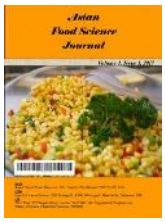

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Natural Toxins in Plant Foods: Health Implications

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Authors' contributions

This work was carried out in collaboration between all authors. All authors read and approved the final manuscript.

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ABSTRACT

This review took a survey of literature on natural toxins in commonly consumed food crops. Food crops are a good source of proteins, carbohydrates, minerals and B-vitamins. However, some foods that are generally consumed possess innate toxins with potential harmful effect in human health. The current study looked at some of the toxins their chemical structures, their mechanism of toxicities, sources of the toxins and the effect of processing on the toxins with focus on saponin, glycosides, toxic protein/amino acids and polyphenols. This paper informs consumers, regulators and researchers of plant origin foods hence help reduce toxicities among consumers.

Keywords: Phytotoxins; glycosides, toxicity; degradation.

1. INTRODUCTION

Plants constitute a vital human diet particularly in developing countries where the price of animal origin food is above the reach of the resource-poor, or in localities where animal-based diets are culturally restricted. In India and some South East Asian countries, and Sub-Saharan countries where there is diet restriction due to religion and poverty, respectively. There are many reported

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cases of plant food toxicities, leading to adverse health effects [1–3]. Plant origin foods are an excellent source of proteins, carbohydrates, minerals and B-vitamins. Natural toxins are present in a wide variety of plants that are consumed as food. More so, when the toxic substances are ingested insignificant amount or when they are not processed appropriately they can be potentially harmful to human health causing illness or even death. Food plants make compounds for functions like inter and intraspecific communication (feromones, defense compounds, kairomones, and synomones) catching prey and metabolic functions. However, these compounds may end up being toxic to human beings at specific doses [4].

Phytochemicals have commercial applications in the food, cosmetic and pharmaceutical sectors [5]. They possess numerous health benefits e.g. ant-diabetic, anticancer, antioxidant, cholesterol-lowering effect etc. [6,7]. There are many types of toxins with either chronic or acute health impact, but it's the dose and vulnerability of the subject exposed that makes them poisonous [8]. There are very few products that are entirely safe at all doses; therefore safety limits are set for various compounds which can be toxic at a given level. Regulatory agencies have the responsibility to protect vulnerable sub-population and general population against overconsumption of poisonous components [8].

Consumption of plants for their known and perceived health benefit has been with us for eternity. Moreover, there are various documented nutritive and health improving effect of plants, e.g. polyphenols as antioxidants, dietary fibres, chlorophyll-rich plants for anaemia and vitamin-rich plants for improved health [9]. The intake of 400-600 g/day of fruits and vegetables is associated with reduced incidences of many forms of cancer, besides diets rich in plant foods are associated with reduced risk of heart diseases and many chronic diseases [10]. These foods contain phytochemicals which confer many health benefits. Indeed, herbal medicine sector has been growing based upon the pharmacological benefits of plant bioactive [11]. There is increased health nutrition consciousness among middle class and affluent populace. This has led to increased consumption of plant products which are perceived to be healthier compared to livestock origin products. Moreover, several people take uncooked food products due to the perception that cooking destroys the healthy food components. Cooking is a well-known method of processing food product and it is a well-established fact that heat treatment of foods lead to the destruction of plant toxicants and pathogens [12].

Plant-based diets have been shown to be beneficial to individuals especially those with high blood pressure, diabetes, cardiovascular disease, or obesity [13]. However, it has been established that most plant-based diets are deficient in some nutrients like calcium , Avitamin D, Vitamin B-12 and n–3 fatty acids [14]. In poor communities where there is lack of balanced diet (protein-energy balance) during dry seasons, drought-resistant crops like grass pea and cassava remain their main diet. In some parts of Ethiopia and India where grass pea is widely consumed during long draughts, lathyrism epidemic was reported during drought which was occasioned by consumption of a single plant diet [15]. Cassava a drought resistant tropical crop has equally been linked to konzo and lathyrism in Mozambique, Cameroon, Tanzania and the Central African Republic during times of severe food shortage, occasioned by dependence on cassava as a single source of diet [1,3]. Vegetarian diets involving some animal products like lacto-vegetarian, lacto-ovo vegetarian and ovo-vegetarian can provide sufficient nutrients without supplements but vegan diet may require a supplement and could lead to phytotoxins toxicities owing to higher exposure to plant toxins compared to the average population. Moreover, uncooked plant foods tend to have a higher level of toxins and anti-nutritional factors than cooked foods [13,16,17].

There are many known phytotoxins, and most of them are generally accepted as safe at the mean levels of daily consumption by average population [18]. Current trends in food consumption by the elite and health-conscious individuals emphasizes on minimally processed and raw foods. The patterns are based on perceptions that food processing mostly destroys essential nutrients, and that consumption of fresh foods will increase intake of such nutrients. Despite this, certain foods in their natural form contain toxins or toxic substances which upon consumption may cause serious adverse health implications to the consumers. It is critical that knowledge of the toxicological aspects of particular foods popularly consumed or promoted for use in raw form is documented and highlighted. Therefore this review brings together knowledge about plant toxins in food plants with

potential to cause harm in human being not adequately addressed.

2. PLANTS AND THEIR ASSOCIATED TOXINS

Some food crops have different toxins distributed in different plant parts. The parts of a crop plant which may be used as food sources include the foliage, buds, stems, roots, fruits and tubers. Typical examples of natural toxins in food plants include lectins in beans such as green beans, red and white kidney beans; cyanogenic glycosides in bitter apricot seed, bamboo shoots, the edible roots of cassava and flaxseeds; glycoalkaloids in potatoes and muscarine in some wild mushrooms which are the agonist for all muscarinic receptors. Some of the known plant toxins categories are discussed below.

2.1 Toxic Amino Acids

Proteins present as amino acids mostly play a defensive role against insects where they antagonise amino acids in the body. The most well-known group is Lathyrogens, which are mainly found in *L. sativus, L. cicera* and *L. clymenum, L. odoratus (*sweet pea*), L. hirsutus, L. pusillus* and *L. roseus, L. sylvestris, L. latifolius* etc. Lathyrus is mainly grown and consumed in Asia and East Africa, where it is used for food and feed. Hence most of the toxicities are experienced in these regions [19]. Another emerging toxin is hypoglycemic amino acids from Sapindaceae family which consist of lychee; they contain hypoglycaemic amino acids that disrupt gluconeogenesis and β-oxidation of fatty acids. Hypoglycemia can be arrested by restoring serum glucose concentrations. In children, however, there are reported cases of cognitive deficits, movement disorder or muscle weakness [20].

2.1.1 Lathyrogens

The toxins cause a disease resulting in motor system, connective tissue and blood vessels walls abnormalities, and it may give rise to the collapse of the vertebral column and central motor conduction deficit. It is caused by prolonged consumption of grass pea seeds [19]. The active compounds in grass pea responsible for this include; L-2, 4-diaminobutyric acid (DABA), Beta-aminopropionitrile (βAPN), β-N-oxalyl-L-α, β-diaminopropionic acid (β-ODAP) (neurotoxin), β-1 and α-1 which causes blood vessels raptures, skeletal disorder by inhibiting the formation of collagen and elastin. βAPN inhibits lysyl oxidase and disrupts collagen formation leading to osteolathyrism and angiolathyrism. Geographically it is common among poor households in Ethiopia, India and other Asian countries who principally consume sweet peas during long draughts [21].

2.1.1.1 Structure of lathyrogens

They are a structurally diverse group of compounds.

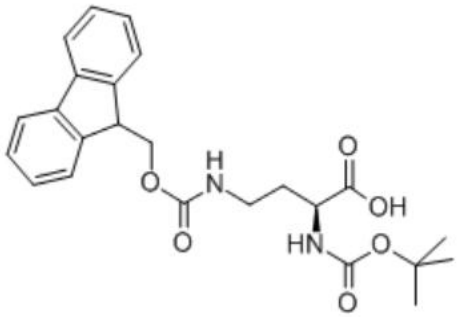

Fig. 1. The structure of L-2, 4-diaminobutyric acid
Source [19]

2.1.1.2 Mechanism of action

ODAP affects the central nervous system (CNS), muscle weakness and stiffness, paralysis of leg muscles, called neurolathyrism [22]. βAPN inhibits the activity of lysyl oxidase, an enzyme of importance in collagen formation. The resulting connective tissue abnormalities may give rise to collapse of the vertebral column, and angiolathyrism (abnormalities of blood vessel walls) [21].

2.2 Toxic Proteins/Peptides

Several types of harmful proteins are in food crops which include lectins, ricin, abrin, modeccin, viscumin and volkensin, ribosome-inactivating proteins, porcelains, protease inhibitors, glycohydrolases among others [23]. The toxicity of proteins depends on their specific native structure and can be denatured to reduce toxicity. Plant toxic proteins mainly belong to glycoproteins of 60 kDa molecular weight consisting of two subunits linked into a dimer by a disulphide bond [24]. Studies have shown the presence of ribosome inactivating proteins (RIP) in many plants and toxicity determined by the

connection to the B-chain which binds cell surface receptors and facilitates transport of A-chain across the cell membrane [23].

Ricin comes from castor oil plant, and the whole plant is known to be poisonous due to the action of A-chain which inactivates the ribosomes of the cell [8]. Abrin is another type of toxic protein found in rosary pea with the same mechanism of action as ricin which exists in two forms, abrin and abrin b. Other highly toxic ribosome-inactivating proteins include viscumin identified as the active ingredient of mistletoe extract, volkensin from the kilyambiti plant, modeccin from the roots of *Adenia digitate* all having the same mechanism of action as ricin [12]. The above-discussed plant toxic proteins are very potent and commonly known as ribosome-inactivating proteins (RIP) due to the mechanism of action [25]. Ribosome-inactivating proteins (RIPs) are mainly produced by plants, classified as type 1 or type 2 ribosome-inactivating protein and act as N-glycosidases (EC 3.2.2.22) [26]. Moreover due to different sizes, structures and functions of the ribosome-inactivating proteins, not all can be grouped into classical type 1 or type 2 ribosome-inactivating proteins [26]. Furthermore, there is no uniform terminology existing for the ribosome-inactivating enzymes. Pathological symptoms associated with ribosome-inactivating proteins include abdominal pain, vomiting, diarrhoea, electrolyte imbalance and hemorrhagic intestinal lesions, tissue necrosis for the postmortem features [12,21].

Mushroom origin toxins are called phallotoxins, they exhibits the typical structure, and the amatoxins, an example of which is amanitin are usual toxins in toxic mushrooms. Varied amounts of amatoxins have been reported in widely consumed edible mushrooms species including some species of *Amanita* such as *Amanita virosa, A. phalloides, A. muscaria, A. silvaticus (Champignon), Cantharellus cibarius (Pfifferling),* and *Boletus edulis (Steinpilz).* Some of these amatoxins are poisonous thermostable the most deadly being α-amanitine with amatoxin poisoning symptoms including motor depression, ataxia, euphoria, dizziness, gastrointestinal disturbances, and muscle twitches [27,28]. Phallotoxins are cyclic heptapeptides containing several novel amino acids and unusual combinations of the more common protein amino acids. The typical structural feature in the phallotoxins is the cystathionine, formed between L-tryptophan and L-cysteine, the amatoxins on the other hand are octapeptides [29].

2.2.1 Health effect

Phallotoxins act quickly and at higher dosages, causing death in 1 to 2 hours. Amatoxins work more slowly and at lower dosages and increasing the dosage does not reduce the time required for lethal effects to less than 15 hours. a-Amanitin is 10 to 20 times more toxic than phalloidin and thus constitutes the primary poison in deadly amanita. The organ affected by both groups of peptides is the liver but in markedly different ways. Phalloidin intoxication is associated with changes in the microsomal fraction of liver cells [30]. Trypsin inhibitors are a protein that inhibits trypsin hence hindering protein digestion. It is mainly found in raw legume seeds. Its pathological symptoms are enlarged pancreas and loss of essential amino acids [31]. Hemagglutinins (lectins) on the other hand, causes agglutination of red blood cells, bind avidly to mucosal cells and interfere with nutrient absorption from the intestine. Moreover, lectins may also cause toxicity by facilitating bacterial growth in the gastrointestinal tract and castor bean lectin called ricin has been implicated in deaths of children [8].

2.2.2 Effects of treatment on digestibility

Germination and cooking were reported to improve *in vitro* protein digestibility of horse gram and mothbean. Generally, most bean sprouts have the low level of toxins and are more nutritious than beans. It is also documented that β-1 in solution transforms the alpha isomer via an unstable intermediate until an equilibrium mixture 3:2 ratio. Dry heating grass pea seed is reported variously to lower, raise, or effect no change on β-1 content. Compound 1 is a water-soluble amino acid that can be leached from seed by soaking in water, increasing water temperature increases leaching rate [23].

2.3 Glycosides

This is a wide group of phytotoxins typically consisting of glycosylated compounds with a non-sugar section. The compounds contains a carbohydrate and a non-carbohydrate residue in the same molecule. The carbohydrate residue is attached by an acetal linkage at carbon atom 1 to a non-carbohydrate residue. Some of the toxic glycosides are cynanogenic glycosides in flaxseed and some cassava species, solaniane and shaonine in potato [1]. Cassava cyanogenic glycosides are associated with toxicities which can be acute or chronic, for instance konzo is

common symptom of cassava toxicity among communities where cassava is the staple food and there is overdependence on e.g. in Northern Mozambique, parts of Congo and India, acute poisoning is also common in these populations. [1,3]. Cassava toxic cyanogens come from toxic species and mostly in bitter cassavas tubers. Glycosides can be grouped broadly as.

2.3.1 Solanine and chaconine

Glycoalkaloids (GAs) consist of α-chaconine and α-solanine which are the main toxins found in *Solanaceae* family which include potatoes (*Solanum tuberosum*), nightshade (*Solanum nigrum)*, tomato (*Solanum lycopersicum*) and eggplant (*Solanum melongena*). They are trisaccharide connected to aglycone called solanidine, a steroid alkaloid. They are natural crop protection agents, produced to protect plant against fungi, insect pests and herbivores [32].

They occur naturally in any part of the plant, including the leaves, fruit and tubers [32]. Glycoalkaloids levels in plants vary within different plant organs and in different plants and varieties. Additionally, these levels are influenced by genetics, pre and post-harvest factors including microbial attack, handling (during harvest or transit), exposure to light which has effect of stimulating chlorophyll synthesis leading to 'greening' of tubers [33]. In the potato tuber glycoalkaloids are concentrated mainly in the outer (1.5~mm) layer, and routine processing usually reduces the content by peeling.

2.3.1.1 Toxicity symptoms

The glycosides toxicity is mainly gastro-intestinal and systemic, by cell membrane disruption and acetylcholinesterase inhibition, respectively. The toxicity on membranes leads to cell disruption caused by the formation of destabilizing complexes of the lipophilic moiety of the GAs with cholesterol in membranes. On the other hand it affects the nervous system by interfering with the body's ability to regulate acetylcholine. They cause gastro-intestinal tract (GIT) irritation, abdominal cramps, vomiting, diarrhea etc. [34]. The maximum level set for safe consumption is 200 mg/kg body weight, and for food safety, an upper limit of 20 mg glycoalkaloid content per 100 g of potato is generally accepted [35]. Different varieties have varying solanin content and breeding techniques can be used to reduce its content. It has been established that glycosides could have teratogenic effects, for instance feeding pregnant dams sufficient potato material caused cranial and facial defects in developing fetuses and sometimes resulted in the death of the dams [36].

2.3.2 Glucosinolates

They are also called goitrogens owing to their ability to bind to thyroid receptor, hence suppressing the function of the thyroid gland by interfering with the uptake of iodine. They are naturally occurring components of Brassica vegetables. The group consists of a class of more than 100 sulfur-containing glycosides that yield thiocyanate, nitrile or isothiocyanate upon enzymatic hydrolysis [37]. Have a pungent smell, it is found in plants such as mustard, cabbage, fern and horseradish. Mustard oil comes from glucosinolates when the plant material is chewed, cut, or damaged. However, studies have shown that some of these compounds may inhibit some carcinogenic processes when consumed as part of the standard diet [11].

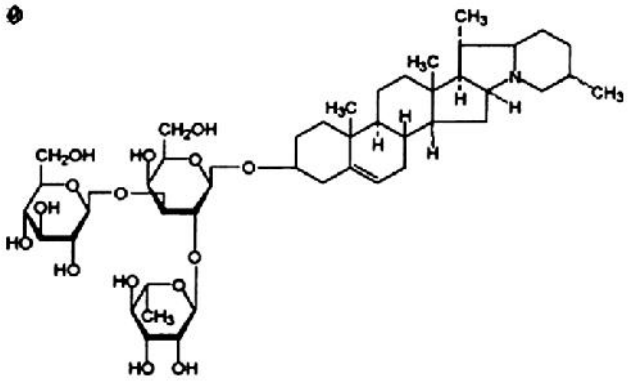

Fig. 2. The chemical structure of α-solanain. Showing tri-glycoside chain and aglycone section,
Source [32].

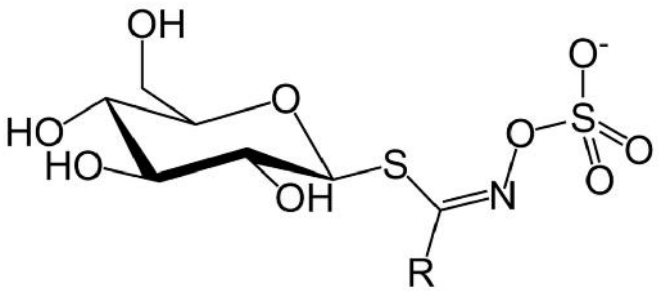

Fig. 3. The chemical structure of glucosinolates, the glycol components having sugar group and thiocyanate containing sulphur groups

2.3.3 Cyanogenic glycosides

Cyanogenic glycosides are chemical compounds listed in certain foods that release hydrogen cyanide when processed, chewed (eaten) or digested. These glycosides are common in certain families of plants such as *Fabaceae, Rosaceae, Euphorbiaceae, Gramineae, Leguminosae, Linaceae* and *Compositae* families [8]. Under normal environmental conditions, many fruits such as apricots, peaches, apples, cherries, plums, quinces contain cyanogenic glycosides mainly in their seeds. These glycosides are also found in bamboo shoots, almonds, cocoyam, lima bean, chickpea, cassava, pome fruit, stone fruit, flaxseed/linseed, cashew and kirsch. Food ingredients containing flavour properties such as almond powder, marzipan, stone fruit, or alcoholic drinks made from stone fruit may present the potential source of hydrogen cyanide [12]. There as more than 2,600 plant species that produce cyanoglycosides [38]. Grinding or maceration of edible parts of plants releases a β-glucosidase enzyme which comes into contact with the cyanogenic glycosides. The enzyme hydrolyses the glycosides to produce hydrogen cyanide, glucose, ketones, or benzaldehyde [4]. Legumes such as lima beans (*Phaseolus lunatus*) have been reported to exhibit toxicity and responsible for human poisoning due to their ability to release hydrogen cyanide. These legumes contain specific cyanogenic glycosides which upon hydrolysis release hydrogen cyanide. They have been observed to yield hydrogen cyanide levels ranging from 210.0-312.0 mg/100 g. Most of the liberated HCN from lima bean is lost during cooking through volatilization and rapid conversion to thiocyanates or other compounds. However, human intoxication and chronic neurological effects were reported even with cooked beans [4].

In a previous study, it was found that sweet and bitter almonds have levels of 25.20±8.24 and 1062±148.70 mg/kg hydrogen cyanide, respectively. These values indicate that both sweet and bitter almonds are classified as cyanogenic foods, owing to the threshold value of less than 10 mg/kg according to ISO 2146-1975 NT standard. In the same study, it was reported that cyanide level in apricot kernels ranged from 540 mg/kg to 1193.40 mg/kg in Tunisia. Comparable levels for hydrocyanic acid were reported at 1175±63.63 mg/kg and 799±19.80 mg/kg in Algeria and Australia, respectively [38]. European Food Safety Authority (EFSA) indicated that the advisable quantity of almond consumed should be from 5 to 10 kernels per day for the general population, while for cancer patients, the promoted portion size reached 60 apricot kernels per day [39].

Amygdalin has been found to be the significant cyanogenic glycoside present in apricot kernels. Physical processes such as chewing or grinding or digestive processes such as microbial activity in the stomach releases cyanide by facilitating mixing of amygdalin and the catabolic enzymes. On complete degradation, a gram of amygdalin can release 59 mg of hydrogen cyanide. The human acute lethal oral dose of cyanide has been reported to be 0.5-3.5 mg/kg body weight [39]. Ingestion of amygdalin preparations and bitter apricot kernels resulted in acute cyanide according to Tunisian study, the almond syrup was found to contain less than 3 mg/kg of hydrocyanic acid therefore it could be considered a product free of cyanogen toxicity [38].

2.3.4 Positive health benefits

Despite amygdalin being a source of toxicity for humans, it has medicinal value. Amygdalin exhibits supporting roles in the treatment of

several diseases such as cancer, diabetes, atherosclerosis, immune suppression, leprosy, therefore there is a need to do risk benefit analysis of the compounds [40]. The amygdalin as active ingredient in drugs occurs in the form of amygdalin and Laetrile [41]. Currently, there is still little in-depth information on the pharmacological mechanisms of action of amygdalin. Cyanide toxicity majorly inhibits oxidative phosphorylation by binding cytochrome oxidase which supply the heart and brain adenosine triphosphate (ATP) [39].

2.3.5 Reducing toxicity

Cyanides are decomposed during fermentation, which has been established to substantially reduce the amount of cyanogenic glycosides in cassava. Heating enhances evaporation rate of hydrogen cyanide and cyanohydrin hence reducing toxicity. When boiled in water for 15 min, about 85% of cyanogenic glucosides are removed by dissolving into water. Hydrogen cyanide is a volatile compound which evaporates rapidly in the air at temperatures over 28ºC and dissolves rapidly in water. Cyanogenic glycosides in cassava have been successfully reduced by up-to 3 fold using genetic engineering [42,43]. The bitter cassava varieties are preprocessed by peeling, dicing, washing, steeping, drying or mould-fermented before drying and milling, this reduces the cyanogenic glycosides up to 98% [44].

It is worth noting that cooking potatoes doesn't destroy α-chaconine and α-solanine. To prevent toxicity it is advisable to keep glycoalkaloid content low by storing potatoes at lower temperatures, such as 7ºC and away from sunlight. The toxins are heat stable, hence are not destroyed during cooking, steaming, baking, frying or microwaving. Moreover, they are not easily soluble in water [35].

2.3.6 Structure of cyanogenic glycosides

Are compounds composed of α-hydroxynitrile aglycone and of a sugar moiety (mostly D-glucose). Despite their diversity in structures, these secondary metabolites of plant origin are mainly derived from six amino acids namely, L-valine, L-isoleucine, L-leucine, L-phenyalanine or L-tyrosine and cyclopentenyl-glycine (a non-protein amino acid) [31]. In plants, the glycosides play critical roles in chemical defense system against insect and pests attack.

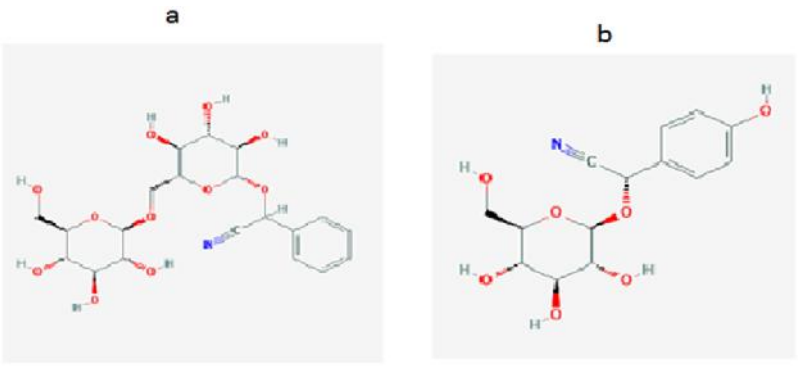

Fig. 4. Structure of amygdalin (a) and thurrin (b). With gentiobiose and -glucose as the sugar moiety, respectively

Source [38]

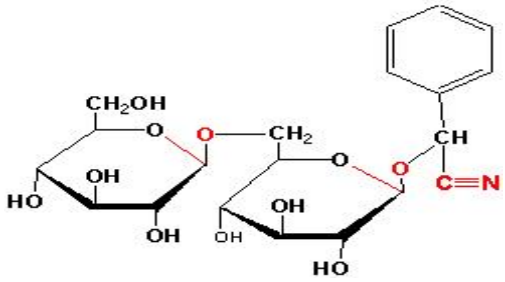

Fig. 5. General structure of Cyanogenic glycosides

Source [45]

2.4 Saponins

Saponins are water-soluble plant constituents, capable of forming soapy foam even at low concentrations. Structurally, they are glycosides with non-sugar polycyclic aglycone attached to one or more sugar side chains. The aglycone part referred to as sapogenin is either steroid (C27) or a triterpene (C30). The foaming ability of saponins is caused by the combination of a hydrophobic sapogenin and a hydrophilic sugar part, some saponins are toxic and are known as sapotoxin [46]. Saponins are distinguished by their bitter taste and ability to hemolysis red blood cells. Commercial saponins are extracted mainly from *Yucca schidigera* and *Quillaja saponaria* [47]. Saponins are widely distributed in the plant kingdom and can occur in all parts of plants, although the concentration is affected by variety and stage of growth. They are particularly abundant in soybeans, sugar beets, peanuts, spinach, asparagus, broccoli, potatoes, lucerne, broad bean, wheat apples, eggplants, alfalfa and ginseng root [4,48]. Bengalgram, soybeans, navy beans, haricot beans and kidney beans have saponin content as follows 56, 43, 21, 19 and 16 g/Kg of dry material, respectively [12].

2.4.1 Positive health impacts

Saponin rich product are mainly consumed due to their positive health effects which include neuroprotective, anti-carcinogenic (myelopoiesis, anti-angiogenic and cytotoxic), anti-inflammatory, hyperlipidemia, anti-fungal and hypocholesterol [31,49]. The most well-known role being cholesterol lowering and reduced lipid metabolism effects [50]. It reduces fat metabolism by inhibiting lipases like pancreatic lipase (PL) and other two carboxyl ester. Pancreatic lipase is the most critical human lipase associated with the hydrolysis of 50%–70% of total dietary fats, and is one of the most effective interventions used by pharmaceutical industries to decrease fat absorption after ingestion [49], and lowers cholesterol following two patterns, the first mechanism involves saponins forming insoluble complexes with cholesterol, thus inhibiting its intestinal absorption. The second mechanism entails saponins forming large aggregates with bile salts (BS) in the intestine and thus inhibits ileum bile salts reabsorption. The latter effect triggers an increased synthesis of BS from cholesterol in the liver, which leads to depletion of serum cholesterol, these effects have been established both *in-vitro* and *in-vivo* [48].

2.4.2 Structure of saponins

They are classified according to the chemical nature of the sapogenin into two major groups: steroidal and triterpenoid saponins. The presence of aglycone and hydrophobic sides make saponins be a suitable surfactant. The sugar chain may have one to three monosaccharides, this coupled with the steroid or triterpenoid aglycone imparts different structural and functional ability to saponins [46].

2.4.3 Deleterious health effects

Saponins are capable of disrupting red blood cells and causing diarrhoea and vomiting. Their toxic effects are related to the reduction of surface tension. Saponins are generally harmless to mammals and other warm-blooded animals except at large doses. Studies done on the toxicity of crude saponin fraction with 70% pure saponin showed some level of toxicity with

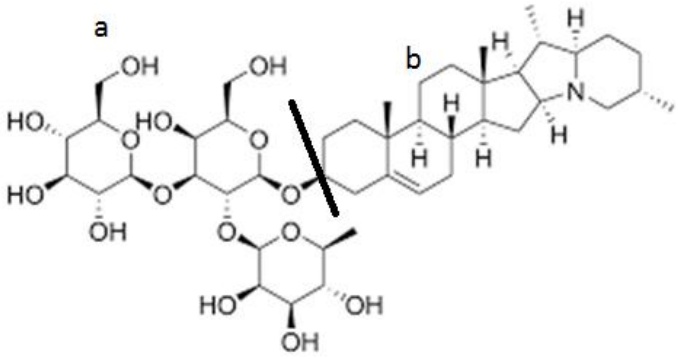

Fig. 6. The structure of saponin showing the sugar chain (a) and saponin section labelled as b. The sugar chain exhibits many hydrophobic groups while aglycone is hydrophobic.

lethal dose approximated at 1100 mg/kg of body weight [51]. The body can detoxify saponin when taken in a small amount since intestinal microflora can destroy them and blood plasma can inhibit their action. However, in large quantities, they can be irritating to the gastrointestinal tract causing vomiting and diarrhoea.

2.4.4 Effect of processing on saponins

Saponins are resistant to cooking or other physical processing but are degraded during fermentation. The saponin level of fermented products like tempeh, doufu, tofu etc. is significantly lower than unfermented soy products [12]. Therefore, it is advisable to consume fermented products from plant materials with high saponin content.

2.5 Polyphenols

Polyphenols are phenol-rich plant products, encompassing a broad group of phytochemicals produced as secondary compounds by plants. Polyphenols are widely distributed in the plant kingdom and are grouped into various classes depending on the number of phenol rings they contain and the structural elements that bind these rings to one another [52]. On this basis, the groups include phenolic acids, anthocyanin, catechin, epicatechin, gallates, flavonoids, stilbenes and lignans. They exist in various chemical forms and aid in the protection of plant tissues from injuries and attacks from insects or animals [53]. The most widely distributed phenolic in plant foods are tocopherolsboth tocopherols (alpha, beta, gamma and delta) and tocotrienols (alpha, beta and gamma) may be present. Phenolic acids are abundantly found in whole grains and oilseeds, particularly in the bran layer. Phenolic acids in foods, such as those of benzoic and cinnamic acid derivatives occur in the free, esterified/etherified and insoluble-bound forms [7,41].

Polyphenols found in foods generally contribute to their astringency [54,55]. Flavonoids and isoflavonoids are other groups of phenolic antioxidants found in foods, of which green tea is a rich source with (25%) of flavonoids of the catechin type. Tea catechins as well as other flavonoid found in plant foods are among the most reliable natural antioxidants found in nature [52]. Also, isoflavonoids, such as those found in soybean in relatively large amounts, e.g. Glycitein, myricitrin, daidzein, equol, genistein also exhibit potent antioxidant activity [56,57].

2.5.1 Structure of some polyphenols

They are rich in phenol groups, as shown in Figure 7 (a) and (b) below, hence the name polyphenols. Owing to numerous functional groups on the phenyl ring the ability to quench radicals and other functional abilities are exerted.

2.5.2 Positive health impacts

Phenolic compounds have been reported to possess multiple biological effects, including antioxidant activity as indicated by crude extracts from fruits, herbs, vegetables, cereals, and other plant materials [53]. By retardation of oxidative degradation of lipids, the phenolic compounds improve the quality and

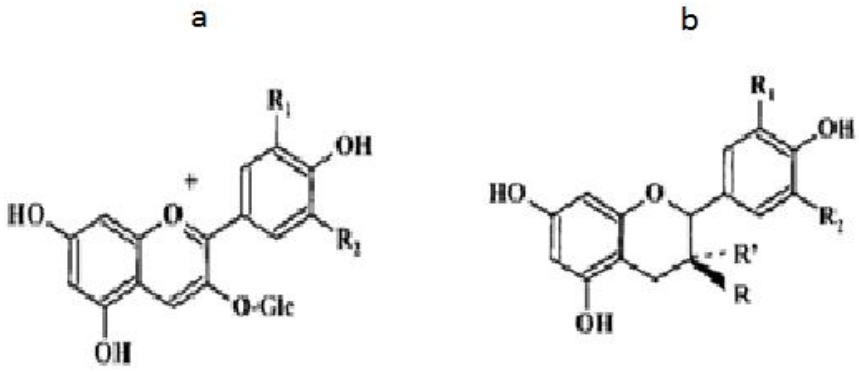

Fig. 7. The structure of anthocyanin (a) and flavanol (b)
Source [52]

nutritional value of food. Given that lipid peroxidation has been linked to atherosclerosis and other degenerative diseases associated with the ageing process including cataracts, it is likely that antioxidants from polyphenols hinders rancidity development in foods and thereby enhances the body's antioxidant defense mechanism especially in older adults. Furthermore, phenolic compounds have been shown to prevent oxidation of LDL-lipoprotein, platelet aggregation, and damage of red blood cells. Catechin, epicatechin and gallates of epicatechin are significant in human health due to their natural antioxidant properties against lipid oxidation, as supplements and antimicrobial agents in foodstuffs [9]. They are good metal chelators, which are beneficial to health and wellbeing due to their ability to get rid of heavy metals in the body, hence preventing their deleterious effects, this can find application in treating heavy metal poisoning [58]. Anticancer agents, through their anti-mutagenic and carcinogenic activity, the health benefit of red wine in colon cancer is linked to its rich polyphenol content, so is tea polyphenols catechin, epicatechin and gallic acid [9,53]. Through epidemiological studies, flavonoids have been linked to decreased risk of coronary heart disease and myocardial infarction. Moreover, model studies have suggested that tea extracts containing flavanols reduce the risk of arthritis as observed in animal models of inflammatory polyarthritis [54].

2.5.3 Deleterious health effect

Despite their many positive health effects, polyphenols have been linked to some adverse health effects. The potentially strong DNA-damaging activities have been shown to occur *in vitro* from dietary chemicals including polyphenols found in coffee, tea, and liquid smoke [59]. Phenolic compounds like gossypol may reduce the availability of certain minerals such as zinc due to their strong chelating activity [60]. Some polyphenols have been shown to exhibit hormonal disruption effects, hence the name phytoestrogens (coumestrol, genistein, daidzein and apigenin). They have the structure that mimics estrogen, consequently they disrupt estrogen receptors thus inducing female-like characteristics in male animals and may make some females to be sterile , indeed gossypol has been associated with decreased fertility [57,61].

Specific polyphenols may have carcinogenic/ genotoxic effects or may interfere with thyroid hormone biosynthesis, compounds like sanguinarine, epigallocatechin-3-gallate, pyro-gallol, and gallic acid etc. [41,62] The carcinogenicity and mutagenicity could be linked to the setup of the experiments. Some polyphenols inhibit non-heme iron absorption and may lead to iron depletion in some people leading to anaemia. Tannins interfere with dietary iron absorption due to decreased feed intake, feed efficiency and digestibility [31,55].

Owing to both positive and (potential) adverse health effects of polyphenols. Moreover, complexity in carrying out a thorough risk assessment for polyphenols, not only because there exist so many different compounds but also because the state of technology is not adequate to do rigorous risk assessment. Hence, risk-benefit analysis remains one possible means of evaluating them [63].

2.5.4 Effect of processing on polyphenols

During thermal processing, phenolic compounds may undergo oxidation and oxidized phenolic so formed, such as quinones, may combine with amino acids, thus making them nutritionally unavailable [64]. Furthermore, such reaction products are generally highly coloured in nature and impart undesirable dark colours to foods. Therefore, in some cases, it might be beneficial to remove phenolic in foods by devising novel processing techniques. In a study by Sun et al., it was shown that heat had a minimal effect on the antioxidant capacity of sweet potato leaves polyphenols, with not more than 9% loss in activity on exposure to up to 50-100^{0}C for 90 minutes [65]. On the other hand, blanching resulted in more than 70% loss of antioxidant capacity of cabbage polyphenols [66].

3. CONCLUSION AND RECOMMENDA-TION

Most of the anti-nutritional factors are present in foods of plant origin. Thus, the presence of cyanogenic glycosides, protease inhibitors, lectins, tannins, alkaloids, and saponins in foods may induce undesirable effects in humans if their consumption exceeds an upper allowable limit. Specific harmful effects might also be due to the breakdown products of these compounds. However, some anti-nutritional factors, as well as their breakdown products, may possess beneficial health effects if present in a given amount.

Owing to the innate presence of the phytotoxins and their variation in different crop varieties, it is advisable to undertake regional research to establish the level and toxic profile of specific phytotoxins. Considering advances in science, particularly molecular breeding, it is essential to breed crops with the low level of toxins to enhance food security particularly in areas where the specific crops are inherent part of diet, e.g. Lathyrus in Ethiopia and India. The overall risk of each plant compound can be established but owing to the complexity of plant constituents and genetic polymorphism it is difficult to determine the safety limits taking into consideration variations in consumption pattern. Public education should be made to inform the public on the health risks associated with anti-nutritional factors in some crops due to lack of knowledge of the tolerance levels to these compounds in the human.

Training and sensitization on viable methods of decontamination should be undertaken. Moreover, encouraging consumption of balanced diet by population should be done. Finally, it is worth noting that with mass use of herbal products as food supplements and adjuvants it is vital to establish their risks taking into consideration genetic polymorphism in population and ability to metabolize the compounds and consumption patterns.

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COMPETING INTERESTS

Authors have declared that there exist no competing interests

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