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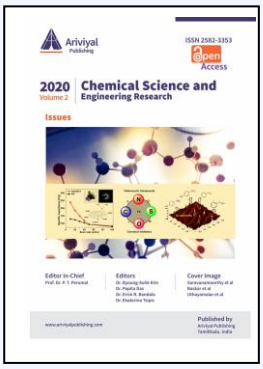

Cyanide Content Analysis in Selected Food Crops (Soya Beans, Maize, White Beans), Nuts (Ground Nut, Tiger Nut) Cultivated and Consumed in Nigeria and, Espouse of Environmental Toxicants as Epigene (s) Modifier

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Abstract: Food crops contain essential nutrients, necessary for proper developments. Hence, as a plant group, they are cultivated for human consumption to provide nutritional supports for the body. Experimental evidence has shown that some food crops/nuts contain Cyanogenic glucosides, capable of producing cyanide upon enzymatic hydrolysis. This current work focuses on the qualitative and quantitative analysis of the cyanide contents of soya beans, maize, white beans, groundnut and tiger nut using different extraction solvents (distilled water, 70% ethanol, acetone and n-hexane). The qualitative analysis shows, all the samples experimented contains hydrogen cyanide in the form of cyanogenic glucosides. Using UV-Vis spectrophotometer, the cyanide concentration for each crops/nut samples was analysed quantitatively after solvent extractions. The tiger nut sample had the least cyanide concentrations (180.67±5.03 mg/kg, 210.67±3.79 mg/kg, 223.33±2.89 mg/kg and 233.33±2.89 mg/kg) in all the solvents used for the extraction process. However, the concentrations were observed to increase as the polarity of the solvents decreases from distilled water to n-hexane. White beans had high concentration of cyanide in distilled water (261.33±3.51 mg/kg), acetone (365.33±2.52 mg/kg) and n-hexane (370.00±3.00 mg/kg). Also, maize presented a high concentration of cyanide (343.33±2.89 mg/kg) in 70% ethanol. Cyanide intakes has serious consequences including; effects on mitochondria bioenergetics, triggering ROS formation, enhance carcinogenesis process mediated by DNA methylation, and it affects other human hormonal functions thought to be involved in infertility, delay pregnancy due to changes in the metabolic demands of the pre-implantation embryo. Thus, it is imperative that, for human consumption, an initial detoxification process is advised as a precaution for food crops with potential for high cyanide concentrations.

Keywords: Cyanide; Food crops; n-hexane; UV-Vis Spectrophotometer; Carcinogenesis; Bioenergetics; Mitochondria; Polarity.

1. Introduction

Some food crops and nuts including soybeans, groundnuts, white beans, maize and tiger nut[1] contain traces of cyanide or cyanogenic glucoside, such as amygdalins and linamarin. Plant Cyanogenic glucosides release cyanide residues enzymatically albeit, food processing reduces cyanide residues. However, the cyanide contents of processed foods are often a problem in most developing economy including Nigeria where hydrocyanic acid gas is permitted for grain fumigation, preventing, against hydrocyanic poisoning from these foods consumption standards for usage are set for inspection.[2]

There are at least two thousand, six hundred and fifty (2650) species of plants that produces cyanogenc glucosides and usually with the corresponding hydrolytic enzyme (β-glucosidase), which are brought together when the cells structure of the plant is disrupted, due to a subsequent break down resulting to the formation of sugar and a cyanohydrins, further decomposed to form hydrogen cyanide, which is an aldehyde or a ketone.[3,4] The combination of cyanogenic glucoside and hydrolytic enzyme form the protective means use by cyanogenic plants, against predators.[3,5] Different species of plants produces one or more related cyanogenic glucosides, whilst some plant species such as cassava (*Manihot esculenta*, Crantz) are cyanogenic which is catalysed by hydroxynitrile lyase (HNL).

However, some species such as white clover, bird's foot trefoil, yarrow, bare-bell and white flax are plants that contain either or both cyanogenic glucosides enzyme (GE). Cyanogenic glucosides without enzyme, is represented as (G-), while enzyme without cyanogenic glucosides is presented as (-E) and neither cyanogenic glucoside nor enzyme is represented as (--).[5-7]

Many methods have been developed for the analysis of the total cyanogenic glucoside (total cyanide) contents of soya beans, Groundnuts, white beans, Maize and Tiger nut in different geographical locations with different soil types.[8,9] This and other related experiments were conducted basically in the North eastern and North western regions of the country thought to have different soil topography and pollution from those of the North central region

of the country where Benue state is located. These and other environmental factors and practices contributes to the possibilities or differences, which may exist on the cyanogenic glucoside content of food crops and nuts cultivated in these different regions of the country.

The picrate method[10] and the Feigl-Anger spot test[11] have been used to analyze for cyanogensis in a wide range of plants. The latter method depends on endogenous enzyme, which catalysed the hydrolysis of cyanogenic glucoside to cyanohydrins, with further break down to hydrogen cyanide.

Using the Bradbury et al. (2004); the cyanide contents of any plant material, can be determined.[12] This does not require the presence of a specific enzyme and would be effective with all cyanogenic glucosides. However, there are few losses of hydrocyanide (HCN) gas during acid hydrolysis at 100 ^{0}C.[12,9]

This research work was designed to first; establish the presence of cyanide in the food crops and nuts (Soybeans, Groundnuts, White beans, Maize and Tiger nuts) by qualitative test, determine how much of the cyanide is present in the plant materials cultivated for consumption in a specific location, Benue State, of the North central geopolitical zone, Nigeria. The current work focuses on the qualitative and quantitative analysis of the cyanide contents of soya beans, maize, white beans, groundnuts and tiger nuts using different extraction solvents such as distilled water, 70% ethanol, acetone and n-hexane. This work is aimed at setting the stage for country wide consumer protection agency to establishing the safety and advice were appropriate the safety of these selected crops and nuts for human consumption. More so, such crops as tiger nuts, groundnuts, soya beans, maize and white beans does not have robust information about their cyanide contents.

2. Experimental Section

2.1. Plants and Nuts materials

Maize (*Zee mays L*), Soya bean (*Glycine max L*), White bean (*Vigna aguiculata L*), and the Ground nuts (*Arachis hypogaea L*), Tiger nuts (*Cyperus esculentus L*), used for this study were sourced locally. All samples were properly washed to remove dirt and other microorganisms to avert spoilage and contaminations.

2.2. Analytical tools

Using the UV-Visible Spectrophotometer (Cole-Parmer UV-7504), the quantitative and qualitative presences of cyanide in all samples were analysed. All reagents used for this study are of analytical grades.

2.3. Sample Preparation

The samples collected were air dried at room temperature (25°C) in the laboratory. After drying, they were grinded into powder using pistle and mortar.

2.4. Qualitative Analysis of Cyanide

To ascertain the presence of cyanide in the samples provided, a qualitative test using the methods previously described by Nkafamiya and Manji (2006).[13] 5 gram of the powdered samples was moistened and placed in a test tube. Moistened sodium picrate paper was inserted in the test tube, taking note that it does not come in contact with the samples. This was followed by the addition of a few drops of chloroform and the tube was stooped tightly. The sodium picrate paper gradually turned to reddish brown colour indicating the presence of HCN in the form of cyanogenic glycoside.

2.4.1. Extraction and Quantitative Determination of Cyanide

Cyanide contents of the samples was determined using the methods previously described by Bradbury et al. (1991) and Adeniran et al. (2013) with slight modification in both the solvent and the sample quantity used for the extraction.[14,15] 2.5 g of each sample was dissolved in 25 ml of distilled water in a corked conical flask to extract cyanide. The cyanide extraction was allowed to stay overnight at room temperature and the extract filtered through a filter paper.

Alkaline picrate solution was prepared by dissolving 1g of picric acid and 5g of sodium carbonate in warm water in a volumetric flask and making up the volume to 200 ml with distilled water. To 1 ml of the sample filtrate, 4 ml alkaline picrate was added and incubated in a water bath (37°C) for 5 mins enabling colour development, the absorbance of the solution, measured at 490 nm using a Cole-Parmer UV-7504 spectrophotometer. From a concentrated potassium cyanide solution standard cyanide solution were prepared with final concentration of the Cyanide in solution expressed in mg/kg.

Other solvent such as 70% ethanol, hexane, and acetone were used to extract the cyanide and the preceding steps was followed to determine the cyanide content of the samples provided.

2.5. Statistical Analysis

The data were analysed using one-way analysis of variance (ANOVA) followed by GraphPad prism version 8.1.2 statistical analytical tools. The least significance differences or probability were carried out when: $F_{cal} > F_{tab}$; using the equation:

$$LSD = (Sx1 - x2)t$$

This was calculated and significance between mean values was determined using the critical values of p at 0.05 being the Pearson correlation coefficient and test of significant level of probability.

3. Results and Discussions

Using qualitative and quantitative analysis methods the present study investigated the cyanide content of soya beans, maize, white beans, ground nut and tiger nut with different extraction solvents including; distilled water, 70% ethanol, acetone and n-hexane, aiming to establishing the presence of cyanide in these food crops and nuts cultivated for human consumption in Benue State, Nigeria.

Cyanogenic glucosides are groups of widely occurring natural substances, which on hydrolysis produce a ketone or aldehyde, a sugar, and the highly toxic cyanide ion.[16] The major food sources of cyanogenic glucoside include bitter almonds, cassava root, sorghum and lima beans. [17]

Table 1. Qualitative determination of cyanide in different samples

Samples	Observations
Soya beans	+
White beans	+
Tiger nut	+
Maize	+
Ground nut	+

The positive sign (+) shows the presence of cyanide in all the samples.

However, such crops as tiger nut, ground nut, soya bean, maize and white beans on the other hand are devoid of robust information about their cyanide contents. The toxicity of cyanide in food crops and nuts is basically due to its liberation from cyanogenic glucoside upon hydrolysis by enzyme. Cyanide release from cyanogenic glucosides occurs readily in the laboratory by acid or base hydrolysis, whilst in chewed/chopped plants they are released via the ingestion of two enzymatic process earlier reported by Bokanga *et al*.[18] and most recently by Oracz *et al*.;[19] Andre *et al*.[20] The qualitative results for the nuts and crops analysed are presented in the Table 1.

3.1. Qualitative Determination of Cyanide

Table 1 presents the qualitative analysis of cyanide. The results indicate that all the samples contain cyanide in the form of cyanogenogenic glucoside. This may be due to the cultivation of the crops in environment containing cyanide or naturally occurring potassium and sodium cyanide in the cultivated soil as previously reported.[21]

Moreover, the results (Table 1), indicates, all samples contain cyanide, which may be due to the use of pesticides containing cyanide, waste contamination, combustion of polyurethane around the environment, catalytic converters that generate cyanide, some industrial activities, industrial waste-water discharged or naturally occurring Sodium or Potassium cyanide in the area. During water runoff, there is usually distribution of waste containing different chemicals resulting to pollution of the soil.

Inadequate wastes disposal pollutes the soil and these are a major problem faced by many regions across the globe. In Nigeria, these problems are more compounded, because until now, efforts to combat waste pollutions and its health consequences were not given the enabling laws and where available, it lacks enforcement. Thus, wastes such as cyanide and other hazards from waste dump sites produces contaminated leachates, which eventually pollutes the groundwater. Plant in contrast absorbs this water contaminated

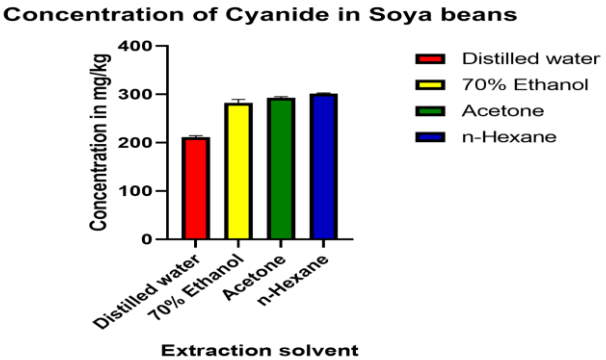

Fig. 1. Concentration of cyanide in soya beans extracted using different solvents. Values are mean ± SD

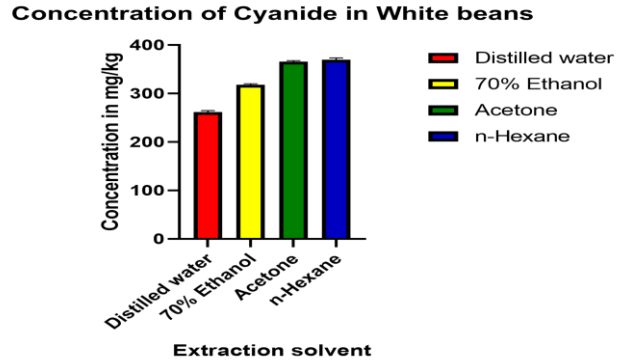

Fig. 2. concentration of cyanide in white beans extracted using different solvents. Values are mean ± SD.

with different chemical effluents from the industry resulting to its susceptibility of environmental toxicants including, cyanide. Similarly, report has been presented by Cho *et al*.[22] and Jaszczak *et al*.[21] showing the presence of cyanide in some edible nuts. Nwaichi *et al*.[23] Orjiekwe *et al*.[24] also reported the presence of cyanogenic glucosides in cassava (*Manihot exculenta L*.).

The differences in the cyanide concentration of the both the nuts and crops are represented in the Figures 1-5. From the results, a decrease in polarity of the extraction solvent leads to a marked increase in the concentration of the cyanide in the sample, indicative that the form, which the cyanide occurs in the sample, is more soluble in nonpolar solvents.

The Fig. 1 represents the mean concentration of cyanide in soya beans when extracted with distilled water, 70% ethanol, acetone and n-hexane. There is an increase concentration of cyanide with n-hexane having the highest concentration (301.33±1.53) and distilled water with the least concentration (211.33±3.21).

The Fig. 2 showed the mean concentration of cyanide in white beans, extracted with distilled water, 70% ethanol, acetone and n-hexane. There is an increase concentration of cyanide with n-hexane having the highest concentration (370.00±3.00) and distilled water with the least concentration (261.33±3.51).

White beans had the highest concentration of 261.33±3.51 mg/kg when extracted with distilled water followed by maize with concentration of 250.00±2.53 mg/kg, with soya beans having the least concentration of 211.33±3.21 mg/kg. The high concentration of cyanide in white beans than other samples may be largely due to its inherent nature (complexation of cyanide and carbohydrate); its exposure to more pesticides than the rest of the samples or due to the difference in the composition of the soil type, implying the soil contains more Sodium or Potassium cyanide. The low concentration of cyanide in soya beans may be due to its naturally low carbohydrate residue, which forms complex with cyanide in cyanogenic glucosides.

However, White beans extracted using acetone had a concentration of 365.33±2.52 mg/kg (see Fig. 2). This may be due to its less polarity and hence high solubility in the solvent than the rest of the samples.

The Fig. 3 is the mean concentration of cyanide in tiger nut, extracted with distilled water, 70% ethanol, acetone and n-hexane. There is an increase concentration of cyanide with n-hexane having the highest concentration (233.33±2.89), distilled water 180.67±5.03

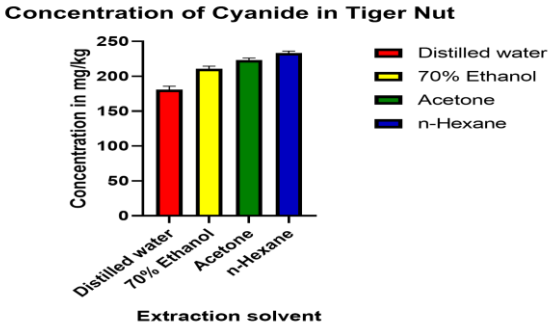

Fig. 3. Concentration of cyanide in tiger nut extracted using different solvents. Values are mean ± SD.

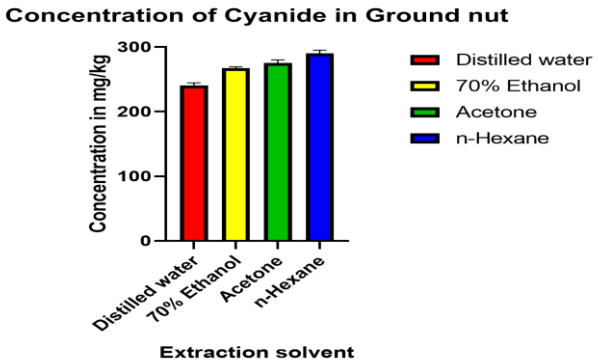

Fig. 5. Concentration of cyanide in ground nut extracted using different solvent. Values are mean ± SD.

concentration, this may be because of its high polarity and hence low solubility in the solvent than the rest of the samples. The concentrations of the cyanide obtained when extracted with n-hexane were higher than the ones extracted with distilled water and the other solvents. This is due to differences in polarity and hence, the difference in solubility of plant samples.[25] Water is a more polar solvent than ethanol, acetone and n-hexane but, most organic compounds with less polarity have high solubility in a less polar solvent and this had earlier been presented in a similar observation based on the polarity of solvent of extraction.[26,27]

The Fig. 4 showed the mean concentrations of cyanide in maize, extracted with distilled water, 70% ethanol, acetone and n-hexane. There is an increase concentration of cyanide with n-hexane having the highest concentration (363.33±2.89) and distilled water with 250.00±2.53.

Fig. 5 represents the mean concentrations of cyanide in soya beans extracted with distilled water, 70% ethanol, acetone and n-hexane. There is an increase concentration of cyanide with n-hexane having the highest concentration (290.00±5.00) and distilled water with 240.67±4.04.

Maize had the highest concentration of 343.33±2.89 mg/kg when extracted with 70% ethanol. White beans with concentration of 317.67±2.52 mg/kg and soya beans with concentration of 282.33±7.02 mg/kg follows. Tiger nut had the least concentration of cyanide (210.67±3.79 mg/kg) when extracted with ethanol, which may be due to its naturally low content in the sample. Similarly, report has been presented by Adeniran *et al.*,[15] presenting the concentration of cyanide in lima beans found in Ibadan, Oyo state, Nigeria. Also, Nwaichi et al.[23] reported the concentrations of

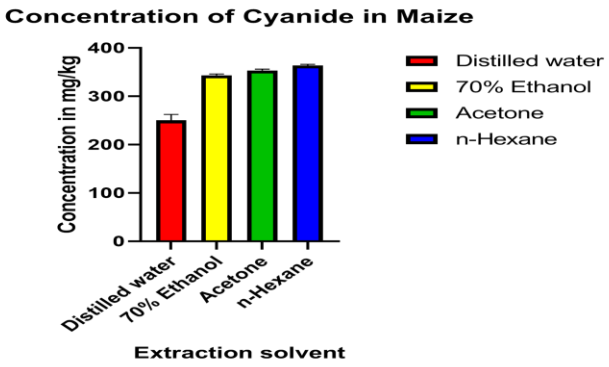

Fig. 4. Concentration of cyanide in maize extracted using different solvents. Values are mean ± SD.

cyanide to be 0.5 mg/kg in maize (*Zee mays L.*), 0.05 mg/kg in soya bean (*Glycine max L.*) and 0.10 mg/kg in cassava (*Manihotexculenta L.*) found in Port–Harcourt, Rivers state, Nigeria. Orjiekwe *et al.*[24] also reported the concentrations of cyanide in cassava (*Manihotexculenta L*) flour, fufu and garri found in Okada town, Edo state, Nigeria to be 30 mg/kg, 50 mg/kg and 25 mg/kg respectively. The concentrations obtained from this work were higher than those previously reported in the literatures. This may be due to a number of factors, which include crop variant, environmental factors, location, season, soil types and most especially the difference in polarity of the extraction solvents as reported by Oluwaseyi*et al.*[28] The results indicates significant presence of the HCN in the form of cyanogenetic glucoside although below the threshold level of 600 mg/kg per day in adult earlier reported.[29]

The result obtained indicates that the concentration of cyanide (in the form of cyanogenic glucoside) is significant although, it is below the acceptable permissible level of 600 mg/kg per day in adult diets as suggested by the World Health Organization (WHO). It is important to note that, although low, the fear of the damage (discussed below) it could cause, lies on the constant exposure and accumulation.

3.2. Cyanide Environmental toxicants to human health

In human biology, cyanide as an enzyme inhibitor is dangerous (see Table 2). It is involved in the covalent modifications of enzymes structure. Moreover, as an irreversible enzymes inhibitor, it can covalently bind to mitochondrial cytochrome oxidase and inhibits the reactions associated with electron transports (Table 2).

Mitochondria are the major source of energy in all eukaryotic cells, producing ATP through oxidative phosphorylation and the citric acid cycle.[37] They regulate calcium homeostasis and modulate apoptosis through release of several cell death-inducing molecules.[38,39] Mitochondrial functions, which includes protein import, ATP generation and lipid biogenesis, depends on the maintenance of the ROS.[21,39] Cyanide poisoning has potential to affects mitochondrial bioenergetics, which may acts as a novel mechanism for the regulation of cell fate and, more importantly, in the reprogramming of cells to pluripotency.[40] These processes may also include potential to block antioxidative agents that are involved in the biochemical processes such as, permeabilization of the outer mitochondrial membrane (allowing biological radicals to interfere with cytochrome C released into the cytosol), with the attendant

Table 2. Some irreversible enzymatic inhibitors with their putative effects

S/N	Irreversible Inhibitors	Binding site	Outcome/Effect	Reference
1	Cyanide	Binds to the iron atom (Fe) in cytochrome C oxidase enzyme in mitochondria cells.	It effect as an irreversible inhibitor prevents cytochrome C oxidase from transporting electron to oxygen in electron transport chain of aerobic respiration. This affects the production of ATP required for metabolism. If not prevented, it could lead to gradual death of muscle cells and nerve cells that required this form of energy. Death of large amount of these cells could lead to the death of individual.	[30]
2	Diisopropylphosphofluoridate (DIFP).	Binds to an active serine-195 residue in the active site of enzymes like acetylcholinesterase and chymotrypsin.	The binding of DIFP to the active serine residue of acetylcholinesterase inactivate the functionality of the enzyme as nerve impulse transmitter.	[31,32]
3	Tosyl-L-phenylalanine chloromethyl ketone (TPCK)	Binds to an active serine, histidine and cysteine in the active site of certain enzymes involved in protein synthesis and alkylation	-Release its alkyl group radicals into biologically active molecules and thereby prevent their proper functioning which subsequently leads to carcinogenesis, mutagenesis, teratogenesis and immunosuppressant actions. -Interrupt peptide-chain elongation, blocking the A-site of ribosome, misreading of the genetic code and prevention of attachment of oligosaccharide side chains to glycoproteins.	[33]
4	Phenelzine (Hydrazine derivative)	Activated phenelzine covalently binds to the N^5 of FAD cofactor of monoamine oxidase resulting to inactivation of the enzyme.	As an irreversible inhibitor, the HN-NH bond is oxidized to give a diazene that can lose an electron to oxygen leaving a reactive radical that alkylates the N^5 of the flavin molecule leading to inactivation of the enzyme and subsequent release of reactive oxygen species (ROS).	[34]
5	Tranylcypromine (Parnate)	Binds to the copper residue in the active site of monoamine oxidase and cytochrome P450 oxidase.	-Inactivate monoamine oxidase leading to epigenetic-modification of flavoprotein enzyme and enzyme LSD-1. This cause hypertensive crisis except there is dietary follow up.	[34,35,36]

early impairment of cell proliferation, severe arrest and accumulations of cells in the G2/M phase, which are followed by apoptosis through the enhanced functions of the pro-apoptotic members of the B-cell lymphoma 2 (Bcl-2) family.[41,42] Thus, such toxicant such as cyanides, could be involved in the biochemical process of transcriptional up regulation of c-myc (transcriptional repressors) via interactions with components of Polycomb repressive complex 2 (PRC2) and induces the acetylation of histones H3 and H4.[43]

Earlier Okoh,[42] discussed the deleterious effects of genetic damage due to exposure to ROS with concomitants consequences on genomic stability. Moreover, environmental metals (see Fig. 6) such as Cadmium (Cd), Mercury (Hg), Lead (Pb) etc, trigger formation of ROS, affecting human health via directing epigenetics signatures of the genome suggesting that epigenetic alterations can be mediated by toxicity from environmental chemicals.[44,45] Moreover, they couldreact with physiological gas controlling a number of physiological processes at low (submicromolar) concentrations.[45]

Cyanides as environmental contaminants (Fig. 6), upon its absorption via food, is rapidly distributed throughout the body, with the highest levels found typically in the liver, lungs, blood, and brain where they elicit effects.[46] Majority of the absorbed cyanide is bio- transformed to thiocyanate by the action of mitochondrial sulfur transferase enzymes and other sulfur transferases.[47] The oxidative stress induction by cyanide involves increases in ROS and nitric oxide, leading to the inhibition of antioxidant systems (inhibition of catalase and superoxide dismutase activities and activation of NADPH oxidase), with co-inhibition of mitochondrial functions.[48]

The primary targets of cyanide toxicity in humans and animals are the cardiovascular, respiratory, and central nervous systems.[44,48] As a function of continued exposure to thiocyanate, endocrine systems are disturbed, preventing the uptake of iodine in the thyroid, acting as a goitrogenic agent.[44] These actions of cyanide ion, affects other human hormonal functions involved in fertility and pregnancy.[49] Its being shown that prolong and over exposure to cyanide results in ovulation disorders in human females, affecting the

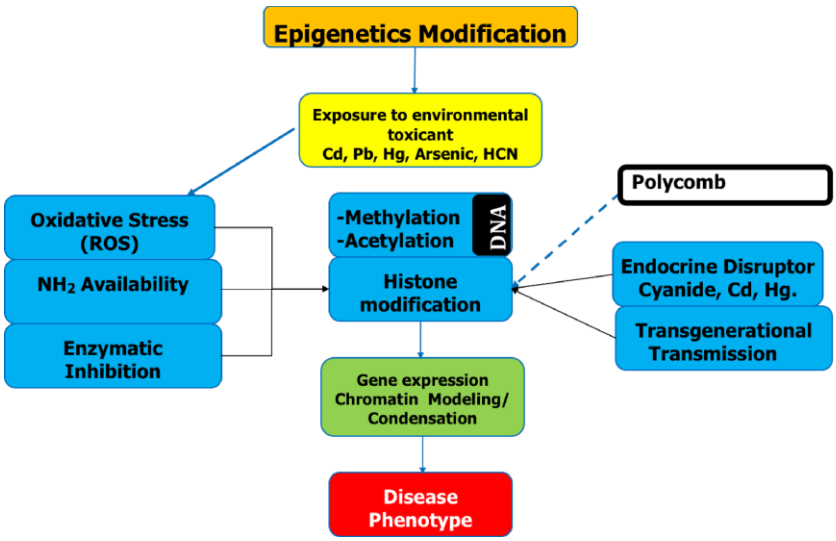

Fig. 6. Postulated cycle of events/cross talk that affects biological processes with consequences on epigenetics leading to disease phenotypes (modified from Baccarelli& Bollati, 2009[44]).

release of eggs from ovaries, due to hormonal imbalance arising from polycystic ovary syndrome, hyperprolactinemia, (a condition in which individual have too much prolactin-the hormone that stimulates breast milk production)-resulting to interferences in ovulation, hyperthyroidism and hypothyroidism, which affects the menstrual cycle or causes infertility-by blocking the major pathway associated with iodine secretion and uptake.[49]

In male, exposure to cyanide through cigarette smoking, pesticide usage, or marijuana, causes low sperm counts,[51] via the inactivation's of cytochrome oxidases and inhibitions of cellular respiration with consequent histo-toxic anoxia in sperm cells.[46] This causes the death of sperm cells available for fertilization of female subject leading to infertility. Moreover, experimental evidence had also showed toxic effects of cyanide on rat sperm were higher dosage of cyanide caused significant decrease in biochemical composition and structural changes in the proteins of rat sperms.[52] Such changes, may be due to the germ cells degeneration, implying most probably cyanide action on sperm follows similar mechanism with nicotine, which causes low intra-testicular concentrations of testosterone, bearing high level of testosterone in testis are essential for the normal spermatogenesis, the maintenance of the structural morphology and the normal physiology of seminiferous tubule.[53] Alternatively, it might be through mechanisms that involved the inactivation of the X-chromosome via epigenetics methylation processes.[54] Cyanide as an endocrine disruptor may be involved in trans-generational transmission of chemically-induced epigenetic changes (Fig. 6), however, more research is needed to validate such postulations.

The inhibition of mitochondrial functions by cyanide is thought to produce ROS and superoxide anion,[39] and these reaction processes has been shown to participate in carcinogenesis that are being mediated by DNA methylation.[55,30] Moreover, such ROS induction, can damage DNA (Fig. 6), interfering with the ability of methyltransferases enzymes to interacts with DNA, resulting in an altered methylation of cytosine residues at the CpG sites,[44,45] usually found at the 5' end of regulatory region of most genes. An established facts are, DNA methylation and histone acetylation are major epigenetic processes (Fig. 6) that alter and modify gene expression resulting in disease state.[44] It is also most probable; cyanide poisoning may targets, the ABC (family of ATP-dependent transporters that pump amino acids and proteins etc., out of cells against a concentration gradients). The genetic variations in the ATP binding cassettes (ABC) genes has been implicated in a wide variety of human disorders with Mendelian and complex inheritance including; neurological disorders, cystic fibrosis, cholesterol and bile transport defects, retinal degeneration, drug response phenotypes and anemia.[55] Thus, understanding the events that causes inactivation of genes are critical to understanding the processes/mechanisms of environmental toxicants in disease pathogenesis.

4. Conclusions

The present study involves the qualitative and quantitative analysis of the cyanide contents of household food crops and nuts. The results indicated the presence of HCN in the selected samples. The quantitative efficiency of cyanide extractions was experimented using water, 70% ethanol, acetone and n-hexane. However, n-hexane gives a better extracts of cyanide from the food crops followed by acetone. Water had the least efficiency when used for cyanide extraction compared to other solvents. This may be due to its high polarity as compared with the other solvents. In general, the cyanide levels of the food crops evaluated in this study are below the maximum acceptable (permissible) level of 600 mg/kg and above the minimum acceptable level of 14-30 mg/kg by the World Health Organization (WHO). Accumulations of small doses of cyanide over a long period of time can be deleterious since cyanide poisoning affects oxygen as it binds to an allosteric site on cytochrome oxidase – a carrier molecule that forms part of the electron transport chain. The effects of cyanide is not limited to its hazardous activities on

human and animal subjects; it also affects plants at the cellular and molecular level, HCN enhances the prevention of plants seed dormancy, however, the mechanism for now remain unclear.

For human, exposure to cyanide from unintentional or intentional consumption of food crops with cyanogenic glycosides may leads to acute intoxications. Thus, precautionary detoxification by food processing method may be the way forward for the consumption of the food crops presented in this study with high cyanide concentration. This data as presented provides useful background for further studies on crops profiling of edible food crops and their nutritional requirement by humans.

Conflicts of Interest

The authors declare no conflict of interest.

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