorbed into the bloodstream, and prevents haemoglobin from releasing oxygen to tissues, means that even the circulating blood is incapable of supplying oxygen to the tissues leading to tissue hypoxia. When tissue hypoxia occurs, the energy requirement of tissues can no longer be met causing a fall in cellular levels of ATP. This deficiency in highly needed cellular ATP deprives the cells the energy to maintain the integrity of their membranes leading to degenerative and necrotic changes (Flood *et al.,* 2023). It is believed that this tissue hypoxia was responsible for the widespread tissue necrosis that occurred in most of the visceral organs of the WAD goat.

The oedema recorded in the WAD goat is believed to be majorly a consequence of congestion, over time. When the oxygen and nutrients in the congested vasculature is exhausted, gaps develop between endothelial cells of the vessels due to weakness in the energy-dependent endothelial junctions. Through these gaps, plasma proteins and fluids leak from the congested vessels into the interstitial spaces, resulting in oedema (Mosier, 2017). The oedema fluids are probably of the exudative form; they were observed to cause increased opacity as well as thickening of the serous membranes and mesenteries associated with the visceral organs. Although this is not particularly an acute event, the non-availability of oxygen in the blood in this case would have hastened the event, causing the oedema observed in the lungs, liver and other organs of this goat.

In summary, this article reported the gross and histopathological findings in some organs of a West African Dwarf goat suspected to have died of cyanide poisoning following consumption of large quantity of cassava root tubers and leaves.

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Conflict of Interest

The author declares no conflict of interests.

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References

Arnold M, Gaskill C, Smith SR and Lacefield GD (2014). Cyanide poisoning in ruminants. *Agriculture and Natural Resources Publications,* 168. https://uknowledge.uky.edu/anr_reports/168.

Bhutta BS, Alghoula F and Berim I (2024). Hypoxia. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing: https://www.ncbi.nlm.nih.gov/books/NBK482316/.

Cereda MP and Mattos MCY (1996). Linamarin: the toxic compound of cassava. *Journal of Venomous Animals and Toxins,* 2(1): 06 - 12. https://doi.org/10.1590/S0104-79301996000100002.

Cope RB (2020). Cyanide Poisoning. *MSD Veterinary Manual.* https://www.msdvetmanual.com/special-pet-topics/poisoning/cyanide-poisoning.

Cope RB (2021). Cyanide Poisoning in Animals. *MSD Veterinary Manual.* https://www.msdvetmanual.com/toxicology/cyanide-poisoning/cyanide-poisoning-in-animals.

FAO (Food and Agriculture Organization) (2004). A cassava industrial revolution in Nigeria The potential for a new industrial crop. In: Proceedings of the Validation Forum on the Global Cassava Development Strategy, Food and Agriculture Organization of the United Nations, Rome, April, 26 – 28, 2000. https://www.fao.org/4/y5548e/y5548e07.htm.

Flood D, Lee ES and Taylor CT (2023). Intracellular energy production and distribution in hypoxia. *The Journal of Biological Chemistry*, 299(9): 105103. https://doi.org/10.1016/j.jbc.2023.105103.

Hamel J (2011). A review of acute cyanide poisoning with a treatment update. *Critical Care Nurse*, 31(1): 72 – 82. https://doi.org/10.4037/ccn2011799.

Ihedioha JI (2002). The clinicopathologic significance of enriching grated cassava mash with red palm oil in the production of garri. *Plants Foods for Human Nutrition,* 57(3 – 4): 295 – 305. https://doi.org/10.1023/a:1021824031799

Ihedioha JI and Chineme CN (2003). Clinicopathologic implications of shortened fermentation periods in the production of toasted cassava granules (garri)**.** *Plant Foods for Human Nutrition,* 58: 1 – 11. https://doi.org/10.1023/B:QUAL.0000040341.87463.be

Kwok J (2008). Cyanide poisoning and cassava. In: Food Safety Focus. https://www.cfs.gov.hk/english/multimedia/multimedia_pub/multimedia_pub_fsf_19_01.

MacIntyre NR (2014). Tissue hypoxia: Implications for the respiratory clinician. *Respiratory Care,* 59 (10): 1590 – 1596.

Mosier DA (2017). Vascular disorders and thrombosis. In: Pathologic Basis of Veterinary Disease, Sixth edition, Elsevier, Inc., pp. 44 – 51.

Ndubuisi ND and Chidiebere ACU (2018). Cyanide in cassava: A review. *International Journal of Genomics and Data Mining.* 118. https://doi.org/10.29011/2577-0616.000118.

Oke OL (1969). The role of hydrocyanic acid in nutrition. *World Review of Nutrition and Dietetics*, 11: 170 – 198.

Stoltenow C and Lardy G (2012). Cyanide poisoning. North Dakota State University Extension, V1150. https://www.ag.ndsu.edu/publications/livestock/cyanide-poisoning/v1150.pdf.

Ufaysa G (2019). Review on cyanide poisoning in ruminants. *Journal of Biology, Agriculture and Healthcare.* 9(6): 1 – 12.